

Southlands Remediation and Development Project

Environmental Assessment

Project Application (MP 06_0191)

Volume 2: Appendices (H)



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Environmental Assessment

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Appendix H: Remediation Action Plan Orica Southlands (with Human Health Risk Assessment)



FINAL REPORT

Remediation Action Plan

Southlands Remediation and Development Project

Prepared for

Orica Australia Pty Ltd & Goodman Pty Ltd

2 October 2008


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Date: 2 October 2008
Reference: FINAL RAP (REV B)
Status: Final

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Appendices

- A. Human Health Risk Assessment

Glossary

ACM	Asbestos Containing Materials
AHD	Australian Height Datum - a standard reference point for the elevation of a location.
ANZECC	Australian and New Zealand Conservation Council
APM	Australian Paper Manufacturers
Asbestos Waste	Under the current (DECC NSW, 2008) Waste Guidelines (refer definition for Waste Guidelines) Asbestos Waste is any waste that contains asbestos. Asbestos Waste is a type of Special Waste.
Aquifer	An underground geological formation that contains water and is capable of yielding water to a well or spring; a water bearing formation.
Aquitard	A low permeability unit that can store groundwater and also transmit it slowly from one aquifer to another.
Attenuation	The removal or reduction of contaminants in groundwater with time and with distance travelled.
Bioremediation	Removal of in situ organic contamination by utilising naturally occurring or specifically engineered or introduced bacteria.
BIP	Botany Industrial Park
Block 1	The portion of the parcel of land known as Orica Southlands located to the east of Springvale Drain.
Block 2	The portion of the parcel of land known as Orica Southlands located to the west of Springvale Drain.
Bore/Borehole	An uncased well drill hole.
Bore Log	See - Geological Log
Botany Groundwater Cleanup (BGC) Project	The project to hydraulically contain and treat CHC contaminated groundwater in a Groundwater Treatment Plant (GTP) on BIP, and its associated infrastructure requirements, including groundwater extraction, effluent disposal and treated water distribution.
Botany Sands	The stratigraphical name given to unconsolidated sediments comprised predominantly of sand which underlie BIP and adjoining areas.
BP	See Bundle Piezometer
BTEX	BTEX is an acronym for benzene, toluene, ethyl benzene, and xylene
Bundle Piezometer	A cluster of narrow diameter piezometers with very short screens at different depths in the same hole.
CEMP	Construction Environmental Management Plan
Central EDC Plume	Plume inferred to originate from the EDC storage tanks
CFM	Trichloromethane (Chloroform)
Chain-of-Custody	Procedure to ensure that samples are traceable from the sample collection through to laboratory analysis and reporting.
CHC	Chlorinated Hydrocarbon
Chemical Reduction	Degradation of chemicals in an oxygen deficient environment.
cis-1,2-DCE	cis-1,2-Dichloroethene
CLM Act	Contaminated Land Management Act 1997
CTC	Tetrachloromethane (Carbon Tetrachloride)
1,2-DCB	1,2-Dichlorobenzene
1,3-DCB	1,3-Dichlorobenzene
1,4-DCB	1,4-Dichlorobenzene
DCP	Development Control Plan
DECC and NSW EPA	Department of Environment and Climate Change (formerly known as the Department of Environment and Conservation (DEC)) and incorporating the NSW Environment Protection Authority.
DEUS	Department of Energy, Utilities and Sustainability. Divisions of this department were combined with divisions of DNR to form the DWE in April 2007.
Dissolved Phase	See Aqueous Phase
DNAPL	Dense Non-Aqueous Phase Liquid - an organic chemical or mixture of organic chemicals that does not readily mix with water and is heavier than water.

Glossary

DNAPL Source Zones	Zones where residual or free phase DNAPL is present
DNR	Department of Natural Resources. Divisions of the DNR were combined with divisions of DEUS to form the DWE in April 2007.
Drawdown	A lowering of the water table of an unconfined aquifer or the potentiometric surface of a confined aquifer caused by pumping from wells.
DQOs	Data Quality Objectives
DWE	Department of Water and Energy. This department was created in April 2007 and combines divisions of the former DNR and DEUS.
EC	Environmental Consultant
EDC	1,2-Dichloroethane (Ethylene Dichloride), an intermediate compound in the production of vinyl chloride.
EMP	Environmental Management Plan
Exclusion Zones	Areas of the Site which either require additional protective measures or may require the adoption of additional occupational health and safety requirements and work practices. The zones primarily correspond to: Areas where there is a potential for exposure to dusts or noxious vapours; and Other areas of the Site which are affected by emissions from the works being undertaken by the Remediation Contractor.
Extraction Well	A well installed to enable groundwater remediation by the extraction of groundwater. Extraction wells assist in the control of a migrating plume.
Extraction Pump	Pump associated with extraction well system.
Field Duplicates	These are a set of two discrete samples collected from the one sampling point. The sample is prepared in the field by splitting a field sample, then submitting both to either the same laboratory (inter-laboratory duplicates) or a different laboratory (inter laboratory duplicate) as two independent samples, which are labelled as two discrete locations, the duplicate sample having no reference to the primary sample.
Flow Lines	Direction of groundwater flow.
Flow Path	The direction in which groundwater is moving.
Free Phase DNAPL	DNAPL saturation exceeding the capillary pressure of the soil.
FSRIB	Full Scale Reactive Iron Barrier
GC/MS	Gas Chromatograph/Mass Spectrometer - Instruments for the measurement of concentrations of organic compounds in soil and water.
GCP	Groundwater Clean up Plan – Plan prepared in response to Notice of Clean Up Action (NCUA).
General Solid Waste (non-putrescible)	Material classified as General Solid Waste (non-putrescible) in accordance with the DECC NSW (2008) Waste Guidelines.
General Solid Waste (putrescible)	Material classified as General Solid Waste (putrescible) in accordance with the DECC NSW (2008) Waste Guidelines.
Geology	The study of the earth as a whole, its origin, structure, composition and history, and the nature of the processes which have given rise to its present state.
Geological Log	A record of the lithology or stratigraphy of the rock or soil encountered in a borehole.
Gradient	The rate of inclination of a slope. The degree of deviation from the horizontal.
Groundwater	Water beneath ground surface.
Groundwater Extraction Exclusion Area (formerly Groundwater Protection Zone 1)	Area of groundwater as defined by DIPNR, during August 2003, for which there is an exclusion on the extraction of groundwater except for remediation purposes.
GTP	Groundwater Treatment Plant- A chemical treatment plant required to be constructed for the ex situ treatment of groundwater from hydraulic containment as required by the Notice of Clean Up Action (NCUA).
Hazardous Waste	Material classified as Hazardous Waste in accordance with the DECC NSW (2008) Waste Guidelines.
HCB	Hexachlorobenzene
HCBD	Hexachlorobuta-1,3-diene
HCE	Hexachloroethane
Heavy Ends	Waste stream from solvent manufacturing, which includes HCB, HCBD and HCE.

Glossary

HHRA	Human Health Risk Assessment
Hydraulic Conductivity	A coefficient of proportionality describing the rate at which water can move through a permeable medium.
Hydraulic Containment	Measures taken to lower the potentiometric surface and/or water table and effect hydraulic capture of the contaminant plume (as defined in the NCUA).
Hydrogeology	The study of the interrelationships of geological materials and processes with water, especially groundwater.
Hydrology	The study of the occurrence, distribution and chemistry of all waters of the earth.
Hydraulic Gradient	The change in total head in an aquifer with the change in distance in a given direction.
Hydrocarbon	Organic chemicals such as benzene or tetrachloroethene that contain atoms of carbon and hydrogen.
Inorganic	A chemical substance that does not contain carbon.
Laboratory Control Sample	Samples prepared by the laboratory by spiking an aliquot of appropriate clean matrix reagent with known concentrations of specific analytes. The control sample is then analysed and the results are used to assess the laboratory performance on sample preparation and analysis procedure.
Laboratory Duplicate	These are prepared within the laboratory by dividing a field sample into two samples and analysing separately.
Lithology	The geological (physical) character of a rock or soil.
LOR	Limit of Reporting. The lowest concentration that an analytical laboratory can report for an analyte with a sufficient degree of confidence. Also commonly referred to as the detection limit.
Matrix Spike/Matrix Spike Duplicate	Samples prepared by the laboratory in duplicate by individually spiking two aliquots of a field sample with known concentrations of specific target analytes. The matrix spike and matrix spike duplicate samples were then analysed and subsequently, the results used to assess the effects of the sample matrix on the accuracy and precision of analyses.
MCS	Maritime Container Services Pty Limited
Microgram (µg)	One thousandth part of a milligram (mg) one millionth part of a gram (g); one billionth part of a kilogram (kg).
Migration	The movement of materials (e.g.. water, gas or contaminants in soil) from one location to another.
Monitoring Well	A well installed to routinely observe groundwater levels or to systematically collect water samples and analyse these for chemical pollution.
NAPL	Non-Aqueous Phase Liquid - An organic chemical or mixture of organic chemicals that does not readily mix with water.
NCUA	Notice of Clean Up Action – Notice issued by the NSW Environment Protection Authority under Section 91 of the Protection of the Environment Operations Act 1997. The notice (No. 1030236) was issued on 26 September 2003.
NEPC	National Environment Protection Council
Organic Compound	A carbon containing compound.
Orica	Orica Australia Pty Limited
PCA	Primary Containment Area – Block 2 Southlands
PCE	Tetrachloroethene (Perchloroethene)
PE	Project Engineers
Piezometer	A well with a short slotted screen for measuring a potentiometric surface or elevation of the water table.
Plume	A mass of contaminated water extending outward from the source of the contamination.
Plume Axis	Inferred centre line of a dissolved phase groundwater contamination.
Porosity	The ratio of the volume of void spaces in a rock or sediment to the total volume of the rock or sediment.
Potentiometric Surface	An imaginary surface representing the total head of groundwater and defined by the level to which water will rise in a well.
Precision	The degree to which a measurement is reproducible.
Primary Containment Area	The primary containment area is defined in the NCUA as Block 2 of Southlands.
Proponent	Orica Australia Pty Limited and Goodman Pty Limited
PSI	Preliminary Soil Investigation

Glossary

PSRIB	Pilot Scale Reactive Iron Barrier
Pure Phase Solubility	Aqueous solubility of a single organic compound.
RAP	Remediation Action Plan
RBSC	Risk Based Soil Criteria
RC	Remediation Contractor
Recharge	Replenishment of an aquifer by a natural process such as addition of water at the ground surface, or by an artificial system such as addition through a well.
Recovered Aggregate	An aggregate source as defined in the Protection of the Environment Operations (Waste) Regulation 2005 – General Exemption Under Part 6, Clause 51 and 51A
REF	Review of Environmental Factors
Restricted Solid Waste	Material classified as Restricted Solid Waste in accordance with the DECC NSW (2008) Waste Guidelines.
Rinsate Blank	Type of field blank used to check specifically for cross-contamination from reuse of the sampling equipment.
RVP	Remediation Validation Plan
Saturated Zone	An underground geologic formation in which the pore spaces or interstitial spaces in the formation are filled with water under pressure equal to or greater than atmospheric pressure.
Screen	Perforation in a well casing and usually located near the bottom of the well or at selected depths to tap perched aquifers.
SCA	Secondary Containment Area - The area defined in the NCUA as “the location where the EPA approved contaminant containment works upgradient of Botany Bay and Penrhyn Estuary, for the interception and containment of contaminant plumes that have migrated or may migrate beyond the primary containment area, are carried out”.
Semi-volatile Compound	An organic compound which has a low potential to form a vapour at room temperature.
SEPP 55	State Environmental Planning Policy No.55 – Remediation of Land
Site	The land parcels to which this RAP refers. The site is also known as Southlands and/or the Southlands site.
SA	Site Auditor
Site Audit	<p>Site auditors review the work of contaminated site consultants. The CLM Act calls these reviews ‘site audits’ and defines a site audit as an independent review:</p> <p>(a) that relates to investigation or remediation carried out (whether under the CLM Act or otherwise) in respect of the actual or possible contamination of land, and</p> <p>(b) that is conducted for the purpose of determining any one or more of the following matters:</p> <p>(i) the nature and extent of any contamination of the land</p> <p>(ii) the nature and extent of the investigation or remediation</p> <p>(iii) whether the land is suitable for any specified use or range of uses</p> <p>(iv) what investigation or remediation remains necessary before land is suitable for any specified use or range of uses</p> <p>(v) the suitability and appropriateness of a plan of remediation, a long-term management plan, a voluntary investigation proposal or a remediation proposal.</p> <p>The main products of a site audit are a ‘site audit statement’ and a ‘site audit report’.</p>
Site Auditor	An independent third party technical reviewer (for land contamination issues) who is accredited by the DECC, NSW under the Contaminated Land Management Act 1997.
Site Audit Statement	<p>A site audit statement is the written opinion by an accredited site auditor, on a DECC-approved form, of the essential findings of a site audit. There are two types of Site Audit Statement (Section A or Section B) that can be prepared.</p> <p>A Section A Site Audit Statement is used where site investigation and/or remediation has been completed and a conclusion can be drawn regarding the suitability of the land use(s).</p> <p>A Section B Site Audit Statement is used when the audit is completed to determine the nature and extent of contamination and/or the appropriateness of an investigation or remediation action or management plan and/or whether the site can be made suitable for a specified land use or uses subject to the successful implementation of a remedial action or management plan.</p>
Southlands	A parcel of land bisected by Springvale Drain and lies to the west of the BIP. Orica purchased the land from Australian Paper Manufacturers (APM) in 1980. The Southlands land parcel forms the subject site (the Site) for this RAP.
Special Waste	Special Waste is Material that is classified under the current (DECC NSW, 2008) Waste Guidelines

Glossary

	(refer definition for Waste Guidelines) with unique regulatory requirements. Special Wastes are: clinical and related waste; asbestos waste; and waste tyres.
SSHSEP	Site Specific Health Safety and Environment Plan
SSU	Steam Stripping Unit
Stratigraphy	The study of rock and soil strata, especially their distribution, deposition and age.
Surrogate Compound	A compound that is introduced into a sample at a known concentration and is used as a system monitoring compound to assess the performance of individual organic analyses.
Surrogate Spike	System monitoring compounds used to assess the performance of the individual analyses. Compounds are spiked into all sample aliquots then undergo normal extraction and analysis procedures. Percent recoveries are calculated for each surrogate, providing an indication of the analytical accuracy.
SVD	Springvale Drain
1,1,2,2-TeCA	1,1,2,2-Tetrachloroethane
1,1,2-TCA	1,1,2-Trichloroethane
1,2,4-TCB	1,2,4-Trichlorobenzene
1,2,4,5-TeCB	1,2,4,5-Tetrachlorobenzene
TCE	Trichloroethene
Trip Blank	Type of field blank used to check if samples have been cross-contaminated with volatile contaminants during handling and transit between the field and laboratory. A trip spike typically comprises a sample of deionised water supplied by the laboratory in a laboratory sample bottle.
TOC	Total Organic Carbon.
Topography	The relief and contour of the land surface.
TPH	See Petroleum Hydrocarbons.
Transmissivity	The transmission rate of water (based on a unit width of an aquifer) as a function of hydraulic conductivity and aquifer thickness.
Unconfined Aquifer	An aquifer occurs when the potentiometric surface is equal to the water table .
Unsaturated Zone	The area between ground surface and the underground water table. Interstitial spaces in this zone contain moisture (water) and air.
VC	Vinyl Chloride (Chloroethene)
VENM	Virgin Excavated Natural Material. This includes natural material (such as clay, gravel, sand, soil and rock) that is not mixed with any other type of waste and that has been excavated from areas of land that are not contaminated as a result of industrial, commercial, mining or agricultural activities and that do not contain sulphidic ores or soils.
VFAs	Volatile Fatty Acids
VOC Scan	Volatile Organic Compound analytical scan.
Volatile Compound	Chemical with sufficiently low vapour pressure to become a gas at room temperature.
Waste Guidelines	DECC, NSW (2008) Waste Classification Guidelines: Part 1 Classifying Waste. During April 2008 the DEC NSW Environmental Guidelines: Assessment, Classification and Management of Liquid and Non-Liquid Wastes (2004) were replaced by these guidelines.

Section 1

Introduction

1.1 Overview

Orica Australia Pty Limited (Orica) and Goodman International (jointly the Proponent) have agreed to seek Development Approval for the staged remediation and development of Southlands, an approximately 20 hectare, vacant and generally disused site owned by Orica and fronting McPherson Street, Banksmeadow (the site). The site location is presented in Figure 1 and the site layout and existing infrastructure is presented in Figure 2.

The Proponent proposes to develop the site for high quality industrial and warehouse purposes. The site is currently zoned 4(a) Industrial within the Botany Bay City Council area. The Proponent intends to seek Development Approval (DA) for remediation and redevelopment works under Part 3A of the Environmental Planning and Assessment Act 1997.

The DA will be supported by an Environmental Assessment (EA) prepared in accordance with Department of Planning requirements. The EA will incorporate a Remediation Action Plan (RAP) prepared to meet the NSW DECC accredited Site Auditor's requirements.

Mr Chris Jewell, a NSW DECC accredited Site Auditor has been appointed by the Proponent to review the appropriateness of the RAP and whether the site can be made suitable for the proposed land use by its implementation.

1.2 General Site Description

Southlands (the site) is divided into two blocks, known as Block 1 and Block 2, by Springvale Drain. Springvale Drain is a north-south trending man-made drainage feature and surface water conduit. Floodvale Drain, a similar drainage feature, forms the western boundary of Block 2 Southlands.

Immediately west of Springvale Drain, there is a disused secondary surface water channel, known as the 'realignment channel'. It was created to temporarily divert flow from Springvale Drain during civil works conducted in 2000. It is considered likely the feature is charged by shallow groundwater, similar to Springvale Drain proper, but is cut off at both the northern and southern ends.

Nant Street is an unsealed roadway that provides access from the McPherson Street site entrance to the Genos Pty Ltd Tank Farm, located to the north of Southlands.

Various infrastructure, including above ground pipe lines and groundwater extraction bores, which form the Primary Containment Area (PCA) of the Orica Botany Groundwater Treatment Plant (GTP), are located on Southlands. These are principally located along the McPherson Street Boundary and the western margin of Springvale Drain. The PCA extracts groundwater, which is pumped via pipeline to the GTP located in the Botany Industrial Park (BIP) to the north east of Southlands.

There are five topographical depressions on Southlands Block 1, which are periodically charged by surface and ground water. It is understood these features, generally referred to as "*Paper Waste Ponds*", are remnants of historical sand and peat mining activities, which during a period of ownership by Australian Paper Manufacturers (APM), were partially backfilled with paper wastes from the manufacturing processes.

Prior to erection of a boundary fence by Orica in 1991, Southlands was subject to a significant volume of uncontrolled dumping. The dumped materials generally comprised demolition wastes and rubble. Subsequent environmental investigations have indicated these materials contain asbestos containing materials (ACM). Since cessation of dumping the various stockpiles of waste have become heavily overgrown with vegetation. As a result, the surface of Southlands Block 2, and to a lesser extent Block 1, has a characteristic "*hummocky*" topography.

A grid based series of access tracks have been cut into the dense vegetation, originally to facilitate a geotechnical survey in the late 1990's. but they now serve as access for GTP maintenance and environmental monitoring activities.

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Introduction

Considerable groundwater monitoring infrastructure is located on Southlands, more detail on which is provided in Section 1.6 and Section 5.4.

1.3 Proposed Development Summary

The site is to be developed for high quality industrial and warehouse purposes. A staged development approach is proposed and is described in the following sections.

1.3.1 Project Staging

The primary stage, Stage 1, generally incorporates development of the area to the west of Springvale Drain, known as Southlands Block 2. This area is bounded by McPherson Street, Port Feeder Road and a Mobil Oil Depot to the south, west and north respectively.

Stage 1 will also include construction of an internal roadway to the east of Springvale Drain (on Block 1), which will provide access to Stage 1 from McPherson Street.

The flooding solution for Stage 1 (CW, 2007) requires some shallow earthworks on the southern portion of Southlands Block 1, and which will be included in the Stage 1 development and therefore the *Project Approval Application*¹. The extent of the required excavation is illustrated on Figure 4².

The area to the east of Springvale Drain, known as Southlands Block 1, will contain later development stages (Stages 2 and 3), on the southern and northern portions of Southlands Block 1 respectively.

Project Approval for Stages 1 and 2 is the focus of the current *Application* and hence of this RAP. Development of Stage 3 will be the subject of a future development application and is not supported by this RAP.

The development master plan is provided on Figure 3, while Figure 4 illustrates the land use scenario for Stage 1. A conceptual lay out for Stage 3 is also included in Figure 3, however, this application does not include this stage.

1.3.2 Stage 1

The *Project* plan for Stage 1 (see Figure 4) makes provision for three major single storey commercial buildings, the largest of which, occupying 24,800 square metres (sqm), is to be located in the northern portion of Stage 1, with main access from McPherson Street. This structure will be divided into two warehouse facilities (Warehouses 5 and 6) of approximately equal area. Office space is also incorporated into the plan and is likely to be located at the southern end of the northern building, occupying approximately 2,000 sqm.

A multi-storey car park is proposed to be constructed to the west of the main warehouse building, with concrete aprons providing surface level car parking to the south and south west. Two loading bays will be located to the south of the building.

Two smaller buildings, occupying approximately 8,200 and 10,000 sqm respectively, will be located in the southern area of Stage 1, with loading access from McPherson Street via the Proposed Access Road and office space at the south. These buildings will be divided into four warehouse facilities (Warehouses 1 to 4).

¹ Italicisation indicates that reference is made to an approval within the context of the Part 3A of The Environmental Planning and Assessment Act 1997.

² Detail in Figure 3 sourced from Goodman Pty Ltd

Section 1

Introduction

The proposed development also includes several "in ground" rainwater detention tanks. These are to be located towards the centre of Stage 1 beneath the hard stand areas, either side of the access roadway.

Other features of Stage 1 include two compensatory flood storage areas along the western margin of Springvale Drain.

Prior to later stages of development (Stages 2 and 3), Southlands Block 1 will remain largely vacant, but will be used as a compensatory flood storage area. As noted above, earthworks to accommodate compensatory flood storage requirements will form part of the Stage 1 works.

1.3.3 Stage 2

Stage 2 will occupy the southern portion of existing Block 1. The development (see Figure 3) makes provision for three single storey buildings, which will accommodate seven warehouse facilities and associated office spaces.

Other features will include car parking spaces and paved access for loading / unloading of goods. Provision is made for a compensatory flood storage area (Basin No. 2) in the south western corner.

1.3.4 Stage 3

Stage 3 will occupy the northern portion of existing Block 1. A conceptual plan for Stage 3 is included in Figure 3, however, as noted above, this RAP is not intended to support Stage 3 development, which it is understood will be part of a separate planning application.

1.4 Planning Context

The *Environmental Planning and Assessment 1979* (EP&A Act), and the *Environmental Planning and Assessment Regulation 2000* (EP&A Regulation), provide the framework for development and environmental assessment in NSW.

In late 2005 a new part (Part 3A) was inserted into the EP&A Act to provide for a new category of development known as Major Projects.

State Environmental Planning Policy (SEPP) 2005 (Major Projects) (SEPP Major Projects) was prepared and gazetted in conjunction with this amendment. SEPP Major Projects identifies categories of development which are considered to be Major Projects to which Part 3A of the EP&A Act applies and identifies projects of State or regional planning significance that are of a kind that the approval and assessment process under Part 3A of the Act should apply.

Group 4 class of development in Schedule 1 of SEPP (Major Projects) declares that development for the purpose of container storage facilities, or storage or distribution centres, with a capital investment value of more than \$30 million are Part 3A projects. The Minister for Planning has formed the opinion that Part 3A of the Act applies to the Southlands Proposal. Consequently the Minister is the approval authority for this project.

1.5 Regional Groundwater Contamination Context

A review of information provided on The NSW Department of Natural Resources (DNR) web site³ indicates that the site is located within one of four '*management zones*' prescribed by the DNR. The site is located in Zone 1 which is the Groundwater Extraction Exclusion Area (GEEA) around the Orica site and the use of groundwater from this zone is prohibited by the DNR.

³ http://www.naturalresources.nsw.gov.au/water/botany_bay.shtml. Note: While this website is still in operation, the DNR has been reformed as the New South Wales Department of Water and Energy (NSW DWE).

Section 1

Introduction

The DNR website identifies the Orica Botany Groundwater website⁴ as a source of further information on the history and nature of groundwater contamination in the area. As shown on Figure 1, the BIP site is located to the northeast of Southlands.

The following information is taken from the Orica website and provides a background to the existing groundwater contamination issues in the area:

“Contamination of groundwater at the Botany Industrial Park dates back over a period of up to 50 years and was first identified in a survey conducted for Orica Australia in 1990.

The environmental problems at what is now known as the BIP date back to the 1940s when manufacturing began at the site. The 1960s saw the introduction of larger manufacturing plants making a wider range of chemicals. This was an era when environmental awareness and standards were far lower than those of today, especially in understanding the impact of chemical use and storage on soil and groundwater.

During this time, chlorinated hydrocarbons (CHCs) were stored on-site in tanks and drums. Some of these stored materials have leaked into the ground and groundwater. It is likely that contamination was also caused by accidental spills.

The main contaminant in the groundwater is ethylene dichloride (EDC). The chemical EDC may pose a serious health risk, but at the levels detected in extensive testing at Botany it has been established as unlikely to result in adverse health effects. A risk assessment of the area has concluded there is no unacceptable risk to human health. Orica still takes this issue very seriously and is addressing the EDC through their Cleanup Plan”.

1.6 Orica Groundwater Treatment Program

1.6.1 Notice of Clean Up Action (NCUA)

In September 2003 (subsequently amended and consolidated in February 2004) the Department of Environment and Conservation (DEC) (now the Department of Environment and Climate Change (DECC), NSW) issued a Notice of Clean Up Action (NCUA) No. 1030236 to Orica Australia Pty Ltd (Orica) requiring remediation of the chlorinated hydrocarbon contaminant plumes emanating from the Botany Industrial Park (BIP) and present in areas to the south and west of BIP.

The NCUA also required the preparation and implementation of a more extensive groundwater monitoring program compared to that which was being undertaken at that time. The expanded monitoring program was documented in the Groundwater Cleanup Plan (GCP, Orica 2003) and included the installation of new groundwater monitoring wells/bundle piezometers and sampling and reporting of analytical data on a quarterly basis.

1.6.2 Groundwater Cleanup Plan (Orica 2003)

The GCP documented the proposed strategies adopted to address groundwater contamination at BIP and downgradient of BIP. The strategy selected to achieve hydraulic containment of groundwater contamination was described in the Botany Groundwater Cleanup (BGC) Project Environmental Impact Statement (EIS) (URS, 2004c) and comprised the installation and operation of three hydraulic containment lines (Primary, Secondary and DNAPL/BIP) and construction of a groundwater treatment plant (GTP) to enable treatment of the groundwater extracted to achieve hydraulic containment. The general layout of this infrastructure is shown on Figure 5.

⁴ <http://www.oricabotanygroundwater.com/>

Section 1

Introduction

The Joint Determining Authority Report (DEC NSW 2005) for the BGC Project EIS included conditions of approval related to groundwater and surface water monitoring. The approval conditions included a number of specific objectives including:

- Monitoring of aquifer water levels (hydraulic monitoring);
- Monitoring of contaminants of concern in groundwater; and
- Monitoring of contaminants of concern in surface water.

1.6.3 GTP Monitoring Program (2005)

To address the conditions of the BGC approval, the Groundwater Treatment Plant (GTP) – Groundwater and Surface Water Monitoring Program (URS 2005a) was developed and submitted to DECC, NSW. This program is regularly reviewed and refined in consultation with the DECC, NSW.

1.6.4 Quarterly Groundwater and Surface Water Monitoring

Quarterly groundwater monitoring programs have been completed and reported by Orica to the DECC, NSW under the NCUA since March 2004. The existing monitoring well network relevant to Orica Southlands is shown on Figure 6.

Focused consideration of the requirements for maintenance of a suitable groundwater monitoring network during and following staged development of the site is the subject of Section 5.4.

1.7 Flood Management

Connell Wagner Pty Ltd (CW) have been engaged by the Proponent to develop a detailed flood investigation of the site and the surrounding area, building upon and adding to the flood modelling prepared for the Port Botany Expansion (Lawson and Treloar 2003). The model prepared by CW assesses potential flood inundation in the area and determines the 1:100 design flood level. Section 5.1 provides more detail on the findings of the report and the implications for the adopted remedial solution for the site.

1.8 RAP Objectives

1.8.1 Development

The Proponent seeks to develop Southlands (the site) for high quality commercial and warehouse purposes.

1.8.2 Remediation

To remediate and/or manage (as appropriate) the identified contamination so as to render the proposed Stage 1 (Block 2) and Stage 2 (southern portion of Block 1) redevelopment areas of the Southlands site as suitable for the proposed commercial / industrial development land use in accordance with the relevant DECC, NSW endorsed guidelines.

Section 1

Introduction

1.8.3 Remediation Action Plan

For the proposed Stage 1 (Block 2) and Stage 2 (southern portion of Block 1) redevelopment areas of the Southlands site:

- To identify remediation requirements in the context of the DECC, NSW endorsed contaminated site assessment framework;
- To identify available remediation technologies and identify a suitable approach for remediation/management of identified contamination;
- To identify suitable remediation validation protocols (as required) for the site;
- To assess and identify the regulatory and legislative frameworks for the remediation program; and
- To identify a framework for preparation of a long term environmental management approach.

Section 2

Site Background

2.1 Site Identification

The site cadastral boundaries are shown on Figure 2 and the property identifiers are summarised as follows:

Table 2-1 Lot and Deposited Plan (DP Details)

Site Area	Development Stage	Lot / DP Number	Area (m ²)
Southlands Block 1	Stages 2 and 3	Lot 1 / DP 85542	61,300
		Lot 11 / DP 109505	23,430
Southlands Block 2	Stage 1	Lot 1 / DP 1078077	95,300
		Lot 1 / DP 254392	2,850

2.2 Site Environs

The site is located approximately 10 km south east of Sydney CBD and approximately 4 km east of Sydney Airport. Access to the site is from McPherson Street. The total area of the site is 182,880 m² (18.2 hectares (ha.)). The site is located in a predominantly industrial / commercial area. Surrounding businesses are identified on Figure 7 and described under the following headings.

2.2.1 North and North East

The site is located to the south west of Botany Industrial Park (BIP), separated by the Sydenham - Botany Goods Railway Corridor. BIP is located to the north and north east of the site. Several chemical manufacturing businesses, including Orica Australia Pty Ltd, have manufacturing and distribution facilities in BIP.

A Qenos Pty Ltd (Qenos) owned, Mobil leased tank farm facility, known as *The Nant Street Tank Farm* is located immediately north of Block 1 Southlands. This site has been used historically to store petroleum hydrocarbons. It appears, on the basis of partial plans provided by the dial-before-you-dig service and anecdotal evidence, that the Caltex Terminal, located approximately 0.5 km to the south of Block 1 is linked with the Nant Street Mobil Terminal via an underground oil pipeline. The pipeline is apparently routed beneath Nant Street and then eastward along McPherson Street towards the Terminal.

The Mobil Botany Oil Distribution Terminal is located immediately to the north of Southlands Block 2. Beyond the Mobil facility, there is a vacant, currently undeveloped site, owned by Valad Property Group.

Located approximately 0.4 km due west of the Mobil Terminal is another chemical business, Nuplex Industries (Aust) Pty Ltd. This Nuplex site has frontage on Stephens Road.

2.2.2 West

A chemical manufacturing facility owned by Solvay Interlox Pty Ltd (Solvay) is located to the west of the site, separated by Floodvale Drain. Beyond Solvay, is an industrial estate.

2.2.3 South

McPherson Street forms the southern boundary to Southlands. Beyond McPherson Street, there is a shipping container storage facility operated by Maritime Container Services (MCS) Pty Ltd. A warehouse owned by Gazal Corporation, an apparel manufacturer, is located to the south of Block 2. Beyond MCS to the south is a Goodman International managed Industrial Estate identified as Discovery Cove.

Section 2

Site Background

2.2.4 East

The Sydenham to Botany railway line is located beyond the eastern boundary. The site on the other (eastern) side of the railway is understood to be occupied by a light engineering works.

2.3 Local Area History

HLA Envirosciences Pty Ltd (HLA) completed a Limited Environmental Investigation⁵ (HLA, 2005) of the site in 2005. The report provided a general history derived from the A G Environmental Engineers Stage 1 Preliminary Investigations (AGEE, 1990) and Woodward-Clyde Stage 2 Investigation Report (WWC, 1996b). The historical information provided in the HLA report is presented below, with additional information from the original source documents provided as appropriate.

The first industries established in the vicinity of east Botany prior to the 1940s included tanneries, fellmongeries, wool scourers and a paper mill. It is not clear whether such industries were established on or near the site.

Chemical Industries, including ICI, AC Hatrick and Crest Chemicals, were initiated in the 1940s. Fuel storage terminals were established between 1956 and 1966. Catoleum and LaPorte Chemicals (now Solvay Interlox) were both established towards the end of this period.

ICI (now Orica) began limited manufacturing in the southern portion of what is now BIP in 1942. The scale of the operation increased in the post war years, shifting through the 1960s into the northern portion of BIP and becoming more predominantly focussed on petrochemical manufacture. Further major developments of the ICI complex continued through the 1970s and 1980s.

The Stage 2 Investigation Report (WWC, 1996b) identifies trichloroethene (TCE), which was manufactured from 1948 through 1977, and solvents (carbon tetrachloride (CTC) and tetrachloroethene (PCE)) which were manufactured during the period 1964 through 1991, as the most notable of the now discontinued manufacturing operations on the ICI (now BIP) site.

2.4 Southlands History

Limited anecdotal and some aerial photographic evidence suggest the earliest uses of the land included some form of intensive agriculture, probably market gardening. The land (both Blocks 1 and 2) was also subject to sand and peat mining in the 1960s.

Australian Paper Manufacturers (APM) owned Southlands prior to Orica's acquisition of the land in 1980. It appears the land was used by APM for storage of paper waste bails and anecdotal evidence suggests paper wastes were disposed of into the excavations that remained following the earlier mining activities, resulting in the shallow, periodically charged depressions known as The Paper Waste Ponds on Block 1.

The Stage 2 Survey (WWC, 1996b) suggests that APM used mercury based fungicides and sulphites in the paper manufacturing processes. Wet solid wastes were reportedly removed from the site by truck and pipeline (WWC, 1996b), however the route or discharge point of this pipeline is not known.

2.4.1 Land Filling

The Southlands site, as well as adjoining properties to the south of McPherson Street and the northern portion of BIP, have been extensively filled; primarily with ash from coal-fired boilers from local industries including the Bunnerong Power Station (HLA, 2005). Based on field observations, it is likely that other materials such as building rubble and demolition wastes were also used in the filling process.

⁵ Factual Report on Limited Environmental Investigation, Orica Botany – Southland, NSW (August 2005)

Section 2

Site Background

Fill thickness has generally been reported to be between 0.75 metres and 2.5 metres across the site, but has been measured in parts at thicknesses of up to 4 metres (WWC, 1996b).

2.4.2 Stockpiles

Prior to erection of a fence around the Southlands perimeter by Orica in the early 1990s, Block 2 and to a lesser extent Block 1, were subject to ad hoc dumping of wastes, predominantly demolition wastes, which currently remain on site. The stockpiles have been heavily overgrown with vegetation over time.

2.4.3 Aerial Photograph Review

The Stage 2 Investigation Report (WWC, 1996b) provided a detailed review of oblique and stereo pair aerial photographs, a précis of which is provided as follows.

May 1951

- Bournes Tannery was located on Springvale Drain (to the west of Springvale Drain and north of Block 2, at the present location of the Mobil Botany Terminal);
- A large pond was present on Block 2 (west of Springvale Drain);
- A pond / excavation was present in the south west corner of Block 1 (east of Springvale Drain);
- A “possible” drain extending from the then ICI plant across Block 1 to Springvale Drain was identified; and
- Waste paper storage (off-site) south of McPherson Street was noted.

August 1955

- Three “possible” ponds were identified on Block 2, adjacent to McPherson Street; and
- Fill (boiler ash) was being spread over a large area of Block 1.

June 1961

- A drain appeared to extend from the ICI Plant across the northern boundary of Southlands Block 1 to Springvale Drain; and
- Extraction / mining of materials was noted to have resulted in a large pond on Block 1

1962 (Month Unknown)

- The area between the railway embankment and the [former] Orica effluent treatment plant, formerly located immediately to the north east of Block 1, appeared to have been receiving direct discharge of effluent to ground before this time. The report (WWC, 1996a) noted that such discharge was understood to have occurred during periods when the effluent treatment plant was off line.

August 1965

- Two tanks were present on the Naptha Tank Farm (now known as the Nant Street Tank Farm);
- The drain noted in the 1951 photograph was no longer present; and
- Numerous excavations and earthworks were clearly visible on Block 1 (assumed to be associated with the sand and peat mining activities).

Section 2

Site Background

1969 (Two photographs, month unknown and September)

- The former Esso tank farm was under construction on the site of the former Bournes Tannery (at the current location of the Mobil Botany Terminal); and
- Several ponds were noted on Block 1, some of which were banded.

July 1970

- Major earthworks, including excavations and stockpiling, were noted on Block 2, including placement of boiler ash as fill;
- A series of ponds were in use on Block 1 and a drain from the railway line onto Block 1 was also noted; and
- Possible placement of paper waste was also identified.

August 1975

- Extensive excavations were noted as being clearly visible on Block 1;
- The central pond (the pond currently referred to as the large Paper Waste Pond) had been in filled; and
- Nant Street appeared to have been built up to act as a bund wall for paper slurry storage.

June 1978

- Excavations on northern boundary appeared to have back filled with ash;
- Ash materials were stockpiled on the southern portion of Block 1, at the former location of ponds that had since been back filled; and
- Paper waste disposal and land spreading / filling of ash material were also evident.

February 1979

- The surface of Block 2 was noted to be markedly reshaped at this stage, being flat and well vegetated;
- Numerous ponds and excavations were still present on Block 1, several of which were noted to have been back filled by the time of the Stage 2 works (1995/1996);
- The large central pond was noted to have been dry and slurried paper waste effluent extended from the pond to Nant Street (to the west).

2.4.4 Site History Summary (After WWC, 1996b)

- Southlands appeared to have been used for waste paper slurry disposal until the late 1960s at the earliest;
- The final shape of the central pond on Block 1 appeared to be the result of backfilling of old peat mining depressions as well as above grade bunding;
- Most of the small ponds appeared to have been formed from above grade bunding and not necessarily sand/peat excavations;
- Both Block 1 and Block 2 were noted to have been extensively filled with boiler furnace ash/coke and in some cases building rubble; and
- Fill thickness was considered to be variable across the site.

Section 2

Site Background

2.4.5 ICI Effluent Disposal Information

The Stage 2 Investigation report (WWC, 1996a) identified that, while recycling and recovery of effluents derived from processes on the ICI site (now BIP) had progressively improved throughout the site's manufacturing history, information provided by ICI had suggested that prior to 1958, effluent disposal was via a stormwater pipe directly into Springvale Drain.

The report also identified that old photographic evidence reviewed as part of the investigation had indicated that disposal directly onto the ground in the location of the former effluent treatment plant, located to the north east of Block 1, had also occurred.

2.5 Topography

The local area is located on an area of former sand dunes and coastal swamps within the Botany Basin. The local topography generally slopes down towards the south west and Penrhyn Estuary, from a relative high point of 20 m Australian Height Datum (AHD) at the eastern side of BIP, down to elevations of less than 4 m AHD at and to the south of Southlands (WWC, 1996a).

The Southlands site forms part of an extensive low lying (< 4 mAHD) area to the west of BIP, which was formerly referred to as the Veterans Swamp, prior to construction of the Springvale and Floodvale Drains during the 1940s (WWC, 1996a). The drains were designed to assist in the drainage of the former swamp and both discharge into Botany Bay via Penrhyn Estuary, which was formed by the reclamation of the Port Botany Container Terminal area. Figure 2 shows the locations and orientations of the drains.

The surface of the site is generally flat, sloping slightly down towards Springvale and Floodvale Drains. However, the heavily vegetated stockpiles have resulted in an uneven or "hummocky" surface across much of the site.

2.6 Local Geology

Botany Basin

The Botany Basin occupies an area of approximately 80 square km and lies to the south of the city of Sydney (see Figures 1 and 7). It is bounded by Centennial Park to the north, Randwick and Matraville in the east, Alexandria and Rockdale to the west and Kurnell Peninsula to the south (WWC, 1996b).

Quaternary sediments in the basin reach thicknesses of up to 80 m in parts, overlying the Hawkesbury Sandstone and infilling palaeodrainage features. One such feature, the "Lakes Valley Palaeochannel" has been inferred by geophysics and confirmed by drilling (WWC, 1996a) to lie to the west of the BIP / Southlands area.

The Quaternary sediments generally comprise predominantly unconsolidated to semi-consolidated permeable sands, interspersed with discrete layers of low permeability peat and peaty sands (WWC, 1996a).

2.7 Local Hydrogeology

Botany Sands Aquifer

The Botany Sands contain a system of unconfined and semi-confined aquifers of varying yields that are referred to as the Botany Aquifer. They are interconnected vertically via leakage through the confining peaty layers (WWC, 1996b).

Section 3

Summary of Previous Investigations

3.1 General

The Southlands site has been subject to environmental investigations of soil, groundwater, sediment and surface water. This section provides a précis of relevant earlier investigation work and presents data collected through completion of the Phase 2 Assessment by HLA Envirosciences (HLA, 2005) and a further detailed soil quality investigation completed by URS in 2006.

It is noted that in this section, the identification of impacts are discussed in the context of the investigations levels adopted in the previous studies. Through the human health risk assessment process (see Sections 4 and 8) other site specific assessment criteria have been adopted and/or derived and applied to the data set. Therefore, the identification of impacts requiring remediation may differ slightly to the summary of impacts discussed here.

3.2 Data Sources (Previous Site Assessments)

The following relevant environmental reports on historical and recent intrusive investigations are identified for the Southlands site:

- Australian Groundwater Environmental Engineers (AGEE) (1990). *ICI Botany Environmental Survey, Stage 1 Preliminary Investigation*, May 1990. State Pollution Control Commission (SPCC);
- (WWC 1996a) *ICI Botany, Groundwater Stage 2 Survey (Contract S2/C1, Springvale Drain)* August 1996;
- (WWC 1996b) *ICI Botany, Groundwater Stage 2 Survey (Contract S2/C3, Water/Soil Phase 2)* August 1996; and
- (HLA 2005) *Factual Report on Limited Environmental Investigation, Orica Botany – Southland, NSW* August 2005.

Based upon the results of the HLA Investigation (HLA, 2005) URS completed a program of detailed soil quality assessment across Southlands in November 2006.

Other reports, relevant to the Southlands site have been referenced. These include:

- URS (2004a). *Orica Botany Environmental Survey Stage 4 – Remediation, Full Scale Reactive Iron Barrier (FSRIB) – Data Gaps*. May 2004.

This report includes details of investigations completed on Southlands as part of a groundwater remediation trial for the establishment of a full scale permeable reactive iron barrier. Trials included the installation in 1999, and subsequent monitoring, of a pilot-scale iron barrier (PSRIB) on Block 1, Southlands.

DNAPL source area investigations, required to satisfy the NCUA requirements, and have also included investigations of a portion of the Southlands site. Investigations have primarily been focussed in the north eastern portion of the site (northern portion of Block 1, Development Stage 3). Reports referenced included:

- URS (2004b). *Orica Botany Environmental Survey Stage 4 – Remediation, 2004 DNAPL Source Area Investigation*. August 2004.

This report summarises all DNAPL source area investigations completed prior to 2004 and the results of 2004 investigation works. Various DNAPL source areas are identified including the Southern DNAPL source areas: S1, S1 and S3. The DNAPL source area located on the Southlands site is known as S1, the source of which is reported to be overflow from the effluent treatment plant from the 1940s to 1970s onto the rail corridor and Southlands, and possible seepage from a former unlined drain in the northern portion of Southlands linking BIP with Springvale Drain possibly up until the 1960s.

Section 3

Summary of Previous Investigations

Area "S1" is located in the Southlands site Stage 3 redevelopment area, the remediation of which is not the subject of this RAP.

Subsequent investigations of the Southern Plumes are reported in the following documents:

- URS (2005a). Orica Botany Environmental Survey *Stage 4 – Remediation*, 2005 DNAPL Source Area Delineation Investigation. April 2005.
- URS (2005b). Orica Botany Environmental Survey *Stage 4 – Remediation*, Progress Report – Southern Plumes DNAPL Source Area Delineation Investigation, August 2005. December 2005.
- URS (2006). Southern Plumes Source Area Delineation Investigation. February 2006.

The following report summarises the results of the all the various intrusive investigations (including DNAPL investigations), risk assessment and remediation trials completed on the BIP and the Southlands site up to 2007:

- URS (2007c). Conceptual Site Model - Botany. November 2007.

3.3 *Stage 1*⁶ Preliminary Investigation - AGEE 1990

With respect to Southlands, this report concluded that the "shallow" groundwater was contaminated, mainly by CHCs. Limited evidence of "deep" groundwater CHC contamination beneath Southlands was also reported. Concentrations of CHC in deep groundwater were markedly higher than in the shallow groundwater.

AGEE (1990) also reported that elevated concentrations of CHC had been detected in the shallow fill materials. It was inferred these concentrations may have been related to discharge of ICI effluent directly onto the ground in the vicinity of the former effluent treatment plant and/or leaks from a pipeline that discharged into Springvale Drain.

Hexachlorobenzene (HCB), Hexachlorobutadiene (HCBd) and Hexachloroethane (HCE) were detected in shallow fill materials, with concentrations decreasing downwards through the fill profile.

Some of the ponds in Southlands were reported to be contaminated with CHCs. Contamination, including CHCs and mercury, was also identified in sediments and surface water sampled from Springvale Drain.

3.4 *Stage 2* Survey - WWC 1996b

The findings and conclusions of the *Stage 2* Investigation with respect to the Southlands area are summarised under the following headings.

3.4.1 Soil Quality

The soil quality investigations undertaken by WWC (1996b) were targeted on the basis of the *Stage 1* Investigation (AGEE, 1990), more specifically on the information taken from the aerial photographs. Overall, the analytical results collected on Southlands indicated that very few volatile or semivolatile compounds were present in the unsaturated soils. The following provides a summary of the analytical results and inferred distribution of soil contamination.

⁶ Where italicised, Stages 1 to 4 refer to previous environmental investigation stages to avoid confusion with the proposed development staging.

Section 3

Summary of Previous Investigations

Block 1

- The soils on Southlands Block 1 were found to contain few volatiles with concentrations greater than 1 mg/kg, with the exception of one sample of peat (SS057) collected from a depth interval of 0.7 – 0.8 m below ground surface level (bgs), in the eastern portion of Block 1. This sample contained concentrations of CHC (including PCE, TCE, 1,1-DCE, cis-1,2-DCE, trans-1,2-DCE and vinyl chloride VC) ranging between 3.5 to 21 mg/kg.
- “Low levels” (<8 mg/kg) of semivolatiles were detected in soils collected from across Block 1. The highest of these concentrations were generally detected in samples collected from a trench excavated immediately east of Springvale Drain in the northern portion of Block 1, in particular from sample SS049, collected from a near surface sample interval.
- HCB, HCD and HCE were the most widely detected semi-volatiles across Southlands.
- An elevated concentration of mercury (70.5 mg/kg) was recorded in a sample collected from the same trench.

Block 2

- CHCs were generally not detected in the fill material layer. CHC were detected at three of the sampling locations, including:
 - VC (0.03 mg/kg) at a depth of 0.7 m at sampling location TP1, located in the mid western portion of Block 2;
 - 1,1-DCE was detected (0.03 mg/kg) at a depth of 0.4 m at sampling location TP3 located at the centre of the northern boundary and was also detected (0.07 mg/kg) at a depth of 1.2 m at sampling location TP4 in the north eastern portion of Block 1.
- The semivolatiles HCB, HCE and HCBd were detected at ‘low’ concentrations (< 2 mg/kg) in samples collected from the northern and eastern sections of Block 2;
- Mercury was detected in most of the soil samples collected from Block 2. The highest concentration (66 mg/kg) was recorded in a sample collected from a depth of 1.4 m in the south eastern portion of the site, within a location identified as being the site of a filled peat excavation.
- Chromium was also detected across the Block 2, with the highest concentration (46.6 mg/kg) recorded at the northern boundary.

3.4.2 Dense Non Aqueous Phase Liquids (DNAPL)

- DNAPL was positively identified at:
 - Monitoring well WG67D, located on the State Rail Authority (SRA) land, adjacent to the former effluent treatment plant to the north east of Southlands;
 - Monitoring well WG82D, located on Southlands, Block 1; and
 - Bundle piezometer SB1 on Block 1 Southlands.
- It was concluded that the most significant potential areal extent of DNAPL was between the former Solvents Plant and Springvale Drain, however, more detailed delineation was not possible at that stage.

Section 3

Summary of Previous Investigations

3.4.3 Groundwater and Surface Water Quality

- Dissolved phase CHCs, in particular 1-2-dichloroethane (EDC), were detected in shallow and deep groundwater; and
- Dissolved phase CHCs were detected in surface water (Springvale Drain).

3.4.4 Sediment Quality

- CHCs, mercury and chromium were detected in sediment collected from Springvale Drain; and
- CHCs and mercury were detected in sediment collected from elsewhere on Southlands.

3.5 Stage 4 Investigations – URS 2004

3.5.1 URS 2004a – Full Scale Reactive Iron Barrier Investigations

URS assessed alignment options for the Full Scale Reactive Iron Barrier (FSRIB). In investigating the geochemical and geotechnical conditions of the north-eastern corner of the site, the following findings related to groundwater contamination were noted:

- TCE was detected and was inferred to be associated with the Central EDC Plume;
- 1,1,2,2-Tetrachloroethane (1,1,2,2-TeCA) concentrations were lower than previously reported; and
- EDC was present at high concentrations (>2,000 mg/L).

Occasional groundwater sampling continues at the PSRIB to monitor the effectiveness of the barrier system in the long term and more recently Orica has examined the possibility of contributing in-situ biological degradation in the barrier wall.

3.5.2 URS 2004b – DNAPL Source Area Investigation

This investigation was aimed at identification of DNAPL source areas to enable assessment of potential containment and remediation options. The investigation comprised bore drilling down gradient of the former Solvents and TCE/PCE Plants.

Report findings in relation to the Southlands site included:

- The lateral extent of the DNAPL zone on the north western portion of Block 1 was relatively well defined, however, the vertical distribution was complex;
- DNAPL in Block 1 was present as thin (<0.1 m) accumulations.

The report concluded that containment would be the most effective means of minimising potential migration of contaminants from a DNAPL source area. Additionally, other primary methods of containment including pumping and secondary treatment methods including flushing, thermal and chemical oxidation degradation were identified.

3.5.3 URS 2005a - DNAPL Source Area Delineation Investigation

Investigations completed indicated the following in respect of the Southlands site:

- Evidence of DNAPL was not identified in core holes located on Southlands site, down gradient of free phase DNAPL identified at BP34 and WG13 and down gradient of the former Solvents Plant. This indicated that the S2 DNAPL source area may not extend as far southwest as Southlands.

Section 3

Summary of Previous Investigations

3.5.4 URS 2005b Progress report – Southern Plumes DNAPL Source Area Delineation Investigation

This investigation did not specifically target the inferred S1 DNAPL source area. The focus of the investigations was the further delineation of the S2 and S3 DNAPL source areas.

3.6 URS 2006 Southern Plumes Source Area Delineation Investigation

The objectives of the URS (2006) investigations included:

- Establishment of the northern boundary of the DNAPL S1 source area located on the Southland site; and
- Better characterisation of the S1, S2 and S3 source areas and/or dissolved phase plumes in the north east corner of Block 1.

Investigations included:

- the installation and sampling of monitoring wells BP103 and BP104 along the northern boundary of Southlands Block 1; and
- the installation and sampling of monitoring wells BP105 and BP106 along the north east boundary of Block 1.

DNAPL was observed in the newly installed location BP105 at various depths along with high dissolved phase CHC concentrations. The range of volatile and semi-volatile CHCs present in groundwater and DNAPL at BP105 included volatile CHCs: CTC, CFM, EDC, 1,1,2,2-TCA, PCE and TCE and semi-volatile CHCs: HCB, HCBD and HCE. The DNAPL was predominantly PCE and the DNAPL contamination composition was indicative of contamination from the former BIP Solvent Plant. The exact mechanism under which DNAPL reached the S1 source area is not known.

Given the age of the source (decades) it is likely that a significant mass of the residual DNAPL in the highly permeable zones has dissolved into the Southern Plumes.

3.7 Preliminary Soil Investigations – HLA 2005

HLA Envirosciences (HLA) was engaged by Orica in 2005 to conduct a Preliminary Soil Investigation (PSI) at Southlands. The sampling locations are shown on Figures 8 and 9.

HLA notes in their report (HLA, 2005) that the sampling regime employed was not designed to meet the requirements of the NSW EPA Contaminated Sites: Sampling Design Guidelines (NSW EPA, 1995).

3.7.1 Scope of Work

The scope of the investigation included the following components:

- Collection of soil / fill samples from approximately 78 test pits, the locations of which are shown on Figures 8 and 9;
- “Broad” characterisation of the fill stockpiles located across the site; and
- A limited program of water leach tests tests under neutral conditions.

3.7.2 Potentially Contaminating Activities

- Historical information was identified which suggested APM had discharged paper waste slurries directly into some of the ponds and pits across the site;

Section 3

Summary of Previous Investigations

- A summary of historical reviews undertaken in previous investigation reports noted that in the past, effluent disposal from the ICI Botany Plant [now BIP] was via a stormwater pipe that discharged directly into Springvale Drain. Prior to installation of the stormwater pipe, some of the effluent may have flowed over parts of Block 1;
- The site was subjected to uncontrolled dumping up until 1991, when a boundary fence was erected;
- Fuel pipelines (noted in the HLA report (HLA, 2005) as jet fuel pipelines) located along the eastern margin of Nant Street, were identified as potential contaminating sources, in the event of leakage;
- An historical diesel spill from a former StateRail Rail Authority (SRA) tank, located immediately north-east of Block 1, was also identified in the report;
- Anecdotal information, suggesting contaminated groundwater from pits located on the EnergyAustralia property located to the north – east of the site, was cited.

3.7.3 Contaminants of Potential Concern (CoPC)

Based on the results of earlier investigations and on consideration of the identified contaminating activities, the following CoPC were identified by HLA:

- Heavy metals (As, Cd, Cr, Cu Ni, Pb, Zn and Hg);
- Volatile chlorinated hydrocarbons;
- Semi-volatile chlorinated hydrocarbons;
- Petroleum hydrocarbons;
- Benzene, toluene, ethylene, xylene (BTEX) compounds;
- Polycyclic aromatic hydrocarbons (PAH);
- Asbestos; and
- Phenols.

3.7.4 PSI (HLA, 2005) Findings – Block 1

Soil Contamination

The report conclusions with regard to soil contamination included the following:

- **Heavy metals**
 - Mercury concentrations above the adopted investigation levels⁷ (IL) were identified in soil in the south western portion of Block 1 and along the eastern side of Nant Street. Limited water leach testing indicated the mercury was leaching under neutral conditions; and
 - An elevated lead concentration (exceeding the adopted site IL) was recorded in soils, near the southern boundary of Block 1.

⁷ The National Environmental Protection Council (NEPC) *National Environmental Protection (Assessment of Site Contamination) Measure, 1999* (NEPC, 1999) Health Based Investigation Level (HIL F) for Industrial Commercial Land uses were used in the HLA assessment.

Section 3

Summary of Previous Investigations

• Petroleum Hydrocarbons

- Concentrations of TPH (Fractions C₆ to C₃₆) and BTEX were detected in samples collected from the south western portion of Block 1; and
- TPH (Fractions C₁₀ to C₃₆) contamination in soils (considered likely to be related the former RailCorp Diesel Tank Spill) was also identified in the eastern portion of Block 1.

• Volatile and Semivolatile CHCs

- Concentrations of semivolatiles, including hexachlorobenzene (HCB), above the adopted site ILs⁸ were recorded in samples collected from the south western and eastern portions of Block 1; within the zone identified (in the HLA report) as the EnergyAustralia pump out zone.

• Asbestos

- Numerous instances of potential asbestos containing materials (ACM) were noted in the fill layer during the investigations.

Stockpile Contamination

With regard to stockpile sampling, the report conclusions included the following:

• Volatile and Semivolatile CHCs

- CHCs (TCE and 1,1,2-TCE) were detected above the adopted investigation levels in two of the stockpiles, located in the north eastern (SP28) and southern portions (SP30) of Block 1 respectively;

• Asbestos

- Asbestos fibres were detected in both a fragment of fibrous cement sheeting and in a soil sample collected from a stockpile (SP15), located in the northern portion of Block 1.

Sediment

Elevated concentrations of metals (As, Cd, Cr, Cu, Ni, Pb, Zn and Hg) and PAHs, which exceeded the ILs⁹ adopted by HLA, were recorded in a limited number of pond sediment characterisation samples. Volatiles (CHCs) were also detected in the sediments, however, screening IL for these compounds were not provided in the adopted guidelines

3.7.5 PSI Findings – Block 2

Soil Contamination

The report conclusions with regard to soil contamination included the following:

• Heavy metals

- A mercury concentration above the adopted site investigation levels was identified in a soil sample collected from a test pit (TP50) located in the in the north eastern portion of Block 2.

⁸ The USEPA Preliminary Remedial Goals – Region IX were adopted for assessment

⁹ For assessment of sediment quality, HLA adopted the guidelines provided in the ANZECC / ARMCANZ (2000) Australian Water Quality Guidelines for the Protection of Aquatic Ecosystems

Section 3**Summary of Previous Investigations**

- **Volatile and Semivolatile CHCs**

- A HCB concentration which exceeded the adopted site investigation level was recorded at test pit TP50, located in the north-east of Block 1.
- Bis (2-chloroethyl) Ether was detected above adopted site investigation level at test pit location TP52, also located in the north eastern portion of Block 2

- **Asbestos**

- Asbestos fibres were detected in a fragment of bonded fibrous cement and in the fill material at test pit TP46 and was detected in the fill soil at test pit TP65, located in the western portion of Block 2.

Stockpile Contamination

With regard to stockpile sampling, the report conclusions included the following:

- **Volatile and Semivolatile CHCs**

- Concentrations of HCB, which were marginally above the adopted site IL were detected in stockpile sample SP34, located in the south-eastern portion of Block 2.

- **Heavy Metals**

- A lead concentration which exceeded the adopted site IL was detected in a single stockpile sample (SP50), located in the centre of Block 2.
- Mercury was identified to be water leachable under neutral conditions for a soil sample collected from stockpile SP30, located in the south-western portion of Block 2.

Sediment Contamination

Sediment sampling was not undertaken on Block 2.

3.7.6 Additional Soil Quality Investigations - URS 2006

URS undertook a program of additional soil investigation in October 2006 based on the findings of the PSI (HLA, 2005). At the time of the investigation, the remediation approach for Southlands was not fully developed and investigations were considered warranted to gain an improved understanding of the nature and extent of shallow soil contamination on Southlands.

It is noted however, that the risk assessment program was not complete at this time. Therefore, the derivation of risk based soil criteria (RBSC) had not been undertaken and not all areas of concern relating to soil contamination had been identified, in particular on Block 2. Figures 8 and 9 show the areas where exceedances of the RBSC have been identified and require remediation.

Figure 8 shows the areas of interest targeted on Block 1 in the additional investigations, which were identified as Zones A, B and C. The sampling locations were primarily selected to investigate areas where shallow soils had been shown (in the PSI) to be contaminated as summarised below. Limited additional investigations were also undertaken on Block 2 and are shown on Figure 9.

Area A (Central Southern Portion of Block 1)

- Semivolatiles (HCB and HCBd);
- Volatiles (CHCs including EDC, TCE, PCE, VC and trichloromethane (CFM));
- Petroleum hydrocarbons (measured as TPH);
- Metals; and

Section 3

Summary of Previous Investigations

- Asbestos.

Area B (South Western Portion of Block 1)

- Petroleum hydrocarbons (measured as TPH);
- Volatiles (CHCs including TCE, 1,1,2-TCE, PCE and VC);
- Semivolatiles (HCB);
- Metals (mercury); and
- Asbestos.

Area C (East of the Northern Portion of Springvale Drain)

- Metals (mercury);
- Semivolatiles (HCB and PAHs); and
- Asbestos.

Other Isolated Areas

- Volatiles (CHCs including VC, TCE and EDC); and
- Semivolatiles (HCB and PAHs).

3.7.7 Methodology

Investigations were undertaken using test pitting techniques, with limited shallow samples in Area A collected using hand augering techniques. The investigations were primarily targeted at the fill materials, however, deeper apparently “*natural*” samples were also collected and analysed.

A total of 115 soil samples were collected from 54 investigation locations and were analysed (as appropriate) for suites of analytes reflecting the potential contaminants identified above.

3.7.8 Site Investigation Levels

The soil quality data were assessed against environmental guidelines appropriate for the proposed development land use (Industrial/Commercial).

The primary guidelines adopted for the assessment were:

- The National Environmental Protection Council (NEPC) *National Environmental Protection (Assessment of Site Contamination) Measure, 1999* (NEPC, 1999) Health Based Investigation Level (HIL F) for Industrial Commercial Land uses.

This guideline (NEPC, 1999) document does not provide appropriate investigation levels for some of the CHCs. Guidelines for these compounds were therefore adopted from:

- The USEPA Preliminary Remedial Goals – Region IX US Environmental Protection Agency (US EPA), 2004.

The NEPM guidelines (NEPC, 1999) provide HILs for total petroleum hydrocarbons (TPH) as aliphatic and aromatic hydrocarbons for a range of land uses. The analysis of aliphatic and aromatic hydrocarbon fractions is not the routine analytical method for TPH. Additionally, the NEPM does not include guidelines for BTEX. Guidelines for TPH (C₆-C₉ and C₁₀-C₃₆ fractions) and BTEX have, therefore, been adopted from:

Section 3

Summary of Previous Investigations

- NSW EPA Contaminated Sites: Service Station Guidelines (NSW EPA, 1994) (the “*Service Station Guidelines*”).

The *Service Station Guidelines* specify ‘Threshold Concentrations for Sensitive Land Use – Soils’ for TPH (C₆-C₉ and C₁₀-C₃₆ fractions) and BTEX.

It is noted that the guidelines adopted by URS (URS, 2006) are the same as the guidelines adopted by HLA in the PSI (HLA, 2005). The URS (2006) soil data are described in the following sections with reference to the adopted guidelines.

3.7.9 Area A

The analytical results for samples collected from across Area A are summarised as follows:

Semivolatiles Compounds

- These compounds were generally recorded in soils at concentrations less than the laboratory detection limits and the respective site adopted investigation guidelines, where available.
- HCB was detected at a concentration of 1.6 mg/kg and exceeded the site adopted guideline (USEPA, 2004) (1.1 mg/kg) in a near surface soil sample collected by hand auger from location HA006. All other HCB results were less than the laboratory detection limit.

Chlorinated Hydrocarbons

CHCs were detected in several soil samples collected from across the area.

- 1,1,2,2-TeCA was detected in soil samples collected in Area A. Concentrations exceeded the site adopted guideline (0.93 mg/kg) (USEPA, 2004) at two of the investigation locations, TP105 and TP106. Soil sample concentrations were otherwise less than the laboratory detection limit.
- EDC was detected in soil at concentrations that marginally exceeded the site adopted guideline (0.6 mg/kg) (USEPA, 2004) at two investigation locations, TP105 and TP107.
- Cis-1,2-DCE was detected in several of the soil samples, with concentrations ranging between 0.5 mg/kg and 62.2 mg/kg. None of the soil concentrations exceeded the site adopted guideline concentration of 150 mg/kg (USEPA, 2004).
- PCE soil concentrations were generally less than the site adopted guideline concentration (USEPA, 2004) (1.3 mg/kg) but was detected at concentrations in excess of the site adopted guideline at sampling locations TP105, TP106 and HA001.
- TCE soil concentrations were detected in excess of the site adopted guideline concentration (USEPA, 2004) (0.11 mg/kg) at several of the locations in Area A, including TP105, TP106, TP107, TP110, TP118, TP120, TP126 and HA001, HA005 and HA006.

Total Petroleum Hydrocarbons

TPH (C₆ to C₉ range) soil concentrations were generally less than the adopted IL (NSW EPA, 1994) (65 mg/kg), with the exception of a soil sample collected from sample location TP105, which contained a concentration of 102 mg/kg. Slightly elevated concentrations, which were less than the site adopted guideline (NSW EPA, 1994), were recorded in several soil samples collected from this area. It is noted that the elevated TPH (C₆ to C₉ range) concentrations may be related to the detection of chlorinated hydrocarbons.

Section 3

Summary of Previous Investigations

TPH (C₁₀ to C₃₆ range) soil concentrations exceeded the site IL (NSW EPA, 1994) (1,000 mg/kg), at several of the sample locations located in the eastern portion of Block 1, including:

- TP110_1.9 (2,640 mg/kg);
- TP117_1.0 (2,530 mg/kg);
- TP118_0.9 (1,170 mg/kg);
- TP120_1.4 (2,110 mg/kg);
- TP121_0.9 (3,770 mg/kg);
- TP123_0.9 (2,970 mg/kg); and
- TP123_1.5 (2,440 mg/kg).

Heavy Metals and Metalloids (As, Cd, Cr, Cu, Ni, Pb, Zn & Hg)

- A lead concentration (7,670 mg/kg) recorded in a soil sample collected from a depth of 1.1 m below ground surface level BGSL at investigation location TP127 exceeded the site IL (NEPC, 1999) for lead (1,500 mg/kg).
- All other soil sample heavy metal and metalloid concentrations were below the respective site adopted guideline concentrations.

Asbestos

Potential asbestos containing materials (ACM) were not identified during site works. Nine of the fill samples were randomly selected and submitted for asbestos analyses. Asbestos fibres were positively identified in one of the nine samples. The sample was collected from a depth of 1.2 m BGSL at sample location TP109, located in the south-east of Block 1.

3.7.10 Area B

The analytical results for samples collected from across Area B are summarised in the following.

Total Petroleum Hydrocarbons

- TPH soil concentrations were generally less than the laboratory detection limits.
- A TPH (C₆ to C₉) concentration of 137 mg/kg, which exceeded the IL of 65 mg/kg (NSW EPA 1994) was recorded in a sample collected from a depth of 2.7 m at investigation location TP087, located in the south western corner of Block 1.
- A TPH (C₁₀ to C₃₆) concentration of 3,530 mg/kg, which exceeded the IL of 1,000 mg/kg (NSW EPA 1994) was recorded in a sample collected from a depth of 1.0 m at investigation location TP089.

It is considered likely the TPH concentrations could be related to localised leakage from the fuel pipeline that runs beneath Nant Street. The RAP makes provision for remediation of the contaminated soils in this area (see Area B on Figure 8). However, assessment of the pipe line integrity should also be undertaken to assess whether a continuing source of fuel related contamination exists in this area. Excavation of the contaminated soil may provide an opportunity for visual assessment of this section of the pipe, however, more detailed integrity testing may also be warranted.

Section 3

Summary of Previous Investigations

Chlorinated Hydrocarbons

- CHCs were not detected in the soil samples collected from Area B.

Semivolatiles (HCB)

- Semivolatiles were not detected in the soil samples collected from Area B.

Heavy Metals and Metalloids (As, Cd, Cr, Cu, Ni, Pb, Zn & Hg)

- A limited number of the samples were submitted for metals/metalloids analysis. For all the samples analysed the heavy metal and metalloid concentrations were below the respective site adopted guideline concentrations.

Asbestos

Potential asbestos containing materials (ACM) were not identified during site works. Three of the fill samples were randomly selected and submitted for asbestos analyses. Asbestos fibres were not detected in these samples.

3.7.11 Area C

Mercury

- Mercury was detected at concentrations in excess of the site adopted guideline concentration (75 mg/kg) for soil samples collected at the following locations and depths:
 - TP094 at a depth of 0.9 m (135 mg/kg);
 - TP098 at a depth of 1.0 m (82 mg/kg);

Polycyclic Aromatic Hydrocarbons

- Benzo(a)pyrene was recorded at a concentration equivalent to the IL (5 mg/kg) for the soil sample collected at sample location TP094 at a depth of 0.9 m.
- A total PAH concentration of 62.5 mg/kg was recorded in the same sample, but this concentration was less than the IL of 100 mg/kg.

Asbestos

Potential asbestos containing materials (ACM) were not identified during site works. Three of the fill samples were randomly selected and submitted for asbestos analyses. Asbestos fibres were positively identified in two of the three samples, collected from sample locations TP094 and TP098. The samples were collected from depths of 0.9 and 1.0 m BGS� respectively.

Section 4

Remediation Requirement

4.1 Preliminary Human Health Risk Assessment

The Southlands risk assessment process followed an iterative approach. The available site data (summarised above) was initially considered in a Preliminary Human Health Risk Assessment¹⁰ (PHHRA, URS, 2007b), a copy of which forms Appendix H of the HHRA. The PHHRA identified a number of key issues that required further assessment as well as consideration of risk management measures prior to completion of the HHRA.

The key issues identified included the potential for shallow groundwater to discharge to Springvale Drain (resulting in emissions to air of volatile chlorinated compounds), potential for shallow groundwater to discharge into proposed compensatory flood storage areas (resulting in emissions to air of volatile chlorinated compounds) and presence of residual impacts in soils on the site.

4.2 Remediation Requirement

As described in Section 1.3, the Proponent proposes a staged development of the site for a commercial / industrial land use, specifically, construction of an industrial estate. The Project development scenarios (Stages 1 and 2) are presented in Figures 3 and 4.

The PHHRA (URS, 2007b) indicated the site is not suitable in its current condition for the proposed development land use. Remediation (and/or management) of contamination was considered to be required to render the site suitable for the proposed land use.

4.3 Remediation Objective

The remediation objective is to remediate (and/or manage as appropriate) identified contamination so as to render the Southlands site suitable for the proposed commercial / industrial development land use, in accordance with the relevant DECC, NSW endorsed guidelines.

4.4 Guidance Framework

The approach outlined in the NEPM (NEPC, 1999) for the development of a remediation strategy is to adopt a risk based approach taking into account the nature of the contaminants on the site and the proposed development land use.

The approach adopted in this RAP is consistent with:

- the strategy outlined in the Australian and New Zealand Guidelines for the Assessment and Management of Contaminated Sites (ANZECC/NHMRC, 1992) and as adopted by NSW Site Auditor Scheme (DEC, NSW, 2006); and
- the attainment of Environmental Outcomes described in the NEPM (NEPC, 1999).

The DECC NSW preferred position on the selection of remediation options is:

- If remediation were likely to cause a greater adverse effect than would occur if the site were left undisturbed, then it should not proceed.

The preferred hierarchy of options for site clean-up and management is:

- On-site treatment of soil, so that the contaminant is either destroyed or the associated hazard is reduced to an acceptable level, and

¹⁰ The PHHRA forms an appendix (Appendix H) to the HHRA (URS, 2008a) which is presented in Appendix A.

Section 4

Remediation Requirement

- Off-site treatment of excavated soil, so that the contaminant is either destroyed or the associated hazard is reduced to an acceptable level, after which the soil is returned to the site.

If these options cannot be implemented, then other options that should be considered include:

- Removal of contaminated soil to an approved site or facility, followed, where necessary by replacement with clean fill; and
- Consolidation and isolation of the soil on site by containing with a properly designed barrier.

The following section outlines a series of site specific constraints to selection of a suitable remediation approach. They include constraints associated with requirements for flood mitigation, the nature of the contaminants and the requirements for accommodation of existing infrastructure. In addition, the site area and distribution of the contaminants introduce further site specific constraints. In this regard, the following section outlines the rationale for the selection of the fourth above mentioned and least preferred option for the site remediation and management.

Section 5

Site Development Considerations

The following section outlines site specific development considerations and constraints. It is noted that there are several site specific requirements arising from:

- the Environmental Assessment Requirements (EAR);
- the specific nature of the contaminants involved; as well as
- Specific land management issues identified by the Proponent for future use and management of the land.

5.1 Site Flooding Solution

Connell Wagner (CW) was engaged by the Proponent to develop a flooding solution for the site. The details and findings of the flooding study are presented in the Connell Wagner report entitled:

- Orica / Goodman Southlands Remediation / Development Project Flood Investigation (2 November 2007).

The study (CW, 2007) determined that the modelled 1:100 year flood design level is higher than the existing mean site elevation. The report indicates the 1% AEP (annual exceedance probability) across the site ranges between 4.4 mAHD and 4.9 mAHD (incorporating a 300 mm freeboard). The existing site surface elevation on Block 2 varies, but reaches relative low points in the order of 3.6 mAHD near the centre.

5.1.1 Stage 1 Development

The main outcome of the flood study with regard to the Stage 1 Development is therefore a requirement to increase the elevation of the Block 2 site surface so as to meet the 1:100 year design flood level, while maintaining compensatory flood storage capacity on other areas of the site. CW proposes this will be achieved through placement of up to 1 metre (m) thickness of imported material across Block 2, while providing a compensatory flood storage area on the southern portion of Block 1 (future Stage 2 area) until further development of that area proceeds and flood retention requirements for the site are met through other means. Earthworks will therefore occur on the southern portion of Block 1 as part of the Stage 1 development.

The flood storage capacity of this area will be augmented by construction of an engineered berm across the southern (McPherson Street) boundary of Block 1 as part of Stage 1.

5.1.2 Stage 2 Development

Development of Stage 2 will require filling of Block 1 to a level similar to that proposed for Block 2 in Stage 1 (CW, 2007).

The Stage 2 development will require additional flood capacity and this is likely to be achieved through development of a new flood storage area on Block 1 and/or works on downstream (off-site) stormwater assets to increase capacities and stormwater flows from the site.

5.2 Vapour Risk Considerations

The PHHRA identified a number of key issues for site development. In particular, issues associated with the generation of vapours where shallow groundwater discharges to the site surface were noted. These issues included the potential for shallow groundwater to discharge to Springvale Drain (resulting in emissions to air of volatile chlorinated compounds) and the potential for shallow groundwater to discharge into proposed compensatory flood storage areas (resulting in emissions to air of volatile chlorinated compounds).

Section 5

Site Development Considerations

5.2.1 Springvale Drain

The results of ambient air monitoring activities undertaken between early 2005 and late 2007 have detected elevated concentrations of volatile organic compounds (VOCs) in ambient air in the vicinity of Springvale Drain.

A series of investigations completed since the mid-1990's have demonstrated that shallow groundwater discharge is the likely primary cause of chlorinated hydrocarbon (CHC) contamination in surface water which is believed to be the main driver for the presence of volatile organic compounds (VOC) in air near Springvale Drain on the Southlands site.

The PHRA (presented as an Appendix to the Southlands HHRA) showed that ambient air concentrations would preclude the proposed land use and that mitigation would be required to render the site suitable for development.

Section 7.1 describes assessment works undertaken to date, which have involved a detailed monitoring program. It also presents a remediation approach aimed at reducing (or precluding) shallow groundwater discharge into the drain to minimise the potential for volatilisation of contaminants from surface water.

The monitoring works completed to date have also shown that based on comparison of CHC concentrations in surface water samples collected from the realignment channel and Springvale Drain respectively, it was considered that ambient air concentrations recorded on Southlands are affected by vapours emanating from the realignment channel. Given the large difference in water concentrations, it was considered likely that the prime contribution to the CHCs in air is the realignment channel.

The remediation strategy will make provision for mitigation of these elevated concentrations.

5.2.2 Compensatory Flood Storage Area

Several investigations undertaken since the late 1980s have shown the depth to groundwater on Block 1 to be quite shallow, in certain instances less than 0.5 metre. Work undertaken by JBS Environmental Pty Ltd (JBS) considered historical standing water level data recorded on Block 1 Southlands as part of the Quarterly GTP Surface and Groundwater Monitoring Program, as well as hydrographs from selected monitoring wells on Block 1, in the context of the proposed finished levels of the compensatory flood storage areas. The report (JBS, 2008) indicated that '*measured water levels have historically exceeded the proposed base of the flood storage basin at a number of locations*'. The report (JBS, 2008) concluded '*there is potential for groundwater to discharge into the proposed storage basin*'.

Ponding of contaminated groundwater within this area (or any area on Southlands) would pose unacceptable risks to human health (URS, 2008a). The site development detailed design will therefore need to include provision for system that precludes mixing of groundwater with the flood waters that will periodically be contained in the flood compensatory area on Stage 1 and/or will site surface waters. More details on these requirements are provided in Section 7.3.

5.2.3 Groundwater

Impacted groundwater within the shallow, intermediate and deep aquifers is present beneath both Block 1 and Block 2 of the Southlands site. The main contaminants identified in groundwater are derived from the central plumes dominated by EDC, PCE, TCE and VC and the southern plumes dominated by CTC, PCE, TCE and VC, however a range of other chemicals have also been reported in groundwater that may be derived from former operations at the BIP as well as surrounding industrial areas. The Stage 2 Investigations (Woodward-Clyde, 1996a) and subsequent DNAPL investigations (URS (2004b) and URS (2006)) also reported the presence of DNAPL (i.e. free phase) at depth within the northern portion of Block 1 (Redevelopment Stage 3).

As discussed in Section 5 of the HHRA (URS, 2008a, refer Appendix A), the most significant exposure pathway associated with the presence of contamination in groundwater that is considered complete is the inhalation of vapours from volatile chemicals that may be present at the surface of the groundwater

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(shallow aquifer), following vapour migration through overlying soils into overlying buildings, outdoor air and excavations.

5.3 Existing GTP Infrastructure

With respect to ongoing BIP groundwater remediation programs, development requirements are chiefly those associated with continued operation of the existing GTP, as well as adequate provision for ongoing monitoring of the hydraulic regime and groundwater quality across Southlands, which forms part of Orica's ongoing GTP Groundwater and Surface Water Quality Program (Orica 2005). The existing GTP infrastructure is shown on Figure 5.

The primary purpose of the GTP is to effect hydraulic containment of contaminated groundwater at strategic locations. This is achieved through large scale groundwater pumping and delivery of the extracted water to the treatment plant, located on Orica BIP, via a series of above and below ground pipelines. .

The pumping infrastructure comprises three main groundwater extraction areas, each of which comprises a series of groundwater extraction bores and groundwater monitoring wells including:

- **The Botany Industrial Park (BIP) Containment Area**, located along the south western boundary of BIP and designed to intercept and contain contaminated groundwater at the western boundary of the BIP site;
- **The Primary Containment Area (PCA)** is located along the southern (McPherson) boundary of Southlands. It is noted the PCA also includes three other extraction bores, located in the central southern portion of Southlands Block 2 (EWB02, EWB06 and EWB14D); and
- **The Secondary Containment Area (SCA)**, located along the centre of Foreshore Road.

Therefore, the site remedial approach needs to make provision for the PCA infrastructure.

5.4 Groundwater Monitoring Infrastructure

As discussed in Section 1.6, quarterly groundwater monitoring is a requirement of the Notice of Cleanup Action (NCUA) issued by the DECC, NSW for BIP. A portion of the required monitoring infrastructure is located on Southlands, as shown on Figure 6. This network of shallow, intermediate and deep groundwater monitoring wells is designed to enable monitoring of both the hydraulic containment performance of the GTP systems as well as periodic (quarterly) 'chemical' monitoring of groundwater quality.

It is noted that the monitoring well network on Southlands comprises wells from a variety of historic investigations as well as those installed for the specific purpose of hydraulic monitoring. The locations of the proposed buildings and infrastructure across Southlands will require replacement of much of the groundwater monitoring infrastructure.

The remedial approach for the site will therefore need to design a replacement well network, which targets key areas for the purpose of hydraulic and chemical monitoring and provide a framework for ensuring that continued access to these wells is achieved. It will also make provision for decommissioning of the existing wells.

5.5 Nant Street Tank Farm Pipelines

Two north-south trending underground pipelines, linking the Nant Street Tank Farm to the north of Southlands with the Caltex Terminal location to the south, run beneath the unformed Nant Street. These Mobil/Caltex shared pipelines include a 6" white oil line and a 10" white oil line. Two AGL gas lines also occupy the Nant Street easement. Soil quality investigations (see Section 3) have shown hydrocarbon related contamination exists in the south eastern portion of Block 2 and it is probable this is related to

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localised leaks from the pipelines. Future access to the pipeline, as well confirmation of whether it is an ongoing source of contamination, needs to be considered in the remediation approach for the site.

5.6 Easements for Groundwater Monitoring and Future Remediation

The Proponent has identified a requirement for continued ownership (after development) of a series of easements at the site for use in both groundwater monitoring and extraction as well as for future remediation technologies, should such become available or relevant to remediation of the site and/or other areas in BIP.

The locations of the proposed easements are indicated on the site development plans (Figures 3 and 4) and are shown in isolation on Figure 10. The easements are generally located at the site boundaries but also include easements that run through the centre of the site (Easements 2 and 9) and Easements 3 and 4, located to the east of Nant Street.

The following table provides a summary of the intended purposes for each of the easements.

Table 5-1 Easement Requirements

Purpose	Easement Numbers (see Figure 10)
To enable continued operation of Primary Containment Area	Easement Nos. 2, 6, 7 and 8
To facilitate future (new or complimentary) remedial works on the site	Easement Nos. 1, 2, 3, 4, 5 and 9
To allow access to the entire site for installation and sampling of monitoring wells as required (post development)	General "easement" or contractual agreement (site access) across the site

5.7 Asbestos Containing Materials (ACM)

Relatively limited site soil investigations (HLA, 2005) (URS, 2006) undertaken to date have shown that the surface fill and stockpiles on Block 2 and (to a lesser extent) Block 1 are impacted with ACM. The ACM generally comprise (and in some cases are derived from) fragments of AC sheeting ('*fibro-board*'). They are considered likely to be a legacy of the period prior to Orica's ownership during which dumping was common practice.

The numbers of positive identifications of ACM have been relatively few, however, in the context of both the land area in question (18 ha.) and the known volume of historically deposited material, the investigations completed to date have been relatively limited. Furthermore, a significant number of potential ACM identifications (i.e. through visual observation rather than laboratory analyses) have been made both during soil investigations and other incidental activities on site.

Given deposition of the stockpile and fill material occurred in an uncontrolled manner, over an extensive period of time, it is not possible to rule out the potential for isolated "*pockets*" of more heavily contaminated soil or buried sheets of ACM to exist.

Insufficient data exists to draw a conclusion that the occurrence of ACM is isolated and it is considered a significant level of further investigation and eventual validation would be required, if a remediation approach based on hotspot removal was proposed.

Therefore, the presence of confirmed ACM in surface or near surface materials across the large area occupied by the site (>18 ha) is considered to be a development constraint in the context of the likely costs involved to render the site as suitable for development through remedial approaches other than on-site containment. The magnitude of this constraint provides further justification for selection of the least preferred site remediation and management option as listed in Section 4.4.

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5.7.1 Asbestos Management Guidance

The following guidance from the DECC, NSW and EnHealth on the development of remedial approaches where management of asbestos contaminated (or suspected asbestos contaminated) materials is required is considered relevant to the development of a suitable remedial approach for the site.

DECC, NSW

The DECC, NSW generally adopts a “zero-tolerance” approach for management of sites contaminated with ACM and an objective of “no asbestos in surface soil” was provided by NSW EPA to Accredited Site Auditors at a Site Auditor’s meeting held on 12 September 2003.

The DECC, NSW has not sanctioned the retention of asbestos or asbestos containing materials at depth.

The DECC, NSW Contaminated Sites: Guidelines for Site Auditor Scheme (DEC, NSW, 2006) notes that *‘until such guidelines become available, auditors must exercise their professional judgment when assessing whether a site is suitable for a specific use in light of evidence that asbestos may be a contaminant of concern’*. Auditors are further advised to consult with NSW Health *on a case by case basis*.

EnHealth 2005

The Department of Health and Ageing Guidelines: Management of Asbestos in the Non-occupational Environment (enHealth, 2005) provides guidance for development of for site-specific approaches for the management of ACM in soils.

In considering risk management options to control exposure to non-occupational sources of asbestos fibres, the document identifies the following points for consideration in development of a suitable approach:

- Proposed and current land use;
- Current or potential child occupancy or access;
- Information from risk assessment;
- The classification of contaminated land and its effect on property values;
- Available technology and cost; and
- Community support or concern.

The document references the preferred options for site clean up identified in the NEPM (NEPC, 1999) (see Section 4.4) and provides the following additional options for management of asbestos:

- Isolating the soil by covering with a properly designed barrier;
- Choosing a less sensitive land use to minimise the need for remedial works;
- Leaving contaminated material “in-situ” providing there is no immediate danger to the environment or community and the site has appropriate controls in place; and/or
- Removing contaminated soils to an approved site or facility followed, where necessary, by replacement of clean fill.

The document identifies advantages and disadvantages associated with adoption of the first management option of covering with a properly designed barrier. The advantages include:

- Such isolation will stabilise material and prevent disturbance and release of asbestos dust;
- Erosion and drainage can be controlled;

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- The area will not significantly disturbed in the future; and
- Removal is difficult or not feasible.

The disadvantages include:

- The hazard remains [on-site];
- Cost for large areas may be near removal cost;
- Management plan and public record required; and
- May affect property values.

The guidance provided in the enHealth (2005) document states that the depth of a barrier should be decided on a case by case basis, taking into consideration several factors including the likelihood of future disturbance, the number and type of services beneath the site, requirement for marker layers etc.

5.8 Remediation Approach (Stage 1)

It is considered a remedial approach based on on-site containment through implementation of a physical barrier layer will form the basis for Stage 1 remediation on Block 2. This approach is based up on consideration of the environmental data present in Section 3, the outcome of the PHRA (URS, 2007b) as well as the development constraints outlined above, in particular those associated with:

- The requirement for filling on Block 2 to meet the site flood design levels; and
- The constraints imposed by the presence of ACM.

In this respect, the remediation approach for Southlands will essentially be achieved '*in tandem*' with the flooding solution for the site.

In addition, Section 7 presents further refinement of the remediation approaches to address the requirement for the mitigation of the issues of risks to human health associated with ambient air quality in the vicinity of both Springvale Drain and the proposed compensatory flood storage area on the southern portion of Block 1. These risk issues currently preclude the proposed development land use.

5.9 Remediation Approach (Stage 2)

Stage 2 of the development will be located in the southern portion of Block 1. This portion of the site will be used for flood storage during Stage 1.

Remediation components pertinent to development of Stage 2, which will be completed as part of Stage 1 works will include:

- Backfilling of the paper waste ponds;
- Removal of impacted soils;
- Mitigation of vapour concentrations in the vicinity of Springvale Drain.

Given these components will be achieved during the Stage 1 Development, based on the conclusions of the HHRA (URS, 2008a, refer Appendix A) the main issues remaining will be:

- Management of potential ACM in fill (and remaining stockpile material, if any); and
- Management of the potential for accumulation in buildings (or other structures) of vapours associated with the contaminated shallow groundwater beneath Block 1.

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The proposed flood solution (CW, 2007) requires importation and placement of up to 1 metre of validated fill to raise the site elevation to meet the 1:100 year flood design level. It is proposed that placement of the fill will provide a physical barrier to prevent exposure to potential ACM impacted soils. Vapour mitigation measures will also need to be incorporated into the building design in this area. More details on the vapour barrier design and the remediation scope for Stage 2 are provided in Section 10.

The following sections provide additional detail of the remediation approaches presented above.

Section 6

Physical Separation Approach - Stage 1

The primary consideration for the Block 2 remediation is the on-site management of the asbestos contamination.

The Phase 2 Investigation (HLA, 2005) identified fragments of asbestos on Block 2, but it is considered that based on the heterogeneous nature of the filling, there is potential for more significant quantities of asbestos to be present, both in stockpiles and in the shallow sub surface.

For this reason, and in light of the flood management requirements (CW, 2007), an on-site containment approach has been adopted for Block 2, as outlined in the remediation strategy in Section 5. The ACM will be contained on site and physically separated from future site users through construction of a *physical separation layer*.

6.1 Separation Layer Thickness

6.1.1 Flood Study (CW, 2007)

As outlined in Section 5.1, a main outcome of the flood study (CW, 2007) with regard to the Stage 1 development was a requirement to increase the elevation of the Block 2 site surface so as to satisfy the 1% AEP flood design criteria.

The flood study (CW, 2007) has indicated that an increase in the site elevation to between 4.4 and 4.9 mAHD across Block 1 will be required to meet the design flood criteria. Based on existing site elevations, the degree to which the site surface needs to be raised will vary across Stage 1. In some areas the proposed finished elevation may be more than 1 metre higher than existing, while in other areas, particularly in the north eastern portion of Stage 1, less than 1 metre increase in elevation will likely be required.

The raising of the site surface level will be achieved through:

- Limited placement onto Block 2 of materials cut from the southern portion of Block 1, as part of Stage 1 (following removal and off-site disposal and/or treatment of contaminated soil) ;
- Importation and placement of VENM; and
- Construction of a concrete slab across the majority of the finished surface.

6.1.2 Site Filling

All materials used for site filling, other than materials cut from the southern portion of Block 1, will be Virgin Excavated Natural Materials (VENM) as described in the NSW DECC (2008) Waste Classification Guidelines: Part 1 Classifying Waste and as per requirements identified in Section 12.

General Approach

It is envisaged that Block 2 will be prepared by levelling the existing stockpiles across Block 2. Further movement of existing materials within Block 2 and materials cut from Block 1 and transferred into Block 2 will be required to enable placement of a minimum thickness of imported VENM across the site to achieve a uniform physical separation layer. Section 9 provides more detail for this general approach.

Section 6.2 outlines a different approach for the southern edges of Southlands where a physical barrier will not be used due to the presence of GTP infrastructure.

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Physical Separation Approach - Stage 1

The preparation of the pre-filling surface shall enable placement of the following VENM thicknesses immediately beneath the finished surface or surface slab.

Table 6-1 Minimum VENM Filling Thickness - Stage 1/Block 2

Stage 1 (Block 2) Area Description	Minimum VENM Thickness
Areas where concrete slab (assumed minimum concrete slab thickness of 0.1 m) will overlie separation layer	Not less than 0.4 m
Areas where the site surface will not be finished with concrete	Not less than 0.5 m

6.1.3 Marker Layer

A marker layer, such as a geotextile fabric or equivalent, will be laid directly beneath the VENM in areas of Stage 1 where there is potential for future excavation. Such areas will include:

- All areas outside of the main building footprints.

The placement of a marker layer forms part of the 'long-term' environmental management plan (EMP) described in Section 14. The intent of the layer is to indicate clearly the interface between the VENM physical separation layer and the underlying impacted fill soil, including ACM, in those areas where there is potential excavation for:

- General maintenance purposes;
- Installation of groundwater monitoring infrastructure;
- Installation of groundwater extraction infrastructure; and/or
- Any future site remediation activities that require excavation.

While this measure is considered sufficient for the purposes of the currently proposed development, it is noted that the targeted placement of a marker layer may make minor changes to the development footprint and/or future site re-development difficult, if not impossible.

The Proponent may therefore consider placing marker layer across the whole of Stage 1 Area (and later Stage 2 Area). This would allow for minor changes to the development plan and for future site developments to occur, without the restrictions imposed by a targeted placement of the marker layer materials.

6.1.4 Other Design Features

The development will incorporate localised depressions of the finished site surface to allow construction of features such as:

- Truck Loading / Unloading Bays; and
- Rainwater Detention Tanks.

There will be approximately 14 truck ramps, servicing the northern and southern warehouses from the central roadway (see Figure 4) and 5 sub surface water detention tanks, located through the centre of Stage 1, as shown on Figure 4.

It is envisaged that construction of these features will occur after completion of the site filling. Therefore, given these facilities will be outside of the main building footprints, the marker layer described in Table 6.1 will be in place at this stage.

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Physical Separation Approach - Stage 1

It is considered that if a sufficient depth for these features can be achieved without penetrating the marker layer, then no further remediation measures will be required to be implemented at these locations. However, provision for maintaining the separation layer integrity at such locations will need to be included in the detailed design. In addition, the long-term EMP will need to note that separation layer thicknesses may be less in these isolated locations and make adequate provision if disturbance (such as upgrades or redesign) is required in the future.

The construction environmental management plan (CEMP) will also be required to make also provision for contamination management requirements during construction if localised penetration of the marker layer is required in constructing the loading ramps or other design features.

6.2 Perimeter Barrier Requirements

6.2.1 General Approach

The physical separation layer margins will generally be finished by constructing a 1:10 batter.

Where the minimum thicknesses of VENM materials prescribed in Table 6.1 cannot be achieved at the edges, additional measures will be required to ensure a sufficient separation is achieved. These could include:

- Additional excavation prior to filling to enable placement of a suitable thickness (as per Table 6.1);
- Additional separation measures such as sealing of the batter slope using “*spraycrete*” or equivalent; and/or
- Additional investigation / remediation (as required) and validation of surveyed perimeter areas, to demonstrate that those specified areas are not impacted by contaminated fill or ACM, prior to filling.

Based on the proposed design it is considered the completion of the perimeter areas can be achieved through a combination of the measures identified above. However, the following sections describe in more detail specific requirements for the physical barrier perimeters.

6.2.2 Boundary Easements

As shown on Figure 10 and summarised in Table 5.1, several easements are to be located at the northern, southern and part of the western site boundaries of Stage 1 as well as through the centre. Specifically the boundary easements include:

Table 6-2 Summary of Boundary Easements

Number	Width (m)	Purpose
Easement 1	10	Future remediation (if required)
Easement 7	10	Continued operation PCA (GTP)

Northern Easements (Easement No. 1)

The purpose of the northern easement is to provide for installation of future groundwater monitoring and/or groundwater extraction wells or implementation of future remediation systems.

While it is considered an effective physical separation layer can be implemented for the purpose of the proposed land use through the measures identified above (Section 6.2.1), the specific future uses of the easement are not currently known. It is, however, likely they would include penetration of the marker layer through drilling or some form of localised excavation.

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Physical Separation Approach - Stage 1

Specific provision for these future activities will therefore need to be made in the long-term EMP and are discussed further in Section 14.

Southern and Western Easement (Easement No. 7)

The southern and western easements will accommodate the GTP PCA infrastructure and rerouted pipeline.

In this case the easement will not be covered by the physical separation layer. Figures 12 and 13 provides sections taken from the landscape architects plan for the site (Habitation, 2007). These details show that the effective southern leading edge of the physical barrier layer will be located 9 m from the southern site boundary.

A 9 m “corridor” shall therefore be defined along the southern portion of the Stage 1 (Block 2). The soils in this area will be assessed and validated on the basis that the site design is not compatible with a physical barrier approach in this area.

Section 13 makes provision for these validation requirements.

6.3 Placement and Compaction of VENM Materials

All materials imported onto the site will be VENM as defined in the DECC, NSW (2008) Waste Classification Guidelines: Part 1 Classifying Waste.

This section addresses the minimum compaction requirements for the separation layer materials. The minimum thicknesses for construction were identified in Section 6.1.

These compaction requirements do not constitute a geotechnical specification for the development, but are intended as a minimum requirement for construction of the separation layer. It is likely that the geotechnical compaction requirements of the eventual detailed site design may be different to those identified here for the physical separation layer, in which case the more stringent should be adopted.

Imported VENM will be placed and compacted in successive horizontal layers for the full width of the cross section. The loose depth of the material in each layer will be such that the loose layer thickness shall be not more than 300 mm. At the time of compaction of each layer the moisture content of the material will be such that the specified compaction is achieved, as indicated by standard tests.

In areas that are not accessible for rolling with power equipment, mechanical hand tampers or vibrators will be used.

If, at any time during the progress of the work, tests show that the specified degree of compaction is not being obtained, equipment and/or methods will be replaced or modified, as required, to obtain the specified results.

Materials shall be compacted as specified in the table below.

Table 6-3 Compaction Standards

Area	Compaction Required
All Areas of Block 2 (Stage 1)	98% of the Standard Maximum Dry Density

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Physical Separation Approach - Stage 1

Compaction will be checked by standard maximum dry density tests and field density tests in accordance with the table provided below. Tests will be undertaken in groups of at least three and compaction of the layer concerned will be considered to be satisfactory if no single result falls below the desired density.

Table 6-4 Frequency of Field Density Tests

Area	Frequency of Tests
All areas of the Block 2 (Stage 1)	Not less than 1 test per 1,000 m ³ distributed reasonably evenly throughout full depth and area

The finished surfaces will be smooth, compacted as specified and free from irregular surface changes except where these changes are required as part of the redevelopment works. Testing for compaction will be undertaken by a qualified geotechnical engineer using National Association of Testing Authorities NATA certified methods to Level 3 standards as specified under AS 3798 – 1996 “Guidelines on earthworks for commercial and residential developments”.

6.4 Stage 2 Area

Development of the southern portion of Block 1 (future Stage 2 Area) as a compensatory flood storage area for Stage 1 shall remove the stockpiles and therefore the bulk of the ACM, however, it is noted there is potential for ACM to be present in the underlying fill. There will be some potential, therefore, for ACM to be present in soils in the flood storage area.

The amount of ACM likely to be present in the remaining soils is unknown, but is not expected to be considerable.

At this stage, it is envisaged that exposure to these materials shall be mitigated through access restriction (erection of a perimeter fence) and maintenance of the surface vegetation.

Should more appreciable amounts of ACM be encountered in the remaining soils, a contingency would need to be developed, which might include localised ACM removal and validation.

Section 7

Management of Vapour Risks – Stage 1

As described in Section 5.2, environmental monitoring activities have demonstrated that the risks to human health associated with vapour emissions from contaminated surface water in Springvale Drain (SVD) are unacceptably high and would restrict the proposed development.

In addition, modelling undertaken in the HHRA (URS, 2008a, refer Appendix A) has identified a scenario where if shallow groundwater discharges into the compensatory flood storage area, it may pose an unacceptable exposure scenario.

This section outlines the proposed measures for mitigation of these issues for the Site.

7.1 Springvale Drain

7.1.1 Environmental Monitoring Program

Based on observations of water levels in the drain since the Botany Industrial Park (BIP) Containment Line of the Orica Groundwater Treatment Plant (GTP) commenced operation in late 2006, it was suggested that the increased rate of GTP operation may be resulting in a lowering of the local groundwater table and hence decreasing the rate of shallow groundwater discharge into Springvale Drain.

URS was engaged to undertake a program of environmental monitoring to measure the apparent response of shallow groundwater levels in the vicinity of the SVD to the operation of the GTP and to assess whether the operation would be sufficient to decrease risks to future occupiers of Southlands and down gradient properties due to air emissions from SVD.

7.1.2 Environmental Monitoring Program

The program included:

- Monitoring of the shallow hydraulic regime in the vicinity of the drain through development of a network of shallow and intermediate monitoring wells. The monitoring network was designed to assess the influence of recharge events, as well as GTP operation on shallow groundwater discharge into the Drain;
- Monthly monitoring of ambient air concentrations in the vicinity of the drain on Southlands and other selected off-site locations;
- Monthly sampling of surface water from SVD (sampling events coinciding with ambient air sampling); and
- Periodic collection of groundwater samples from the shallow well network.

The first round of data (ambient air and surface water) was collected on the 11 May 2007. Groundwater monitoring wells were installed in early June 2007 and the hydraulic monitoring network established soon thereafter.

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Management of Vapour Risks – Stage 1

7.1.3 Monitoring Results

The findings of the Springvale Drain monitoring program were reported in a draft report¹¹ entitled:

- URS (2008b). *Surface Water and Ambient Air Monitoring Program for Springvale Drain May 2007 to June 2008. Draft Report September 2008;*

and are summarised below.

Hydraulic Monitoring Data

The URS (2008b) review of data collected indicated that operation of the BIP is likely to have decreased shallow groundwater levels on Southlands, thereby reducing overall discharge to Springvale Drain. However, the hydrographs suggested that extraction from the deep aquifer at the PCA had limited influence on shallow groundwater levels (monitoring well location WG224S, WG225S and WG226S) along Springvale Drain.

The data suggested that recharge of the shallow aquifer from rainfall appears to significantly influence water table elevation, and groundwater extraction from BIP and the deep aquifer at PCA is not effective in reducing and maintaining shallow water levels to below the level of Springvale Drain, particularly during rainfall events.

During events when pumping temporarily ceased during routine maintenance of the GTP, occurring in June 2007 and November 2007, a minor but rapid change in surface water levels was noted, particularly within the intermediate and deep aquifers. Water levels within the shallow aquifer were also observed (in November 2007) to increase during the shutdown period, with water levels in some wells increasing to levels above the invert level of SVD (where discharge to groundwater is expected to occur).

Recharge from rainfall events has been observed to have an influence on shallow groundwater levels, and hence the potential for shallow groundwater to discharge into SVD. Significant increases in shallow water levels have been observed following rainfall events with increased concentrations of CHCs reported in surface water and ambient air during sampling events occurring just after such rainfall events.

Surface Water Data

There is a strong correlation between groundwater levels and contaminant concentrations in the surface water and ambient air near SVD. Generally, higher groundwater levels correspond with higher surface water concentrations as, groundwater levels in excess of the SVD invert level lead to contaminated groundwater discharging into SVD. Accordingly it has been observed that reducing the groundwater level at Southlands (through pumping at the BIP and PCA containment lines) has decreased CHC concentrations in the surface water in SVD. Similarly a strong relationship between recharge from rainfall events and shallow groundwater levels has been identified. Surface water concentrations within SVD have been observed to increase following such rainfall events.

Higher CHC concentrations in surface waters have been reported in the SVD Re-alignment Channel (SW062) than have been recorded in the main channel of SVD (SW062_East). This is consistent with observations made in the GTP Quarterly Monitoring Program, where more elevated concentrations have historically been recorded in the western realignment channel, albeit at different locations along the drain. This is also consistent with observations made in the field where the water surface is usually higher in the realignment channel than in the main channel of SVD and where odours and visual evidence of contamination are normally more obvious in the realignment channel. Similarly, SW005, located at the southern end of the main channel of SVD recorded concentrations of EDC similar to those at SW062_East during the monitoring period presented in URS 2008b.

¹¹ At the time of RAP preparation, this report was in the process of being finalised.

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Management of Vapour Risks – Stage 1

The elevated concentrations detected in surface water in the realignment channel are considered likely to be related to groundwater discharge (as is the case in the main channel). However, the realignment channel is no longer in use and has been cut off at the McPherson Street end. The closed nature of the channel limits the flow through of cleaner water (both surface and groundwater).

Ambient Air Monitoring Data

With regard to ambient air quality in the vicinity of SVD, the URS (2008b) report concluded that:

- Concentrations of EDC (and other CHCs) reported are; in general, lower than reported during previous rounds (ie to the end of 2006 prior to operation of GTP) with many samples not reporting concentrations of CHCs above the analytical limit of reporting. Concentrations in air reported since August 2007 are consistently lower than the level considered in the Consolidated HHRA (URS,2005c) as indicative of acceptable risks;
- Concentrations in some locations (particularly within the Southlands site) reported prior to August 2007 are highly variable with concentrations of EDC reported up to values around 1,000 $\mu\text{g}/\text{m}^3$. The higher concentrations were collected after rainfall events, and generally coincident with elevated concentrations in surface water. It is noted that since August 2007, higher concentrations of EDC have also been reported after rainfall events, however the peaks reported are lower (maximum of 15 $\mu\text{g}/\text{m}^3$) than reported prior to August 2007; and
- Concentrations downstream of the Southlands site (i.e. off-site) are generally lower and displayed less variability than samples collected from within the Southlands site.

It was considered that the operation of the GTP has effectively reduced CHC concentrations within the Southlands site, north of the Southlands site, MCS and Discovery Cove such that long-term exposures to these chemicals that may be derived from volatile emissions to air from SVD are considered low and acceptable. Some degree of variability remains within the potential emissions to air adjacent to SVD within the Southlands site such that the potential for peak exposures (associated with groundwater discharge into the drain after rainfall events or shutdown of the GTP) should be further managed.

Based on comparison of CHC concentrations in surface water samples collected from the realignment channel and Springvale Drain respectively, it was considered that ambient air concentrations recorded on the Southlands site are affected by vapours emanating from the realignment channel. Given the large difference in water concentrations, it was considered likely that the prime contribution to the CHCs in air is the realignment channel.

Summary

- The GTP has sufficient mitigating effects to mitigate vapour levels in offsite areas and long-term risk to workers outside of Southlands are acceptable; and
- Provided the discharge of shallow groundwater into SVD is appropriately managed such that concentrations of CHCs in surface water (and subsequently in air) remain at levels reported since August 2007, risks to workers who may occupy Southlands in the future are considered to be low and acceptable. Some degree of variability remains with potential concentrations of CHCs in air close to SVD within Southlands such that the potential for peak exposures (associated with groundwater discharge into the drain after rainfall events or shutdown of the GTP) should be further managed.

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7.2 Springvale Drain Mitigation Approach

The proposed SVD mitigation approach is twofold and is summarised as follows:

1. Infilling of the Realignment Channel; and
2. Installation of a shallow groundwater extraction system.

A description of the proposed works is provided below:

7.2.1 In filling the Realignment Channel

The Re-alignment Channel runs north south and is located parallel to SVD within Block 2 (Lot 2) of the Southlands site. The Re-alignment Channel was originally excavated when maintenance works, consisting of sediment removal, were being completed for SVD in the late 1990's. The SVD Re-alignment Channel was left unfilled upon completion of the SVD maintenance works. The proposed infilling of the Re-alignment Channel involves the filling of the channel to the surrounding ground surface levels, depending upon availability of suitable VENM / Recovered Aggregate back fill material.

The infilling of the channel is the first component of actions to mitigate the emissions from SVD and the Re-alignment Channel. Stage 2 of the mitigation approach is to intercept shallow groundwater which is described in Section 7.2.2 below.

7.2.2 Shallow Groundwater Extraction System

The proposed system will extract groundwater from targeted points adjacent to the Springvale Drain, creating a localised and controlled draw down of the shallow water table. The shallow water table will be maintained beneath the invert of Springvale Drain, preventing groundwater discharge.

This proposed system comprises a shallow groundwater extraction system utilising a series of relatively small diameter (<50 mm diameter) wells (spear points) that are manifolded to a single surface mount pump. The surface mounted pump extracts water from all wells simultaneously with control on individual well flow rates achieved by adjustment of valves. The extracted groundwater would be transferred to the GTP for treatment or may be reinjected in the event of a long term GTP failure.

The shallow groundwater extraction system option would involve the following components:

- Trial groundwater extraction points implemented prior to full scale installation. The trial would consist of 5 wells at 5 m intervals. Depending on results, the detailed design for the full scale installation would be completed.
- Construction of approximately 28 groundwater extraction points at 5 m intervals, starting at Nant Street Tank Farm and extending 140 m south (may be extended past McPherson Street).
- The pipes from the wells would be manifolded into one line and then fed into the header and ultimately to the GTP.
- The pipes from each of the wells would be placed in an adjacent gutter with a grill cover.
- A vacuum pump would be used to extract the groundwater from the wells.
- The Groundwater Extraction Points would only operate when required, such as when the level of shallow groundwater is elevated.

Based on the results of hydraulic monitoring undertaken to date (URS, 2008b), it appears that groundwater recharge following rainfall is the key factor influencing shallow aquifer water levels on the Southlands site and hence discharge into the Springvale Drain. Therefore, it is likely that during periods

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of low rainfall, operation of the proposed shallow groundwater extraction system may not be required. Field observations during dry periods when Springvale Drain and other groundwater charged features such as the paper waste ponds have been dry suggest the system may only operate 30-50% of the time.

The system will be located within easements of the Southlands site and will be able to operate independently of the GTP. The operation and maintenance of the system will be Orica's responsibility until remedial activities have rendered it no longer necessary. Planning approval for these works are being sort separately under the umbrella of the NCUA, as part of the Botany Groundwater Clean-up Project.

7.3 Compensatory Flood Storage Area

As discussed in Section 5.1 the construction of a compensatory flood storage area on the southern portion of Block 1 (Refer Figure 4) is required to mitigate site flooding for development.

In considering historical and more recent water table elevations and the proposed flood storage design level (CW, 2007), JBS (JBS, 2008) have suggested potential for groundwater to discharge into the proposed flood storage area.

Based on the modelling described in the JBS Report (JBS, 2008) groundwater levels could “*exceed the proposed excavation surface at MWB12S (located at the south-eastern corner of Block 1 Southlands) for an average of 18% of the time each year. In addition, groundwater levels remain within 300 mm of the proposed excavation surface approximately 40% of the time each year*”.

7.3.1 Risk Assessment

Historical shallow chemical groundwater quality data and estimates of the likely frequency and duration of groundwater discharge into the flood compensatory area calculated by JBS (JBS 2008) were used to assess risks to human health of future site users during a scenario where groundwater discharges to the storage area as well as where groundwater remains just beneath the surface. The assessment by JBS assumed there would be no engineering or mitigating factors that would minimise or prevent groundwater entering the compensation area.

The HHRA (URS, 2008a) concluded that risks to workers (site users) were considered unacceptable (without consideration of other exposures on Southlands) based on the scenario where groundwater discharges directly to the compensatory flood storage area.

The assessment indicated that if an appropriate drainage system were installed beneath the flood storage area to capture and drain shallow groundwater before it discharged to the surface, risks would be sufficiently low to ensure workers in areas adjacent to the area would be protected (URS, 2008a).

Any system designed to mitigate the potential risks to human health would therefore also need to prevent mixing of contained flood waters with the underlying groundwater as well as prevent discharge of contaminated waters to Springvale Drain.

7.3.2 Preliminary System Concept

The flood compensation area will be designed and engineered in order to achieve the following:

1. Minimise ingress of contaminated groundwater into the compensation area;
2. Prevent mixing of contaminated groundwater with flood waters that will discharge to Springvale Drain (a possible offence under the Protection of the Environment and Operations Act 1997); and
3. If practicable, allow for capture of floodwaters in high rainfall events for treatment by the GTP and eventual re-use in the local area.

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The detailed design of the flood compensation area is yet to be completed; however there are a number of proven techniques that may assist in achieving these objectives outlined above. These include:

1. The installation of subsurface groundwater drainage / reticulation system to prevent groundwater discharge into the compensatory flood storage area and to prevent mixing of groundwater with flood waters. This may consist of a network of 'ag-lines' (or equivalent collection conduits) will be installed at an appropriate depth for this purpose. The materials to be used in the design will need to be specified in the context of the chemically aggressive subsurface conditions;
2. The use of vegetation to provide a passive lowering of the shallow groundwater table in the area and prevent ingress into the compensation area. Trees such as genus *melaleuca* have proven to be very effective at lowering shallow groundwater table levels;
3. Selecting materials such that their effective porosity minimises ingress of rainfall to the shallow groundwater, thereby reducing recharge and minimising shallow groundwater levels locally;
4. Construction of vertical pathways to allow increased connectivity between the shallow and deeper aquifers and further reduce potential for contaminated water to enter the compensation area from the shallow aquifer;
5. Connection of compensation area to the GTP pumping network to allow for pumping of collected groundwater (from the reticulation system) or collected stormwater, if required.

The system is likely to incorporate several of the above design elements in order to achieve the outlined objectives. The design process will include elements of hydraulic modelling, detailed engineering review and possibly field trials. The completed design documents will be required to contain sufficient information to demonstrate to the Site Auditor that the design objectives are achievable.

As the detailed design of the compensation area has not been completed the management of soils and/or spoils generated during its installation have not been addressed in this RAP and will need to form part of the remediation contract specification documents. Contamination management measures described in Section 11 will also apply to this area of the site.

7.4 System Contingency

Section 8 presents the findings and conclusions of the HHRA program (refer Appendix A) for the site. The assessment presented assumed that the systems described in Section 7.2 and 7.3 would be effective in maintaining groundwater levels below the invert level of Springvale Drain and preventing discharge of groundwater into the compensatory flood storage area, thereby eliminating the risks to health of the site occupants posed by vapours associated with contaminated shallow groundwater discharge to the site surface. It has been noted that verification of the effectiveness of these systems is an integral part of the site development.

7.4.1 Primary Contingency

Temporary Aquifer Storage and Recovery (TASR)

In response to NCUA, an EIS was prepared for the Botany GCP by URS dated 12 November 2004 for the construction and operation of the GTP (URS, 2004c).

The Joint Determining Authority Report prepared for the Project dated February 2005 (DEC (NSW), 2005), specified under Section 2.3.5 that:

“Orica will maintain the Steam Stripping Unit (SSU)[used for interim treatment] in standby mode, for recommissioning in a controlled and timely manner in the event of a long term shutdown (for example, catastrophic failure of key equipment) [of the GTP]. This unit would be used to provide ongoing containment of the highest concentration contaminated groundwater and protection of receiving environments while GTP operational issues are rectified.”

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Towards the completion of construction of the GTP, the SSU was showing evidence of severe corrosion and was requiring constant maintenance and repairs. Its capacity and reliability was barely adequate to meet the requirements of treatment.

Although it served the purpose of temporary treatment of groundwater whilst the GTP was being constructed, its reliability as a long term back up to the operation of the GTP is uncertain. In order to maintain it in standby mode, extensive equipment replacement and repair would be required and significant expense incurred.

Subsequently Orica has proposed the use of temporary aquifer storage and recovery (TASR) as a viable backup alternative in order to protect aspects of human health and the environment that are most sensitive to medium / long term failures of the GTP.

This system will be located on BIP and involves injection of extracted groundwater into the subsurface, on the basis that such waters will eventually be intercepted by the hydraulic containment extraction bores, located down gradient of BIP. This system will provide a contingency for the existing GTP system, where in the event the GTP goes off line for a significant period of time, groundwater pumped from the hydraulic containment lines can be diverted to TASR.

Orica has submitted an Review of Environmental Factors (REF) to the NSW DECC for their consideration detailing a trial injection and preliminary designs for the full-scale system. The results of the trial planned for late 2008 will be required to demonstrate that the injection and recovery can occur on a sustainable basis.

The REF outlines the intent to use this system as a backup for the proposed shallow groundwater extraction system along Springvale Drain

Following the completion of the detailed TASR design, an assessment will be made regarding the contingency requirements of the flood compensation area. It is anticipated that with the appropriate design and material selection, the volume of groundwater entering the drain will diminish relatively quickly due to reduced recharge and initial dewatering during and following construction.

7.4.2 Environmental Management Plan

7.4.3 Additional Contingency

The alternate system (TASR) for receiving water pumped from the Springvale Drain groundwater extraction system is intended to be the primary contingency measure. In addition, the HHRA (URS, 2008a, refer Appendix A) considers the unlikely event where the GTP and secondary systems fail for sufficient periods of time to allow groundwater to enter the drain. Additionally, in order to be conservative, it has been assumed that groundwater may also accumulate in the flood compensation area.

Allowable exposure frequencies have been estimated for emissions from the Springvale Drain under such circumstances, taking the distance of the receptor from the drain into account. The distances considered ranged from 10 m to 50 m. The following table is taken from the HHRA (refer Appendix F of URS 2008a) and presents the estimated allowable exposure frequency.

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Table 7-1 Estimated Exposure Frequencies

Distance from Springvale Drain (Workplace as building or outdoors)	Estimated Exposure Frequency Allowable based on Protection of Human Health	
	Average ¹	Estimated Maximum ²
20m (current proposal)*	360 days over 30 years (an average of 12 days each year for 30 years)	225 days over 30 years with no more than 90 days during any one year
Average for 20-50m from Drain (considered relevant for exposures onsite)	1060 days over 30 years (or an average of 35 days each year for 30 years)	650 days over 30 years with no more than 270 days during any one year

Notes:

- 1 Estimated exposure frequency for the average scenario presented as a total number of days over a working lifetime as the calculated risks are dominated by non-threshold carcinogenic risks. In the event that all the days of exposure occurred in only one year then the threshold HI would remain acceptable and hence no additional limit on maximum number of days per year has been set.
- 2 Estimated exposure frequency for the maximum scenario presented as a total number of days over a working lifetime as the calculated risks are dominated by non-threshold carcinogenic risks. In addition for distances up to 30m from the drain the total number of days during any one year is also limited by the calculated threshold HI. Hence an additional limit on the maximum number of days each year where peak exposures occur has been set.

*In considering such a scenario, it is noted that it is very unlikely that a worker would be located at 20 m from the drain for a whole day and it is more likely that the scenario estimated for an average of 20 -50 m is more relevant.

As a result of the above, a setback distance of 20 m from the Springvale Drain has been factored into the development plan (see Figure 4) in order to be conservative. The 20 m buffer zone either side of the drain will be fenced and access by workers will be precluded. However consideration of work areas adjacent to the drain indicates that the average exposure that may occur 20-50m from the drain is considered representative of potential exposures that may occur following development of the site. The focus of the assessment is associated with longer duration exposures (over a number of days, weeks or months), not short-term peak exposures. Therefore, in the event that both the GTP and its primary contingency, the TASR system, are both off line and rainfall levels at that time are sufficient to result in discharge of groundwater into the drain, the allowable exposure (based on the average for 20-50m from the drain) is in the order of 650 to 1060 days over 30 years with no more than 270 days occurring in any one year.

The long term Environmental Management Plan (EMP) will be required to outline strategies to manage this risk scenario. This may include the use of vapour mitigation measures in buildings closest to the Springvale Drain and provision of actions in the event of failure of the GTP and its backup systems. Such actions may include:

1. Communication of risk issues to the tenants and landowners to allow for modification of worker behaviour; and
2. Air monitoring in work areas to assess actual exposures and risks,

The above exposure frequency for workers near Springvale Drain when the GTP and secondary systems are potentially off-line is based on the protection of worker health during an intermediate duration event (over a number of days, weeks or months that cover less than a year) not a short peak event (hours).

It should be noted that these risks were estimated using data collected when the re-alignment channel was open. This channel will be filled for the development. The effects of the infilling of this channel on the calculated risk factors is unknown, but will be reassessed following an extended period of sampling over a variety of conditions.

The proposed content of the additional contingency measures in the long term EMP will be finalised following further air sampling after the infilling of the re-alignment channel.

Section 7**Management of Vapour Risks – Stage 1****7.5 Verification Testing**

In addition to the environmental data collected to date (Section 7.1.2) environmental monitoring will continue in the months prior to system commissioning to provide a baseline for comparison with data collected during operation.

A period of detailed environmental monitoring will follow commissioning to verify the effectiveness of the system. Details of the proposed validation program framework, with regard to vapour concentrations in ambient air, are provided in Section 13.6.

Verification will also include collection of hydraulic data to confirm the shallow water table is maintained beneath the invert of the drain. It is likely the existing hydraulic monitoring network, as well as additional monitoring wells to be installed between the wells installed for the shallow groundwater extraction system, will be employed for this purpose.

In addition, surface water samples will be collected from the drain and ambient air monitoring will be undertaken in the immediate vicinity of the drain and the results will be compared with data collected prior to system implementation. Water (if present) will also be collected from the Compensatory Flood Storage Area and tested as required in Section 13. Further detail of the verification process is provided in Section 12.

Section 8

Human Health Risk Assessment

URS were engaged to undertake a site specific Human Health Risk Assessment (HHRA) program for the Southlands Remediation and Development Project. The findings of the program are reported in the URS report entitled:

- Human Health Risk Assessment, Southlands Remediation and Development Project, March 2008 (URS, 2008a).

A copy of the report is presented in Appendix A.

8.1 Risk Assessment Approach

The process followed for the completion of the HHRA was an iterative approach. The available site data and development proposal was considered initially in a Preliminary Human Health Risk Assessment (PHHRA, URS, 2007b).

8.1.1 Preliminary Risk Assessment

The PHHRA identified a number of key issues that required further assessment and risk management measures prior to completion of the HHRA. The key issues identified included the potential for shallow groundwater to discharge to Springvale Drain (resulting in emissions to air of volatile chlorinated compounds), potential for shallow groundwater to discharge into proposed compensatory flood storage areas (resulting in emissions to air of volatile chlorinated compounds) and presence of residual impacts in soils on the site.

Further investigations were undertaken and risk management measures were identified to address these issues. The outcome of these investigations and the identified risk management measures have been discussed in Sections 4 through 7 and the requirements arising are incorporated into the remediation scope, developed in the following sections.

8.1.2 Site Development Risk Assessment

The risk management measures proposed were then further considered in the completion of the HHRA. The HHRA was undertaken as a site-specific assessment that considered key aspects of the proposed development, flood management requirements and risk management measures. These included the filling in of existing ponds on Block 1, construction of compensatory flood storage areas on the southern portion of Block 1, the placement of fill on Blocks 1 and 2 prior to construction of any buildings to address flood issues and the restriction of access to the northern portion of Block 1 (proposed Stage 3 development).

With respect to risk management measures the assessment has considered proposed measures designed to intersect shallow groundwater prior to discharge into Springvale Drain (with groundwater directed back to the GTP) and the drainage system proposed to control shallow groundwater in the area of the compensatory flood storage area on Block 1.

The performance of these measures will need to be monitored through the collection of appropriate data for some time after commissioning. A framework for validation is provided in Section 13.6. It is noted that should any of these assumptions change, or the proposed risk management measures do not achieve the stated objectives, then the assessment of risks to human health presented in the report will need to be reviewed and potentially revised.

The following table presents a summary of the risk issues and the site specific risk mitigation measures that were modelled in the HHRA (URS, 2008a) associated with the development of Block 1 and Block 2 of the Southlands site.

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Human Health Risk Assessment

Table 8-1 Summary of Risk Issues (HHRA, 2008a, Refer Appendix A)

Issue	Issues Identified	Risk Mitigation Measures
All Aspects of the Proposed Development		
Intrusive workers in all areas including the maintenance of GTP infrastructure	Potential for elevated exposures to chemicals in shallow groundwater and inhalation of volatile chlorinated chemicals (particularly within confined spaces)	All works to be undertaken under a long term environmental management plan [EMP] that ensures development of job specific safe work practices including addressing any issues associated with works that breach the areas covered with barriers.
Workers involved in maintenance of Springvale and Floodvale Drains	Potential for elevated exposures to chemicals in surface water and inhalation of volatile emissions	All works to be undertaken under a long term environmental management plan that ensures development of job specific safe work practices
Presence of asbestos fibres in soil	Potential for generation of dusts containing asbestos fibres.	Placement of appropriate barrier. Where no physical barrier is present a sound ground cover should be maintained to minimise generation of dusts. These areas need to be considered in a long term environmental management plan [EMP].
Inhalation of volatile chlorinated chemicals associated with emissions to air from Springvale Drain	Potential for significant exposures by all users of the site on basis of measurements completed to date.	Surface water concentrations within the drain would need to be lowered such that air concentrations are reduced by 100 to 1000 fold. This may require a reduction in the discharge of shallow groundwater into Springvale Drain. Proposed mitigation measure involves a shallow groundwater extraction system adjacent to Springvale Drain to intercept groundwater prior to discharge into the drain, with groundwater directed to the GTP. Exposures are expected to be lower once system is installed, however the effectiveness requires verification. Risk issues when the GTP is offline need to be reviewed in the final design and management of such a system.
Stage 1 Development (also relevant to Stage 2 development)		
Elevated concentrations identified in soil and sediment in the ephemeral ponds – that are moved across the site during earthworks	Potential for elevated exposure by workers in area not covered with barriers, including gardening activities (ingestion, dermal contact and vapour intrusion into buildings)	Remediation of assumed hot-spots to a level protective of all exposures including vapour intrusion (refer to details in HHRA). Remediation to derived risk-based concentrations (derived and presented in the HHRA to assist in this process for some key chemicals) or other guidelines that can be justified on the basis of risks to human health. Further sampling could be undertaken to better define extent of area affected by elevated concentrations
Inhalation of volatile chemicals in outdoor air (emissions from soil and groundwater)	No issues identified for outdoor air if the compensatory flood storage area is appropriately designed to prevent groundwater discharge to the surface.	Effectiveness of the proposed drainage system for the compensatory flood storage areas on Block 1 requires verification.
Inhalation of volatile chemicals in outdoor air and within buildings constructed on Block 2 (emissions from some subsurface soil and groundwater)	No issues identified (subject to findings of sub slab investigations).	Review of vapour intrusion issues that may arise as a result of the consideration of issues associated with the presence of a large slab.
Stage 2 Development		
Inhalation of volatile chemicals within buildings constructed on the southern portion of Block 1 (emissions from some subsurface soil and groundwater)	Potential for elevated exposure to volatile chlorinated chemicals, present in shallow groundwater, that may migrate into buildings (including buildings associated with GTP infrastructure)	If buildings are constructed in the area vapour mitigation measures should be incorporated into the building design. Such measures may include passive barriers, sub-slab depressurisation or building pressurisation.

Section 8

Human Health Risk Assessment

8.1.3 Additional Considerations

The HHRA has indicated that accumulation in buildings of vapours related to emissions from shallow groundwater is an issue for Stage 2 development. This was not identified as an issue for Stage 1 Development (i.e. development of Block 2), because as described in Section 7.1, the results of historical and more recent monitoring suggest that Springvale Drain at Southlands intercepts the most shallow contaminated groundwater before it reaches Block 2.

Section 7.2.2 described installation of a shallow groundwater extraction system at Springvale Drain. Modelling of pumping requirements is currently being undertaken by the Proponent's consultants. The proposed system will be designed to create sufficient draw-down to maintain groundwater levels below the invert level of Springvale Drain.

URS has flagged with the Proponent design team that creating such drawdown may allow migration beneath the drain of shallow contaminated groundwater that before system installation was intercepted by the drain.

If this were the case, then the issues of potential vapour accumulation in buildings that were identified for Block 1 (see Section 10.2.2) may become pertinent for Block 2.

It is unlikely that the development timeframe will allow testing or verification of these effects, as it is likely it would take some time for migration to occur to an extent that would be detectable on Block 2. The options for management of these potential effects would include:

- Detailed modelling to establish if the design draw-down levels could cause vapour issues on Block 2 and the timeframe over which it is expected to occur;
- Incorporation of vapour mitigation measures, as proposed for Stage 2 development (refer Section 10 and HHRA) into building design for Stage 1 (Block 2 Area).

In order to take a conservative approach, the Proponent has advised that vapour mitigation measures will be implemented in the construction of all buildings on Southlands.

8.2 Soil Quality

The HHRA (URS, 2008a) (Appendix A) calculated site specific risk based soil criteria (RBSC) for some of the volatile contaminants of concern in soils on the site. The RBSCs have been used in the identification of impacted soil, located mainly on Block 1 (Stage 2 Area) that requires remediation. The RBSCs are included in the validation criteria presented in Table 13.1. Areas of soil contamination that require remediation in the context of the proposed land use have been highlighted on Figures 8 and 9.

It is noted that in Area C (see Figure 8) remediation is required due to the potential for leaching of mercury contamination rather than risks to human health.

Section 9

Remediation Scope - Stage 1

This section outlines the scope of works required to achieve the remediation strategy developed in Sections 4 through 7. These components will broadly occur in the order presented below.

9.1 Asbestos Management

Disturbance of site fill/soil materials, including stockpiles and existing subsurface filling has the potential to expose ACM and potentially generate asbestos fibres.

All works described herein will be undertaken in accordance with the asbestos management component of the CEMP and the site specific health and safety and environment plan (SSHSEP), the framework for both of which is described in Section 11.

The CEMP and SSHSEP will make provision for health and safety protocols for workers involved in the earthworks program, describe mitigation measures to minimise generation of dusts, describe protocols for removal and disposal of asbestos finds during the work program and prescribe site and boundary monitoring programs to be undertaken for the duration of the works.

9.2 Preliminary Works

The following preliminary works are relevant to the entire site and will occur prior to bulk earthworks in the Stage 1 and 2 Development Areas.

9.2.1 Existing Infrastructure

General Infrastructure

There are no buildings on the site and, as outlined in earlier sections, the site is generally covered with vegetation.

On Southlands Block 2, there are two areas, one in the south western corner and the other near the northern boundary (see Figure 9), where vegetation has been cleared and the surface has been prepared for use as staging areas for GTP maintenance operations. These areas are serviced by unsealed roads and are used primarily as staging areas for GTP maintenance operations.

In addition, the GTP above ground pipe line, running northwards along the western side of the Springvale Drain, is supported on concrete pavement.

Any redundant concrete hard stands and/or pads remaining after any required rerouting of GTP pipe work will be demolished and removed from the site.

Remediation Infrastructure

In addition to the general infrastructure outlined above, there are two existing (and largely redundant) pilot scale in-situ remediation systems on Southlands.

The pilot scale reactive iron barrier (PSRIB) is located in the northern portion of Block 1 and is not affected by the development described herein.

Another pilot scale bio-remediation system (known as the '*bio-trial*' system), has infrastructure located in two area of Block 1, located in the north and south-west. These areas will be fully decommissioned during preliminary works.

The above ground infrastructure will be demolished and removed and the injection points / monitoring wells will be decommissioned in accordance with the guidance for well abandonment provided in the Minimum Construction Requirements for Water Bores in Australia (LWBC, 2003).

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Remediation Scope - Stage 1

Groundwater Monitoring Well Decommissioning

As noted in Section 5.3 and 5.4, given much of the existing monitoring well infrastructure will be destroyed as a result of the development, a replacement monitoring well network will be required to meet the ongoing requirements of the NCUA. While the proposed replacement will likely occur as a phased program, based on the sequence of works being undertaken, the requirement is included in the preliminary works.

The proposed well replacement locations are shown on Figure 11. Section 11.3 provides a framework for a groundwater monitoring well replacement program. Existing wells shall be decommissioned in accordance with the program requirements and in accordance with well abandonment guidance provided in the Minimum Construction Requirements for Water Bores in Australia (LWBC, 2003). The plan shall include replacement of GTP extraction bores located on the southern portion of Block 2 Southlands.

9.2.2 Service Conduits and Corridors

There are existing underground services beneath Southlands. These include an underground fuel pipeline beneath Nant Street that connects the Nant Street Tank farm to the north of Southlands (see Figure 1) with the Caltex Oil Terminal to the south east of the site. In addition, there are service easements, including Energy Australia and Qenos Pty Ltd easements, located beyond the eastern boundary of Block 1.

All underground services beneath, and in the environs of, Southlands will be mapped and documented in detail. This is required to avoid intercepting services during remediation works that require excavation, such as hotspot removal, as well as to enable incorporation of service and easement locations into the long term EMP (see Section 14).

The existing services beneath, and in the environs of the site, will be mapped using:

- Available information from the site owner / occupiers;
- Dial before you dig underground service locations services; and
- A suitably qualified service location provider.

In addition, invasive service location techniques, such as hand digs and/or air-knifing may be required to verify the specific locations of services.

It is noted that provision has been made in the site design for incorporation of subsurface easements for underground services. However, should other services be located through the measures identified above, assessment of available management options for future access to these easements would be required, through liaison with the service owner and/or Council. Measures such as incorporation of easements into the barrier design or termination and diversion of the services may be required. Future management of site services will be incorporated into the long term EMP.

Overhead cables (if present) will also need to be identified and managed in accordance with the service owner's requirements.

9.2.3 Vegetation Reduction

This component will likely apply to the whole site (i.e. including Stage 3). While remediation of Stage 3 forms part of a later development, the proponent will likely address visual amenity issues such as vegetation reduction and stockpile levelling during Stage 1 works.

The site is covered with vegetation and scrub. The bulk of the surface vegetation will be removed and disposed off-site at suitably licensed green waste facility.

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Remediation Scope - Stage 1

ACM may be mixed in the shallow soils beneath the surface vegetation and there is potential that vegetation removal, using traditional clearing and grubbing techniques, would disturb the soils and generate dusts that could potentially contain asbestos fibres requiring specialised waste management requirements.

The vegetation removal approach will therefore need to consider management of the shallow soils associated with the root systems of the vegetation. This issue will be addressed in more detail in the CEMP, with mitigating measures including a preliminary reduction of the vegetation by cutting above ground surface rather than ripping / grubbing. Weedicide could also be used to reduce the root systems further, following the above surface cutting.

Any vegetation clearance thereafter (including scrub and root systems etc.) will be treated as part of the bulk earthworks, for which the SSHSEP, will incorporate measures to mitigate dust generation and handling of ACM (see Section 11) will be included.

9.2.4 Stockpiles - Preliminary Screening and Segregation

Following vegetation reduction, a detailed visual inspection will be undertaken to identify (where possible) where oversized materials are contained within stockpiles.

An area within the site boundaries will be designated and prepared for temporary storage of oversized materials, prior to their disposal off site.

Oversized materials

Oversized materials including (but not limited to) concrete fragments, masonry, boulders, scrap metal etc. will be removed from the stockpiles and transported¹³ to the designated staging area for off-site disposal or appropriate off-site management.

It may be required that very large fragments of concrete, masonry etc. are further broken down prior to movement using pneumatic breaking equipment mounted on an excavator.

Disposal of such materials from the designated area will be tracked and documented in accordance with requirements identified in Section 13 and the details will be presented in the Validation Report.

These works will be undertaken in accordance with the specific guidance for earthworks provided in the CEMP and SSHSEP (see Section 11). There is potential that during this preliminary screening, localised "pockets" or piles of buried ACM or bonded sheeting will be uncovered and/or disturbed. Specific protocols, including "step by step" guidance for management of such finds will be provided in the CEMP (see Section 11).

9.3 Shallow Soil Contamination Management

This section describes management of impacted soils located on the site, however, the majority are located within the Stage 2 area and need to be managed prior to Stage 1 development of the area as a compensatory flood storage area.

The locations of the identified hotspots are shown on Figures 8 and 9 and discussed in Section 3. Figures 8 and 9 show the inferred lateral extent of the impacted materials based on available data.

A preliminary waste classification for these materials is provided in Section 12.

¹³ In certain instances, depending on the size of the objects involved, it may be preferable to load such materials directly onto a truck for disposal off-site.

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Remediation Scope - Stage 1

9.3.1 Further Assessment

Figures 8 and 9 present the inferred extent of impacted soils based on currently available data.

The inferred lateral extent of the contamination is generally based on the distance to the nearest investigation point where concentrations were less than nominated assessment criteria. In the cases of sediment samples where impacts have been identified, the lateral extent is not known.

It is considered further assessment of soil quality is required to:

- Further constrain those areas where there is uncertainty as to the extent of the impacts;
- Assess whether impacts identified in sediments are localised or characteristic of a wider area of sediment.

Such investigations should adopt the criteria presented in Table 13.1 for assessment of soil quality and refinement of the areas of impact presented in Figures 8 and 9.

9.3.2 Excavation

Following refinement of the lateral extents of the contamination, the impacted materials will be excavated and loaded directly onto trucks which are appropriately licensed by the NSW DECC to transport the respective waste categories identified for each hotspot area (refer to the preliminary waste classifications provided in Section 12).

Where interim stockpiling of excavated materials is required, the environmental management measures identified in Section 11 will be implemented.

The impacted soils in areas A and B (Block 1) are contaminated predominantly with volatile and semi-volatile compounds. Given the status of groundwater quality beneath Southlands, in which there are significant dissolved phase concentrations of volatile compounds, excavation will be terminated immediately above the ground water table ('*smear zone*') in these areas.

At Area C, where inorganic contamination (mercury) is the principal contaminant of concern and the Phase 2 investigation (HLA, 2005) has indicated that the mercury contamination in soil is likely to leach (potentially into groundwater), remediation (excavation) will continue (beyond the water table as required) to a depth of 2 mBGL¹⁴, where validation sampling will be undertaken in accordance with protocols provided in Section 13.

9.3.3 Disposal

Excavated contaminated materials will be disposed off-site at a facility licensed by the NSW DECC to receive the waste types identified in Section 12 (or the waste types identified through subsequent and more detailed waste assessment). Detailed records of the materials movements will be maintained by the remediation contractor in accordance with framework provided in Section 11.

Where wastes contain HCB, they will be transported and treated under a separate application, which at the time of RAP preparation, was being processed by NSW DECC.

9.3.4 Excavation Validation

The floors and walls of the remediation excavations will be validated in accordance with the sampling protocols and frequencies identified in Section 13.

¹⁴ Mercury concentrations in soil samples collected from 1.9 mBGL in Area C were less than investigation levels (NEPC, 1999).

Section 9

Remediation Scope - Stage 1

9.3.5 Excavation Reinstatement

Reinstatement shall not occur until the analytical results from the validation sampling (8.3.4) have been received and assessed and it has been confirmed that the extent of the impacted soils have been reached.

The validated excavations will be reinstated with VENM. The VENM reinstatement materials will be tracked as per protocols identified in Section 13 and placed and compacted in accordance the protocols identified in Section 6.3.

9.4 Block 2 - Preparatory Works

Preparatory works are required on Block 2 to enable movement and placement of materials from Block 1. These will include the following.

9.4.1 Levelling of Stockpiled Materials

The stockpile materials that remain on Block 2, following removal of oversized components and/or gross ACM contamination, and management of impacted soils will be levelled using bulk earth moving equipment.

Further oversized materials, if encountered during this process, will be removed and transported to the designated staging area..

9.4.2 Receiving Areas

Areas for receipt and placement of materials from the Stage 2 Area will be identified and prepared (as required, i.e. levelled, impact rolled etc).

9.5 Stage 1 Works in Stage 2 Area

9.5.1 Excavation of Stockpiled Materials

The stockpiled materials in the Stage 2 area (following screening operations as per Section 9.2.4) will be progressively excavated and transported directly to the identified receiving areas on Block 2.

Further oversized materials encountered during this process will be transported to the designated staging area, located in the Stage 3 Area, for off site disposal or management.

9.5.2 Landscaping for Flood Retention Requirements

Further bulk earthworks will then be undertaken to prepare the Stage 2 area as an interim (Stage 1) flood retention area, in accordance with the findings of the site flood model (CW, 2007).

Any materials generated during this process can be transferred to Block 2, or if aesthetically unsuitable or surplus to fill volume requirements, disposed off-site in accordance with requirements identified in Section 11).

9.5.3 Back Filling of Paper Waste Ponds

Section 1.2 identified five depressions on Block 1 (Stage 2) that are periodically charged by shallow groundwater and to a lesser extent surface water by overland flow.

In Stage 1 of the development, these depressions (known as 'paper waste' ponds) will be backfilled to finished site surface levels to mitigate the potential for generation of vapours from periodic groundwater inundation.

Section 9

Remediation Scope - Stage 1

It is noted that, notwithstanding the backfilling of the ponds as part of Stage 1, later Stages 2 and 3 incorporate bulk filling to increase the site elevation to address site flood criteria (CW, 2007).

The geotechnical assessment undertaken by URS (URS, 2007a) indicated that removal of the 'soft' waste materials from the bases of the ponds will be a requirement, in areas where future development (Stages 2 and/or 3) requires structural loading.

The large paper waste pond (Pond number 1 on Figure 8) is located in the southern portion of the Stage 3 development area. While Stage 3 is not included in the current application, it is noted that concept design incorporates construction of a compensatory flood storage area (Basin 3 on Figure 3) above the current location of the paper waste pond (following the bulk filling referred to above). It is considered therefore that within the context of the current development proposal, removal of the paper waste materials for geotechnical considerations is not warranted.

The southern ponds (Numbered 2 to 5) on Figure 8 are located within Stage 2 of the development. Construction of buildings in this area is incorporated into the Stage 2 plan. Removal of the paper wastes from the ponds will be a requirement at these locations. This removal can, however, occur either prior to Stage 1 backfilling of the ponds, or prior to the eventual Stage 2 development.

It is noted that contaminated sediment has been identified in some of the ponds and requires further assessment and remediation. Such work would occur before any bulk removal of sediments or paper waste material from the ponds.

9.6 Stage 1 Filling

Following completion of the components outlined above, importation of validated VENM materials onto Stage 1 will commence.

All materials imported will be VENM and will meet the VENM specification requirements described in Section 13. They will also be required to meet the placement and compaction requirements outlined in Section 6.3.

Detailed tracking records will be maintained by the Remediation Contractor as outlined in Section 13 and will form the basis of the Validation Report, the framework for which is provided in Section 13.7.

Section 10

Stage 2 Remediation

10.1 Barrier Approach

As discussed in Section 5, it is required that the remediation approach for Stage 2 provides both a physical barrier to ACM impacted materials as well as a vapour barrier to prevent the accumulation in buildings and structures of vapours derived from contaminated groundwater.

To achieve a physical barrier against ACM impacted materials, it is proposed to construct a “*physical barrier*” across the entire site to prevent contact between site users and underlying ACM impacted contaminated materials. Given the thickness of filling (> 1m) required to meet the flood design levels (CW, 2007), it is considered that the engineered fill required to meet these flood levels and associated concrete paving will achieve this objective.

In other areas of the site where there is a risk of vapour accumulation beneath structures (i.e. confined spaces such as buildings, sheds, pits etc) an additional physical “*vapour barrier*” is proposed. Typically a vapour barrier would comprise either a plastic liner (i.e. high density polyethylene (HDPE) membrane or similar) or combination of a gas drainage layer and overlying low permeability barrier (e.g. compacted clay, GCL or plastic liner). Such a barrier would be required to be installed beneath the footprint of proposed site structures extending to the external footprint edge. Further consideration during detailed design will be required to determine if a gas drainage medium is required and if passive or active gas venting is necessary.

10.2 Barrier System

The following section provides a preliminary general specification. It is aimed at eliminating exposure pathways to control potential risks to human health. The specification is not designed to address the foundation/geotechnical requirements for specific elements of the future development including uses such as buildings / facilities etc.

A detailed barrier design will be required prior to implementation of the remedial approach which takes into consideration specific building engineering requirements whilst incorporating necessary vapour barrier/gas drainage layers and venting. Additional requirements such as ensuring all electrical equipment, machinery etc is intrinsically safe and that appropriate Australian Design Standards are met and detailed foundation/geotechnical requirements would be incorporated into the detailed barrier design.

10.2.1 Barrier Design Guidelines

The ANZECC Guidelines for Assessment of On-site Containment of Contaminated Soil 1999 (ANZECC, 1999) have been used as a guide in the specification of the remediation barrier design. Although these guidelines have not been officially endorsed by DECC,NSW (under section 105 of the Contaminated Land Management Act 1997) they do provide examples of a variety of barriers and suggest a minimum total barrier thickness of 500 mm as a conservative standard.

In addition, the guidance provided in The Department of Health and Ageing Guidelines: Management of Asbestos in the Non-occupational Environmental (enHealth, 2005) (see detailed discussion provided in Section 5.7) has been considered.

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Stage 2 Remediation

10.2.2 Physical Barrier

The physical barrier shall consist of compacted engineered fill with a minimum thickness of 500 mm after compaction. Engineered fill shall consist of a well graded granular or cohesive material capable of being compacted to meet the required compaction levels (dependent on pavement requirements but, as a minimum, 98% of the Standard Maximum Dry Density). The material shall not contain any oversize (fragments >100 mm in size). It shall not contain any material susceptible to volume change or excessive shrink/swell effects upon wetting and drying. Soils should have a liquid limit of <40% and Plasticity Index of <30%. The material shall also not contain any vegetation, peat, timber, organic, soluble, perishable or construction materials.

For areas beneath proposed structures and building footprints, it will be necessary to design and install a vapour barrier comprising either a plastic liner (e.g. HDPE membrane or similar) or a combination of a gas drainage layer and overlying low permeability barrier (e.g. compacted clay, geo-synthetic clay liner (GCL) or plastic liner). The final barrier specification will depend on the type/size of building/structure, with appropriate measures incorporated into the design such as adequate venting of vapours if required, details of sealing systems for structures required to penetrate the vapour barrier (e.g. foundations, conduits, pipes etc.) and ensuring all electrical, machinery etc. is intrinsically safe and that appropriate Australian Design Standards are incorporated into the design.

10.2.3 Upper Protection Surface Layer

The majority of the site area will be protected by either concrete paving (or similar) or built structures. Specific sub-grade requirements beneath buildings and concrete paved areas shall be designed and incorporated into the surface cover (e.g. road-base layer). In all other areas that are neither paved nor covered by structures (for example landscaped areas and easements), a protective surface layer shall be placed which includes 100 mm of topsoil and 100 mm of loose soil to accommodate seeding and growth of grasses.

To promote effective stormwater runoff, the completed surface layer shall be appropriately designed such that the grading and level provide a uniform slope with no irregularities or depressions that would allow ponding of rain water.

10.2.4 Drainage

The site design shall make provision for a stormwater control and management system to collect and convey runoff, where necessary via constructed detention systems, to the Council stormwater drain system in accordance with applicable regulations.

10.2.5 Imported Materials

All imported barrier material shall be verified as clean Virgin Excavated Natural Material (VENM) as defined in the NSW DECC (2008) Waste Classification Guidelines: Part 1 Classifying Waste and Section 13.3.

10.3 Marker Layer

The measures outlined in Section 6.1.3 with regard to the requirement for marker layer shall apply to construction of the barrier on Stage 2.

10.4 Other Design Features

The measures outlined in Section 6.1.4 with regard to loading / unloading bays and construction of stormwater water detention tanks, or any other sub surface design feature, shall apply to the Stage 2 area.

Section 10

Stage 2 Remediation

10.5 Perimeter Features

10.5.1 General Approach

The physical separation layer margins will generally be finished by constructing a 1:10 batter.

Where the minimum thicknesses of validated fill materials prescribed in Table 6.1 cannot be achieved at the edges, additional measures will be required to ensure a sufficient physical separation is achieved. These might include:

- Additional excavation prior to filling to enable placement of a suitable barrier thickness (as per 9.2.1);
- Additional separation measures such as sealing of the batter slope using “*spraycrete*” or equivalent; and/or
- Additional investigation / remediation (as required) and validation of surveyed perimeter areas, to demonstrate that those specified areas are not impacted by ACM, prior to filling.

Based on the proposed physical barrier design it is considered the completion of the perimeter areas can be achieved through a combination of the measures identified above. However, the following sections describe in more detail specific requirements for the physical barrier perimeters.

10.5.2 Boundary Easements

As shown on Figure 10 and summarised in Table 5.1, several easements are to be located at the northern, southern and eastern site boundaries of Stage 2. Specifically these include:

Table 10-1 Boundary Easements

Number	Width (m)	Purpose
Easement 4	3	Future Remediation (if required)
Easement 6	10	Continued operation PCA (GTP)
Easement 7	10	Continued operation PCA (GTP)
Easement 9	10	Future remediation (if required)

Northern Easement (Easement No. 9)

The purpose of the northern easement is to provide for installation of future groundwater monitoring and/or groundwater extraction wells or implementation of future groundwater remediation systems. Easement No. 9 is located along the northern edge of the Stage 2 area and will therefore lie directly beneath the 1:10 batter slope.

As no immediate use is envisaged for this easement, it is considered an effective physical separation layer can be implemented for the purpose of the proposed land use through the measures identified above (Section 10.5.1). The specific future uses of the easement are not currently known. It is, however, likely they would include penetration of the marker layer through drilling or some form of localised excavation.

Specific provision for these future activities will therefore need to be made in the EMP and are discussed further in Section 14.

Section 10**Stage 2 Remediation*****Southern and Eastern Easements (Easements Nos 6 and 7)***

The southern Easement No. 7 will accommodate the GTP SCA infrastructure and associated pipelines. Easement No. 6 will accommodate the GTP pipeline. The pipeline, which runs from the SCA to the GTP, returns northwards at the south eastern corner of Southlands through Easement 6.

These easements will not be covered by the physical separation layer and will occupy 10 metre wide land corridors. The site barrier design at these perimeters will be similar to that defined for the southern perimeter of Stage 1, as outlined in Section 6.2 and as shown on Figures 12 and 13. In these cases, the edges of the physical barrier layer will be located 10 metres from the site boundary.

A 10 metre land "corridor" will be defined along the southern and eastern portions of the Stage 2 (Block 1). The soils in these areas will be assessed and validated on the basis that the site design is not compatible with a physical barrier approach in this area.

Section 13 makes provision for these additional validation requirements.

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Site Management

The site management procedures provided in this section apply to works proposed to be undertaken during both Stages 1 and 2. The proposed site management procedures during the remediation works include the following:

- Identification of regulatory approvals required for works to commence;
- Environmental management procedures presented as an outline Construction Environmental Management Plan (CEMP) to be adopted during remediation works; and
- Occupational health and safety (OH&S) procedures presented as an outline Site Specific Health, Safety and Environment Plan (SSHESP) during remediation works.

11.1 Regulatory Approval Requirements

All required regulatory approvals, permits, licenses and notices will be obtained prior to commencement of the works. The following approval requirements have been identified for the project.

11.1.1 Environmental Planning and Assessment Act 1979

As outlined in Section 1, the *Environmental Planning and Assessment (EP&A Act) 1979* and the *Environmental Planning and Assessment Regulation (EP&A Regulation) 2000*, provide the framework for development and environmental assessment in NSW.

In late 2005 a new part (Part 3A) was inserted into the EP&A Act to provide for a new category of development known as Major Projects.

State Environmental Planning Policy (SEPP) 2005 (Major Projects) (SEPP Major Projects) was prepared and gazetted in conjunction with this amendment. SEPP Major Projects identifies categories of development which are considered to be Major Projects to which Part 3A of the EP&A Act applies and identifies projects of state or regional planning significance that are of a kind that the approval and assessment process under Part 3A of the Act should apply.

Group 4 class of development in Schedule 1 of SEPP (Major Projects) declares that development for the purpose of container storage facilities, or storage or distribution centres, with a capital investment value of more than \$30 million are Part 3A projects. The Minister for Planning has formed the opinion that Part 3A of the Act applies to the Southlands Proposal. Consequently the Minister is the approval authority for this project.

11.1.2 SEPP 55, Remediation of Land

The State Environmental Planning Policy No. 55 (SEPP 55), Remediation of Land, provides a state wide planning approach for the remediation of contaminated land. In particular, SEPP 55 defines remediation as either Category 1 or Category 2. Remediation defined as Category 1 requires development consent, while remediation defined as Category 2 does not require development consent.

As Part 3A of the EP&A Act applies to the Southlands Proposal, the consent requirements of SEPP 55 are not considered further here.

11.1.3 Local Council Contaminated Land Policy

Whilst Part 3A of the Act triggers ministerial approval, it is recommended that consideration also be given to the specific requirements of the City of Botany Bay (CoBB) Council under Development Control Plan (DCP) No. 34 – *Contaminated Land (Version 5)*, dated February 2003 (CoBB, 2003). CoBB are a stakeholder in the approval process and the specific requirements of DCP No.34 should be consulted, particularly in during the preparation of the site specific Construction Environmental Management Plan (CEMP).

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In Section 4 of DCP No.34 (CoBB, 2003), the following requirements, that are considered relevant to this project, are specified:

- Hours of operation;
- Soil and water management;
- Stockpiles;
- Site access, security and signage;
- Excavation pump-out;
- Landscaping and rehabilitation;
- Bunding;
- Noise,
- Vibration;
- Air quality, dust and odour control;
- Groundwater;
- Transport;
- Hazardous materials;
- Disposal of contaminated soil;
- Containment and capping of contaminated soil;
- Importation of fill;
- Community communications; and
- Occupational health and safety.

11.1.4 Licenses and Other Requirements

All works are to be carried out by appropriately licensed operators. This would include and would not be limited to:

- The Contractor carrying out works associated with asbestos is required to hold a current friable AS1 asbestos licence from WorkCover NSW. The contractor must also make an application to WorkCover for the remediation, in accordance with WorkCover requirements; and
- All NSW WorkCover codes of practice and regulations relating to asbestos, including the Protection of the Environment Operations (Waste) Regulation, 2005, the National Occupational Health and Safety Commission (2002) Code of Practice for Safe Removal of Asbestos and all other requirements of the NSW Occupational Health and Safety Act 2000 and the NSW Occupational Health and Safety Regulation 2001, are to be complied with. The WorkCover Authority NSW is to be notified prior to the commencement of work.

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In addition the requirements specified in the following legislation is relevant:

- Waste Avoidance and Resource Recovery Act 2001;
- Protection of the Environment and Operations Act 1997;
- Environmentally Hazardous Chemicals (EHC) Act 1985, in particular the requirements of the Scheduled Chemical Wastes Control Order (2004) made under the EHC Act.
- Contaminated Land Management Act 1997.

11.2 Outline Construction Environmental Management Plan

The CEMP shall be prepared in accordance with the CoBB Development Control Plan No.34 (CoBB, 2003) and will detail, as a minimum, the requirements during the remediation works in relation to the management of soil, stormwater, groundwater, noise, dust, odour and waste management.

The objectives of the CEMP will be to perform the remediation works:

- in accordance with the appropriate statutory environmental and occupational health and safety requirements;
- in accordance with good environmental practice;
- in such a way as to minimise the likelihood of environmental degradation;
- such that all employees engaged in the works comply with the requirements of the plan;
- in response to changes in environmental conditions through review of the monitoring and control programs and liaison with regulatory authorities; and
- such that corrective actions are performed in a timely manner.

11.2.1 Fencing and Security

The Site is currently fenced, however the prior to commencement of works the appointed Remediation Contractor may be required to erect temporary fencing in additional areas within the currently fenced site if required., so as to minimise the potential for unauthorised access to excavation areas and to assist in environmental management and establishment of Exclusions Zones required for control of occupational health and safety exposures and potential cross contamination of clean/remediated areas during the works.

During the period of the Remediation Contractor would be responsible for the maintenance and repair of the fences surrounding and within the Site.

11.2.2 Soil and Erosion Management

Objectives

The CEMP will include procedures for management of soil and erosion to meet the objectives of the following guidance documents:

- Department of Conservation and Land Management: CALM (1992) Urban Erosion Control and Sediment Control; and
- Landcom: Managing Urban Stormwater: Soils and Construction (2004);

The CEMP will also include the following generic project objectives:

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Site Management

- to control soils (and potentially asbestos fibres) released as a result of site excavations and the handling, including stockpiling, loading and/or placement of site and imported materials;
- to control erosion and potential sediment transport from the site; and
- to minimise the extent of soil transport and erosion by heavy machinery.

Soil and Erosion Control Measures

Soil and erosion management during the remediation activities may be controlled by:

- construction of earth bunds and similar diversion drains around the perimeter of remediation areas where surface disturbance occurs, to prevent surface water entering these areas;
- erection of silt fences or straw bales at strategic locations (around stockpiles etc.) to prevent the migration of fines;
- construction of temporary sediment retention ponds;
- installation of water recycling equipment for dust suppression as needed;
- minimising the surface area disturbed by remediation activities at any one time;
- regular inspection and maintenance of erosion control structures;
- protecting and retaining surface cover where possible in order to avoid erosion which may result from their removal;
- placement of an erosion protection barrier (eg grassing) at the completion of works;
- protecting and retaining surface cover in order to minimise potential for fibre release from asbestos impacted soils;
- restricting vehicles to designated access roads and paths where possible;
- washing trucks and plant (bodies, wheels and undercarriage) moving from asbestos impacted areas to other areas of the site or off site; and
- removing soil adhering to the wheels and undercarriage of trucks (eg by wheel wash) prior to departure from the site;

In addition, every effort will be made to limit both the size of any stockpile footprints and the time between excavation and removal off-site of materials.

11.2.3 Water Management

The two potential sources of contaminated water at the site include stormwater which comes in contact with contaminated soil and contaminated groundwater within excavations.

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Objectives

The CEMP will include procedures for management of water to meet the following objectives:

- to prevent surface run off from becoming contaminated as a result of soil disturbance or contact with site contaminants;
- to control erosion and sediment transport from the site, and thereby minimise the potential for off-site contamination of surface water by sediment;
- to control and manage run off in sediment control ponds prior to discharge from the site; and
- to minimise potentially contaminated groundwater entering the excavations.

Water Control Measures

Given the known contamination status of shallow groundwater beneath Block 2, the CEMP shall make provision to preclude (or at least significantly minimise the potential for) interception of shallow groundwater during excavations in this area. The letter report prepared by JBS (JBS, 2008) shall be referred to in preparation of this plan (see Appendix E of the HHRA). The CEMP will provide contingency planning for the potential interception of shallow groundwater during excavation works.

The safeguards which will be implemented to prevent sediment entering surface waters are similar to those listed for the soil and erosion controls. Additional safeguards are listed as follows:

- in the event that groundwater is intercepted through excavation, it shall not be purged from the excavations for disposal. The excavation shall be backfilled such that the base of the excavation is reinstated to above the groundwater table;
- installation of sediment ponds and silt traps, as necessary, to manage sediment in stormwater run-off from the remediation activities;
- routine clearance of silt in sediment pond(s) used for wet weather runoff containment in order that their capacity is not reduced more than 10% by volume. Sediment ponds will be regularly inspected for storm damage and where necessary cleaning and/or repairs will be undertaken as soon as practicable;
- water in sediment ponds will either be pumped out for off-site disposal or allowed to infiltrate through the underlying sands depending on the water quality;
- inspection of ponds on a weekly basis and after rain to ensure correct operation; and
- any overland and diffuse drainage paths will be addressed by the installation of straw bales or filter fences that will be inspected at least weekly and extended, relocated or repaired as necessary.

11.2.4 Noise and Vibration Control

Objectives

The CEMP will include procedures for management of noise and vibration to meet the following objectives:

- to identify best practice techniques to minimise unnecessary noise and vibration; and
- to adhere to regulatory limits for noise emissions.

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Noise and Vibration Control Measures

The following control measures will be used to minimise the impact of noise on neighbouring properties:

- neighbouring premises will be informed of the work programme, potential impacts, and measures taken to minimise the impacts;
- a minimum of 48 hours notice will be given to directly affected premises prior to remediation works commencing;
- advising neighbouring premises of any variation to work hours prior to the works occurring;
- the contractor shall sequence the remediation works in such a manner as to take into consideration the requirement to minimise noise impacts to neighbouring properties;
- all equipment will be selected on the basis of its noise performance and will comply with regulatory standards for noise generation;
- equipment will be operated in a proper, efficient and correct manner which includes proper maintenance;
- Vibrations generated on the site shall be within DECC noise control guidelines as specified in the *Noise Guide for Local Government*, DEC, NSW 2007

11.2.5 Air Quality Management

Objectives

The CEMP will include procedures for air quality management, including dust and odour, to meet the following objectives:

- to conform with WorkCover, NSW, DECC, NSW and Council requirements for the management of dust and ACM;
- to minimise dust emissions from the site which could adversely affect air quality or the amenity of the local area.

Works would be conducted with reference to the following relevant guidance documents:

- National Environment Protection Council (1998): National Environment Protection Measure (NEPM) on Ambient Air Quality;
- DEC, NSW (2005): Approved Methods for the Modelling and Assessment of Air Pollutants in NSW;
- DEC, NSW (2007): Approved methods for the Sampling and Analysis of Air Pollutants in NSW; and
- National Occupational Health and Safety Commission, 2nd Edition [NOHSC: 2002 (2005)]: Code of Practice for the Safe Removal of Asbestos.

Dust Control Measures

Dust management is of vital importance to the project, given the potential for ACM across the site. Dust generation and transport off-site will be controlled by:

- provision of *shade cloth* along the site boundary fences. This can probably be limited as required to the area of the current area of works;
- confining vehicle movements to controlled and managed haul roads;
- restricting the speed of vehicle movements;

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- use of water sprays to suppress dust generation;
- watering of active work areas including haul roads and stockpiles by water cart and/or sprinklers to suppress dust in dry conditions when necessary (the water will be applied across ground surfaces to prevent the surface drying out and generating visible levels of dust, either by the operation of equipment over the surface or by wind);
- following completion of remedial works, stabilise surface as soon as practical by means of seeding, hydromulching, capping or suitable alternative;
- covering truckloads of materials as necessary; and
- should dust become a problem, corrective action shall be undertaken which may include alteration of operations, restriction of dust generating activities to low wind speed conditions or the augmentation of dust suppression equipment.

Dust Monitoring

The effectiveness of dust control measures will be monitored by:

- qualitative assessment (i.e. visual inspection); and
- air quality monitoring.

Ambient air quality monitoring would be required to be conducted throughout the remediation earthworks program to monitor compliance with ambient air quality standards at the boundary of the remediation site. This program will be implemented to demonstrate the works are not adversely impacting the surrounding environment.

The monitoring program would likely include:

- Fine particulate monitoring using a portable PM10 monitor. This monitor provides real time instantaneous results which can show compliance with the relevant air quality criteria. Real time monitoring would be undertaken on a daily basis for the duration of the remediation earthworks. Specified acceptance criteria which would trigger “stop work” orders would be developed for the works; and
- Dust deposition gauges would be located at locations on the boundary of the work site to measure total particulates. These gauges would measure dust deposition over 1 month periods. It is proposed that dust gauges be present over the course of the remediation and reinstatement works (including landscaping).

11.2.6 Odour/ Vapour Management

Objectives

The overall objective of odour management is to control the amount of odours/vapours generated from the proposed works, and ensure minimal impact on the air quality of the local area, and health of on-site workers and the general community.

Odour/Vapour Management Measures

The following odour/vapour management measures will be implemented:

- the area of contaminated soils exposed at any one time would be minimised wherever possible by a localised staged program; and
- the application of odour and volatile suppressing agents (such as Biosolve or similar product).

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Odour/Vapour Monitoring

Odour monitoring would be undertaken prior to, during and following the proposed works. Monitoring of volatile organic compounds at the site boundary will be undertaken using suitable field screening equipment (e.g. photoionisation detector (PID)) fitted with a suitable lamp for monitoring of CHCs.

11.2.7 Waste Management

Objectives

The objectives of the CEMP in relation to waste management are as follows:

- to comply with all relevant regulatory requirements;
- to minimise volumes of materials disposed off-site; and
- to recover or recycle materials where possible.

Waste Management Control Measures

The following waste management control measures will be implemented:

- preparation of a comprehensive waste materials handling and recycling plan (Waste Management Plan) by the appointed Remediation Contractor. This would involve the pre-commencement identification of all waste streams to allow development of a comprehensive WMP which will minimise wastes from the site and wherever possible and reasonably practicable, recommend recovery and recycling;
- periodic assessment and review of WMP;
- identification of appropriately licensed waste transporters and waste management facilities for each waste stream; and
- implementation of a system for tracking of all waste movements including contaminated soils.

11.2.8 Traffic Management

Objectives

The objective of the traffic management within the CEMP will be to undertake the works with regard to the safety and welfare of the general public and to alleviate the impact of additional traffic volumes on site access roads and traffic flow in the surrounding area.

Traffic Control Measures

The Remediation Contractor would be required to develop and implement a Traffic Management Plan (TMP) for the works. This would as a minimum include the following traffic control measures:

- establishment of designated haul routes;
- utilisation of transport routes approved in the development consent conditions (if any) when travelling to and from the site as required;
- all access to, and egress from, the site will be forwards from and to McPherson Street;
- access to neighbouring premises will be maintained at all times;
- if appropriate, erection of hazard warning signs to warn traffic on McPherson Street of potential traffic hazards associated with traffic entering the roads;

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- if necessary, stockpile materials on-site temporarily to regulate and control the truck movements;
- selection of roads which are to be used for the transport of excavated materials on the basis that they have adequate capacity to accommodate the increased traffic volumes;
- retention of heavy equipment on-site where practicable; and
- provision for a staging area for vehicles on site to prevent queuing on public roads outside of the site.

All truck drivers carting materials from the site will be given a safety instruction brief. The brief will be concise and shall detail the procedures to be followed by the truck driver should spillage of loads occur. These will include, but not be limited to:

- vehicle accident;
- mechanical breakdown;
- rain commencing during transportation, and
- payload (or other) loss.

11.3 Well Replacement Program Plan

A monitoring well replacement plan shall be prepared by Orica (or their consultants).

A preliminary well replacement network is presented in Figure 11, however, it is noted the locations may be refined during later detailed site planning. Included on this plan are the proposed locations for two replacement GTP extraction bores in the southern portion of Block 2 (Stage 1). These wells will replace the existing GTP extraction bores located in the central and southern portions of Block 2 Southlands. These wells include EWB02, EWB06 and EWB14D.

The plan will provide a detailed well replacement program, which will be phased on the basis of the proposed sequence of site works and the groundwater quality reporting requirements of the NCUA.

The overall objective of the well replacement plan will be to provide a documented framework that ensures Orica's ongoing responsibilities under the NCUA for hydraulic and chemical monitoring can be met both during the construction stages of the development and during the future land use.

The plan shall be prepared in accordance with The Minimum Construction Standards for Waterbores in Australia (LWBC, 2003). The plan shall include (as a minimum) the following components:

- Presentation of the NCUA requirements;
- Review of the proposed sequence of works and identification of the order in which existing monitoring wells will be destroyed or rendered inaccessible;
- Identification of requirements for interim measures (where appropriate);
- Demonstration of the proposed phasing for installation of permanent replacement wells;
- Presentation of proposed well designs;
- Presentation of well decommissioning measures to include (as a minimum):
 - Removal and disposal of above ground infrastructure such as monuments and standpipes etc;
 - Removal of underground infrastructure where practical and/or practicable;
 - Measures to prevent surface water reaching the water table such as backfilling, grouting and/or cementing of the remnant standpipe or annulus.

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11.4 Outline Site Specific Health, Safety and Environment Plan

The outline Site Specific Health, Safety and Environment Plan (SSHSEP) provides a general description of the chemical and physical hazards associated with the work to be undertaken as part of the remediation works. The primary objective of the SSHSEP will be to establish the health and safety requirements and protection procedures to minimise the potential for unsafe exposures and injuries to field personnel.

The SSHSEP will describe the procedures to be followed and the protective equipment to be used by personnel working on-site.

11.4.1 Site Health and Safety and Environment Inductions

All personnel involved in remediation, monitoring and validation activities will be required to attend a site specific occupational health and safety and environmental management induction prior to commencing work on the site. During the induction, an overview of the project will be presented including:

- scheduled remediation activities and personnel responsibilities;
- site control procedures;
- contaminant and hazard identification and precautions;
- exposure risks;
- warning symptoms from exposure to contaminants;
- personal protective equipment usage and maintenance;
- decontamination facilities and procedures;
- prohibitions;
- general hygiene; and
- emergency response procedures.

The occupational health and safety and environmental management induction will be provided to new personnel on arrival at the site. On-site safety meetings/tool box talks will be completed on a minimum daily basis to allow the review of safety and environmental management requirements and discuss modifications to the SSHSEP.

11.4.2 Compliance Agreements

All personnel involved in the remediation activities at the site will be required to sign a compliance agreement after attending the occupational health and safety and environmental management induction and prior to commencing work on site.

11.4.3 Site Mobilisation

Prior to commencement of remediation activities, the following activities will be undertaken:

- establishment of monitoring program and equipment;
- installation of decontamination facilities;
- establishment of site amenities;
- establishment of exclusion zones;

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Site Management

- site survey;
- installation of safety equipment;
- installation of pollution control measures;
- establishment of area fencing/bunding and impacted area exclusion zones;
- access road improvements; and
- project signage.

Exclusion Zones

As part of the Occupational Health & Safety requirements, access within the Site shall be controlled. The Remediation Contractor will be expected to implement varying controls depending on the personnel and whether the proposed work area is in an Exclusion Zone.

Exclusion Zones are areas of the Site that either require additional protective measures or may require the adoption of additional occupational health and safety requirements and work practices. The Exclusion Zones are anticipated to primarily correspond to where there is a potential for exposure to excavated or stockpiled materials. In each case, the Exclusion Zone will incorporate a buffer area along the boundary of the zone.

Access of personnel into and out of Exclusion Zones will be controlled, with these controls varying depending on the personnel classification.

The boundary of all Exclusion Zones will be defined by construction of hazard tape fences. Safety signs shall also be erected at regular intervals around each Exclusion Zone warning on-site personnel of the boundary of the Exclusion Zone, the nature of the hazard associated with it and access restrictions that apply to entry into the Zone. Maintenance of the Exclusion Zones will be the responsibility of the appointed Remediation Contractor.

11.4.4 Occupational Monitoring

The following occupational monitoring is specified for the project:

Asbestos Air Monitoring

The purpose of this monitoring is to assess the effectiveness of controls implemented to minimise the production of airborne asbestos fibres and dusts during the remediation program, for the protection of all site workers and the public. **An appropriately qualified independent asbestos sub-consultant will be engaged to design and implement the asbestos air-monitoring program.**

Volatile Emissions and Potential Explosive Atmospheres

Atmospheric monitoring for organic vapours using a field portable photo-ionisation detector (PID) should be undertaken periodically in the breathing zone during excavations and assessment of impacted soils. The results of the monitoring will direct the appropriate personal protection equipment that will be required.

Periodic monitoring for potential explosive atmospheres should be undertaken using combustible gas and oxygen meters in the vicinity of hydrocarbon impacted soils (specifically in Areas A and B on Southlands Block 1. It is not expected that confined space entry will be required as part of the remediation activities.

11.4.5 Decontamination Procedures

The SSHSEP will outline the procedure for decontamination of personnel leaving the Exclusion Zone prior to breaking for eating, drinking, smoking, use of ablution facilities and before leaving the site.

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All tools, vehicles or equipment which have been in contact with contaminated materials will be decontaminated after leaving the Exclusion Zone.

Section 12

Preliminary In-Situ Waste Assessment

12.1 General

Areas of impacted soils are highlighted on Figures 8 and 9. These include Zones A, B and C and some other more isolated areas where excavation of impacted material for off-site treatment and/or disposal has been nominated.

Based on the number of available samples and the volume of material involved, the available data is generally not adequate for a detailed waste assessment and classification. Typically a sampling density of 1 per 100 m³ would apply. The following section provides a preliminary, in-situ, waste classification for each of the areas where excavation and disposal is required.

The soil quality data for the highlighted areas have been compared with the threshold concentrations provided in the DECC, NSW (2008) Waste Classification Guidelines: Part 1 Classifying Waste. Table 1 presents the preliminary assessment.

12.2 Block 1

12.2.1 Area A

It is likely that the inferred extent of the soil contamination in Area A can be reduced through additional investigation. Several contaminants of potential concern were not detected during the most recent investigations undertaken by URS in December 2006. The inferred extent of the impacted material as shown on Figure 8 may therefore be smaller, in which case the amount of waste requiring treatment and/or disposal would be reduced.

Some materials from Area A would be classified as Scheduled Wastes, for which handling and disposal are controlled under the Environmentally Hazardous Chemicals (EHC) Act 1985.

Orica may consider treatment of HCB impacted materials excavated from Area A using the Direct Thermal Desorption (DTD) plant proposed for establishment on BIP for treatment of other HCB impacted materials. This project is currently subject to assessment by the DoP under Part 3A of the Environmental Planning and Assessment 1979 (EP&A Act). Given the timeframe for planning and establishment of that plant, it is considered provision is required for storage and containment of excavated wastes off-site (on BIP), if they are to be treated on BIP.

The following sections provide a summary waste assessment for each of the analytes tested, however, it is noted that the *Scheduled Waste* classification would be the main driver in management of these materials.

Inorganics

In-situ total concentration data for several metals, in particular lead and mercury, would classify the soil as *Hazardous*¹⁵ Waste following its excavation. This classification may be able to be lowered if toxicity characteristics leaching procedure (TCLP) testing data is acquired for metals.

Chlorinated Aliphatic Compounds

In-situ concentrations of tetrachloroethene (PCE), trichloroethene (TCE) and vinyl chloride (VC) would currently classify the soil as *Restricted Solid* (and in one location *Hazardous*) Waste following its excavation. The *Waste Guidelines* do, however, provide TCLP threshold concentrations for these compounds and collected of additional data may result in a lower waste classification.

¹⁵ Italics indicate a waste assessment and classification completed in accordance with the *Waste Guidelines* (NSW DECC, 2008).

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Preliminary In-Situ Waste Assessment

URS further notes that the concentration of VC in sample TP37_1.2-1.3 would be result in a classification as *Hazardous Waste* irrespective of the availability of TCLP data.

URS also notes that elevated concentrations of compounds (in particular cis-1,2-dichloroethene) for which no threshold concentrations are provided in the *Waste Guidelines* were recorded. In accordance with the *Waste Guidelines* advice should be sought from the DECC, NSW for assistance in classification of wastes containing such compounds.

Chlorinated Compounds

Elevated concentrations of hexachlorobenzene (HCB) and hexachlorobutadiene (HCBd) and in one case (HA02) pentachlorobenzene were reported in the samples assessed.

While the *Waste Guidelines* do not provide threshold concentrations for these compounds in Tables 1 and 2, HCB and pentachlorobenzene are included in the Table 2 notes as Scheduled Chemicals.

Within the framework provided in the Scheduled Chemical Wastes Chemical Control Order 2004 (NSW EPA, 2004), wastes that contain scheduled chemicals at a combined concentration above two milligrams per kilogram (mg/kg) are classified and regulated as *Scheduled Wastes*.

The Chemical Control Order (NSW EPA, 2004) imposes specific conditions for the conveying, storage, treatment and disposal of scheduled chemical wastes.

Asbestos

While asbestos was not tested for in any of the samples collected from Area A, as noted in Section 5 there is potential for ACM to be present in both stockpiled and buried fill materials. The presence of asbestos in the materials would result in the waste being classified as "Special Waste" in accordance with DECC, NSW (2008) *Waste Guidelines*. This class of waste has unique regulatory requirements which specify how the waste should be handled, transported and disposed of.

12.2.2 Area B

Inorganics

In-situ total inorganic concentrations for soil sampled in Area B would classify the materials, following excavation as *Restricted Solid* (and in some cases *Hazardous*) *Waste*. The availability of TCLP test data may allow a lower classification. However, that at least two total mercury concentrations (94.6 mg/kg at TP01_1.4-1.5 and 75.6 mg/kg at TP02_0.8-0.9) would be classify the wastes as *Restricted Solid Waste*, irrespective of TCLP test concentrations.

Total Petroleum Hydrocarbons

In-situ concentrations of TPH (C₆ to C₉ range) would, for two of the samples from Area B, classify the materials, following excavation as *Restricted Solid Waste* and in one of the samples (TP82_1.4-1.5) as *Hazardous Waste*.

URS recommends, as for Area A, that the an additional sampling and analytical program be undertaken to refine (if possible) the volume of materials requiring remediation and to provide a better assessment of the likely waste classifications and volumes of material requiring treatment and/or disposal, prior to commencement of development.

Chlorinated Compounds

URS notes that the in-situ total PCE concentration in TP02 would mean that materials at this location would be classified, following excavation, as *Restricted Solid Waste*. The collection of additional TCLP test data could however, reduce this classification.

Section 12**Preliminary In-Situ Waste Assessment*****Nitrosamines***

An elevated concentration (268 mg/kg) of N-Nitrosodiphenyl & Diphenylamine was detected at sampling location TP02_0.8-0.9. No threshold concentrations are provided in the *Waste Guidelines* for this compound.

12.2.3 Area C***Inorganics***

As for Areas A and B, the total in-situ inorganic concentrations for samples collected to date would classify the materials, following excavation, as *Hazardous Waste*. Additional TCLP data may however provide a lower classification. The total mercury concentrations would, irrespective of TCLP data, classify the excavated materials as *Restricted Solid Waste*.

Polycyclic Aromatic Hydrocarbons

The in-situ benzo(a)pyrene concentrations would currently classify the Area C material, following excavation, as *Hazardous Waste*. Additional TCLP data may, however, provide a lower classification.

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Site Validation

Validation and characterisation of the remediated site is required to ensure that remediation works have been conducted in accordance with requirements of this RAP.

Validation results and documentation also provide a record of the remediation works completed for the review of the DECC, NSW Accredited Site Auditor (*the Auditor*) as part of the Site Audit process.

13.1 Roles and Responsibilities

A suitably qualified and experienced environmental consultant (EC) should be engaged by the Proponent to undertake validation of the remediation and characterisation program. The EC shall report directly to the proponent and will liaise with the Auditor as required. The EC shall liaise with (and direct as appropriate) the remediation contractor (RC) engaged by the Proponent for the works.

The EC will undertake the validation works in accordance with the framework and protocols provided in this RAP. The EC will oversee and document the following remediation components:

- Supervision and validation of impacted materials;
- In-situ and, as necessary ex-situ (stockpile) assessment and waste classification of contaminated materials nominated for excavation;
- Review of outputs from the material tracking system and spot checks (as required) during bulk movement of materials within the site;
- Assessment of VENM source areas (it is noted the remediation contractor may engage another consultant to undertake the assessment of the VENM sources or may rely on validation data provided by the material supplier, however, the EC shall be responsible for collation and presentation of such inputs into the Validation Report);
- Audit / checking / presentation of RC's tracking records for all material movements within the site and importation of VENM onto the site; and
- Verification of physical separation implementation. This will include (but not be limited to) verification of:
 - The barrier layer thickness;
 - The barrier layer compaction;
 - Oversight and documentation of marker layer installation.
- Verification of the ambient air quality in the vicinity of Springvale Drain following commissioning of the proposed shallow groundwater extraction system (see Section 7 and 13.6).

13.2 Validation Documentation

A site specific Remediation Validation Plan (RVP) will be prepared by the EC for review by the Auditor prior to the commencement of works. The framework for the RVP is provided in this section. The RVP will incorporate Sampling, Analysis and Quality Procedures for each of the required remediation validation activities.

The EC will be responsible for documentation of the remediation program through maintaining comprehensive records of site activities for inclusion in a Validation Report (VR). The intent of the VR is to present material tracking and validation data in a consolidated form to enable the Auditor's review.

The VR will form one of the supporting documents for the site audit process.

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Site Validation

The sequence of the proposed remediation validation components is described in Section 9. The following section presents site adopted assessment and/or validation criteria that will be applicable to each of the remediation components.

13.2.1 Assessment of Excavated Impacted Materials as Waste for Off-site Disposal

The HHRA (URS, 2008a, refer to Appendix A) identified areas on Block 1 and Block 2 that require remediation prior to bulk earth works in Stage 1. The areas are identified and addressed in Section 3 and the locations and approximate extents¹⁶ are presented in Figures 8 and 9.

Preliminary waste classifications for the impacted materials to be removed from Blocks 1 and 2 are provided in Section 12 by applying the DECC, NSW¹⁷ *Waste Guidelines* (NSW DECC, 2008).

Where required, waste assessment and classifications during the works would also be undertaken in accordance with the *Waste Guidelines* (NSW DEC, 2008).

13.2.2 Validation of Excavation Surfaces (Following Removal of Impacted Materials Removal)

This section identifies environmental guidelines that will be adopted for the validation of residual soils (walls and floors of excavations) following removal of contaminated materials. The areas requiring remediation are identified on Figures 8 and 9. Investigations of soil quality undertaken to date have shown the contaminants of concern (COC) vary between the identified areas.

13.2.3 Assessment Guidelines

Validation sample results will be assessed against the Health Investigation Levels (HIL) provided in **Table 5-A – Soil Investigation Levels** in Section B (1) of the National Environmental Protection Council, National Environmental Protection (Assessment of Site Contamination) Measure (NEPC, 1999) and the NSW EPA (1994), Contaminated Sites: Guidelines for Assessing Service Station Sites (the *Service Station Guidelines*). For other COC where guidelines are not available from these sources, criteria will be adopted from other sources as outlined below.

National Environmental Protection Council, 1999

NEPC (1999) provides HILs for a variety of exposure settings. It is considered those provided for industrial/commercial land use scenario (HIL F) are appropriate for site validation. These values are provided in Column 1 of Table 13.1.

NSW EPA Service Station Guidelines, 1994

The NEPC (1999) guidelines do not include guidelines for BTEX compounds. The *Service Station Guidelines* (1994) provide suitable human health and ecologically-based guidelines for assessment of these compounds in soils and are adopted as site validation guidelines for this purpose. These values are presented in Column 2 of Table 13.1.

¹⁶ Lateral extent based on the distance to the nearest available location, where sampling has shown concentrations are less than adopted criteria.

¹⁷ The DEC, NSW was renamed The Department of Environment and Climate Change (DECC) in April 2007.

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Site Validation

Site Specific Risk Based Criteria

Risk Based Soil Criteria (RBSC) have been derived for key COPC identified in the HHRA (URS, 2008a, refer Appendix A). The RBSC are preliminary values derived on the basis of assumptions and models presented in the HHRA. The preliminary RBSC are site-specific with respect to exposure parameters as well as the mix of chemicals. These are presented in Column 3 of Table 13.1.

US EPA Preliminary Remediation Goals

For other COPC that were not identified as key chemicals with respect to risk, and for which there are no criteria in either the NEPC (1999) or the *Service Station Guidelines* (1994), assessment criteria have been adopted from the US EPA Preliminary Remediation Goals (PRGs) (2004). The Industrial Soil PRGs are considered appropriate for the intended use of the site and are presented in Columns 4 and 5 of Table 13.1.

The USEPA PRGs for volatile compounds in Column 4 of Table 13.1 have been divided by a factor of 100. The HHRA (URS, 2008a, refer Appendix A) adopts these modified values for the purpose of undertaking a screening level assessment of volatile compound concentrations in soils for use across the site. The reduction factor has been applied as the PRGs have been calculated on the basis of potential for contaminants in soil to affect outdoor air quality and not indoor air quality. As a consequence, a factor of 100 has been applied when comparing soil concentrations to the PRG values for volatile compounds.

Table 13-1 Validation Criteria (mg/kg)

Contaminants of Concern	NSW EPA (1994) Service Station Guidelines	NEPC (1999) HIL "F"	Preliminary RSBC	US EPA (2004) PRGs	US EPA (2004) PRGs (modified for volatiles)
Column No.	1	2	3	4	5
Metals and Metalloids					
Arsenic (As)	-	500	-	-	-
Cadmium (Cd)	-	100	-	-	-
Chromium III	-	60%	-	-	-
Chromium VI	-	500	-	-	-
Copper (Cu)	-	5,000	-	-	-
Lead (Pb)	-	1,500	-	-	-
Mercury (Hg)	-	75	-	-	-
Nickel (Ni)	-	3,000	-	-	-
Zinc (Zn)	-	35,000	-	-	-
Hydrocarbons					
TPH C ₆ -C ₉	-	-	100	-	-
TPH C ₁₀ -C ₁₄	-	-	2000	-	-

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Contaminants of Concern	NSW EPA (1994) Service Station Guidelines	NEPC (1999) HIL "F"	Preliminary RSBC	US EPA (2004) PRGs	US EPA (2004) PRGs (modified for volatiles)
<i>Column No.</i>	1	2	3	4	5
TPH C ₁₅ -C ₃₆	-	-	5000	-	-
Benzene	1	-	-	-	-
Toluene	130 ^a	-	-	-	-
Ethyl Benzene	50 ^a	-	-	-	-
Xylene	25 ^a	-	-	-	-
<i>Poly-aromatic Hydrocarbons</i>					
Total PAHs	-	100	-	-	-
Benzo(a)pyrene	-	5	-	-	-
Naphthalene	-	-	-	-	1.9
<i>Chlorinated Aliphatic Compounds</i>					
1,1,1,2-Tetrachloroethane	-	-	-	-	0.073
1,1,1-Trichloroethane	-	-	-	-	69
1,1,2,2-Tetrachloroethane	-	-	20	-	-
1,1,2-Trichloroethane	-	-	9	-	-
1,1-Dichloroethane	-	-	45	-	-
1,1-Dichloroethene	-	-	-	-	4.1
1,2-Dichloroethane	-	-	4	-	-
Carbon Tetrachloride	-	-	0.2	-	-
Chloroethane	-	-	-	-	0.065
Chloroform	-	-	8	-	-
cis-1,2-Dichloroethene	-	-	4	-	-
Tetrachloroethene	-	-	12	-	-
trans-1,2-dichloroethene	-	-	5	-	-
Trichloroethene	-	-	12	-	-
Vinyl chloride	-	-	0.3	-	-

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Contaminants of Concern	NSW EPA (1994) Service Station Guidelines	NEPC (1999) HIL "F"	Preliminary RSBC	US EPA (2004) PRGs	US EPA (2004) PRGs (modified for volatiles)
Column No.	1	2	3	4	5
Chlorinated Aromatic Compounds					
1,2,4-Trichlorobenzene	-	-	-	-	2.2
1,3,5-Trichlorobenzene	-	-	15	-	-
1,2-Dichlorobenzene	-	-	-	-	41
1,3-Dichlorobenzene	-	-	-	-	21
1,4-Dichlorobenzene	-	-	-	-	0.079
1,2,4,5-Tetrachlorobenzene	-	-	-	-	1.8
Chlorobenzene	-	-	-	-	5.3
Semi-Volatile Chlorinated Hydrocarbons					
Hexachlorobenzene (HCB)	-	-	-	1.1	-
Hexachlorobutadiene	-	-	-	22	-
Hexachlorocyclopentadiene	-	-	-	3700	-
Hexachloroethane	-	-	-	123	-
Pentachlorobenzene	-	-	-	492	-

- No guidelines specified

a Human Health and ecologically based protection levels used (NSW EPA, 1994).

13.2.4 Contaminants of Concern

The soils in each of the identified areas (see Figures 8 and 9) will be validated against the following contaminants of concern.

Area A

- Chlorinated Aliphatic Compounds; and
- Semi-volatile Chlorinated Hydrocarbons.

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Area B

- Mercury;
- TPH;
- BTEX;
- PAH;
- Chlorinated Aliphatic Compounds; and
- Semi-volatile Chlorinated Hydrocarbons.

Area C

- Mercury; and
- PAHs.

13.2.5 Sampling Frequency

Excavation Floor

The floors of the excavations will be sampled on a 25 m systematic grid based pattern.

Excavation Side Walls

Side wall sampling will achieve a sampling density of 1 sample per 25 m² of wall area. Samples will be collected from the 'mid-point' of the excavation walls, or where excavation depths exceed 1 m, at 1 m depths.

If 1 m vertical intervals are not sufficient to characterise individual layers of fill or natural materials exposed through excavation, the vertical sampling frequency will be increased appropriately. If present samples will be collected to represent each identified layer.

Sample Location Identification

The location of each validation sampling location will be recorded by a registered surveyor or by an appropriately accurate global positioning system (GPS).

13.3 Validation of Imported VENM

All material imported to the site to be used in construction of the physical separation barrier, shall be virgin excavated natural materials (VENM) as defined in the *Waste Guidelines (NSW DECC, 2008)*.

Some of the unsealed, finished surface areas may require other materials such as manufactured or blended growing medium, which will also meet the adopted validation criteria.

The EC will have overall responsibility for validation of VENM, but it is noted the remediation contractor may engage another consultant to undertake the assessment of the VENM sources or may rely on validation data provided by the material supplier. However, the EC shall be responsible for collation and presentation of such inputs into the Validation Report.

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13.3.1 Source Characterisation

For VENM proposed to be imported to the site, the following will be undertaken:

- The site history of the proposed VENM source will be reviewed and the source area will be inspected by the EC. The site history review and inspection will assess whether any activities (historic or current) undertaken at the site may potentially have caused contamination of the source material; and
- Samples will be collected from the proposed source area in accordance with the RVP and the analytical results will be assessed against the criteria provided in Table 13.2. The details of the source area review, inspection and sampling will be documented by the EC and included in the VR.

Samples (at least five discrete samples) from each source will be tested for:

- Inorganics (arsenic, cadmium, chromium, copper, lead, zinc, nickel, mercury);
- Total petroleum hydrocarbons (TPH); and
- Benzene, toluene, ethyl benzene, xylenes (BTEX) compounds; and
- Organochlorine and organophosphorous (OC/OP).pesticides

Dependant on the source history of the VENM source, laboratory analysis of the material will also include a selection of the following analyte list:

- Polycyclic aromatic hydrocarbons (PAH);
- Polychlorinated biphenyls (PCB);
- Asbestos; and
- Any other analytes that may be specifically required depending on the source of the materials. A source specific assessment as to additional analyte requirements will be made by the EC.

13.3.2 Site Verification

After the VENM source is approved for use as fill, the materials will be visually verified upon delivery at the site and inspected to confirm there are no suspicious staining and/or odours. The visual inspection will confirm the materials do not contain any of the following:

- Marine mud, peat, vegetation, timber, organic, soluble or perishable materials;
- Dangerous or toxic material or material susceptible to combustion;
- Metal, rubber, plastic or synthetic material or other forms of general rubbish; and/or
- Construction debris.

The imported VENM materials will be sampled at the following frequency:

- 1 sample per 250 m³ of material.

Where a specific source of material is less than 1,000 m³ in volume, 5 samples shall be collected and analysed.

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13.3.3 VENM Validation Criteria

Section 4.2.1 of the DEC NSW Contaminated Sites Guidelines for the NSW Site Auditor Scheme (2nd Edition) (DEC NSW 2006) indicates HILs and PILs are not appropriate and should not be used for the assessment of imported fill and identifies Sections 4.1.1 and 4.2.2 of the Sampling Design Guidelines (NSW EPA, 1995) as providing suitable guidance.

Table 13.2 provides criteria for inorganic compounds which are taken from the Waste Guidelines (NSW DECC, 2004¹⁸) and background ranges taken from ANZECC/NHMRC¹⁹ Guidelines for the Australian and New Zealand Assessment and Management of Contaminated Sites, 1992 (ANZECC/NHMRC, 1992).

The adopted guideline levels for assessing imported fill materials will be as follows:

Table 13-2 Criteria for VENM for Filling

Analyte	Inert Waste Criteria (NSW DEC, 2004) mg/kg	ANZECC 1992 Background Concentrations mg/kg
Arsenic	10 ^a	0.2-30
Cadmium	2 ^a	0.04-2
Lead	10 ^a	<2-200
Nickel	4 ^a	2-400
Mercury	0.4 ^a	0.001-0.1
Chromium	10 ^a (Cr VI)	0.5-110
Copper	No Guideline	1-190
Zinc	No Guideline	2-180

a = CT1 values taken from Table A3 of DEC Waste Guidelines (2004) – Contaminant threshold values for waste classification of non-liquid wastes without doing a leaching test.

All other compounds analysed for source characterisation and site verification should be below the respective laboratory detection limits.

Any deviations from this protocol (such as trace level concentrations of TPHs due to, for instance, naturally occurring hydrocarbon substances) would merit justification and possible specific discussion with the Site Auditor.

13.3.4 Tracking of Imported VENM

A materials tracking system (MTS) will be implemented at the site for the duration of the remediation program. The RC will be responsible for the operation of the MTS, but the outputs will be collated and presented in the VR. The system will track materials coming onto the site during the filling process as well as tracking of materials, such as wastes generated through hotspot removal, being exported from the site.

¹⁸ These guidelines have been superseded by the DECC, NSW (2008).

¹⁹ Australian and New Zealand Environment and Conservation Council and the National Health and Medical Research Council

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The system will enable maintenance of records for all material movements onto and off the site as well as material movements within the site.

In addition, the RC will complete the following components, reporting the outcomes to the EC for inclusion in the VR:

- Monitor and document the movement of vehicles carrying VENM onto the site. The RC will verify the deliveries on the basis of the dispatch weigh bridge dockets from the provider;
- Visually verify the suitability of pre-sampled materials as described above;
- Oversee placement of the materials on the site (EC to perform spot checks and liaise with the RC as required); and
- Document the process through maintaining daily logs, photographic records and other relevant documentation. The various records and documents will form the basis of the validation report.

13.4 Analytical Methods

The Proponent will engage a principal project contract laboratory for the validation program²⁰. The laboratory will be accredited by the National Association of Testing Authorities (NATA) for all validation analyses.

A secondary NATA accredited laboratory will be engaged to perform all inter-laboratory analyses, such as field triplicate samples. The laboratory methods to be adopted for validation analyses are summarised in the following table. Any proposed deviations from these methods would need to be approved by the Auditor.

Table 13-3 Analytical Methods

Analyte	Method Code	LOR (mg/kg)
<i>Metals and Metalloids</i>		
As, Cd, Cr, Cu, Ni, Pb, Zn	USEPA 200.7, ICP-AES	1
Hg	USEPA 7471A, FIMS	0.1
<i>Hydrocarbons</i>		
TPH (C ₆ -C ₉)	USEPA 8015B purge & trap, GC/FID or GC/MS.	5
TPH (C ₁₀ - C ₃₆)	USEPA 8015B, GC/FID	10, 50, 50
BTEX	USEPA 8270, purge & trap, GC/ MS.	0.2, 1, 1, 3
<i>Poly-Aromatic Hydrocarbons</i>	USEPA 8270 GC/MS-SIM (Scan)	0.5 – 1
<i>Volatile Organic Compounds</i>	USEPA 8270, purge & trap, GC/MS.	0.1-0.2

²⁰ An alternate contractual arrangement may be preferable (i.e. the laboratory may be contracted by the EC), but the process would need to be agreed with the Auditor

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Site Validation

Analyte	Method Code	LOR (mg/kg)
<ul style="list-style-type: none"> Chlorinated Aliphatic Compounds 	USEPA 8270 GC/MS-SIM (Scan)	0.5-2
Semi-Volatile Organic Compounds		
<ul style="list-style-type: none"> Chlorinated Aromatic Compounds Semi-Vol Chlorinated Hydrocarbons 		

13.5 Validation of Filling Thickness

Following vegetation clearance and preparation, the Block 2 site surface level will be surveyed and documented, prior to the commencement of filling works.

The 'pre-barrier placement' surface levels will be surveyed and photographed such that the barrier thickness across the site can be confirmed through comparison with surface levels recorded after completion of Physical Separation Layer construction. The EC will undertake these works.

The finished surface levels should be surveyed by a registered surveyor once the fill material has been placed and compacted. The pre-filling and post-filling survey data sets will form part of the site validation.

The results of compaction testing (see Section 6.1) will also be presented as part of the validation data set.

13.6 Validation of Vapour Management Measures

A number of issues were identified in the HHRA (URS, 2008a, refer to Appendix A) that required specific management measures to be implemented as part of the development. These measures were aimed at reducing exposure by future site users to vapour concentrations chiefly associated with the interaction of contaminated shallow groundwater with site features including Springvale Drain and the compensatory flood storage area.

It is required that following implementation of these specific measures, but prior to development and occupation of the Site that the effectiveness of these systems and measures is verified through collection of suitable validation data. The process shall provide sufficient data to support the effective operation of the proposed systems.

This section provides identifies the validation requirements. The validation/verification process shall be developed in the RVP.

13.6.1 Springvale Drain Groundwater Extraction System

The system proposed to address discharge of shallow groundwater and associated emissions to air of volatile chlorinated compounds was outlined in Section 7.2.

Verification of the operation of the system shall involve the collection of surface water samples from the drain within Southlands as well as ambient air samples adjacent to (close to) the drain within Southlands.

Samples should be collected on at least three occasions (number and frequency to be agreed in consultation with the Site Auditor) with at least one occasion sampled after a significant rainfall event (to ensure the proposed system adequately addresses the rise of the groundwater table following such an event).

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Site Validation

13.6.2 Compensatory Flood Storage Area on Block 1

The system designed to prevent groundwater seepage to the surface of the flood storage area will be designed to ensure emissions to air of volatile chlorinated compounds are minimised. A system concept was provided in Section 7.3.

Verification of the operation of the system should involve:

- Inspection of the flood storage area to determine if any groundwater seeps are present (with sampling and analysis of any seeps identified); and
- Collection of flux emission rates from at least four locations on the surface of the flood storage area (including over any seeps if present).

The frequency of sampling is to be agreed through consultation with the Site Auditor. It will, however, comprise a minimum of three sampling events.

13.6.3 Buildings

Vapour mitigation measures were identified to prevent/mitigate vapour intrusion issues.

Verification requires the sampling of sub slab and indoor air from the buildings constructed with such mitigation measures to demonstrate vapour intrusion is not occurring and the mitigation measures are adequate.

The sampling should be undertaken following construction of the buildings with mitigation measures, but before furnishing to minimise any cross contamination of the data with chlorinated emissions from new carpets/furnishing etc. All post construction vapour monitoring results will be required to be provided to the Site Auditor to allow confirmation that the vapour mitigation measures are adequately achieving their exposure goals.

13.7 Validation Reporting

Following the remediation and validation stages of the work a Validation Report will be prepared by the EC that includes the results of all validation components for presentation to the Proponent and the Auditor.

The Validation Report will include the following:

- Executive summary;
- Site identification;
- A summary site history and discussion of the site's environmental context;
- A description of the scope of works undertaken;
- A summary of the site's geological and hydrological context;
- Sampling and analysis plan and methodology;
- Field and laboratory QA/QC and data evaluation;
- Assessment basis;
- Assessment results;
- Site characterisation (validated data supporting the residual soil assessment following hotspot removal and imported materials);
- Tabulated pre- and post- filling level survey and compaction results;
- Presentation and discussion of verification assessments outlined above (Section 13.6);
- Requirements for long term environmental management; and

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Site Validation

- Conclusions.

The report will be prepared in accordance with *Guidelines for Consultants Reporting on Contaminated Sites* (NSW EPA, 1997). As such the final report will be in a format suitable for review by the Site Auditor and to support the Site Audit process.

Section 14

Environmental Management

14.1 Background

The NSW DECC Contaminated Sites Guidelines for the NSW Site Auditor Scheme (2nd Edition) (NSW DEC, 2006) indicate that “within the context of contaminated sites management, a long term environmental management plan (EMP), means a plan which addresses the integration of environmental mitigation and monitoring measures for soil and groundwater throughout an existing or proposed land use. A long term EMP succinctly describes the nature and location of contamination remaining on-site and states what the objectives of the plan are, how contaminants will be managed, who will be responsible for the plan’s implementation and over what time frame actions specified in the plan will take place.

A long term EMP can be an effective means of ensuring the environment is protected, users of the site are not exposed to contamination remaining on-site and the site remains suitable for the specified use when:

- complete clean-up of contamination affecting an area is not practicable;
- contaminants are being capped or contained on-site
- remediation is likely to cause a greater adverse impact than would occur if the site were left undisturbed”.

14.2 Environmental Management Plan (EMP)

The objective of the RAP is to render the site suitable for future industrial /commercial land uses through:

- Creation of a physical barrier to prevent site users accessing, and potentially being exposed to, ACM impacted and other contaminated fill and soils;
- Implementation of vapour barriers (as required) beneath and in the vicinity of site buildings/structures located in Stage 2 Area; and
- Implementation of a targeted shallow groundwater extraction system to mitigate vapour concentrations from surface waters in Springvale Drain.

A long term Environmental Management Plan (EMP) will be required to ensure activities which could potentially result in exposure of future land users to the contained ACM impacted soils, contaminated groundwater beneath the site, and/or vapours from Springvale Dain are precluded or limited / controlled.

In addition, the DCP requires that where capping is carried out on a site and further maintenance is required, Council will require the placement of a covenant on the title to the land. This may also be a condition of the Site Audit Statement.

Given the staged nature of the development and the requirement for audit statements to be prepared for each stage, a long term EMP will initially be prepared to cater for the requirements related to Stage 1 land use scenario, including the use of the Stage 2 area for Stage 1 compensatory flood water detention.

Following completion of Stage 2, the plan will either be updated to cater for Stage 2 specific requirements, or a separate plan shall be prepared.

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Environmental Management

14.3 Site Auditor Requirements

The NSW DECC Contaminated Sites Guidelines for the NSW Site Auditor Scheme (2nd Edition) (NSW DEC, 2006) identify requirements for long term EMPs which include: implementation of an EMP must not be included by a site auditor as a condition on a Site Audit Statement; nor accepted by the auditor as a means of managing contamination of a site, unless the following conditions have been met:

- The EMP has been reviewed by the auditor;
- The EMP can reasonably be made to be legally enforceable, for example because compliance with it is a requirement of a notice under the CLM Act or of development consent conditions issued by the relevant planning authority. The relevant authority (DEC or the local council in these cases, respectively) should be asked their view on the legality of the draft EMP;
- There will be appropriate public notification of any restrictions applying to the land to ensure that potential purchasers or other interested individuals are aware of the restrictions, for example appropriate notations on a planning certificate issued under section 149(2) of the Environmental Planning and Assessment Act 1979 or a covenant registered on the title to the land under section 88B of the Conveyancing Act 1919;
- There is no off-site migration of contamination from the site which is the subject of the site audit or, where there is off-site migration or its potential, that contamination within the site is managed or monitored so that it does not present an unacceptable risk to either the on-site or off-site environments.

14.4 Ongoing Site Management

The site owner shall be responsible for implementation of the long term EMP following completion of development. The site owner may delegate responsibility to future site occupiers by appending the long term EMP to future lease agreements. This may require the development of more area (or building) specific EMPs to be incorporated into lease agreements related to specific land parcels within the site.

14.5 Future Development

The DCP requirement for a covenant on the land title will preclude site development applications for land use scenarios which involve any excavation and / or barrier removal, or will trigger the requirement for further environmental assessment should such activities be proposed.

Such activities would include but not be limited to:

- Any site redevelopment or works that require excavation including for example the construction of any buildings, in particular those with proposed basement space; and/or
- Any proposed site development that requires excavation for the purpose of building foundation installation.

Section 15

Community Consultation

15.1 Background

A series of public workshops were used as the primary tool to share information and seek feedback during the development of the project. Local residents, industrial neighbours and wider stakeholders expressed an interest in the project, particularly with respect to the remediation approach.

Outlined below are the various methods of communication proposed between the proponents, the contractors undertaking the remediation works and members of the community (including neighbouring businesses, residents, and other interested stakeholders) during the remediation works outlined in this RAP.

In preparing the proposed consultation process, the following factors have been considered:

- the consultation work undertaken for the project to date and the feedback received;
- the location of the Southlands site and the surrounding land uses; and
- the requirement to keep the general public informed of the progress of the remediation works.

15.2 Consultation Objectives, Scope and Duration

Open communication, allowing all stakeholders access to information, is necessary during the course of the remediation works and it is essential that all stakeholders have a means of providing feedback and identifying concerns.

The objective of the consultation process is to disseminate clear and factual information that meets the needs of the various interested parties and to reduce the potential for uninformed objections to the works.

The following objectives have been identified:

- To advise the community of the project objectives and benefits of the remediation works proposed at the Southlands site;
- To provide up-to-date information on the staging of remediation and development works and the progress of the project on a regular basis;
- To provide opportunities for all stakeholders to comment and identify concerns during the course of the works;
- To pass on information relating to stakeholder issues and concerns to the remediation project team; and
- To respond to comments and requests for information in a timely manner.

The scope of information released in the consultation process will include the following:

- Any changes in key components of the site works proposed during the course of the remediation and site development works;
- Any changes in the timetable and timeframes proposed over the course of the remediation and site development works;
- Expected effects of the proposed works on the surrounding community;
- Measures to be taken to manage and mitigate these effects;
- Potential risks to the community; and
- Realistic project outcomes.

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Community Consultation

15.3 Consultation Methods and Tools

Over the course of the soil remediation and development works, ongoing consultation will be undertaken to inform stakeholders of the progress of the project and to provide an avenue to receive and respond to feedback and concerns. The proponent will develop a Communications Plan, following receipt of project approval, to ensure effective and efficient consultation with stakeholders for the duration of the soil remediation and site development works.

It is envisaged that the proponents would invite all those on the “Southlands Interest” contact list (refer to Section 15.3.1 below) to attend a planning session to identify preferred communications tools and methods prior to the commencement of remediation and site development works. This session would aid the development of the project specific Communications Plan. This Plan will include the following key components as agreed with project stakeholders.

15.3.1 “ Southlands Interest” Consultation Database

The Proponent has developed a “Southlands Interest” group database during preparation of the EA to identify all stakeholders in the proposed remediation works. The key parties identified to date are:

- the Department of Planning (as the project determining authority);
- NSW DECC (as regulator responsible for licensing of the proposed remediation);
- CoBB Council,
- Other government agencies,
- local residents, industrial neighbours, local businesses and other interested community groups.

Over the course of the project, the database will be updated regularly and will be used to document communications with stakeholders.

15.3.2 Contact Prior to Works Commencing

Appropriate communications tools will be used to distribute information to all identified stakeholders prior to establishment of soil remediation and site development works at Southlands.

The tools will contain information relating to the proposed remediation and site development works, a project program showing the anticipated duration of works, the opportunities available for stakeholder participation, invitation to a communications planning session and means for providing feedback or seeking information.

15.3.3 Project Specific Community Meetings

A series of public workshops were held during preparation of the EA and provided a means for sharing project information and seeking stakeholder feedback. Similar meetings could be held during the course of the remediation works for the various stages of the project.

These meetings would focus on those community members that are most likely to be impacted by the remediation and site development works and their specific information requests. Involvement by State government authorities with a statutory role on the project or an interest in the works and interested environmental groups would be facilitated.

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Community Consultation

15.3.4 Dedicated Telephone Number

A 1800 telephone number/service will be used to receive and respond to queries on the project and comments and complaints. It is likely that this will be the existing Orica Community Hotline number: 1800 025 138.

15.3.5 Information Board

A notice board will be established at the main entrance to the site. The notice board will identify the works underway, present contact information and advise of the available methods of communication with the proponents and remediation team.

15.3.6 Website

Information about the project, including progress updates and relevant contact details will be posted on Orica's Botany Transformation website (<http://oricabotanytransformation.com/>) and updated as required. The website will be advertised to key stakeholders. E-mail inquiries will be available through this web site.

15.3.7 Newspaper Columns

Information about the project and consultation opportunities will also be shared through Orica's regular columns in local newspapers.

15.3.8 Site Tours

Open days or site tours could be arranged to enable interested stakeholders to view the site if a level of interest exists. Visitors to the site would be supervised and appropriate precautions taken to ensure safety.

Section 16

Secondary Information Summary

The RAP identifies several requirements that include preparation of specific plans or other documents as part of its implementation. This section presents a preliminary summary of the various documents and plans identified in the RAP and presents the roles and responsibilities for their preparation and review. It is intended that this list shall be updated and augmented as the site development proceeds.

Section 16

Secondary Information Summary

Table 16-1 Preliminary Summary of Secondary Documents

	Plan / Document / Design	Prepared by	Reviewed by	Content / Comment	
Pre Remediation Works					
1	<ul style="list-style-type: none"> • Review of Environmental Factors: Springvale Drain shallow groundwater extraction system Detail Design; and • Review of Environmental Factors: Springvale Drain Realignment Channel. • Review of Environmental Factors: Temporary Aquifer Storage and Recovery (TASR) 	Proponent / PE / EC	SA	<ul style="list-style-type: none"> • Review of environmental factors for the in filling of the Springvale Drain re-alignment channel and construction of a shallow groundwater extraction system. Both of these measures will assist in the effective mitigation of possible vapours emanating from Springvale Drain. • Approval for these works will be sort under Part 5 of the Environmental Planning and Assessment Act and will be subject to separate Environmental Assessments and Review. They are not subject to the Southlands remediation and development Project Application, but the completion of these works is relevant to this Application. • The TASR REF proposes the use of the use of re-injection of contaminated groundwater up-gradient of the site in the event of long term GTP failure. This is not part of the proposed Southlands site redevelopment as such, but will have relevance to the SA in assessing GTP reliability. • DECC will be the consent authority. 	Prior to Auditor Section B Site Audit Statement and prior to implementation.

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Secondary Information Summary

	Plan / Document / Design	Prepared by	Reviewed by	Content / Comment	
2	Compensatory Flood Storage Area Groundwater Reticulation System Detail Design	Proponent / PE / EC	SA	<ul style="list-style-type: none"> Detailed description of modelling, design, and field trial programs. Design elements and verification sampling program to demonstrate the Flood Storage Area meets vapour emission control requirements. 	Prior to Tender
3	<ul style="list-style-type: none"> Vapour Intrusion Conceptual Model and Preliminary Remedial Options Study Detailed Vapour Intrusion Mitigation Measures for Site Structures 	Proponent / PE / EC	SA	<ul style="list-style-type: none"> Creation of vapour intrusion conceptual model Identification of possible vapour migration pathways Selection of preferred options 	Prior to Construction

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Secondary Information Summary

	Plan / Document / Design	Prepared by	Reviewed by	Content / Comment	
4	Interim Verification Data Plan / Verification Data Report (following installation and testing of 1 and 2). ²¹	EC	SA	<ul style="list-style-type: none"> Summary of validation results from sampling verification plans detailed in 1, 2 and 3. Baseline sampling works will build on existing data and will commence prior to installation of 1, 2 and 3. 	<p>Plan for completion prior to commencement of works in 1, 2 and 3.</p> <p>Verification Report –detailing results from verification sampling in 1, 2 and 3. Likely to be staged, with final report prior to occupancy.</p>
5	<ul style="list-style-type: none"> Detailed Barrier Design Perimeter Area Detailed Design²² 	Project Design Team	Proponent / SA	<ul style="list-style-type: none"> Will contain details of the perimeter finishes to ensure integrity of the filled surface. 	Prior to Tender
6	Groundwater Monitoring Well Replacement Program Plan	Proponent / EC	Proponent / SA / NSW DECC	<ul style="list-style-type: none"> Contains details on the staging and replacement of monitoring wells during the construction phase. Preliminary recommended well replacement locations have been included in the RAP. 	Prior to Tender

²¹ This may include testing carried out during installation and developmental stages.

²² This detail design will likely form part of the overall site detail design.

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Secondary Information Summary

	Plan / Document / Design	Prepared by	Reviewed by	Content / Comment	
7	Further Assessment of the Extent of Impacted Soils Investigation Report	EC	SA	<ul style="list-style-type: none"> To better characterise the extent of hotspots and minimise to extent practical the volume of fill requiring excavation. 	Prior to Tender or Part of tender
8	Waste Classification Report and Management Plan	EC	SA	<ul style="list-style-type: none"> To characterise fill requiring disposal offsite to licensed facilities or possible transport, storage and processing within the BIP (eg. HCB waste items). 	Prior to Tender
9	Construction Environmental Management Plan (CEMP)	RC	EC / SA	<ul style="list-style-type: none"> Will detail environmental controls and procedures that will be implemented during the construction phase of the project. Will include runoff / sediment management and dust control. 	Prior to commencement of works
10	Site Specific Health, Safety Plan (SSHSP)	RC	EC / Proponent	<ul style="list-style-type: none"> Will include details of measures required for worker health and safety, with particular emphasis on management of potential risk presented by existing site contaminants. 	Prior to commencement of works
11	Remediation Communications Plan	Proponent	SA	<ul style="list-style-type: none"> A plan to ensure effective and efficient consultation with stakeholders for the duration of the soil remediation and site development works as provided in the Southlands RAP. All communications will be managed by the Proponent's communications teams using established communication tools and forums. 	Prior to commencement of works
12	Remediation Validation Plan (RVP)	EC	SA	<ul style="list-style-type: none"> A plan for SA approval, that will determine how the validation data will be collated / presented and reviewed (including quality assurance measures) in the Validation Report. 	Prior to commencement of works

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Secondary Information Summary

	Plan / Document / Design	Prepared by	Reviewed by	Content / Comment	
Post Remediation Works					
13	Validation Report (VR) (to include verification data from vapour risk mitigation measures)	EC	SA	<ul style="list-style-type: none"> A presentation of the relevant data demonstrating the effectiveness of remedial works in rendering the site suitable for the proposed landuse. 	Prior to Auditor Section A Site Audit Statement and site occupancy
14	Long term Environmental Management Plan (EMP) – including full implementation of long term mitigation measures	EC	Proponent / SA	<ul style="list-style-type: none"> A report detailing the ongoing obligations and requirements of the Proponent with respect to the maintenance and ongoing verification of the implemented remedial measures. 	Prior to Auditor Section A Site Audit Statement and site occupancy

Notes:

SA Site Auditor
 EC Environmental Consultant
 Proponent Orica Australia Pty Ltd and Goodman Pty Ltd
 RC Remediation Contractor
 PE Project Engineers

Section 17**References**

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- CALM 1992** Department of Conservation and Land Management: Urban Erosion Control and Sediment Control
- NSW EPA, 1994** *Contaminated Sites: Guidelines for Assessing Service Station Sites*
- NSW EPA, 1995** *Contaminated Sites: Sampling Design Guidelines*
- WWC, 1996a** *ICI Botany, Groundwater Stage 2 Survey. Contract S2/C1, Springvale Drain Woodward-Clyde* August 1996.
- WWC, 1996b** *ICI Botany, Groundwater Stage 2 Survey. Contract S2/C3, Water/Soil Phase 2 Woodward-Clyde* August 1996.
- AS 3798 – 1996** *Australian Standard: Guidelines on earthworks for commercial and residential developments*
- NEPM 1998** National Environment Protection Council (1998): *National Environment Protection Measure (NEPM) on Ambient Air Quality*
- ANZECC, 1999** *Guidelines for the Assessment of On-site Containment of Contaminated Soil*
- NEPC, 1999** National Environmental Protection Council (NEPC) *National Environmental Protection (Assessment of Site Contamination) Measure, 1999 Health Based Investigation Level.*
- ANZECC/ARMCANZ 2000** *Australian and New Zealand Guidelines for Fresh and Marine Water Quality.* Australian and New Zealand Environment and Conservation Council and Agriculture and Resource Management Council of Australia and New Zealand.
- NOHSC 2002** National Occupational Health and Safety Commission (2002) Code of Practice for Safe Removal of Asbestos
- LWBC, 2003** Land and Water Biodiversity Committee, *Minimum Construction Requirements for Water Bores in Australia.* Edition 2, Revised September 2003
- CoBB, 2003** City of Botany Bay Council *Development Control Plan No.34*
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- Lawson and Treloar Pty Ltd, 2003** *Proposed Expansion of Container Port Facilities in Botany Bay, NSW – Coastal Process and Water Resource Issues, Volume 1: Hydrology and Hydraulic Studies*
- URS 2004a** *Orica Botany Environmental Survey Stage 4 – Remediation, Full Scale Reactive Iron Barrier.* URS Australia Pty Ltd
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- US EPA, 2004** *Preliminary Remedial Goals – Region IX.*

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References

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- HLA, 2005** *Factual Report on Limited Environmental Investigation, Orica Botany – Southland, NSW* August 2005. HLA Envirosiences Pty Ltd.
- Orica, 2005** Proposed 2005 Groundwater Cleanup Plan Groundwater and Surface Water Monitoring Program (EN1591-61-321)
- URS, 2005a** *Orica Botany Environmental Survey Stage 4 – Remediation, 2005 DNAPL Source Area Delineation Investigation. April 2005.*
- URS, 2005b** *Orica Botany Environmental Survey Stage 4 – Remediation, Progress Report – Southern Plumes DNAPL Source Area Delineation Investigation, August 2005. December 2005.*
- URS, 2005c** Orica Botany Environmental Projects. Consolidated Human Health Risk Assessment 2005. URS Australia Pty Ltd
- URS, 2006** *Southern Plumes Source Area Delineation Investigation. February 2006.*
- CW, 2007** *ORICA / Goodman Southlands Remediation / Development Project Flood Investigations DRAFT.* Connell Wagner Pty Ltd
- DEC, NSW, 2007** *Approved Methods for the Sampling and Analysis of Air Pollutants in NSW*
- Habitation, 2007** *Drawing File Name 07011 Stg 1 LC04.dwg & LC06.dwg dated 22 08 2007*
- URS, 2007a** *Geotechnical Assessment Southlands – Remediation and Development 8 May 2007*
- URS, 2007b.** *Preliminary Human Health Risk Assessment for Proposed Southlands Development, PHHRA, August 2007.*
- URS, 2007c** Conceptual Site Model – Botany, November 2007
- DEC, NSW, 2008** *Waste Classification Guidelines. Part 1: Classifying Wastes.*
- JBS, 2008** *Southlands Development – Groundwater Level Assessment Proposed Stage 2 Flood Storage Area* JBS Environmental Pty Ltd, 19 February 2008
- URS, 2008a** *Human Health Risk Assessment, Southlands Remediation and Development Project. March 2008, URS Australia Pty Ltd*
- URS 2008b** *Surface Water and Ambient Air Monitoring Program for Springvale Drain May 2007 to June 2008. Draft Report September 2008.*

Section 18

Limitations

URS Australia Pty Ltd (URS) has prepared this Report in accordance with the usual care and thoroughness of the consulting profession for the use of Orica Australia Pty Ltd and Goodman Pty Ltd (The Proponent) and only those third parties who have been authorised in writing by URS to rely on the report. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report. It is prepared in accordance with the scope of work and for the purpose outlined in the Proposal dated 17 May 2006 and subsequent revised URS Proposal dated 7 June 2006.

All information in this Report is provided strictly in accordance with and subject to the following limitations and recommendations:

- a) This Report should be read in full and no excerpts are to be taken as representative of the findings. No responsibility is accepted by URS for use of any part of this Report in any other context.
- b) This Report is based solely on the scope of work agreed between URS and Orica Australia Pty Ltd (Orica) and Goodman and described in **Section 1.8** ("Objectives and Scope of Works") of this Report.
- c) This Report has been prepared for the sole benefit of Orica Australia Pty Ltd (Orica) and Goodman Pty Limited (The Proponent). Neither the whole nor any part of this Report may be used or relied upon by any party other than Orica Australia Pty Ltd (Orica) and Goodman Pty Limited. It is acknowledged that this Report may be used by the following parties for the purpose of assessing and approving activities described in this Report: where this Report is to be submitted to an accredited site auditor under the Contaminated Land Management Act 1997, that auditor; and where this Report is to be submitted to a public or regulatory authority pursuant to a requirement under applicable planning or environmental controls, that authority.
- d) This Report is dated October 2008 and is based on the conditions encountered during limited site investigations conducted, and information reviewed, from August 2006 to October 2008. URS accepts no responsibility for any events arising from any changes in site conditions or in the information reviewed that have occurred after the completion of the site investigations.
- e) Where this Report indicates that information has been provided to URS by third parties, URS has made no independent verification of this information except as expressly stated in the Report.
- f) Except as specifically stated above, URS makes no warranty, statement or representation of any kind concerning the suitability of the site for any purpose or the permissibility of any use, development or re-development of the site.
- g) URS makes no determination or recommendation regarding a decision to provide or not to provide financing with respect to the site.
- h) Investigations undertaken in respect of this Report are constrained by the particular site conditions, such as the location of services and vegetation. As a result, not all relevant site features and contamination may have been identified in this Report.
- i) Except as otherwise specifically stated in this Report, URS makes no warranty or representation as to the presence or otherwise of asbestos and/or asbestos containing materials ("ACM") on the site. If fill has been imported on to the site at any time, or if any buildings constructed prior to 1970 have been demolished on the site or materials from such buildings disposed of on the site, the site may contain asbestos or ACM. Even if asbestos was tested for and those test results did not reveal the presence of asbestos at specific points of sampling, asbestos may still be present at the site if fill has been imported at any time, or if any buildings constructed prior to 1970 have been demolished on the site or materials from such buildings disposed of on the site.
- j) Subsurface conditions can vary across a particular site and cannot be exhaustively defined by the investigations described in this Report. It is unlikely therefore that the results and estimations expressed in this Report will represent conditions at any location removed from the specific points of sampling.

Tables

**Table 1 - Southlands Soil Delineation
Orica & Macquarie Goodman Pty. Ltd.**

Analytical Results

Sample ID	TP15_0.3-0.4	TP15_1.6-1.7	TP45_1.5-1.6
Date Sampled	8/04/2005	8/04/2005	11/04/2005

Analyte	LOR	Units	Inert	Solid	Industrial			
Metals (Total)								
Arsenic	5	mg/kg	10	100	400	<5	<5	<5
Cadmium	1	mg/kg	2	20	80	<1	<1	<1
Chromium	2	mg/kg	10	100	400	14	8	<2
Copper	5	mg/kg				10	40	<5
Lead	5	mg/kg	10	100	400	47	61	10
Mercury	0.1	mg/kg	0.4	4	16	0.2	0.4	<0.1
Nickel	2	mg/kg	4	40	160	<2	5	<2
Zinc	5	mg/kg				52	54	<5
Total Petroleum Hydrocarbons								
C6-C9 fraction	2	mg/kg	650	650	2600	-	-	-
C10-C14 fraction	50	mg/kg				-	-	-
C15-C28 fraction	100	mg/kg				-	-	-
C29-C36 fraction	100	mg/kg				-	-	-
Total C10-C36 Fraction			5000	10000	40000			
BTEX Compounds								
Benzene	0.5	mg/kg	1	10	40	-	<0.5	<0.5
Toluene	0.5	mg/kg	28.8	288	1152	-	<0.5	<0.5
Ethylbenzene	0.5	mg/kg	60	600	2400	-	<0.5	<0.5
m&p-Xylene	0.5	mg/kg				-	<0.5	<0.5
o-Xylene	0.5	mg/kg				-	<0.5	<0.5
Total Xylene	Calculated	mg/kg	100	1000	4000			
Chlorinated Aliphatic Compounds								
1,1,1,2-Tetrachloroethane	0.5	mg/kg	20	200	800	-	<0.5	<0.5
1,1,1-Trichloroethane	0.5	mg/kg	60	600	2400	-	<0.5	<0.5
1,1,2,2-Tetrachloroethane	0.5	mg/kg	2.6	26	104	-	<0.5	<0.5
1,1,2-Trichloroethane	0.5	mg/kg	2.4	24	96	-	<0.5	0.8
1,1-Dichloroethane	0.5	mg/kg				-	<0.5	<0.5
1,1-Dichloroethene	0.5	mg/kg	1.4	14	56	-	<0.5	<0.5
1,1-Dichloropropylene	0.5	mg/kg				-	<0.5	<0.5
1,2,3-Trichloropropane	0.5	mg/kg				-	<0.5	<0.5
1,2-Dibromo-3-chloropropane	0.5	mg/kg				-	<0.5	<0.5
1,2-Dichloroethane	0.5	mg/kg	1	10	40	-	<0.5	0.6
1,3-Dichloropropane	0.5	mg/kg				-	<0.5	<0.5
Bromobenzene	0.5	mg/kg				-	<0.5	<0.5
Bromomethane	5	mg/kg				-	<5	<5
Carbon Tetrachloride	0.5	mg/kg	1	10	40	-	<0.5	<0.5
Chloroethane	5	mg/kg				-	<5	<5
Chloromethane	5	mg/kg				-	<5	<5
cis-1,2-Dichloroethene	0.5	mg/kg				-	0.7	1
cis-1,4-Dichloro-2-butene	0.5	mg/kg				-	<0.5	<0.5
Dibromomethane	0.5	mg/kg				-	<0.5	<0.5
Dichlorodifluoromethane	5	mg/kg				-	<5	<5
Hexachlorobutadiene	0.5	mg/kg				-	<0.5	1
Iodomethane	0.5	mg/kg				-	<0.5	<0.5
Pentachloroethane	0.5	mg/kg				-	<0.5	<0.5
Tetrachloroethene	0.5	mg/kg	1.4	14	56	-	<0.5	1.6
trans-1,2-Dichloroethene	0.5	mg/kg				-	<0.5	<0.5
trans-1,4-Dichloro-2-butene	0.5	mg/kg				-	<0.5	<0.5
Trichloroethene	0.5	mg/kg	1	10	40	-	<0.5	2.6
Trichlorofluoromethane	5	mg/kg				-	<5	<5
Vinyl chloride	5	mg/kg	0.4	4	16	-	<5	<5
Chlorinated Aromatic Compounds								
1,2,3-Trichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
1,2,4-Trichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
1,2-Dichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
1,3-Dichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
1,4-Dichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
2-Chlorotoluene	0.5	mg/kg				-	<0.5	<0.5
4-Chlorotoluene	0.5	mg/kg				-	<0.5	<0.5
Chlorobenzene	0.5	mg/kg	200	2000	8000	-	<0.5	<0.5
Chlorinated Hydrocarbons								
1,2-Dichlorobenzene	0.5	mg/kg	8.6	86	34.4	-	<0.5	<0.5
1,3-Dichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
1,4-Dichlorobenzene	0.5	mg/kg	15	150	600	-	<0.5	<0.5
Hexachlorobenzene	1	mg/kg				-	<1	<1
Hexachlorobutadiene	0.5	mg/kg				-	<0.5	0.7
Hexachlorocyclopentadiene	2.5	mg/kg				-	<2.5	<2.5
Hexachloroethane	0.5	mg/kg				-	<0.5	<0.5
Hexachloropropylene	0.5	mg/kg				-	<0.5	<0.5
Pentachlorobenzene	0.5	mg/kg				-	<0.5	<0.5
Trihalomethanes								
Bromochloromethane	0.5	mg/kg				-	<0.5	<0.5
Bromodichloromethane	0.5	mg/kg				-	<0.5	<0.5
Bromoform	0.5	mg/kg				-	<0.5	<0.5
Chloroform	0.5	mg/kg	12	120.00	480	-	<0.5	1.9
Dibromochloromethane	0.5	mg/kg				-	<0.5	<0.5

**Table 1 - Southlands Soil Delineation
Orica & Macquarie Goodman Pty. Ltd.**

Analytical Results

Sample ID	TP15_0.3-0.4	TP15_1.6-1.7	TP45_1.5-1.6
Date Sampled	8/04/2005	8/04/2005	11/04/2005

Analyte	LOR	Units	Inert	Solid	Industrial			
Fumigants								
1,2-Dibromomethane	0.5	mg/kg				-	<0.5	<0.5
1,2-Dichloropropane	0.5	mg/kg				-	<0.5	<0.5
2,2-Dichloropropane	0.5	mg/kg				-	<0.5	<0.5
cis-1,3-Dichloropropylene	0.5	mg/kg				-	<0.5	<0.5
trans-1,3-Dichloropropylene	0.5	mg/kg				-	<0.5	<0.5
Polynuclear Aromatic Hydrocarbons								
2-Chloronaphthalene	0.5	mg/kg				-	<0.5	<0.5
2-Methylnaphthalene	0.5	mg/kg				-	<0.5	<0.5
3-Methylcholanthrene	0.5	mg/kg				-	<0.5	<0.5
7,12-Dimethylbenz(a)anthracene	0.5	mg/kg				-	<0.5	<0.5
Acenaphthene	0.5	mg/kg				-	<0.5	<0.5
Acenaphthylene	0.5	mg/kg				-	<0.5	<0.5
Anthracene	0.5	mg/kg				-	<0.5	<0.5
Benzo(a)anthracene	0.5	mg/kg				-	<0.5	<0.5
Benzo(a)pyrene	0.5	mg/kg	0.08	1	3.2	-	<0.5	<0.5
Benzo(a)pyrene TCLP	0.002	mg/L				-	-	-
Benzo(b)&(k)fluoranthene	1	mg/kg				-	<1	<1
Benzo(g,h,i)perylene	0.5	mg/kg				-	<0.5	<0.5
Chrysene	0.5	mg/kg				-	<0.5	<0.5
Dibenzo(a,h)anthracene	0.5	mg/kg				-	<0.5	<0.5
Fluoranthene	0.5	mg/kg				-	<0.5	<0.5
Fluorene	0.5	mg/kg				-	<0.5	<0.5
Indeno(1,2,3,cd)pyrene	0.5	mg/kg				-	<0.5	<0.5
N-2-Fluorenyl Acetamide	0.5	mg/kg				-	<0.5	<0.5
Naphthalene	0.5	mg/kg				-	<0.5	<0.5
Phenanthrene	0.5	mg/kg				-	<0.5	<0.5
Pyrene	0.5	mg/kg				-	<0.5	<0.5
PAH (total)	Calculated	mg/kg	200	200	800	-	0	0
Anilines and Benzidines								
2-Nitroaniline	1	mg/kg				-	<1	<1
3,3'-Dichlorobenzidine	0.5	mg/kg				-	<0.5	<0.5
3-Nitroaniline	1	mg/kg				-	<1	<1
4-Chloroaniline	0.5	mg/kg				-	<0.5	<0.5
4-Nitroaniline	0.5	mg/kg				-	<0.5	<0.5
Aniline	0.5	mg/kg				-	<0.5	<0.5
Carbazole	0.5	mg/kg				-	<0.5	<0.5
Dibenzofuran	0.5	mg/kg				-	<0.5	<0.5
Haloethers								
4-Bromophenyl phenyl ether	0.5	mg/kg				-	<0.5	<0.5
4-Chlorophenyl phenyl ether	0.5	mg/kg				-	<0.5	<0.5
Bis(2-chloroethoxy) methane	0.5	mg/kg				-	<0.5	<0.5
Bis(2-chloroethyl) ether	0.5	mg/kg				-	<0.5	<0.5
Monocyclic Aromatic Hydrocarbons								
1,2,4-Trimethylbenzene	0.5	mg/kg				-	<0.5	<0.5
1,3,5-Trimethylbenzene	0.5	mg/kg				-	<0.5	<0.5
Ethylbenzene	0.5	mg/kg	60	600	2400	-	<0.5	<0.5
Isopropylbenzene	0.5	mg/kg				-	<0.5	<0.5
m&p-Xylene	0.5	mg/kg				-	<0.5	<0.5
n-Butylbenzene	0.5	mg/kg				-	<0.5	<0.5
n-Propylbenzene	0.5	mg/kg				-	<0.5	<0.5
o-Xylene	0.5	mg/kg				-	<0.5	<0.5
p-Isopropyltoluene	0.5	mg/kg				-	<0.5	<0.5
sec-Butylbenzene	0.5	mg/kg				-	<0.5	<0.5
Styrene	0.5	mg/kg	6	60	240	-	<0.5	<0.5
tert-Butylbenzene	0.5	mg/kg				-	<0.5	<0.5
Toluene	0.5	mg/kg	28.8	288	1152	-	<0.5	<0.5
Nitroaromatics and Ketones								
1,3,5-Trinitrobenzene	0.5	mg/kg				-	<0.5	<0.5
1-Naphthylamine	0.5	mg/kg				-	<0.5	<0.5
2,4-Dinitrotoluene	1	mg/kg	0.26	3	10.4	-	<1	<1
2,6-Dinitrotoluene	1	mg/kg				-	<1	<1
2-Picoline	0.5	mg/kg				-	<0.5	<0.5
4-Aminobiphenyl	0.5	mg/kg				-	<0.5	<0.5
4-Nitroquinoline-N-oxide	0.5	mg/kg				-	<0.5	<0.5
5-Nitro-o-toluidine	0.5	mg/kg				-	<0.5	<0.5
Acetophenone	0.5	mg/kg				-	<0.5	<0.5
Azobenzene	1	mg/kg				-	<1	<1
Chlorobenzilate	0.5	mg/kg				-	<0.5	<0.5
Dimethylaminoazobenzene	0.5	mg/kg				-	<0.5	<0.5
Isophorone	0.5	mg/kg				-	<0.5	<0.5
Nitrobenzene	0.5	mg/kg	4	40	160	-	<0.5	<0.5
Pentachloronitrobenzene	0.5	mg/kg				-	<0.5	<0.5
Phenacetin	0.5	mg/kg				-	<0.5	<0.5
Pronamide	0.5	mg/kg				-	<0.5	<0.5

**Table 1 - Southlands Soil Deliniation
Orica & Macquarie Goodman Pty. Ltd.**

Analytical Results

Sample ID	TP15_0.3-0.4	TP15_1.6-1.7	TP45_1.5-1.6
Date Sampled	8/04/2005	8/04/2005	11/04/2005

Analyte	LOR	Units	Inert	Solid	Industrial			
Nitrosamines								
Methapyrilene	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosodibutylamine	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosodiethylamine	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosodi-n-propylamine	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosodiphenyl & Diphenylamine	1	mg/kg				-	<1	<1
N-Nitrosomethylethylamine	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosomorpholine	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosopiperidine	0.5	mg/kg				-	<0.5	<0.5
N-Nitrosopyrrolidine	1	mg/kg				-	<1	<1
Organochlorine Pesticides (OC)								
4,4-DDD	0.5	mg/kg				-	<0.5	<0.5
4,4-DDE	0.5	mg/kg				-	<0.5	<0.5
4,4-DDT	1	mg/kg				-	<1	<1
a-BHC	0.5	mg/kg				-	<0.5	<0.5
Aldrin	0.5	mg/kg				-	<0.5	<0.5
b-BHC	0.5	mg/kg				-	<0.5	<0.5
d-BHC	0.5	mg/kg				-	<0.5	<0.5
Dieldrin	0.5	mg/kg				-	<0.5	<0.5
Endosulfan 1	0.5	mg/kg				-	<0.5	<0.5
Endosulfan 2	0.5	mg/kg				-	<0.5	<0.5
Endosulfan sulfate	0.5	mg/kg				-	<0.5	<0.5
Endrin	0.5	mg/kg				-	<0.5	<0.5
g-BHC	0.5	mg/kg				-	<0.5	<0.5
Heptachlor	0.5	mg/kg				-	<0.5	<0.5
Heptachlor epoxide	0.5	mg/kg				-	<0.5	<0.5
Organophosphorus Pesticides (OP)								
Chlorfenvinphos	0.5	mg/kg				-	<0.5	<0.5
Chlorpyrifos	0.5	mg/kg				-	<0.5	<0.5
Chlorpyrifos-methyl	0.5	mg/kg				-	<0.5	<0.5
Diazinon	0.5	mg/kg				-	<0.5	<0.5
Dichlorvos	0.5	mg/kg				-	<0.5	<0.5
Dimethoate	0.5	mg/kg				-	<0.5	<0.5
Ethion	0.5	mg/kg				-	<0.5	<0.5
Fenthion	0.5	mg/kg				-	<0.5	<0.5
Malathion	0.5	mg/kg				-	<0.5	<0.5
Pirimphos-ethyl	0.5	mg/kg				-	<0.5	<0.5
Prothiofos	0.5	mg/kg				-	<0.5	<0.5
Oxygenated Compounds								
2-Butanone (MEK)	5	mg/kg				-	<5	<5
2-Hexanone (MBK)	5	mg/kg				-	<5	<5
2-Propanone	6	mg/kg				-	-	-
4-Methyl-2-pentanone	5	mg/kg				-	<5	<5
Vinyl Acetate	5	mg/kg				-	<5	<5
Phenolic Compounds								
2,3,4,6-Tetrachlorophenol	0.5	mg/kg				-	<0.5	<0.5
2,4,5-Trichlorophenol	0.5	mg/kg	800	8000	32000	-	<0.5	<0.5
2,4,6-Trichlorophenol	0.5	mg/kg	4	40	160	-	<0.5	<0.5
2,4-Dichlorophenol	0.5	mg/kg				-	<0.5	<0.5
2,4-Dimethylphenol	0.5	mg/kg				-	<0.5	<0.5
2,6-Dichlorophenol	0.5	mg/kg				-	<0.5	<0.5
2-Chlorophenol	0.5	mg/kg				-	<0.5	<0.5
2-Methylphenol	0.5	mg/kg				-	<0.5	<0.5
2-Nitrophenol	0.5	mg/kg				-	<0.5	<0.5
3- & 4-Methylphenol	0.5	mg/kg				-	<0.5	<0.5
4-Chloro-3-Methylphenol	0.5	mg/kg				-	<0.5	<0.5
Pentachlorophenol	1	mg/kg				-	<1	<1
Phenol	0.5	mg/kg	28.8	288	1152	-	<0.5	<0.5
Phthalate Esters								
bis(2-ethylhexyl) phthalate	5	mg/kg				-	<5	<5
Butyl benzyl phthalate	0.5	mg/kg				-	<0.5	<0.5
Diethyl phthalate	0.5	mg/kg				-	<0.5	<0.5
Dimethyl phthalate	0.5	mg/kg				-	<0.5	<0.5
Di-n-butyl phthalate	0.5	mg/kg				-	<0.5	<0.5
Di-n-octylphthalate	0.5	mg/kg				-	<0.5	<0.5

**Table 1 - Southlands Soil Deliniation
Orica & Macquarie Goodman Pty. Ltd.**

Analytical Results

Sample ID	TP15_0.3-0.4	TP15_1.6-1.7	TP45_1.5-1.6
Date Sampled	8/04/2005	8/04/2005	11/04/2005

Analyte	LOR	Units	Inert	Solid	Industrial			
Miscellaneous Compounds								
1,2,4,5-Tetrachlorobenzene	0.5	mg/kg				-	<0.5	<0.5
1,3,5-Trichlorobenzene	0.5	mg/kg				-	<0.5	<0.5
cis-Isosafrole	0.5	mg/kg				-	<0.5	<0.5
Diallylate	0.5	mg/kg				-	<0.5	<0.5
Methanesulfonate ethyl	0.5	mg/kg				-	<0.5	<0.5
Methanesulfonate methyl	0.5	mg/kg				-	<0.5	<0.5
Safrole	0.5	mg/kg				-	<0.5	<0.5
trans-Isosafrole	0.5	mg/kg				-	<0.5	<0.5
Naphthalene								
Naphthalene	0.5	mg/kg				-	-	-
Natural Attenuation Parameters								
Total Organic Carbon	0.5	%				-	-	-
Sulfonated Compounds								
Carbon disulfide	0.5	mg/kg				-	<0.5	<0.5
Asbestos								
Asbestos						-	-	-
Miscellaneous								
Moisture Content	1	%				-	-	-

Waste Classification without TCLP

Soid Solid Solid

Legend:

Total concentration less than or equal to inert waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS INERT WASTE
Total Concentration less than or equal to solid waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS SOLID WASTE
Total Concentration less than or equal to industrial waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS INDUSTRIAL WASTE
Total Concentration exceeds industrial waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS HAZARDOUS WASTE

PS - Primary Sample
LD - Laboratory Duplicate
ND - Not Detected
D - Detected
LOR - Limit of Reporting

FD - Field Duplicate
FT - Field Triplicate
- - Not Analysed
mg/kg - milligrams per kilogram
Concentrations which cummulatively may classify the material as "Scheduled Waste"

**Table 1 - Southlands Soil Deliniation
Orica & Macquarie Goodman Pty. Ltd.
Analytical Results**

Blck 2 Blck 2 Blck 2

Sample ID
Date Sampled

TP50_0.4-0.5	TP50_0.9-1.0	TP50_1.2-1.3
12/04/2005	12/04/2005	12/04/2005

Analyte	LOR	Units	Inert	Solid	Industrial			
Metals (Total)								
Arsenic	5	mg/kg	10	100	400	10	6	41
Cadmium	1	mg/kg	2	20	80	<1	<1	2
Chromium	2	mg/kg	10	100	400	19	9	20
Copper	5	mg/kg				50	<5	150
Lead	5	mg/kg	10	100	400	177	13	251
Mercury	0.1	mg/kg	0.4	4	16	16.7	0.2	123
Zinc	5	mg/kg				220	<5	917
Total Petroleum Hydrocarbons								
C6-C9 fraction	2	mg/kg	650	650	2600	-	-	-
C10-C14 fraction	50	mg/kg				-	-	-
C15-C28 fraction	100	mg/kg				-	-	-
C29-C36 fraction	100	mg/kg				-	-	-
Total C10-C36 Fraction			5000	10000	40000			
BTEX Compounds								
Benzene	0.5	mg/kg	1	10	40	-	-	<0.5
Toluene	0.5	mg/kg	28.8	288	1152	-	-	<0.5
m&p-Xylene	0.5	mg/kg				-	-	<0.5
o-Xylene	0.5	mg/kg				-	-	<0.5
Total Xylene	Calculated	mg/kg	100	1000	4000			
Chlorinated Aliphatic Compounds								
1,1,1,2-Tetrachloroethane	0.5	mg/kg	20	200	800	-	-	<0.5
1,1,1-Trichloroethane	0.5	mg/kg	60	600	2400	-	-	<0.5
1,1,2,2-Tetrachloroethane	0.5	mg/kg	2.6	26	104	-	-	<0.5
1,1,2-Trichloroethane	0.5	mg/kg	2.4	24	96	-	-	<0.5
1,1-Dichloroethane	0.5	mg/kg				-	-	<0.5
1,1-Dichloroethene	0.5	mg/kg	1.4	14	56	-	-	<0.5
1,1-Dichloropropylene	0.5	mg/kg				-	-	<0.5
1,2,3-Trichloropropane	0.5	mg/kg				-	-	<0.5
1,2-Dibromo-3-chloropropane	0.5	mg/kg				-	-	<0.5
1,2-Dichloroethane	0.5	mg/kg	1	10	40	-	-	<0.5
1,3-Dichloropropane	0.5	mg/kg				-	-	<0.5
Bromobenzene	0.5	mg/kg				-	-	<0.5
Bromomethane	5	mg/kg				-	-	<5
Carbon Tetrachloride	0.5	mg/kg	1	10	40	-	-	<0.5
Chloroethane	5	mg/kg				-	-	<5
Chloromethane	5	mg/kg				-	-	<5
cis-1,2-Dichloroethene	0.5	mg/kg				-	-	<0.5
cis-1,4-Dichloro-2-butene	0.5	mg/kg				-	-	<0.5
Dibromomethane	0.5	mg/kg				-	-	<0.5
Dichlorodifluoromethane	5	mg/kg				-	-	<5
Hexachlorobutadiene	0.5	mg/kg				-	-	<0.5
Iodomethane	0.5	mg/kg				-	-	<0.5
Pentachloroethane	0.5	mg/kg				-	-	<0.5
Tetrachloroethene	0.5	mg/kg	1.4	14	56	-	-	0.5
trans-1,2-Dichloroethene	0.5	mg/kg				-	-	<0.5
trans-1,4-Dichloro-2-butene	0.5	mg/kg				-	-	<0.5
Trichloroethene	0.5	mg/kg	1	10	40	-	-	<0.5
Trichlorofluoromethane	5	mg/kg				-	-	<5
Vinyl chloride	5	mg/kg	0.4	4	16	-	-	<5
Chlorinated Aromatic Compounds								
1,2,3-Trichlorobenzene	0.5	mg/kg				-	-	<0.5
1,2,4-Trichlorobenzene	0.5	mg/kg				-	-	<0.5
1,2-Dichlorobenzene	0.5	mg/kg				-	-	<0.5
1,3-Dichlorobenzene	0.5	mg/kg				-	-	<0.5
1,4-Dichlorobenzene	0.5	mg/kg				-	-	<0.5
2-Chlorotoluene	0.5	mg/kg				-	-	<0.5
4-Chlorotoluene	0.5	mg/kg				-	-	<0.5
Chlorobenzene	0.5	mg/kg	200	2000	8000	-	-	<0.5
Chlorinated Hydrocarbons								
1,2-Dichlorobenzene	0.5	mg/kg	8.6	86	34.4	-	-	<0.5
1,3-Dichlorobenzene	0.5	mg/kg				-	-	<0.5
1,4-Dichlorobenzene	0.5	mg/kg	15	150	600	-	-	<0.5
Hexachlorobenzene	1	mg/kg				-	-	8.8
Hexachlorobutadiene	0.5	mg/kg				-	-	7.7
Hexachlorocyclopentadiene	2.5	mg/kg				-	-	<2.5
Hexachloroethane	0.5	mg/kg				-	-	5.2
Hexachloropropylene	0.5	mg/kg				-	-	<0.5
Pentachlorobenzene	0.5	mg/kg				-	-	<0.5
Trihalomethanes								
Bromochloromethane	0.5	mg/kg				-	-	<0.5
Bromodichloromethane	0.5	mg/kg				-	-	<0.5
Bromoform	0.5	mg/kg				-	-	<0.5
Chloroform	0.5	mg/kg	12	120.00	480	-	-	<0.5
Dibromochloromethane	0.5	mg/kg				-	-	<0.5
Fumigants								
1,2-Dibromomethane	0.5	mg/kg				-	-	<0.5
1,2-Dichloropropane	0.5	mg/kg				-	-	<0.5
2,2-Dichloropropane	0.5	mg/kg				-	-	<0.5
cis-1,3-Dichloropropylene	0.5	mg/kg				-	-	<0.5
trans-1,3-Dichloropropylene	0.5	mg/kg				-	-	<0.5

**Table 1 - Southlands Soil Deliniation
Orica & Macquarie Goodman Pty. Ltd.
Analytical Results**

Blck 2 Blck 2 Blck 2

Sample ID
Date Sampled

TP50_0.4-0.5	TP50_0.9-1.0	TP50_1.2-1.3
12/04/2005	12/04/2005	12/04/2005

Analyte	LOR	Units	Inert	Solid	Industrial	TP50_0.4-0.5	TP50_0.9-1.0	TP50_1.2-1.3
Organochlorine Pesticides (OC)								
4,4-DDD	0.5	mg/kg				-	-	<0.5
4,4-DDE	0.5	mg/kg				-	-	<0.5
4,4-DDT	1	mg/kg				-	-	<1
a-BHC	0.5	mg/kg				-	-	<0.5
Aldrin	0.5	mg/kg				-	-	<0.5
b-BHC	0.5	mg/kg				-	-	<0.5
d-BHC	0.5	mg/kg				-	-	<0.5
Dieldrin	0.5	mg/kg				-	-	<0.5
Endosulfan 1	0.5	mg/kg				-	-	<0.5
Endosulfan 2	0.5	mg/kg				-	-	<0.5
Endosulfan sulfate	0.5	mg/kg				-	-	<0.5
Endrin	0.5	mg/kg				-	-	<0.5
g-BHC	0.5	mg/kg				-	-	<0.5
Heptachlor	0.5	mg/kg				-	-	<0.5
Heptachlor epoxide	0.5	mg/kg				-	-	<0.5
Organophosphorus Pesticides (OP)								
Chlorfenvinphos	0.5	mg/kg				-	-	<0.5
Chlorpyrifos	0.5	mg/kg				-	-	<0.5
Chlorpyrifos-methyl	0.5	mg/kg				-	-	<0.5
Diazinon	0.5	mg/kg				-	-	<0.5
Dichlorvos	0.5	mg/kg				-	-	<0.5
Dimethoate	0.5	mg/kg				-	-	<0.5
Ethion	0.5	mg/kg				-	-	<0.5
Fenthion	0.5	mg/kg				-	-	<0.5
Malathion	0.5	mg/kg				-	-	<0.5
Pirimphos-ethyl	0.5	mg/kg				-	-	<0.5
Prothiofos	0.5	mg/kg				-	-	<0.5
Oxygenated Compounds								
2-Butanone (MEK)	5	mg/kg				-	-	<5
2-Hexanone (MBK)	5	mg/kg				-	-	<5
2-Propanone	6	mg/kg				-	-	-
4-Methyl-2-pentanone	5	mg/kg				-	-	<5
Vinyl Acetate	5	mg/kg				-	-	<5
Phenolic Compounds								
2,3,4,6-Tetrachlorophenol	0.5	mg/kg				-	-	-
2,4,5-Trichlorophenol	0.5	mg/kg	800	8000	32000	-	-	-
2,4,6-Trichlorophenol	0.5	mg/kg	4	40	160	-	-	-
2,4-Dichlorophenol	0.5	mg/kg				-	-	-
2,4-Dimethylphenol	0.5	mg/kg				-	-	-
2,6-Dichlorophenol	0.5	mg/kg				-	-	-
2-Chlorophenol	0.5	mg/kg				-	-	-
2-Methylphenol	0.5	mg/kg				-	-	-
2-Nitrophenol	0.5	mg/kg				-	-	-
3- & 4-Methylphenol	0.5	mg/kg				-	-	-
4-Chloro-3-Methylphenol	0.5	mg/kg				-	-	-
Pentachlorophenol	1	mg/kg				-	-	-
Phenol	0.5	mg/kg	28.8	288	1152	-	-	-
Phthalate Esters								
bis(2-ethylhexyl) phthalate	5	mg/kg				-	-	<5
Butyl benzyl phthalate	0.5	mg/kg				-	-	<0.5
Diethyl phthalate	0.5	mg/kg				-	-	<0.5
Dimethyl phthalate	0.5	mg/kg				-	-	<0.5
Di-n-butyl phthalate	0.5	mg/kg				-	-	<0.5
Di-n-octylphthalate	0.5	mg/kg				-	-	<0.5
Miscellaneous Compounds								
1,2,4,5-Tetrachlorobenzene	0.5	mg/kg				-	-	0.8
1,3,5-Trichlorobenzene	0.5	mg/kg				-	-	1.9
cis-Isosafrole	0.5	mg/kg				-	-	<0.5
Diallate	0.5	mg/kg				-	-	<0.5
Methanesulfonate ethyl	0.5	mg/kg				-	-	<0.5
Methanesulfonate methyl	0.5	mg/kg				-	-	<0.5
Safrole	0.5	mg/kg				-	-	<0.5
trans-Isosafrole	0.5	mg/kg				-	-	<0.5
Naphthalene								
Naphthalene	0.5	mg/kg				-	-	-
Natural Attenuation Parameters								
Total Organic Carbon	0.5	%				-	-	-
Sulfonated Compounds								
Carbon disulfide	0.5	mg/kg				-	-	<0.5
Asbestos								
Asbestos						-	-	-
Miscellaneous								
Moisture Content	1	%				-	-	-

Classification without TCLP

Hazardous Solid Hazardous




Legend:

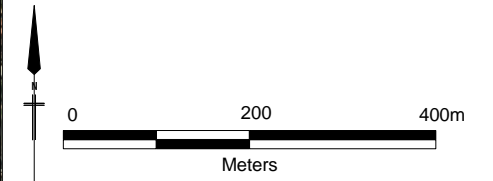
Total concentration less than or equal to inert waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS INERT WASTE
Total Concentration less than or equal to solid waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS SOLID WASTE
Total Concentration less than or equal to industrial waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS INDUSTRIAL WASTE
Total Concentration exceeds industrial waste criteria as outlined in the NSW DEC 2004 Waste Classification - CLASSIFIED AS HAZARDOUS WASTE

PS - Primary Sample FD - Field Duplicate
LD - Laboratory Duplicate FT - Field Triplicate
ND - Not Detected - - Not Analysed
D - Detected mg/kg - milligrams per kilogram
LOR - Limit of Reporting

Figures



- Legend:
-  INVESTIGATION AREA
 -  SPRINGVALE DRAIN
 -  FLOODVALE DRAIN



Datum: GDA94, Projection: UTM, Grid: MGA Zone 56
 Map compiled using SKM Aerial Imagery, January 2005,
 Mapinfo StreetPro (and CadastralPlus) © 2005 and PSMA
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Drawn: AJW | Approved: FINAL | Date: 31-07-06

Job No: **43217542** | File: 43217542-019.wor

Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
SITE LOCATION PLAN

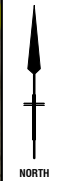
Figure: 1



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- Legend:
- SOUTHLANDS PROPERTY BOUNDARY
 - CADASTRAL BOUNDARIES
 - GTP MONITORING WELL
 - GTP EXTRACTION WELL
 - +— GTP TRANSFER PIPELINE (ABOVE GROUND; UNDERGROUND)
 - - - ELECTRICITY



Datum: GDA94, Projection: UTM, Grid: MGA Zone 56
 Map compiled using SKM Aerial Imagery, January 2005, Mapinfo StreetPro (and CadastralPlus) © 2005 and PSMA Australia Ltd.

Drawn: AJW	Approved: FINAL	Date: 2/10/08
------------	-----------------	---------------

Job No: 43217542	File: 43217542-020.wor
-------------------------	------------------------

Client

ORICA AUSTRALIA PTY LIMITED/
GOODMAN PTY LIMITED

Project

SOUTHLANDS REMEDIATION AND
DEVELOPMENT PROJECT

Title

**SITE LAYOUT AND
EXISTING INFRASTRUCTURE**

Figure: 2



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LEGEND
 DT Inground Detention Tank



Refer Plans SRD DA006 & SRD DA011

Subject to Future Project Approval

SOUTHLANDS AREA SCHEDULE	
Lot 1 DP 254382	0.285 Ha
Lot 1 DP 528680	8.530 Ha
Lot 1 DP 85542	6.130 Ha
Lot 11 DP 109506	2.343 Ha
TOTAL SITE AREA	18.288 Ha
Springvale Drain	0.288 Ha
Nant Street	0.347 Ha

MASTERPLAN DEVELOPMENT AREA SCHEDULE	
Total Site Area	18,288 Ha
Access Road	0.743 Ha
Detention Basins	1.048 Ha
TOTAL DEV. AREA	16,498 Ha
Total Warehouse	73,200 sqm
Total Office	6,750 sqm
Cafe Amenities	240 sqm
Total Floor Area	79,190 sqm
Total Arming	2,000 sqm
Possible Multi-deck Parking	4,666 sqm

FLOOR AREA	79,190 SQM
FOOTPRINT (incl. arming, parking deck)	83,355 SQM
FSR	0.48:1
SITE COVER	50.5%
LANDSCAPE AREA	41,380 SQM (25%)

Carparking Provided -on grade	620
Possible Future Carparking (2 lev)	300
Total Carparking	1120

STAGE 1	
TOTAL SITE AREA	98,150 sqm
DEV. SITE AREA	82,890 sqm
Total Warehouse	43,000 sqm
Total Office	4,000 sqm
Total Facility	47,000 sqm

Carparking Provided -on grade	440
Possible Future Carparking (2 lev)	300
Total Carparking	740

STAGE 2	
TOTAL SITE AREA	48,300 sqm
DEV. SITE AREA	40,310 sqm
Total Warehouse	14,800 sqm
Total Office	1,400 sqm
Cafe Amenities	240 sqm
Total Facility	16,440 sqm

Carparking Provided -on grade	280
-------------------------------	-----

STAGE 3	
TOTAL SITE AREA	38,430 sqm
DEV. SITE AREA	31,780 sqm
Total Warehouse	14,300 sqm
Total Office	1,400 sqm
Total Facility	15,700 sqm

Carparking Provided -on grade	120
-------------------------------	-----

design Southlands Remediation & Development Project **Masterplan** 1:1000 @ A1 1:2000 @ A3 SRD DA005 (B)
 McPherson Street, Botany Part 3A Project Application 23 Jan 08 VK

Drawn: AJW Approved: FINAL Date: 21.02.08

Job No: 43217542 File: 43217542.031.wor

Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
 STAGE 2 SITE PLAN

Figure: 3





LEGEND

- DT Inground Detention Tank
- AW Awning
- HS Hermland

Development Area Schedule

STAGE 1	
Total Site Area	98,150 sqm
less Detention Basins	1,820 sqm
less Access Road	3,440 sqm
Dev. Site Area	92,890 sqm
Total Warehouse	43,000 sqm
Total Office (2 & 3 levels)	4,000 sqm
Total Floor Area	47,000 sqm
Awning	1,840 sqm
Future Parking Deck	4,655 sqm
Footprint (incl awning & parking deck)	50,935 sqm
Site Cover	55 %
FSR	51 %
Landscape Area - 23%	20,950 sqm
Carparking on grade	442
Future Carparking (2 lev)	300
Minor Earthworks Area	45,210 sqm

Drawn: AJW	Approved: FINAL	Date: 21.02.08
Job No: 43217542	File: 43217542.032.wor	
Client		
ORICA AUSTRALIA PTY LIMITED/ GOODMAN PTY LIMITED		
Project		
SOUTHLANDS REMEDIATION AND DEVELOPMENT PROJECT		
Title		
PROJECT APPROVAL (STAGE 1) LANDUSE SCENARIO		

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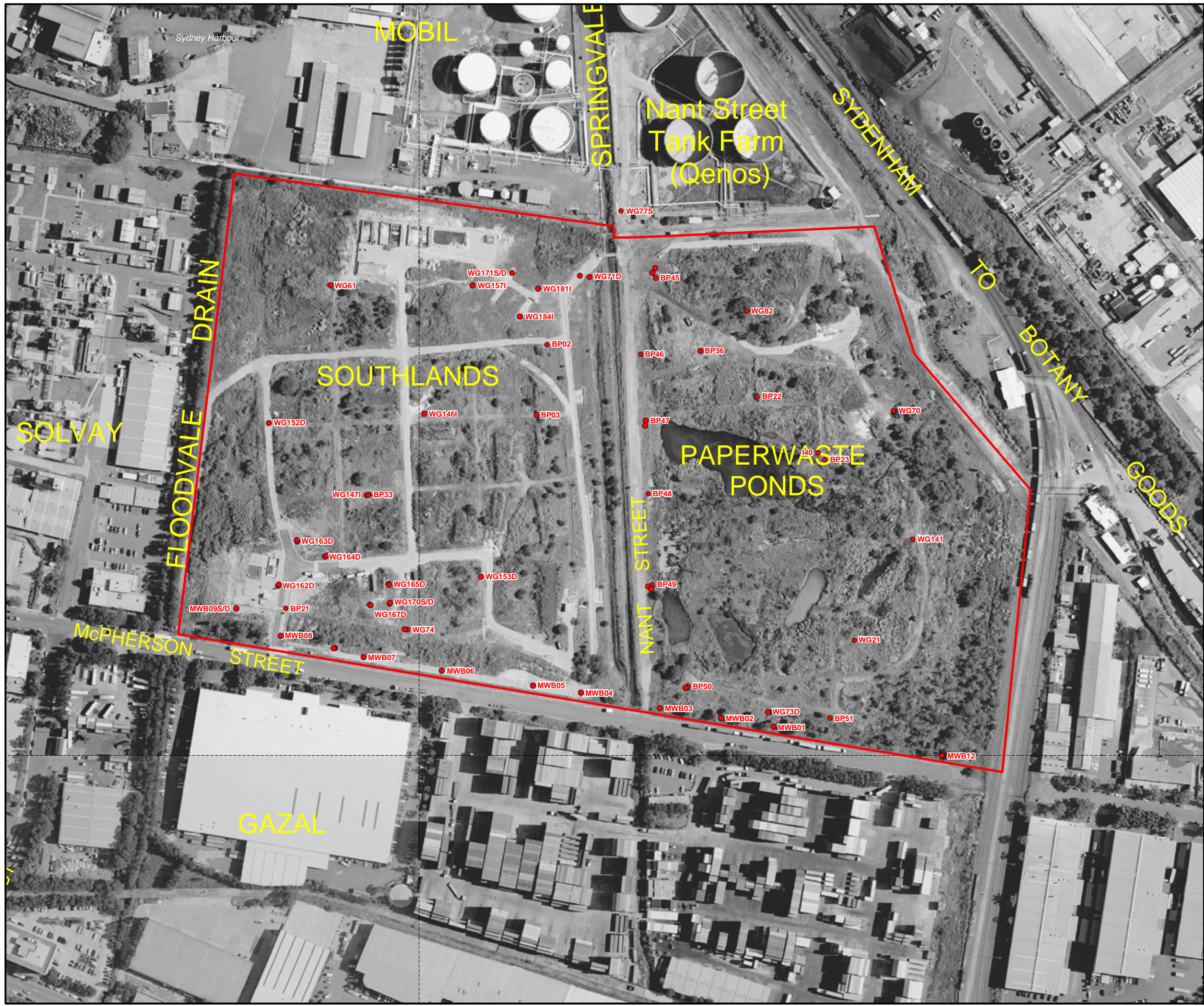


Legend:

- Hydraulic Containment Extraction Well
- Hydraulic Containment Monitoring Well

Datum: GDA94, Projection: UTM, Grid: MGA Zone 56
 Map compiled using SKM Aerial Imagery, January 2005, Mapinfo StreetPro (and CadastralPlus) © 2005 and PSMA Australia Ltd.

Drawn: AJW	Approved: FINAL	Date: 2/10/08
Job No: 43217542	File: 43217542-025.wor	
Client		
ORICA AUSTRALIA PTY LIMITED/ GOODMAN PTY LIMITED		
Project		
SOUTHLANDS REMEDIATION AND DEVELOPMENT PROJECT		
Title		
ORICA GTP HYDRAULIC CONTAINMENT AREAS		
Figure: 5		



Legend:

- Bundle Piezometer/Groundwater Monitoring Well

Datum: GDA94, Projection: UTM, Grid: MGA Zone 56
 Map compiled using SKM Aerial Imagery, January 2005, Mapinfo StreetPro (and CadastralPlus) © 2005 and PSMA Australia Ltd.

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Job No: 43217542	File: 43217542-033.wor	
Client		
ORICA AUSTRALIA PTY LIMITED/ MACQUARIE GOODMAN PTY LIMITED		
Project		
ORICA SOUTHLANDS REMEDIACTION ACTION PLAN		
Title		
SOUTHLANDS EXISTING MONITORING WELLS		
Figure: 6		



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Source: Orica, 2007

Drawn: AW | Approved: FINAL | Date: 2/10/08

Job No: **43217542** | File: 43217542-023.wor

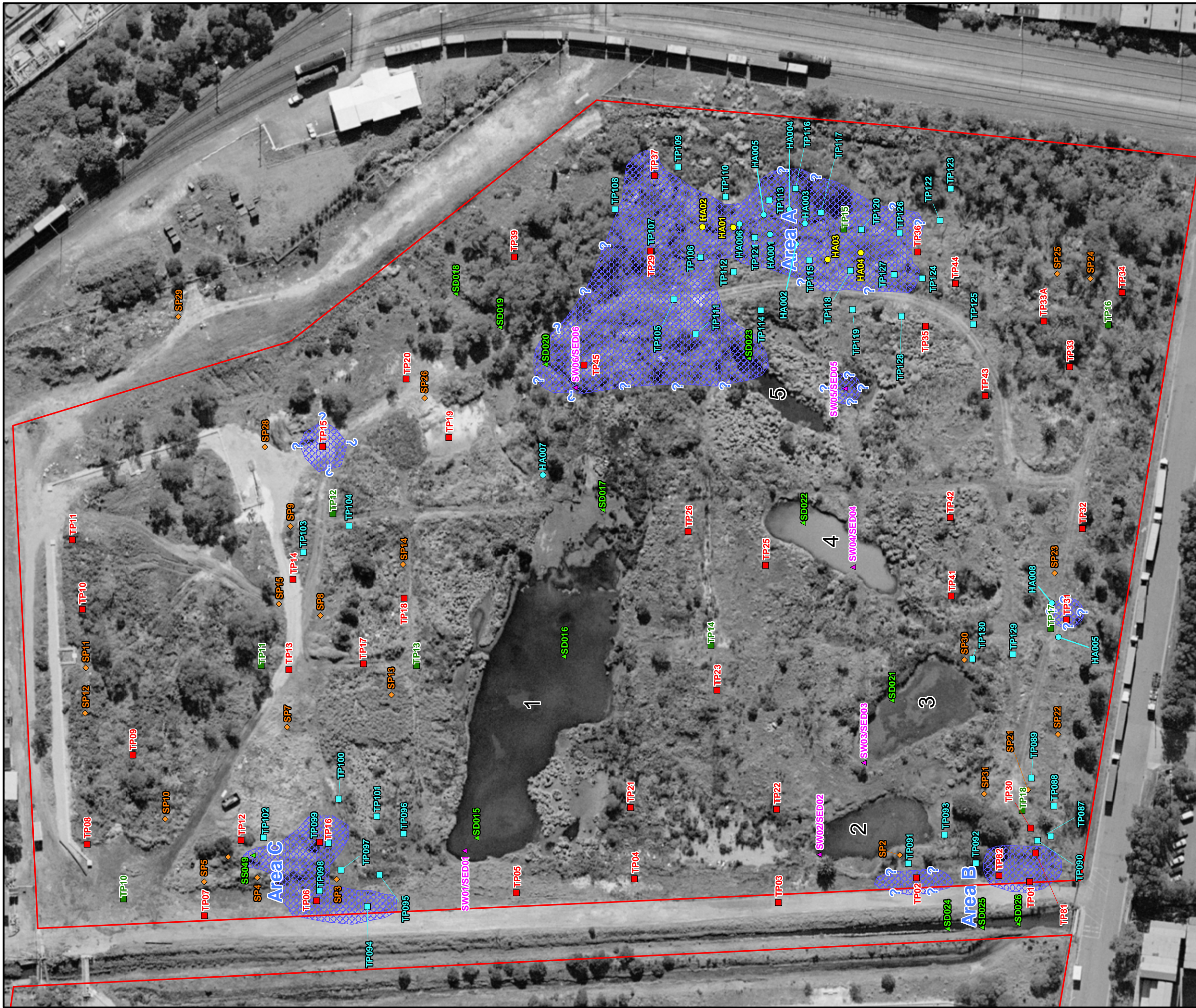
Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
 SURROUNDING ENVIRONMENT

Figure: 7





Legend:

- ORICA PROPERTY BOUNDARY
- URS DATA**
 - TEST PIT LOCATIONS
 - HAND AUGER LOCATIONS
- HLA DATA**
 - ▲ SEDIMENT SAMPLE LOCATIONS
 - ◆ STOCKPILE SAMPLE LOCATION
 - HAND AUGER LOCATIONS
 - TEST PIT LOCATION
- WOODWARD CLYDE DATA**
 - TEST PIT LOCATIONS
 - HAND AUGER LOCATIONS
 - ▲ SEDIMENT SAMPLE LOCATIONS
 - ▨ IMPACTED SOILS

Scale

Source: SKM Ausimage, 2001, 2005
 Datum: GDA94, Projection: UTM, Grid: MGA Zone 56

Drawn: BH	Approved: GB	Date: 2/10/2008
Job No: 43217542	File No: 43217542-027.wor	

Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
TEST PIT LOCATIONS

Figure: 8

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Legend:

— ORICA PROPERTY BOUNDARY

URS DATA

- TEST PIT LOCATIONS
- HAND AUGER LOCATIONS

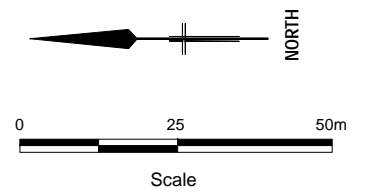
HLA DATA

- ▲ SEDIMENT SAMPLE LOCATIONS
- ◆ STOCKPILE SAMPLE LOCATION
- HAND AUGER LOCATIONS
- TEST PIT LOCATION

WOODWARD CLYDE DATA

- TEST PIT LOCATIONS
- HAND AUGER LOCATIONS
- ▲ SEDIMENT SAMPLE LOCATIONS

■ IMPACTED SOILS



Source: SKM Ausimage, 2001, 2005
 Datum: GDA94, Projection: UTM, Grid: MGA Zone 56

Drawn: BH | Approved: GB | Date: 2/10/2008

Job No: 43217542 | File No: 43217542-027.wor

Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

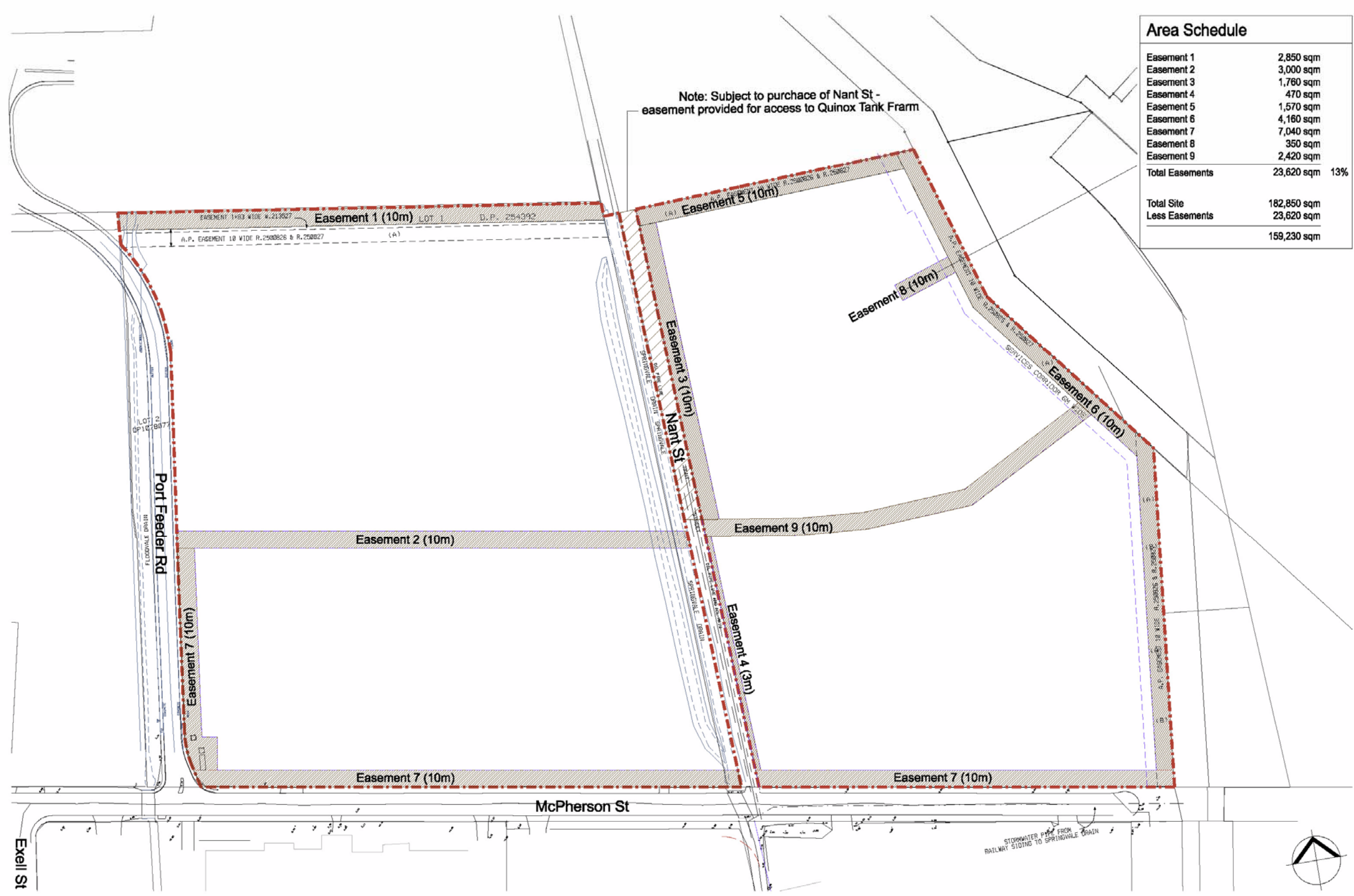
Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
TEST PIT LOCATIONS

Figure: 9



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Note: Subject to purchase of Nant St -
easement provided for access to Quinox Tank Fram

Area Schedule	
Easement 1	2,850 sqm
Easement 2	3,000 sqm
Easement 3	1,760 sqm
Easement 4	470 sqm
Easement 5	1,570 sqm
Easement 6	4,160 sqm
Easement 7	7,040 sqm
Easement 8	350 sqm
Easement 9	2,420 sqm
Total Easements	23,620 sqm 13%
Total Site	182,850 sqm
Less Easements	23,620 sqm
	159,230 sqm

Source: Connell Wagner, 2007

Drawn: AW Approved: FINAL Date: 2/10/08

Job No: **43217542** File: 43217542-028.wor

Client
ORICA AUSTRALIA PTY LIMITED/
GOODMAN PTY LIMITED

Project
SOUTHLANDS REMEDIATION AND
DEVELOPMENT PROJECT

Title
PROPOSED EASEMENTS PLAN

Figure: 10



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Refer Plans SRD DA006 & SRD DA011

Subject to Future Project Approval

LEGEND

DT Inground Detention Tank



SOUTHLANDS AREA SCHEDULE

Lot 1 DP 254382	0.285 Ha
Lot 1 DP 528680	9.530 Ha
Lot 1 DP 85542	6.130 Ha
Lot 11 DP 109505	2.343 Ha
TOTAL SITE AREA	19.288 Ha
Springvale Drain	0.289 Ha
Narr Street	0.347 Ha

MASTERPLAN DEVELOPMENT AREA SCHEDULE

Total Site Area	16.288 Ha
Access Road	0.743 Ha
Detention Basins	1.049 Ha
TOTAL DEV. AREA	16.498 Ha
Total Warehouse	72,200 sqm
Total Office	6,750 sqm
Cafe/ Amenities	240 sqm
Total Floor Area	79,190 sqm
Total Awning	2,666 sqm
Possible Multi-deck Parking	4,666 sqm

FLOOR AREA	79,190 SQM
FOOTPRINT (incl. awning, parking deck)	83,355 SQM
FSR	0.48:1
SITE COVER	50.5%
LANDSCAPE AREA	41,360 SQM (25%)

Carparking Provided -on grade	820
Possible Future Carparking (2 lev)	300
Total Carparking	1120

STAGE 1

TOTAL SITE AREA	98,150 sqm
DEV. SITE AREA	82,890 sqm
Total Warehouse	43,000 sqm
Total Office	4,000 sqm
Total Facility	47,000 sqm

Carparking Provided -on grade	440
Possible Future Carparking (2 lev)	300
Total Carparking	740

STAGE 2

TOTAL SITE AREA	48,300 sqm
DEV. SITE AREA	40,310 sqm
Total Warehouse	14,850 sqm
Total Office	1,400 sqm
Cafe/ Amenities	240 sqm
Total Facility	16,490 sqm

Carparking Provided -on grade	280
-------------------------------	-----

STAGE 3

TOTAL SITE AREA	38,430 sqm
DEV. SITE AREA	31,760 sqm
Total Warehouse	14,350 sqm
Total Office	1,400 sqm
Total Facility	15,750 sqm

Carparking Provided -on grade	120
-------------------------------	-----

- Legend**
- MONITORING WELL REPLACEMENT LOCATION
 - EXTRACTION WELL REPLACEMENT LOCATION
 - ⊕ EXISTING EXTRACTION WELLS TO BE RETAINED
 - EXISTING MONITORING WELLS TO BE RETAINED

Drawn: AJW | Approved: FINAL | Date: 21.02.08

Job No: 43217542 | File: 43217542.034.wor

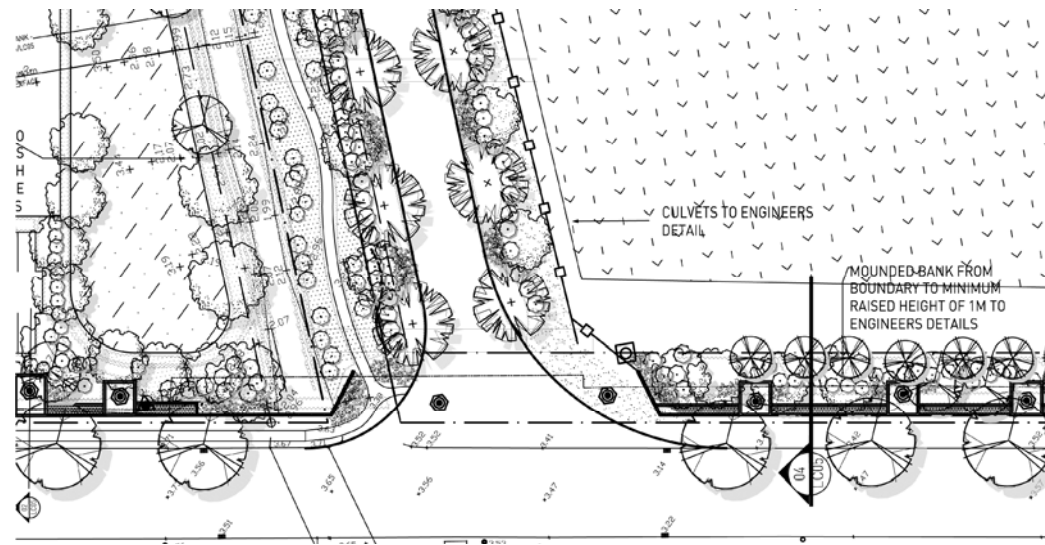
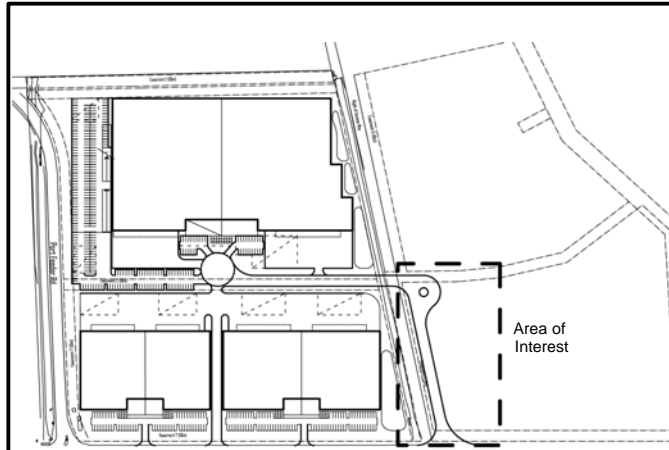
Client
ORICA AUSTRALIA PTY LIMITED/
GOODMAN PTY LIMITED

Project
SOUTHLANDS REMEDIATION AND
DEVELOPMENT PROJECT

Title
LOCATION OF REPLACEMENT
MONITORING WELLS

Figure: 11





ESTATE ROAD



SITE BOUNDARY

STREET TREE TO COUNCIL REQUIREMENTS

NEW CONCRETE FOOTPATH

SECTION 04



EXISTING GROUND

300MM PLANTING MIX TYPE A AND 300 TYPE B MIX FOR LANDSCAPE BANK. FILL UNDER TO ENGINEERS DETAILS

PIPE TRENCH WITH WIRE GRATE TO FUTURE DETAIL

600HT WALL WITH 1200HT PALLISADE FENCE.

BOUNDARY PLANTING TO SOFTEN SITE BOUNDARY AND ASSIST IN SCREENING OF PIPE TRENCH AND WELLS.

Source: Habitation, 2007. 07011 STG 1 LOC5-22-8-07.pdf and 07011 Stg 1 LC04-22-8-07.pdf

Drawn: AW Approved: FINAL Date: 2/10/08

Job No: 43217542 File: 43217542-029.wor

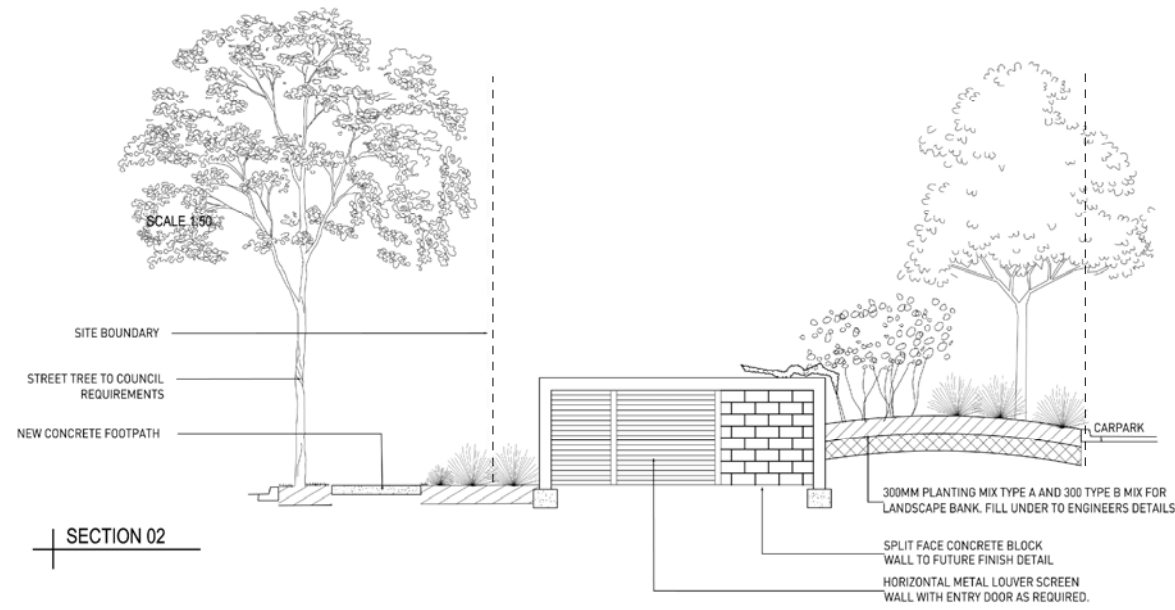
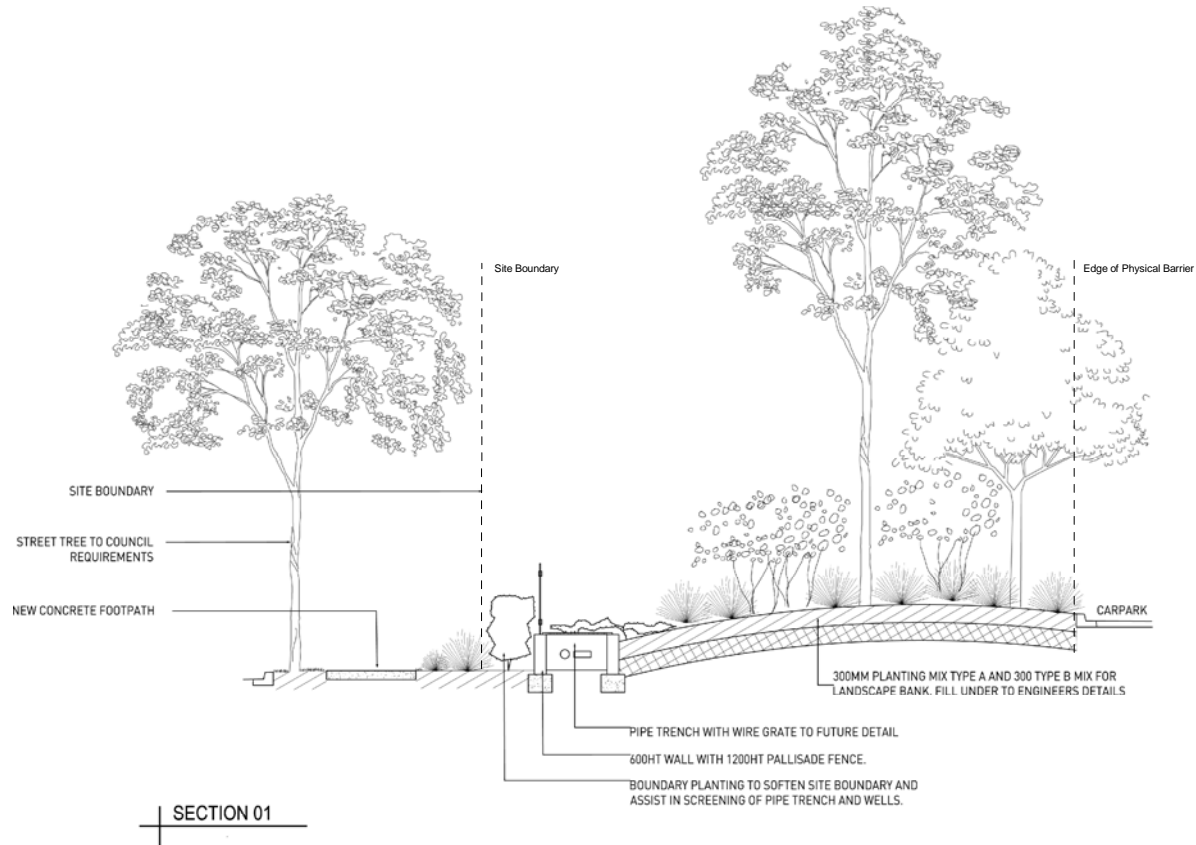
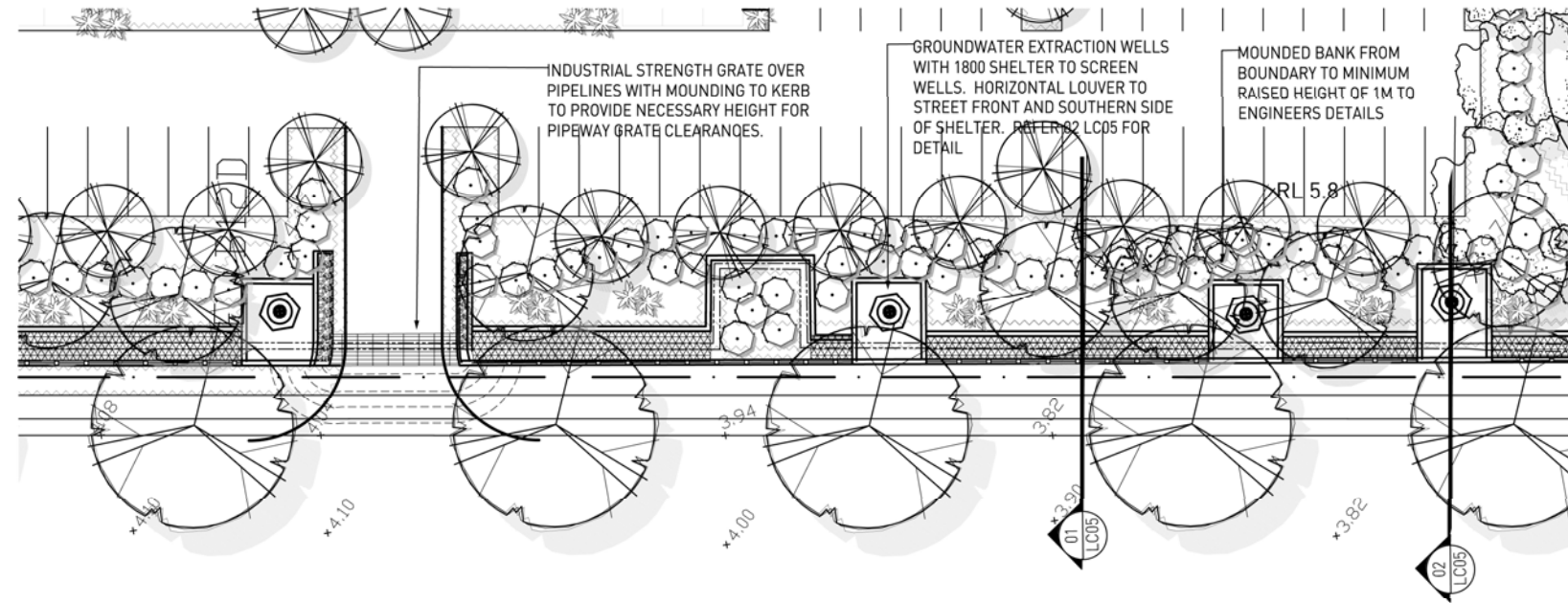
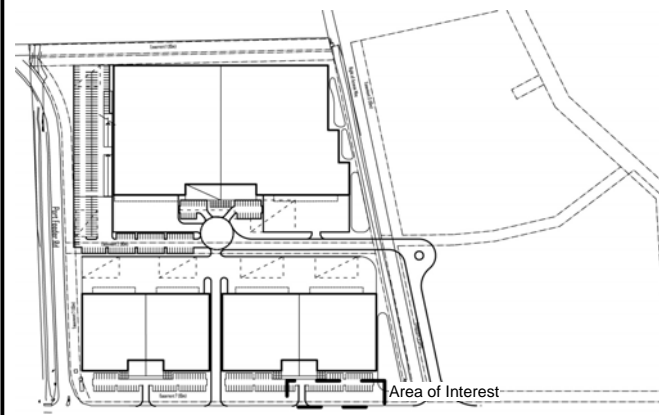
Client
ORICA AUSTRALIA PTY LIMITED/
GOODMAN PTY LIMITED

Project
SOUTHLANDS REMEDIATION AND
DEVELOPMENT PROJECT

Title
McPHERSON STREET BOUNDARY
DETAIL - BLOCK 1 (STAGE 1)

Figure: 12





Source: Habitation, 2007. 07011 STG 1 LOC5-22-8-07.pdf and 07011 Stg 1 LC04-22-8-07.pdf

Drawn: AW Approved: FINAL Date: 2/10/08

Job No: 43217542 File: 43217542-030.wor

Client
ORICA AUSTRALIA PTY LIMITED/
GOODMAN PTY LIMITED

Project
SOUTHLANDS REMEDIATION AND
DEVELOPMENT PROJECT

Title
McPHERSON STREET BOUNDARY
DETAIL - BLOCK 2 (STAGE 1)

Figure: 13



Appendix A

Human Health Risk Assessment

FINAL REPORT

Human Health Risk Assessment, Southlands Remediation and Development Project

Prepared for

Orica Australia Pty Ltd & Goodman Pty Ltd

2 October 2008

43217543

Project Manager:



.....
Gavan Butterfield
Senior Associate
Environmental Scientist

URS Australia Pty Ltd

Level 3, 116 Miller Street
North Sydney
NSW 2060
Australia
Tel: 61 2 8925 5500
Fax: 61 2 8925 5555

Project Director:



.....
Jackie Wright
Principal Environmental
Scientist

Author:

(Optional)



.....
Jackie Wright
Principal Environmental
Scientist

Date: 2 October 2008
Reference: Southlands HHRA Rev D
Status: Final

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Glossary of Terms

Absorption - The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acceptable Daily Intake (ADI) - The amount of a chemical a person can be exposed to on a daily basis over an extended period of time (usually a lifetime) without suffering deleterious effects.

Acute exposure - Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Additive effect - A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

Adverse health effect - A change in body function or cell structure that might lead to disease or health problems.

AHD - Australian Height Datum - a standard reference point for the elevation of a location.

Antagonistic effect - A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

ANZECC - Australian and New Zealand Environment and Conservation Council.

Background level - An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation - Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Body burden - The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

Carcinogen - A substance that causes cancer.

Chemical of Potential Concern (COPC) – Chemical present in environmental media at a concentration sufficiently high or there is a sufficiently high degree of uncertainty to warrant further assessment in relation to risks.

Chronic exposure - Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].

Dermal contact - Contact with (touching) the skin [see route of exposure].

Detection limit - The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

DECC and NSW EPA - Department of Environment and Climate Change (formerly known as the Department of Environment and Conservation (DEC)) and incorporating the NSW Environment Protection Authority.

Dose - The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Exposure - Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].

Exposure assessment - The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure pathway - The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as chemical leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching); and a receptor population (people potentially or

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actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Groundwater - Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

GTP - Groundwater Treatment Plant- A chemical treatment plant required to be constructed for the ex situ treatment of groundwater from hydraulic containment as required by the Notice of Clean Up Action (NCUA).

Guideline Value - Guideline value is a concentration in soil, sediment, water, biota, or air (established by relevant regulatory authorities, such as the DEC or institutions such as the NHMRC, ANZECC and WHO), that is used to identify conditions below which no adverse effects or nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter- and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines will have different names such as investigation level, trigger value, ambient guideline, etc.

Hazard Index and Hazard Quotient – Hazard quotient is the ratio of daily chemical calculated for a specific receptor and exposure pathway, to the acceptable or safe dose (ADI, TDI, RfD, etc.) for that chemical. A value less than 1 indicates that the intake is less than the safe intake. A hazard index is the sum of the hazard quotients for all exposure pathways for a receptor.

Ingestion - The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation - The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure - Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

Lowest-observed-adverse-effect level (LOAEL) - The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

MRL – The Maximum Residue Limit (MRL) is the maximum residue concentration from the legal use of an agricultural or veterinary chemical that is recommended as the acceptable maximum concentration in a food.

Metabolism - The conversion or breakdown of a substance from one form to another by a living organism.

NHMRC - National Health and Medical Research Council.

No-observed-adverse-effect level (NOAEL) - The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

Orica - Orica Australia Pty Limited

Plume - A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or the envelope of a contaminant moving with groundwater.

Point of exposure - The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population - A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

RAP – Remediation Action Plan

Receptor population - People who could come into contact with hazardous substances [see exposure pathway].

Reference dose (RfD) – Specifically refers to a toxicity value identified by the USEPA. The RfD is similar to an ADI or TDI and incorporates uncertainty or safety factors to identify a safe dose assuming daily lifetime exposure to a substance that is unlikely to cause harm in humans.

Reasonable Maximum Exposure (RME) - The RME represents an exposure scenario based on a set of exposure parameters that is representative of expected maximum exposure for that receptor and activity. The RME would not be expected to be exceeded except under highly specific and exceptional circumstances.

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Reference concentration (RfC) - The concentration of a specific chemical in air to which a human population may be exposed to without appreciable risk to their health. RfC's are identified by the USEPA.

Risk - The probability that something will cause injury or harm.

Risk reduction - Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Route of exposure - The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Surface water - Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

Synergistic effect - A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Tolerable Concentration (TC) - A TC (established by WHO) is an airborne concentration to which it is believed that a person can be exposed continuously over a lifetime without deleterious effects. The TC is based on non-carcinogenic effects and is usually calculated by applying uncertainty factors to a NOAEL or LOAEL. As such, the TC is similar to the USEPA reference concentration for inhalation exposures and ADI, TDI or RfD for oral exposures.

Tolerable Daily Intake (TDI) - The term tolerable daily intake (TDI) is used by the International Program on Chemical Safety (IPCS) to describe exposure limits of toxic chemicals and the term acceptable daily intake (ADI) is used by the World Health Organization (WHO) and other national and international health authorities and institutes.

Toxicological profile - An assessment that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology - The study of the harmful effects of substances on humans or animals.

Uncertainty factor - Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

WHO – World Health Organisation.

Executive Summary

URS Australia Pty Ltd (URS) have been commissioned by Orica Australia Pty Ltd (Orica) and Goodman Pty Ltd (jointly the Proponent) to undertake a quantitative human health risk assessment (HHRA) as part of the Environmental Assessment (EA) being prepared for the staged remediation and development of the property referred to as Southlands. The EA will incorporate a Remediation Action Plan (RAP) prepared to meet the NSW DEC accredited Site Auditor's requirements. Southlands is a disused site owned by Orica that is vacant, approximately 20 hectares in size, which fronts onto McPherson Street, Banksmeadow (the site).

The Proponent proposes to develop the site for high quality industrial and warehouse purposes. The current proposal addresses development in stages, with Stages 1 and 2 specifically addressed in this HHRA. The site is currently zoned 4(a) Industrial within the Botany Bay City Council area and is to be developed for commercial purposes with no more sensitive uses (such as childcare) allowed.

Review of data available for Southlands indicates the presence of contamination in groundwater, soil, sediment, surface water and air. On the basis of the available data and understanding of the proposed development, risks to human health associated with the proposed development have been quantified in accordance with guidance from enHealth (Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards, 2004).

The process followed for the completion of the HHRA has been an iterative approach. The available site data and development proposal was considered initially in a Preliminary Human Health Risk Assessment (PHHRA, URS, 2006c). The PHHRA identified a number of key issues that required further assessment and risk management measures prior to completion of the HHRA. The key issues identified included the potential for shallow groundwater to discharge to Springvale Drain (resulting in emissions to air of volatile chlorinated compounds), potential for shallow groundwater to discharge into proposed compensatory flood storage areas (resulting in emissions to air of volatile chlorinated compounds) and presence of residual impacts in soils on the site. Further investigations were undertaken and risk management measures were identified and discussed to address these issues. The outcome of these investigations and discussions and identified risk management measures were incorporated into the RAP. The risk management measures proposed were then further considered in the completion of the HHRA.

The HHRA presented has therefore been undertaken as a site-specific assessment that considers key aspects of the proposed development, flood management requirements and risk management measures. These include the filling in of existing ponds on Block 1, construction of compensatory flood storage areas on the southern portion of Block 1, the placement of fill on Blocks 1 and 2 prior to construction of any buildings to address flood issues and the restriction of access to the northern portion of Block 1 (proposed Stage 3 development). With respect to risk management measures the assessment has considered proposed measures designed to intersect shallow groundwater prior to discharge into Springvale Drain (with groundwater directed back to the GTP) and the drainage system proposed to control shallow groundwater in the area of the compensatory flood storage area on Block 1. The performance of these measures will need to be monitored through the collection of appropriate data for some time after commissioning. Should any of these assumptions change, or the proposed risk management measures do not achieve the stated objectives, then the assessment of risks to human health presented in the report will need to be reviewed and potentially revised.

The following table presents a summary of all the risk issues identified in the HHRA for the proposed Stage 1 and Stage 2 developments.

Executive Summary

Table ES1 Summary of Risk Issues

Issue	Issues Identified	Risk Mitigation Measures
All Aspects of the Proposed Development		
Intrusive workers in all areas including the maintenance of GTP infrastructure	Potential for elevated exposures to chemicals in shallow groundwater and inhalation of volatile chlorinated chemicals (particularly within confined spaces)	All works to be undertaken under a long term environmental management plan [EMP] that ensures development of job specific safe work practices including addressing any issues associated with works that breach the areas covered with barriers.
Workers involved in maintenance of Springvale and Floodvale Drains	Potential for elevated exposures to chemicals in surface water and inhalation of volatile emissions	All works to be undertaken under a long term environmental management plan that ensures development of job specific safe work practices.
Presence of asbestos fibres in soil	Potential for generation of dusts containing asbestos fibres.	Placement of appropriate barrier. Where no physical barrier is present a sound ground cover should be maintained to minimise generation of dusts. These areas need to be considered in a long term environmental management plan [EMP].
Inhalation of volatile chlorinated chemicals associated with emissions to air from Springvale Drain	Potential for significant exposures by all users of the site on basis of measurements completed to date.	Surface water concentrations within the drain would need to be lowered such that air concentrations are reduced by 100 to 1000 fold. This may require a reduction in the discharge of shallow groundwater into Springvale Drain. Proposed mitigation measure involves a shallow groundwater extraction system adjacent to Springvale Drain to intercept groundwater prior to discharge into the drain, with groundwater directed to the GTP. Exposures are expected to be lower once system is installed, however the effectiveness requires verification. Risk issues when the GTP is offline need to be reviewed in the final design and management of such a system.
Stage 1 Development (also relevant to Stage 2 development)		
Elevated concentrations identified in soil and sediment in the ephemeral ponds – that are moved across the site during earthworks	Potential for elevated exposure by workers in area not covered with barriers, including gardening activities (ingestion, dermal contact and vapour intrusion into buildings)	Remediation of assumed hot-spots to a level protective of all exposures including vapour intrusion (refer to details in HHRA). Remediation to derived risk-based concentrations (derived and presented in the HHRA to assist in this process for some key chemicals) or other guidelines that can be justified on the basis of risks to human health. Further sampling could be undertaken to better define extent of area affected by elevated concentrations
Inhalation of volatile chemicals in outdoor air (emissions from soil and groundwater)	No issues identified for outdoor air if the compensatory flood storage area is appropriately designed to prevent groundwater discharge to the surface.	Effectiveness of the proposed drainage system for the compensatory flood storage areas on Block 1 requires verification.
Inhalation of volatile chemicals in outdoor air and within buildings constructed on Block 2 (emissions from some subsurface soil and groundwater)	No issues identified (subject to findings of sub slab investigations).	Review of vapour intrusion issues that may arise as a result of the consideration of issues associated with the presence of a large slab.
Stage 2 Development		
Inhalation of volatile chemicals within buildings constructed on the southern portion of Block 1 (emissions from some subsurface soil and groundwater)	Potential for elevated exposure to volatile chlorinated chemicals, present in shallow groundwater, that may migrate into buildings (including buildings associated with GTP infrastructure)	If buildings are constructed in the area vapour mitigation measures should be incorporated into the building design. Such measures may include passive barriers, sub-slab depressurisation or building pressurisation.

Executive Summary

Following this process the HHRA presented has identified a number of risk issues that warrant further consideration within the RAP (and presented in the table above) particularly in relation the following issues:

- Emissions to air from Springvale Drain associated with the discharge of shallow groundwater into the open drain particularly during periods of rainfall. A risk management system has been proposed to intersect shallow groundwater prior to discharge into the drain. The proposed system involves a shallow groundwater extraction system adjacent to Springvale Drain to intercept groundwater prior to discharge into the drain, with groundwater directed to the GTP (refer to the RAP for details). Exposures are expected to be lower once system is installed; however the effectiveness requires verification through the collection of appropriate data for some time after commissioning. In addition, risk issues when the GTP is offline need to be reviewed in the final design and management of such a system;
- Emissions to air from the compensatory flood storage area proposed for Block 1 associated with discharge of shallow groundwater into the area and the design and management of the proposed drainage system to prevent shallow groundwater discharge at the surface (refer to RAP for details). The effectiveness of the drainage system requires verification through the collection of appropriate data for some time after commissioning;
- Management of residual asbestos fibres in soils through the placement and management of clean fill;
- Management of residual soil contamination (using preliminary risk based criteria derived in the HHRA to assist in the removal of residual contamination);
- Mitigation of vapour intrusion into commercial buildings (on Block 1 in particular, and possibly Block 2); and
- Requirement for a long term environmental management plan that addresses intrusive works across the site and access to open drains and the northern portion of Block 1.

Section 1

Introduction

1 Introduction

1.1 Background

URS Australia Pty Ltd (URS) have been commissioned by Orica Australia Pty Ltd (Orica) and Goodman (jointly the Proponent) to undertake a quantitative human health risk assessment (HHRA) as part of the Environmental Assessment (EA) being prepared for the staged remediation and development of the property referred to as Southlands. Southlands is a disused site owned by Orica that is vacant, approximately 20 hectares in size, which fronts onto McPherson Street, Banksmeadow (the site). The site location is presented in **Figure 1**.

The Proponent proposes to develop the site for high quality industrial and warehouse purposes. The site is currently zoned 4(a) Industrial within the Botany Bay City Council area. The Proponent intends to seek Development Approval (DA) for remediation and redevelopment works under Part 3A of the Environmental Planning and Assessment Act 1997. The DA will be supported by an Environmental Assessment (EA) prepared in accordance with Department of Planning requirements. The EA will incorporate a Remediation Action Plan (RAP) prepared to meet the NSW DEC accredited Site Auditor's requirements.

1.2 General Site Description

Orica Southlands is divided into two blocks, known as Block 1 and Block 2 (refer to **Figure 1**), separated by Springvale Drain, a north-south trending man-made drainage feature and surface water conduit. Floodvale Drain, a similar drainage feature, forms the western boundary of Block 2 Southlands.

Nant Street is an unsealed roadway that provides access from the McPherson St. site entrance to the Genos Pty Ltd Tank Farm, located to the north of Southlands.

Various infrastructures, including above ground pipe lines and groundwater extraction bores, which form the Primary Containment Area (PCA) of the Orica Botany Groundwater Treatment Plant (GTP), are located on Southlands, principally along the McPherson Street Boundary and the western margin of Springvale Drain. The PCA extracts groundwater, which is pumped via pipeline to the treatment facility located in the Botany Industrial Park (BIP), located to the north east of Southlands.

There are five depressions on Southlands Block 1, which are periodically charged by surface water as well as groundwater at times of high watertable elevation. It is understood these features, generally referred to as *Paper Waste Ponds*, are remnants of historical sand and peat mining activities, which during a period of ownership by Australian Paper Manufacturers (APM), that were partially backfilled with paper wastes from the manufacturing processes.

Prior to erection of a boundary fence by Orica in the 1980s, Southlands was subject to a significant volume of uncontrolled dumping. The dumped materials generally comprise demolition wastes and rubble. Subsequent investigations have indicated these wastes include asbestos containing materials (ACM). Since cessation of dumping the various stockpiles have become heavily overgrown with vegetation. As a result, the surface of Southlands Block 2, and to a lesser extent Block 1, has a characteristic "*hummocky*" topography.

A grid based series of tracks has been cut into the dense vegetation to enable access for GTP maintenance and environmental monitoring activities.

Block 1 (and to lesser extent Block 2) is underlain by a chlorinated hydrocarbon (CHC) groundwater plume that is the subject of a current NSW DEC Clean Up notice. Considerable groundwater monitoring infrastructure is located on Southlands (as outlined in detail in the RAP).

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1.3 Proposed Development Summary

The site is to be developed for high quality industrial and warehouse purposes. A staged approach to development is proposed and is summarised in the following sections. Further detail of the development and remediation is presented in the RAP and EA with respect to the Concept Master Plan and details on the level of approvals being sought for each development stage.

1.3.1 Project Staging

The primary stage, Stage 1, generally incorporates development of the area to the west of Springvale Drain, known as Southlands Block 2 and bounded by McPherson Street, Port Feeder Road and a Mobil Oil Depot to the south, west and north respectively and Floodvale Drain to the west (refer to **Figure 1**). Stage 1 will also include construction of an internal (private) roadway to the east of Springvale Drain, which will provide access to Stage 1 from McPherson Street.

Later development stages (Stages 2 and potentially Stage 3) will be located on the area to the east of Springvale Drain, known as Southlands Block with Stage 2 on the southern and Stage 3 (proposed) on the northern portion of Southlands Block 1.

While the site is proposed to be developed on a staged approach (with a mix of Project and Concept Approvals being sought in the DA), the RAP and hence the HHRA have been undertaken on the basis of remediation and use of the whole site for the proposed industrial use, as required.

1.3.2 Stage 1

The *Project* plan for Stage 1 (refer to **Figure 2**) makes provision for three major single storey commercial buildings, the largest of which, occupying approximately 24,800 square metres (sqm), is to be located in the northern portion of Stage 1, with main access from McPherson Street. This structure will be divided into two warehouse facilities (Warehouses 5 and 6) of approximately equal area. Office space is also incorporated into the plan, and is likely to be located at the south of the northern building, occupying approximately 2,000 sqm.

A multi-storey car park is proposed to be constructed to the west of the warehouse building, with concrete aprons providing surface level car parking to the south and south west. Two loading bays will be located to the south of the building.

Two smaller buildings, occupying approximately 8,500 and 10,000 sqm respectively, will be located in the southern area of Stage 1, with loading access from McPherson Street via the Proposed Access Road and office space at the south. These buildings will be divided into four warehouse facilities (Warehouses 1 to 4).

All buildings on Block 2 will be set back from Springvale Drain at least 20m with the area between the warehouse buildings and drain to be used for compensatory flood storage or frog ponds. No work areas (storage areas or other outdoor facilities that may be accessed and used on a regular basis) will be located between the warehouse buildings and Springvale Drain. The area will remain accessible, however such access is expected to be infrequent and of short duration (visiting or passing through).

Other features of the Stage 1 development include the placement of clean fill (as outlined in the RAP) across Block 2 prior to the construction of the proposed buildings, filling of the ponds on Block 1 (but no placement of clean fill) and construction of compensatory flood storage areas along the western margin of Springvale Drain (within the southern portion of Block 1).

Prior to later stages of development (Stages 2 and potentially Stage 3), Southlands Block 1 will remain largely vacant, but will be used as a compensatory flood storage area that will require some shallow earthworks on the southern portion of Southlands Block 1. The nature and extent of the required excavation is outlined in the RAP and Flood Management Study (Connell Wagner Pty Ltd, 2007).

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1.3.3 Stage 2

Stage 2 will occupy the southern portion of existing Block 1 (refer to **Figure 3**). The *Project* plan makes provision for three single storey buildings, which will accommodate seven warehouse facilities and associated office spaces.

Other features will include car parking spaces and paved access for loading / unloading of goods. It is expected that prior to the construction of buildings as part of the Stage 2 development clean fill will be placed over the area. No additional works are expected to be undertaken on the northern portion of Block 1 during this Stage. Provision is expected to be made for compensatory flood storage areas the south western corner of the site or in an off-site location (to be determined).

1.3.4 Stage 3 Development

While development of Stage 3 is not included in the current proposal, the area identified for development as part of Stage 3 is the northern portion of existing Block 1. The final plans will be considered once development approval is sought.

It is noted that the Stage 3 area is associated with a number of DNAPL sources for groundwater contamination that are under investigation. It is unsure whether development will be possible in this area due to the presence of these sources and the need to provide an area that may be used to remove and treat DNAPL in the future.

Currently, the only works proposed as part of the development of Stages 1 and 2 that affect the northern portion of Block 1 (proposed Stage 3) are the filling in of the existing ponds that extend into this area. No further works are proposed at this time.

1.4 Objectives and Scope of HHRA

Based on the available information on the proposed development of Southlands, the overall objectives of the HHRA are:

- To provide a quantitative assessment of potential risks to human health associated with the presence of chemicals in soil, groundwater, surface water and air relevant to the proposed **Stage 1** development; and
- To provide a quantitative assessment of potential risks to human health associated with the presence of chemicals in soil, groundwater, surface water and air relevant to the proposed **Stage 2** development; and
- Depending on the outcome of the HHRA, identify issues that require risk mitigation measures to be incorporated into the RAP with the development risk based soil, groundwater and/or vapour concentrations that can be used at a screening level for further investigation or remediation end-points (if required).

The HHRA presented follows on from a Preliminary HHRA (PHHRA, URS 2006c provided in **Appendix H** for reference) undertaken to provide an initial assessment of risks to human health (based on no specific development details and no remediation being undertaken) which identified key issues that require consideration prior to completion of the HHRA and RAP. This HHRA provides a detailed assessment of risks to human health based on the proposed development based on design details understood to be correct at the end of January 2008 (as outlined in **Section 1.3**). The HHRA draws on and provides input into the RAP to enable key issues relevant to the remediation of the site to be adequately identified and addressed as required.

While the HHRA has considered the general design details as at the end of January 2008, the assessment has aimed at inclusion of assumptions applicable to a more general industrial development of the site to provide sufficient flexibility to encompass design modifications that may be reasonably expected and incorporated following completion of the EA.

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The assessment presented does not address off-site issues as these have been addressed separately in the Consolidated HHRA (URS, 2005a) and subsequent monitoring reports prepared for Orica as part of the wider groundwater program.

1.5 Approach to Human Health Risk Assessment

The approach taken to the assessment of human health risks is generally in accordance with the protocols/ guidelines recommended by enHealth (Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards, 2004). These guidelines draw on and are supplemented by those provided by ANZECC and NH&MRC and detailed in the documents:

- The NEPC (Schedule B(4), Guideline on Health Risk Assessment Methodology, 1999);
- “The Health Risk Assessment and Management of Contaminated Sites” (CSMS 1991, 1993, 1996 and 1998 and enHealth 2002); and
- ANZECC/NH&MRC (1992).

The above currently provide only general guidance for the completion of these tasks and, as such, the more detailed protocols and guidelines developed by the US EPA (1989 and 2001) have been used to provide supplementary guidance.

In addition the HHRA will be undertaken to be consistent with other key risk assessment undertaken in the area, in particular, the Consolidated Human Health Risk Assessment (Consolidated HHRA, URS 2005) that provided an assessment of risks to human health associated with issues derived from chlorinated groundwater plumes in off-site areas, including commercial areas surrounding Southlands.

In following this approach and in accordance with these types of risk assessments, the human health risk assessment presented is not an epidemiological study (which is a study of the distribution and causes of existing health related issues in the community), nor does this assessment provide a statistical analysis of the existing health status of the community. Rather, the assessment provides an evaluation of the potential impact of potential exposure to chemicals identified in soils, water and air based on the proposed use of the site using guidance recommended and endorsed by Australian regulators in particular the DECC and NSW Health.

The conduct of a HHRA can be divided into the following four prime tasks:

- Issue Identification;
- Hazard/Toxicity Assessment;
- Exposure Assessment; and
- Risk Characterisation.

The following presents further detail on the approach adopted in the assessment of risks to human health.

1.5.1 Issue Identification

This involves a review of the available data relevant to the development of Southlands. The aim of this review is to identify key issues and chemicals of potential concern (COPC) that warrant more detailed assessment within the HHRA to address potential exposures on the site. This is presented in **Section 3**.

1.5.2 Hazard/Toxicity Assessment

The objective of the toxicity assessment is to identify toxicity values for the COPC identified that can be used to quantify potential risks to human health associated with calculated intake. Toxicity can be defined as “the quality or degree of being poisonous or harmful to plant, animal or human life” (NEPC 1999b).

The steps involved in this process include the following:

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- Obtain relevant qualitative and quantitative toxicity information relevant to the significant exposure pathways being assessed (namely oral, dermal or inhalation); and
- Identify the appropriate toxicity values for assessing both threshold effects and non-threshold carcinogenic effects (if relevant).

Non-Threshold Response

Non-threshold toxicity values assume that any amount of exposure to the chemical has the potential to result in an increased risk. These chemicals are typically carcinogens with their toxicity values referred to as cancer risk slope factors. The World Health Organisation (WHO) assigns slope factors to chemicals identified as genotoxic carcinogens with other carcinogens identified evaluated on the basis of a threshold response relationship (refer below). A slope factor is an upper bound estimate of the probability of a response occurring following the intake of a chemical over a lifetime via a specific exposure pathway (such as ingestion or inhalation). Therefore the higher the slope factor, the higher the risk that may be associated with a given exposure.

Threshold Response

This relationship assumes that there is a level of exposure below which there is no (or no appreciable) risk of an adverse health effect. This is in contrast to the non-threshold relationship where there is an increased risk associated with any exposure. The WHO identifies threshold chemicals as those which are not suspected of exhibiting carcinogenic effects (non-carcinogens) or those which exhibit non-genotoxic carcinogenicity. Toxicity factors for these chemicals are referred to as an acceptable daily intake (ADI, by the WHO) or reference dose (RfD, by the USEPA) for oral exposures (in units of mg per kg body weight per day) and a tolerable concentration (TC, by WHO) or reference concentration (RfC, by USEPA) for inhalation exposures (in units of mg per cubic metre of air). The lower the ADI, RfD, TC or RfC, the more toxic the chemical and the lower the concentration above which there exists a potential for an adverse health effect.

Identification of Toxicity Values

The identification of toxicity values undertaken in this risk assessment has followed guidance provided by enHealth (2004) and NEPC (1999). EnHealth (2004) provides a list of toxicological data sources. These are classified as Level 1, 2 or 3 data, with Level 1 sources recommended. In order of preference the Level 1 sources are:

- 1) National Health and Medical Research Council (NHMRC) documents and documents from other joint Commonwealth, State and Territory organisations.
- 2) ADI List from the Therapeutic Goods Administration.
- 3) WHO documents.
- 4) enHealth Council documents.
- 5) National Environmental Health Forum (NEHF) documents.
- 6) International Agency for Research on Cancer (IARC) monographs.
- 7) WHO/Food and Agriculture Organisation (FAO) Joint Meeting on Pesticide Residues (JMPR) monographs.
- 8) National Industrial Chemicals Notification and Assessment Scheme (NICNAS) Priority Existing Chemical (PEC) reports.
- 9) US Agency for Toxic Substances and Disease Registry (ATSDR) documents.
- 10) National Toxicology Program (NTP) carcinogenicity appraisals.

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11) Organisation for Economic Co-operation and Development (OECD) Standard Information Data Sets (SIDS) and SID Initial Assessment Reports (SIAR).

12) USEPA Reference Doses.

Level 2 sources include peer-reviewed journals and industry publications and reference to Level 2 sources is considered warranted where Level 1 sources do not provide applicable criteria. Level 3 sources are other sources not covered in Levels 1 or 2. The use of Level 3 sources requires justification that no other data are available and that the appraisal presented meets the required level of conservatism as required.

This is presented in **Section 4** where additional discussion is also presented in relation to the application of occupational inhalation exposure standards and issues associated with the assessment of exposure to mixtures.

1.5.3 Exposure Assessment

This task draws on the evaluation undertaken as part of the "Issue Identification" stage and involves a detailed evaluation, identification and quantification (where required) of the potential exposure pathways and all significant population groups.

The exposure assessment is undertaken to be representative of a particular population and does not calculate the exposure for a given individual. Populations are grouped so as to reflect common activities undertaken by that group (such as workers or children) or by the location of the population in relation to the contaminant distribution. For this reason it is important that the exposure assessment be undertaken in such a way that the most sensitive individuals within the potentially exposed population are adequately protected. The exposure assessment has been structured in the following way:

- Identification of the population that may be exposed to COPC identified;
- Identification of the activities by which exposure may take place for each population;
- Identification of parameters which define activity (such as time spent indoors) and physiological exposure parameters (such as body weight and inhalation rate); and
- Identification of the chemical concentration at the point of exposure. This may include the identification and use of models to estimate chemical concentrations for receptors and exposure pathways that cannot be measured directly.

Key Pathways and Receptors

Receptor populations are similar groups of people who may work or use the site following completion of the development.

An exposure pathway describes a unique mechanism by which an individual or population may be exposed to chemicals or physical agents at or originating from a source. Each exposure pathway includes:

- a source or release from a source;
- a transport/exposure medium or exposure route; and
- an exposure point.

If any one of these mechanisms is missing (such as transport mechanism or exposure point) then the pathway is considered to be incomplete. An exposure pathway can be considered to be less significant if the potential for a receptor or population to be exposed to the COPCs identified is considered to be low. This may be due to a number of factors, which may include dilution during the transport from the source to the point of exposure or limited time for exposure.

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Quantification of Chemical Intake

When quantifying chemical intake or exposure, the risk assessment process focuses on exposure occurring over a prolonged period, that is chronic exposure that occurs over years and possibly a lifetime. Whilst an activity may occur infrequently (i.e. several days a year), it may occur regularly over a long period and therefore have the potential to increase long term or chronic intake of the chemical.

The assessment presented has addressed potential worst-case exposure to COPCs and exposure has been calculated for a **Reasonable Maximum Exposure (RME)** scenario estimated by using intake variables and chemical concentrations that define the highest exposure that is reasonably likely to occur in the area assessed. The RME is likely to provide a conservative or overestimate of total exposure and therefore health risk. This approach follows guidance from enHealth (2004) and NEPC (1999), supplemented by USEPA guidance (USEPA 1989).

The following steps have been followed to estimate chemical intake:

- Identification of **exposure parameters** for each of the identified exposure pathways and receptors. These are values that describe the physical and behavioural parameters relevant to the potentially exposed population and the pathway of exposure. Some examples include ingestion rate (e.g. amount of soil or dust ingested at work each day), inhalation rate (volume of air inhaled during different activities), exposure frequency (i.e. hours per day or days per year), exposure duration (e.g. number of years at work) and body weight. Where available, exposure parameters have been obtained from Australian sources (enHealth 2002, CSMS, 1991, 1993, 1996 and 1998, and NEPC 1999).
- Calculation of **intake factors**. An intake factor is calculated using the exposure parameters defined above and provides a site specific and receptor specific value which, when multiplied by the concentration of each COPC, provides an estimate of the daily chemical intake of the COPCs for each receptor and pathway.
- Estimation of the **chemical concentration** in each medium relevant to the receptor groups and exposure pathways. This involves the estimation of potential concentrations in air and other media such as soil and groundwater (where relevant to the assessment of a complete exposure pathway) and
- Calculation of the **daily chemical intake** using the intake factor and the chemical concentration.

Assumptions and calculations relevant to the quantification of chemical intake are presented within the assessment.

The exposure assessment is incorporated with the risk characterisation (refer to **Section 1.5.4**) assessment and is presented in detail in **Section 5** of this report.

1.5.4 Risk Characterisation

This task, presented in **Section 5**, provides either a qualitative or quantitative (as required) evaluation of potential risks to human health. The characterisation of risk draws on the “exposure assessment” and “hazard assessment”. The determination of potential health impacts will be evaluated on the basis of commonly accepted measures of acceptable risk and discussion on potential implications. The risk characterisation will draw on the data presented in the preceding sections and provide an assessment chronic risks associated with potential exposure to COPC identified on the site.

Hazard Index for Threshold Effects

The potential for adverse threshold effects, resulting from exposure to the COPC identified has been evaluated by comparing an exposure level, expressed as a daily chemical intake, with the adjusted ADI or equivalent threshold value (tolerable daily intake (TDI), RfD or Time Weighted Average (TWA)). The resulting ratio is referred to by the USEPA as the hazard quotient (USEPA, 1989) and is derived in the following manner:

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$$\text{Hazard Quotient} = \frac{(\text{Daily Intake})}{(\text{ADI}) - (\text{Background Intake})}$$

The evaluation of risk associated with threshold chemicals involves a comparison of the total daily intake with an adjusted ADI (or equivalent threshold value). The adjusted ADI is that which has been adjusted for background intake from all other sources (taken to refer to typical background sources such as food, water and air that would be the same in all areas of Australia) so that the hazard quotient calculated compares the chemical intake derived from the site with the ADI allowable from sources other than background. If the total daily chemical intake exceeds the adjusted ADI, TDI, RfD or TWA (i.e. if the hazard quotient exceeds one), then this would indicate potentially unacceptable chemical intakes. The hazard quotient does not represent a statistical probability of an effect occurring.

To assess the overall potential for adverse health effects posed by simultaneous exposure to multiple chemicals, the hazard quotients for each chemical and exposure pathway have been summed. The resulting sum is referred to by the USEPA as the hazard index (HI) (USEPA, 1989). The HI approach assumes that multiple sub-threshold exposures to several chemicals could result in a cumulative adverse health effect, and exposures are summed over all intake routes.

If the HI is less than one, cumulative exposure to the site chemicals is judged unlikely to result in an adverse effect. If the index is greater than one, a more detailed and critical evaluation of the risks (including consideration of specific target organs affected and mechanisms of toxic action of the chemicals of concern) would be required to ascertain if the cumulative exposure would in fact be likely to harm exposed individuals.

Risk for Non-Threshold Effects

The potential for unacceptable non-threshold carcinogenic risks associated with exposure to COPC has been evaluated using US EPA methodology.

Non-threshold carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential non-threshold carcinogen. The numerical estimate of excess lifetime cancer risk is calculated as follows:

$$\text{Carcinogenic Risk} = \text{Daily Chemical Intake} \bullet \text{Cancer Slope Factor}$$

The total non-threshold carcinogenic risk is the sum of the risk for each chemical for each pathway.

Deciding whether the calculated cancer risk is of concern or not requires identification of an acceptable cancer risk value. The calculation of a cancer risk implies that any exposure to these chemicals may result in an increased risk or probability of contracting cancer over a lifetime. The cancer risk value is expressed as a probability such as 1 in 10,000 (1×10^{-4}) or 1 in 1,000,000 (1×10^{-6}). At the simplest level these probability values can be converted to population risks as follows:

An incremental lifetime cancer risk of 1×10^{-6} , means that in a population of 1 million people which has been exposed to the chemical for their lifetime one additional cancer is predicted over and above the background incidence of cancer in that population (1 million people). For the same population a cancer risk of 1×10^{-4} implies that 100 additional cancers are predicted over and above the background incidence (for 1 million people).

These values are extremely low when compared to the background incidence of cancer in our society. The background incidence is in the order of 1 in 4 to 1 in 3 (Fitzgerald, in CSMS 1993). This means that for a population of 1,000,000 around 250,000 individuals are expected to contract cancer over a lifetime. An additional 1×10^{-6} , risk predicts 1 additional individual may develop cancer.

Specific Australian guidance related to the significance of cancer risk estimates is not currently available. Current US EPA policy states that: "Where the cumulative site risk to an individual based on reasonable maximum exposure for both current and future land use is less than 10^{-4} ,...action is generally not warranted unless there are adverse environmental impacts" (US EPA, 1991). If risks are found to be

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greater than the 10^{-4} probability, then the US EPA recommends that a preliminary remediation goal of 10^{-6} cancer risk be developed as the point of departure (ibid).

A review of the origins of the 10^{-6} cancer risk number has been undertaken by Kelly (1991) and a review of the development of an Australian approach to the assessment of carcinogenic contaminants has been prepared for discussion by Fitzgerald (in CSMS, 1993). Both these reviews indicate that the 10^{-6} was suggested by the United States Food and Drug Authority (USFDA) in 1961, as representing the *de minimis* legal risk. That is, the level of risk that can be identified, in a legal sense, as being representative of negligible or trivial risk. As the more recent US EPA policy (quoted above) indicates, the application of cancer risks has seen the acceptance of higher risk values i.e. 10^{-4} or 1 in 10,000 in the assessment of contaminated sites.

The application of cancer risk values in Australia and elsewhere is generally consistent with the US EPA policy. That is, the 10^{-6} risk value is commonly identified as the point of departure from negligible risk and the 10^{-4} risk value is commonly adopted as indicative of unacceptable risks. The 10^{-6} risk value is sometimes used as the basis for defining ambient standards applicable to wide scale population exposure. For example, the NHMRC and the Agricultural and Resources Management Council of Australia and New Zealand (NHMRC/ARMCANZ 2004) have used the 10^{-6} value for the derivation of the Australian drinking water guidelines for genotoxic carcinogens. The WHO, on the other hand, have used the 10^{-5} risk as the basis for the derivation of the WHO drinking water guidelines (WHO 2004) and the Dutch use the 10^{-4} lifetime cancer risk as the basis for the derivation of human Intervention Values for soil and groundwater for genotoxic carcinogens.

Adopted Risk Targets

Based on the above discussion URS consider that the following guidance with respect to incremental lifetime cancer risks is representative of current practice in NSW:

- Calculated incremental risks below 1×10^{-6} would be considered to be effectively zero;
- Calculated incremental risks between 1×10^{-6} and 1×10^{-5} would be considered acceptable; and
- Calculated risks greater than 1×10^{-4} would be considered to warrant some form of action or management to reduce the risk.

Where risks fall between 1×10^{-5} and 1×10^{-4} , then this may warrant further evaluation of the risks to determine whether action is required to reduce the risks.

URS have adopted a Target Risk value of 1×10^{-5} as indicating conditions that would warrant further assessment. Risks values below 1×10^{-5} are representative of acceptable risks.

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Environmental Setting

2.1 Introduction

This section presents a summary of the environmental setting of Southlands. The information presented has been drawn from previous investigations as summarised in the RAP.

The site is located approximately 10 km south east of the Sydney CBD and approximately 4 km east of Sydney Airport. Access to the site is from McPherson Street. The total area of the site is 182,880 m² (18.2 ha). The site is located in a predominantly industrial / commercial area. Surrounding businesses include a range of industrial and commercial premises including:

- The Sydenham to Botany rail way line is located beyond the eastern boundary of Block 1;
- The Botany Industrial Park (BIP), separated by the Sydenham - Botany Goods Railway Corridor, is located to the north and north east of the site. Several chemical manufacturing businesses, including Orica Australia Pty Ltd, have manufacturing and distribution facilities in BIP;
- A Qenos Pty Ltd (Qenos) owned, Mobil leased tank farm facility, known as *The Nant Street Tank Farm* is located immediately north of Block 1 Southlands that has been historically used to store hydrocarbons;
- The Mobil Botany Oil Distribution Terminal is located immediately to the north of Southlands Block 2;
- A chemical manufacturing facility owned by Solvay Interox Pty Ltd (Solvay) is located to the west of the site, separated by Floodvale Drain. Beyond Solvay, is an industrial estate; and
- McPherson Street forms the southern boundary to Southlands. Beyond McPherson Street, there is a shipping container storage facility owned by Maritime Container Services (MCS) Pty Ltd. A warehouse owned by Gazal Corporation, an apparel manufacturer, is located to the south of Block 2.

2.2 Topography

The local area is located on an area of former sand dunes and coastal swamps within the Botany Basin. The local topography generally slopes down to the south west towards Botany Bay and Penrhyn Estuary. A relative high point of 20 mAHD is located at the eastern side of BIP with elevations of less than 4 mAHD at and to the south of Southlands (Woodward-Clyde, 1996a).

The Southlands site forms part of an extensive low lying (< 4 mAHD) area to the west of BIP, which was referred to as the Veterans Swamp prior to construction of the Springvale and Floodvale Drains during the 1940s (Woodward-Clyde, 1996a). The drains, which were designed to assist in the drainage of the swamp, discharge into Botany Bay via Penrhyn Estuary. Penrhyn Estuary was formed by reclamation during the construction of the Port Botany Container Terminal area in the 1970's. **Figure 1** shows the locations and orientations of the drains.

The surface of Southlands is generally flat, sloping slightly down towards Springvale and Floodvale Drains and MacPherson Street. However, the heavily vegetated stockpiles have resulted in an uneven or "hummocky" surface across much of the site.

2.3 Geology

2.3.1 Regional Geology

In general, the site and surrounding areas are underlain by the Botany Sands, a sequence of predominantly unconsolidated to semi-consolidated permeable sands. These are interspersed with lenses and layers of peat, peaty sands, silts and clay which become more common in the lower part of the sequence. The sand sequence which is generally 30 to 60 m thick is underlain by sandstone rock (Hawkesbury Sandstone) which has a very low permeability compared to the sand deposits. Extensive peat layers occur at or close to the surface throughout Southlands and the adjoining low lying areas, and

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Environmental Setting

again at approximately 8 to 10 m below ground surface. Peat layers have also been noted in many shallow foundation boreholes drilled over wide areas of the site

2.3.2 Site Geology

A detailed description of the site geology is presented by Woodward-Clyde (1996a) and HLA (2005) and is summarised as follows:

Table 2-1 Generalised Soil Profile for Southlands

Approximate Depth	Soil Type
0 – 0.7m to 3.6m	<p>Block 1: FILL: predominantly silty or clayey sand fill noted to be slightly moist, grey brown and containing boiler ash (in most areas and more prevalent on northern portion) , slag, brick, metal pipes, timber and sandstone with smaller areas of clay and sand materials.</p> <p>Block 2: FILL: comprises mainly clayey material underlain by bricks, concrete, rubble, steel, wood and a variety of other materials. Some ash materials were noted in the central portion of Block 2.</p>
0.7m to 3.6m – 10m	SAND: (natural) grey/brown, medium to high porosity, fine to medium grain, loose. Minor quantities of peat were noted in Block 1

2.4 Hydrogeology

2.4.1 Regional Hydrogeology

In general, the Botany Sands contain a system of unconfined and semi-confined aquifers that are referred to as the Botany aquifer. Water table gradients indicate that groundwater flows predominantly in a westerly and southwesterly direction under the Southlands area towards and then into Botany Bay. The main recharge areas are in the higher sandy country to the north and east of the site. There is evidence of temporary groundwater flow direction changes from the late 1960s until the late 1980s due to a combination of heavy pumping to the north of the site and two periods of below average rainfall.

The Botany aquifer was one of the early sources of water for Sydney and an important source of industrial water in the Botany area. Previous work indicates that there is considerable variation in the groundwater yield of the sand, suggesting that there are discrete high yielding layers, or aquifers, within the sequence. These layers are interconnected vertically via leakage through the confining peat and clay layers and laterally by the discontinuous geometry of most of the confining units.

A review of information provided on The NSW Department of Natural Resources (DNR) web site¹ indicates that the site is located within one of four '*management zones*' prescribed by the DNR. This zone (Zone 1) is the Groundwater Extraction Exclusion Area (GEEA) around the Orica site and the use of groundwater from this zone is banned by the DNR. The DNR website identifies the Orica Botany Groundwater website² as a source of further information on the history and nature of groundwater contamination in the area.

¹ http://www.naturalresources.nsw.gov.au/water/botany_bay.shtml

² <http://www.oricabotanygroundwater.com/>

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2.4.2 Site Hydrogeology

The following hydrogeological details relevant to Southlands have been noted from Woodward-Clyde (1996a):

- The shallow, intermediate and deep groundwater aquifers flow in a south-westerly direction beneath Southlands towards Botany Bay;
- The shallow aquifer flow is affected by Springvale and, to a lesser extent, Floodvale Drains. Springvale Drain intersects and acts as a collector of shallow groundwater; and
- The depth to the shallow aquifer is variable, however it has been noted to be typically around 0.5 to 1.5m below ground surface (m bgs) on Block 1 and 1.5 to 2.5m bgs on Block 2.

2.4.3 Receiving Waters

Receiving waters on and in the vicinity of Southlands include the following:

- Springvale Drain, which bisects the site;
- Floodvale Drain, located immediately west of the site; and
- Penrhyn Estuary (Botany Bay) located 0.5 km south of the site to which both Springvale and Floodvale Drain discharge.

2.5 Southlands History

The following presents a summary (from the RAP) of significant activities relevant to Southlands and the identification of areas of potential contamination:

- Limited anecdotal and some aerial photographic evidence suggest the earliest uses of the land included some form of intensive agriculture, probably market gardening. The land (both Blocks 1 and 2) was also subject to sand and peat mining in the 1960s.
- Australian Paper Manufacturers (APM) owned Southlands prior to Orica's acquisition of the land in 1980. It appears the land was used by APM for storage of paper waste bails and anecdotal evidence suggests paper wastes were disposed of into the excavations that remained following the earlier mining activities, resulting in the shallow, periodically charged depressions known as The Paper Waste Ponds on Block 1.
- The Stage 2 Survey (Woodward-Clyde, 1996a) suggests that APM used mercury based fungicides and sulphites in the paper manufacturing processes. Wet solid wastes were reportedly removed from the site by truck and pipeline, however the route or discharge point of this pipeline is not known.
- The Southlands site, as well as adjoining properties to the south of McPherson Street and the northern portion of BIP, have been extensively filled; primarily with ash from coal-fired boilers from local industries including the Bunnerong Power Station (HLA, 2005). Based on field observations, it is likely that other materials such as building rubble and demolition wastes were also used in the filling process.
- Fill thickness has generally been reported to be between 0.75 m and 2.5 m across the site, but has been measured in parts at thicknesses of up to 4m (Woodward-Clyde, 1996a).
- Prior to erection of a fence around the Southlands perimeter by Orica in the early 1990s, Block 2 and to a lesser extent Block 1, were subject to ad hoc illegal dumping of rubbish, predominantly demolition wastes, which remain on site. The stockpiles have been heavily overgrown with vegetation.

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- The Stage 2 Investigation report (Woodward-Clyde, 1996a) identified that, while recycling and recovery of effluents derived from processes on the ICI site (now BIP) had progressively improved throughout the site's manufacturing history, information provided by ICI had suggested that prior to 1958, effluent disposal was via a stormwater pipe directly into Springvale Drain.
- Groundwater contaminated with chlorinated hydrocarbons (CHCs) derived from historical operations at the BIP has been identified beneath both Blocks 1 and 2 of Southlands. The groundwater contamination dates back over a period up to 50 years.

Review of the history of Southlands and surrounding areas by HLA (2005) identified a range of activities that may have resulted in the contamination of soil and/or groundwater on the site. These include the following:

- APM Waste Disposal - Historical information suggest that paper waste slurries containing mercury-based fungicides may have been directly discharged into some of the ponds and pits predominately on Block 1.
- ICI Waste Disposal – Historical information and previous investigations indicate that in the past effluent disposal from the ICI Botany Plant was discharged directly to Springvale Drain via a stormwater pipe. Prior to the installation of the stormwater pipe, some of the wastewater may have flowed over parts of Block 1.
- Uncontrolled Disposal – Up until 1991 the site has been used for uncontrolled dumping of construction debris and fill materials, which are evident by the numerous stockpiles across both Block 1 and Block 2.
- Jet Fuel Pipes - The jet fuel pipelines are a potential source of contamination, if leakage were to occur.
- Diesel Spill - The historical diesel spill from the former SRA diesel tank, located immediately north-east of Block 1.
- Contaminated Water Disposal - Based on anecdotal information, contaminated groundwater from Energy Australia pits were pumped onto the surface of an area on the north-eastern portion of the site. The pits are understood to have been located to the north-east of the site
- Contaminated Groundwater - Extensive investigations of groundwater conducted in and around the Orica Botany site have identified that groundwater is contaminated with volatile and semi-volatile CHCs. Woodward-Clyde (1996a) reported that free-phase DNAPL was present beneath the northern portion of Block 1 (Stage 3, Woodward-Clyde 1997) and dissolved phase CHCs were present within the shallow, intermediate and deep aquifers beneath Southlands.

2.6 Orica Groundwater Treatment Program

In September 2003 (subsequently amended and consolidated in February 2004) the Department of Environment and Conservation (DEC) (now NSW Department of Environment and Climate Change (DECC)) issued a Notice of Clean Up Action (NCUA) No. 1030236 to Orica Australia Pty Ltd (Orica) requiring remediation of the chlorinated hydrocarbon contaminant plumes emanating from the Botany Industrial Park (BIP) and present in areas to the south and west of BIP.

The NCUA also required the preparation and implementation of a more extensive groundwater monitoring program compared to that which was being undertaken at that time. The expanded monitoring program was documented in the Groundwater Cleanup Plan (GCP, Orica 2003) and included the installation of new monitoring wells/bundle piezometers and sampling and reporting of analytical data on a quarterly basis. This program is regularly reviewed and refined in consultation with the DECC, NSW.

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2.6.1 Groundwater Cleanup Plan (Orica 2003)

The GCP documented the proposed strategies adopted to address groundwater contamination at BIP and downgradient of BIP. The strategy selected to achieve hydraulic containment of groundwater contamination was described in the Botany Groundwater Cleanup (BGC) Project Environmental Impact Statement (EIS) (URS, 2004b) and comprised the installation and operation of three hydraulic containment lines (Primary, Secondary and DNAPL/BIP) and construction of a groundwater treatment plant (GTP) to enable treatment of groundwater extracted to achieve hydraulic containment.

The Joint Determining Authority Report (DEC 2005) for the BGC Project EIS included conditions of approval related to groundwater and surface water monitoring. The approval conditions included a number of specific objectives including:

- Monitoring of aquifer water levels (hydraulic monitoring);
- Monitoring of contaminants of concern in groundwater; and
- Monitoring of contaminants of concern in surface water.

To address the conditions of approval, the Groundwater Treatment Plant (GTP) – Groundwater and Surface Water Monitoring Program (URS 2005b) was developed and submitted to DEC. The GTP Monitoring Program superseded the previous GCP Monitoring Program (Orica 2003).

2.6.2 Quarterly Groundwater and Surface Water Monitoring

Quarterly groundwater and surface water monitoring programs have been completed and reported by Orica to the DECC under the NCUA since March 2004. The existing monitoring well network includes a number of locations that are relevant to Southlands.

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3.1 Introduction

Review of previous investigations and available data indicate that a number of contaminants are present in soil, groundwater, surface water, sediment and air within and surrounding the Southlands property. This section presents a review of available data with the aim of defining the known nature and extent of contamination and identifying issues that are relevant to the quantification of risks to human health in relation to the proposed development. In addition the review aims to identify chemicals of potential concern (COPC) that require detailed evaluation within the HHRA. The assessment undertaken has built on the key issues identified in the PHHRA (URS, 2006c, refer to **Appendix H**) as well as taking into account the details of the proposed development as at the end of September 2007.

While the HHRA aims to focus on risks to human health associated with the Stage 1 and 2 developments, the available data has been reviewed on the basis of proximity within the Southlands site, namely Block 2 (Stage 1), Block 1 (Stage 2 and proposed Stage 3) and other key features such as Springvale and Floodvale Drains (relevant to both Stage 1 and Stage 2).

3.2 Screening Level Guidelines Adopted for Identification of COPC

Chemicals of potential concern (COPC) are those chemicals, which are known or suspected to be present at concentrations high enough to warrant inclusion in the assessment of risks to human health. The prime objective of identifying COPC is to focus the risk assessment on assessing chemicals that have the potential to significantly contribute to risks to human health. The identification of COPC is based on the assessment of the nature and extent of these chemicals in the environment. The following presents a summary of the guidelines used for the purpose of identifying COPC in soils, sediments, water and air.

It is noted that application of the adopted guidelines also requires some consideration of the potential for cumulative (additive) effects of mixtures, particularly those with similar health endpoints. Hence where relevant (particularly where the exposure concentration is close to the adopted guideline) the potential for cumulative effects has been considered in the identification of COPC.

3.2.1 Soil and Sediment

Soil and sediment concentrations from all areas of Southlands have been screened against the following guidelines to identify COPC for evaluation in the HHRA:

- **National Environment Protection Measure (NEPM)**, Assessment of Site Contamination, Schedule B1, Health Based Soil Investigation Levels, Level F - Industrial Sites (Table 5A) (enHealth 2001); and
- **NSW EPA Guidelines for Assessing Service Station Sites**, 1994, Table 3 - Human Health Levels for BTEX and TPH compounds in soils (no HILs defined in NEPM, 1999).

Where guidelines are not available from the above sources for specific chemicals, screening levels have been obtained from the following sources. These sources are from the United States and while not recognised in Australia, the values provide a screening level for a wide range of chemicals in soils for the purpose of identifying COPC:

- **Region IX Preliminary Remediation Goals, 2004**. US EPA Region IX Preliminary Remediation Goals (PRGs) are conservative risk-based values for soil, tap water and air. The values presented for industrial soil (considering exposure via ingestion, dermal contact, inhalation of particulates and inhalation of volatiles outdoors, based on target risk levels of 10^{-6} for carcinogenic effects and 1 for non-carcinogenic effects) have been considered within this assessment.

For some PAH compounds, no guidelines are available and hence soil screening levels have been adopted on the basis of considering other PAH compounds as surrogates. The surrogate compounds

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have been selected based on consideration of toxicity equivalent factors (TEFs) discussed and presented by Fitzgerald (in CSMS, 1998)³. These are noted where considered. Some other chemicals also have no data available and hence surrogate compounds have also been identified based on the similarity of the compounds and likelihood that the surrogate selected is more toxic than the chemical for which no data is available.

It is noted that the adopted screening levels are relevant for commercial/industrial use of the site, consistent with proposed development and land use of the site. Additional considerations with respect to vapour intrusion have been presented in **Section 3.5.2**.

3.2.2 Groundwater and Surface Water

The screening level guidelines adopted for the assessment of groundwater (where relevant) and surface water data are:

- **Australian Drinking Water Guidelines (ADWG), 2004.** The National Health and Medical Research Council (NHMRC) and the Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ) have developed the Australian Drinking Water Guidelines. The guidelines provide health-based and aesthetic values for a range of micro-organisms, physical quality, inorganic chemicals, organic chemicals, radiological quality and pesticides. The health-based guideline values, which have been used to identify COPC in the groundwater, are concentrations, which based on present knowledge, do not result in any significant risk to the health of a consumer of the water over a lifetime. These guidelines are recognised within the NEPM (Schedule B(6) Guideline on Risk Based Assessment of Groundwater Contamination, 1999) and EPHC⁴ as relevant Groundwater Investigation Levels (GILs) for the assessment of human health issues at the point of extraction (for use as drinking water – protection of human health issues associated with use of water as domestic supply within households⁵). This approach is conservative for the assessment of groundwater, as groundwater in the area is not used as a source of drinking water or as domestic supply within a residence. These guidelines are more current and extensive than the ANZECC 2000 Guidelines for Recreational Water Quality and Aesthetics (relevant to lower levels of exposure than drinking water) and hence have been used in preference to the recreational guidelines.
- **World Health Organisation Drinking Water Guidelines (WHO DWG), 2004 (and rolling revisions).** The WHO have also developed drinking water guidelines using the same approach as in the ADWG. The health-based guideline values, which have been used to identify COPC in the groundwater, are concentrations, which based on present knowledge, do not result in any significant risk to the health of a consumer of the water over a lifetime.
- **Region IX Preliminary Remediation Goals (PRGs), 2004.** Where no guideline value was available from the above sources, the US EPA Region IX Preliminary Remediation Goals (PRGs) have been

³ Fitzgerald J., 1998. *The benchmark dose approach and health-based investigation level for polycyclic aromatic hydrocarbons (PAHs)*, presented in Contaminated Sites Monograph Series, No. 7, 1998.

⁴ National Chemical Reference Guide, Australian Department of the Environment and Heritage, Environment Protection and Heritage Council. Supporting documentation and online database available at:
http://hermes.erin.gov.au/pls/crg_public/!CRGPPUBLIC.pStart

⁵ Australian Drinking Water Guidelines (NHMRC, 2004) provide guideline values for water that are considered to be safe for “*human consumption, either directly, as supplied from the tap, or indirectly, in beverages, ice or foods prepared with water. Drinking water is also used for other domestic purposes such as bathing and showering*”. The guidelines apply to any water intended for drinking irrespective of the source (municipal supplies, rainwater tanks, groundwater bores etc.). The methodology used to derive the guidelines allows for exposures other than ingestion (dermal contact and inhalation including inhalation of volatiles during activities such as showering in heated water). Hence the guidelines are considered relevant for the assessment of pathways of exposure that may be associated with use of groundwater.

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used. The PRGs are conservative risk-based values for soil, tap water and air. It should be noted that the PRGs are currently not recognised in Australia. However, the PRGs have been used in this assessment to provide a screening level for the purpose of identifying the COPC within groundwater for further assessment.

3.2.3 Ambient Air

Numerous guidelines exist for VOCs in air. Some guidelines are designed to protect all individuals from chronic or long term exposure, whilst others are designed to protect workers during an 8 hour working day. For the purpose of this assessment, the focus is on long term exposure by workers at the site and the general public who may visit the site. These exposures are anticipated to be associated with chemicals not expected to be used or stored in the specific work place and as such, site workers may be unaware of the presence of these chemicals. Hence guidelines relevant to the assessment of inhalation exposures by the general public have also been considered relevant for the purpose of screening concentrations for workers in place of occupational exposure standards.

The guidelines relevant for VOCs in ambient air are contained in the National Environment Protection Measure (NEPM) Air Investigation Levels (NEPC, 2004), however, these investigation levels are limited with respect to the specific compounds included. Where a NEPM investigation level was not available for individual VOCs detected, reference has been made in the first instance to World Health Organisation (WHO) ambient air guidelines and then the US EPA Region IX (2004) Preliminary Remediation Goals (PRG) for ambient air followed by alternate US guidelines such as those established by the Californian EPA. All of the air guidelines adopted for screening have been derived to be protective of health effects associated with inhalation exposures by all members of the public on a daily basis for a lifetime. A discussion of these guidelines is provided below.

NEPC Air Toxics Measure

In June 1998, the National Environment Protection Council (NEPC) released a National Environment Protection Measure (NEPM) for Ambient Air Quality, setting out national standards and investigation levels for criteria pollutants. In addition, air investigation levels based on a number of averaging times for high priority VOCs were established in 2004. It should be noted however, that these guidelines are designed for use as regional goals and are not intended to be used as near-source or site boundary criteria from operating facilities with known air emissions.

WHO Air Quality Guidelines

Where individual VOCs detected during the air sampling did not have NEPC Air guidelines, the WHO air quality guidelines (2000 and 2000b) have been referenced. In some cases air quality goals have been updated by the WHO in relevant review documents (available from the WHO). The WHO Air Quality Guidelines provides guidelines for non cancer effects and carcinogenic effects for a specific averaging time. The selection of the WHO guidelines following those published by the NEPC is in accordance with the NEPM guideline hierarchy.

US EPA Region IX Preliminary Remediation Goals

Where individual VOCs detected during the air sampling have neither NEPM guidelines or WHO guidelines, US EPA Region IX (2004) Preliminary Remediation Goals (PRG) values have been used. The PRGs are conservative human health risk-based values for soil, tap water and ambient air. The values available for air represent concentrations that the general public may inhale every day for a lifetime without adverse health effects. It should be noted that the PRGs are currently not endorsed in Australia, however, the PRGs have been used in this assessment to provide a screening level for the purpose of identifying the presence of any individual VOCs in air that may be of concern with respect to human health.

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Californian Office of Environmental Health Hazard Assessment (OEHHA)

The Californian Office of Environmental Health Hazard Assessment (COEHHA⁶) Chronic and Acute Reference levels have been adopted in the absence of NEPM, WHO, USEPA and TNRCC sources.

Use of Air Criteria

For the purpose of identifying COPC an annual average has been adopted. It is noted that the NEPM investigation annual average levels consist of the mean of 24-hour data collected from monitoring locations and hence use of the annual average levels is considered relevant in comparison with the mean or average 24-hour concentrations. For samples collected over a shorter period of time such as an 8 hour period, the data collected over the 8 hours (considered representative of a workday) has been assumed to be representative of concentrations that may be present over the whole day. This approach has been adopted for consideration of other guidelines that are presented as annual averages such that they can be directly compared against the air samples collected. As a limited amount of data has been collected, the ambient air concentrations have not been averaged, with each concentration and the maximum reported compared directly against the annual average screening criteria. This provides a conservative (i.e. overly protective of human health) approach to screening the ambient air data.

3.3 Surface Water

Surface water features within the Southlands property that require further assessment include Springvale Drain, Floodvale Drain and the ponds located on Block 1. It is noted that the data reviewed for these areas relates to shallow groundwater as shallow groundwater, particularly on Block 1, interacts with the existing ponds as well as Springvale Drain. Hence the review presented in this section also draws on shallow groundwater data where relevant.

3.3.1 Springvale and Floodvale Drains

Springvale Drain, and to a lesser extent Floodvale Drain, have in addition to the original purpose of draining the swamps, been historically used for the purpose of waste disposal resulting in the discharge of a range of contaminants from the former ICI operations that included mercury and CHCs. In addition, the shallow groundwater aquifer is intersected by Springvale Drain, and to a lesser extent by Floodvale Drain, resulting in the discharge of CHCs present within the shallow aquifer into the drains. Both drains are also impacted by surface water runoff and discharges derived from a range of other industries surrounding and up gradient from the Southlands site. Hence the surface water quality within Springvale and Floodvale Drains has and continues to be affected by a range of contaminants derived from shallow groundwater as well as other sources/industries that contribute to surface water runoff or discharge.

Surface water within Springvale and Floodvale Drains discharge directly into Penrhyn Estuary. Risks to human health associated with drain water quality downstream from Southlands including Penrhyn Estuary has been conducted as part of the Consolidated HHRA (URS, 2005a) and subsequent summary report (URS 2007b). Hence the focus of the Southlands HHRA is the risks to human health within the Southlands property only for the proposed development.

As discussed in the Exposure Assessment (**Section 5**), the only potentially significant exposure pathway associated with the presence of contamination in surface water in the drains that is considered complete is the inhalation of vapours from volatile chemicals that may be present in the surface water within the drains. Vapours (associated with the COPC identified) may disperse into adjacent areas (outdoors) and into buildings. Hence the identification of COPC considered relevant to the presence of impacts in Springvale and Floodvale Drains and the inhalation pathway have been identified using air data collected adjacent to the surface water of both these drains (refer to **Section 3.8**). The surface water data has not

⁶ Refer to <http://www.oehha.ca.gov/air.html>

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been further assessed or screened with respect to potential exposure via direct contact with drain water within the Southlands property (refer to **Section 5**).

Further Review of Springvale Drain

The calculation of risk presented in the PHHRA (URS, 2006c, refer to **Appendix H**) identified emissions to air from Springvale Drain as an issue that requires risk management or remediation as part of the development. While consistent operation of the GTP containment system results in drawdown of the shallow aquifer to at or below Springvale Drain level on Southlands, groundwater extraction from BIP and PCA is insufficient to maintain these levels during and following rainfall events and GTP shutdowns (URS, 2008).

As a consequence, in order to address discharge of shallow groundwater into Springvale Drain a shallow groundwater extraction system along the eastern side of the drain has been proposed (refer to the RAP for further detail on the proposed system and potential backup systems). Discharge from the containment line is proposed to be connected into the existing GTP (or other backup system). It is noted, however that as part of the maintenance program the GTP needs to be offline during short periods. The period of time that the GTP will be offline does not affect containment of the groundwater plumes, however it has the potential to cause groundwater levels in the vicinity of the drain to rise and shallow groundwater may discharge into the drain (as per current conditions) resulting in increased vapour emissions from the drain.

While Orica is in the process of identifying and evaluating appropriate backup systems to address the operation of containment lines when the GTP is not operating, the potential risk issues associated with down time of the GTP (with no backup system operating) have therefore been considered. COPC have been identified following review of current groundwater concentrations by URS (refer to **Appendix A**) in the vicinity of Block 1 relevant to the assessment of current concentrations in the drain. Based on the available data the average⁷ groundwater concentration reported (in the data set identified by URS) in wells screened from 2-8m bgs and those installed as shallow (S) and intermediate (I) wells has been assumed to be representative of concentrations that may discharge to the drain at some point should the GTP plant be offline⁸. The use of groundwater data is conservative as this assumes that no dilution takes place in the drain after discharge.

Surface water data from a period when the GTP was not operating has been reviewed for comparative purposes. Data from March 2004 to December 2006 have been reviewed with the average (detected) and maximum concentrations identified.

The average groundwater concentration that may discharge into the drain for each analyte reported has been screened against relevant human health based screening level guidelines (as presented in **Section 3.2.2**) such that key chemicals, or chemicals of potential concern (COPC) can be identified.

Table 3-1 presents a summary of the analytes detected, concentrations in surface water and groundwater that are considered relevant to concentrations that may be present in the drain during down time of the GTP, the relevant screening level guideline adopted and the COPC identified (shaded rows).

There are limitations to the approach used to identify COPC in the drain if the GTP is offline. There may be a range of issues that arise from the redevelopment of Southlands (such as filling in the realignment channel and constructing compensatory flood storage areas) that affect the interaction of shallow

⁷ Arithmetic average of concentrations reported. Where a concentration was not reported above the analytical limit of reporting, the reporting limit has been used in the calculation.

⁸ The groundwater/surface water interaction relevant to the assessment of Springvale Drain is presented in further detail in the Stage 2 Report (Woodward-Clyde, 1996). In particular a conceptual model of the effect of the drain on the water table elevation within Southlands is presented that suggests shallow groundwater to a depth of -4.0 m ADH, or approximately 7m bgs interacts with the drain and potentially discharges as seeps into the drain.

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groundwater with the drain. These effects cannot be predicted. In addition, in general, sampling of surface water from the drain has not aimed to target days when maximum concentrations may be expected.

Appendix A presents further detail on the groundwater concentrations considered in this assessment.

Table 3-1 COPC Identified for Assessment of Potential Issues in Springvale Drain (GTP Offline)

Chemicals Detected (COPC highlighted in shaded rows)	Concentration in Surface Water ¹ (mg/L)		Concentration in Shallow Groundwater ² (mg/L)	Screening Level Guideline ³ (mg/L)
	Average	Maximum		
carbon tetrachloride	0.78	5.1	17.8	0.003 (ADWG)
chloroform	0.74	4.5	4.8	0.25 (ADWG)
methylene chloride	0.021	0.027	0.14	0.004 (ADWG)
1,1,2,2-tetrachloroethane	0.069	0.311	0.35	0.37 (PRG) ^{T,C}
1,1,1-trichloroethane	Only detected once	0.182	0.057	2 (WHO)
1,1,2-trichloroethane	0.13	0.42	0.92	0.024 (PRG) ^T
1,1-dichloroethane	0.071	0.287	0.15	0.81 (PRG)
1,2-dichloroethane (EDC)	24.7	83.1	34.9	0.003 (ADWG)
chloroethane	Not reported	Not reported	0.089	8.6 (PRG) ^T
tetrachloroethene (PCE)	1.8	10.5	13.3	0.05 (ADWG)
trichloroethene (TCE)	0.87	6.6	3.3	0.02 (WHO)
1,1-dichloroethene	0.063	0.43	0.15	0.03 (ADWG)
cis-1,2-dichloroethene	0.60	2.7	4.3	0.06 (ADWG)
trans-1,2-dichloroethene	0.036	0.18	0.38	0.06 (ADWG)
vinyl chloride	1.9	7.6	11.5	0.0003 (ADWG)
1,2-dichlorobenzene	Not detected in limited analysis		0.013	1.5 (ADWG)
1,3-dichlorobenzene	Not detected in limited analysis		0.013	0.04 (ADWG for 1,4-dichlorobenzene)
1,4-dichlorobenzene	Not detected in limited analysis		0.014	0.04 (ADWG)
hexachlorobutadiene (HCBd)	0.0045	0.006	0.065	0.0007 (ADWG)
hexachloroethane (HCE)	Only detected once	0.003	0.099	0.036 (PRG) ^T
1,3,5-trichlorobenzene	Not detected in limited analysis		0.0025	0.03 (ADWG)

Notes:

- T – PRG based on threshold approach as chemical not considered genotoxic carcinogen (refer to toxicity summaries in **Appendix B** and IARC evaluations).
- C – Maximum concentration reported in water close to the adopted guideline. Based on information available about the chemical, there is the potential for cumulative risks to be of concern as the health endpoints are similar to a range of other chlorinated compounds also identified as COPC (refer to toxicity summaries in **Appendix B**). On this basis this chemical has also been selected as a COPC for further assessment. Note that the assessment of inhalation exposures adjacent to Springvale Drain has been based on measured air data where this compound was not reported above the LOR, therefore it is not considered a COPC in air adjacent to the drain.
- 1 Concentrations reported in Springvale Drain at locations SW046, SW049 and SW005 from data collected from March 2004 to December 2005. Concentrations reported based on detected concentrations as an average and maximum during the period of time considered (chemicals not detected above LOR were not considered in calculation of average).
- 2 Concentrations in shallow groundwater that may discharge into Springvale Drain calculated as average concentration from wells screened at depths of 2-8m and shallow and intermediate wells (refer to **Appendix A**).
- 3 Screening level guidelines adopted based on drinking water guidelines (refer to **Section 3.2.2**)

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3.3.2 Surface Water in Ponds on Block 1

A number of ponds are present within Block 1. Four of the ponds are deep enough to be permanent features as they extend below the level of water table, while the remaining ponds are ephemeral in nature and include some shallow surface drainage lines. Contaminants identified within the surface water of the ponds include some metals and CHCs (in particular vinyl chloride (VC), 1,2-dichloroethane (EDC), trichloroethene (TCE), tetrachloroethene (PCE), cis-1,2-dichloroethene (cis-1,2-DCE), chloroform and hexachlorobutadiene (HCBDD)). As the ponds were originally associated with paper pulp wastes, there is the potential for the water in the ponds to be odorous. Shallow groundwater through Southlands is also a source of odour in the ponds that intersect groundwater.

The PHRA (URS, 2006c, refer to **Appendix H**) identified the potential for elevated exposures to contaminants in surface water and sediments by workers involved in the maintenance of the ponds in these areas, should they remain after development. However, the proposed development of Stage 1 requires the filling in of the ponds and installation of compensatory flood storage areas. Stages 2 and 3 require the filling of Block 1 to a level similar to that proposed for Block 2 in Stage 1. As such the existing ponds are to be filled in as part of the development removing the potential for exposure to surface water in the existing ponds. Hence the quality of water currently reported within the ponds is not considered to be representative of concentrations that will be present on the site following development and the data has not been further reviewed or assessed in relation risks to human health.

Issues relating to contaminants identified in sediments that may be moved across the site are addressed in **Section 3.6**.

3.3.3 Surface Water in Compensatory Flood Storage Area Proposed for Block 1

The Flood Management Design prepared by Connell Wagner has proposed the installation of a compensatory flood storage area on the southern portion of Block 1 included in the Stage 1 works. The compensatory flood storage area is currently proposed to extend to a depth below the shallow groundwater level. This has the potential to result in discharge of shallow groundwater into the compensatory flood storage area during rainfall events associated with elevated groundwater levels and when the GTP is offline (also associated with elevated groundwater levels). Exposures associated with the discharge of shallow groundwater (contaminated with chlorinated hydrocarbons) the basin will be greater than if no discharge occurs. Hence, further assessment of potential risk issues associated with the construction of the compensatory flood storage area is required.

While the potential for discharge and subsequent exposure is expected to be managed with the use of specific construction methods and subsurface drainage (refer to the RAP for details), groundwater concentrations that may seep into the compensatory flood storage area have, nonetheless, been reviewed to identify COPC.

A review of groundwater concentrations in the vicinity of Block 1 relevant to the quantification of current concentrations has been undertaken (refer to **Appendix A**). While limited data is available regarding the quality of groundwater in the top metre of the groundwater (which is more likely to discharge into a flood storage area), the average⁹ groundwater concentration reported (in the data set identified by URS) in wells screened from 2-3m and those installed as shallow (S) wells is considered representative of concentrations that may discharge to the proposed compensatory flood storage area.

The average shallow groundwater concentration for each analyte reported has been screened against relevant human health based screening level guidelines outlined in **Section 3.2.2** to identify COPC. These are chemicals that are present in groundwater that may discharge into the compensatory flood

⁹ Arithmetic average of concentrations reported. Where a concentration was not reported above the analytical limit of reporting, the reporting limit has been used in the calculation.

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storage area at concentrations high enough to contribute to the total risk and warrant detailed assessment. The screening level guidelines are drinking water guidelines. While the water that seeps into the compensatory flood storage area will not be used for drinking or potable water, no guidelines are available that specifically relate to inhalation exposures from surface water bodies. Drinking water guidelines consider ingestion of 2 litres of water every day for a lifetime, with other exposures (dermal contact and inhalation) accounted for in the derivation of the guidelines. The use of drinking water guidelines for this purpose is considered a conservative basis for identifying chemicals that warrant detailed assessment.

Table 3-2 presents a summary of the chemicals detected in shallow groundwater, average shallow groundwater concentrations, the relevant screening level guideline and the COPC identified (shaded in the table). **Appendix A** presents a summary of the groundwater data identified and considered relevant for shallow groundwater in this assessment.

Table 3-2 COPC and Concentrations for Assessment of Compensatory Flood Storage Area

Chemicals Detected (COPC highlighted in shaded rows)	Concentration in Shallow Groundwater ¹ (mg/L)	Screening Level Guideline ² (mg/L)
carbon tetrachloride	2.3	0.003 (ADWG)
chloroform	0.95	0.25 (ADWG)
methylene chloride	0.056	0.004 (ADWG)
1,1,2,2-tetrachloroethane	0.15	0.37 (PRG) ^T
1,1,1-trichloroethane	0.041	2 (WHO)
1,1,2-trichloroethane	0.21	0.024 (PRG) ^T
1,1-dichloroethane	0.085	0.81 (PRG)
1,2-dichloroethane (EDC)	9.9	0.003 (ADWG)
chloroethane	0.070	8.6 (PRG) ^T
tetrachloroethene (PCE)	1.8	0.05 (ADWG)
trichloroethene (TCE)	1.2	0.02 (WHO)
1,1-dichloroethene	0.056	0.03 (ADWG)
cis-1,2-dichloroethene	4.8	0.06 (ADWG)
trans-1,2-dichloroethene	0.54	0.06 (ADWG)
vinyl chloride	6.8	0.0003 (ADWG)
hexachlorobutadiene (HCBd)	0.036	0.0007 (ADWG)
1,2-dichlorobenzene	0.029	1.5 (ADWG)
1,3-dichlorobenzene	0.029	0.04 (ADWG for 1,4-dichlorobenzene)
1,4-dichlorobenzene	0.030	0.04 (ADWG)
hexachloroethane (HCE)	0.020	0.036 (PRG) ^T
1,3,5-trichlorobenzene	0.0046	0.03 (ADWG)

Notes:

T – PRG based on threshold approach as chemical not considered genotoxic carcinogen (refer to toxicity summaries in **Appendix B** and IARC evaluations)

1 Concentrations in shallow groundwater that may discharge into the compensatory flood storage area has been calculated as average concentration from wells screened at depths of 2-3m and shallow wells, refer to **Appendix A**.

2 Screening level guidelines adopted based on drinking water guidelines (refer to **Section 3.2.2**)

No additional COPC have been identified based on consideration of potential cumulative effects of mixtures.

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3.4 Groundwater

Impacted groundwater within the shallow, intermediate and deep aquifers is present beneath both Block 1 and Block 2 of the Southlands site. The main contaminants identified in groundwater are derived from the central plumes dominated by EDC, PCE, TCE and VC and the southern plumes dominated by CTC, PCE, TCE and VC, however a range of other chemicals have also been reported in groundwater that may be derived from former operations at the BIP as well as surrounding industrial areas. The Stage 2 Investigations (Woodward-Clyde, 1996a) also reported the presence of DNAPL (i.e. free phase) at depth within the northern portion of Block 1 (Stage 3) (URS, 2005c).

As discussed in the Exposure Assessment (**Section 5**), the most significant exposure pathway associated with the presence of contamination in groundwater that is considered complete is the inhalation of vapours from volatile chemicals that may be present at the surface of the groundwater (shallow aquifer), following vapour migration through overlying soils into overlying buildings, outdoor air and excavations. The identification of COPC considered relevant to the presence of groundwater contamination and inhalation has therefore been based on data from soil gas and surface flux emissions investigations undertaken above the area of contaminated shallow groundwater (refer to **Section 3.7**). However it should be noted that the assessment of exposures on the site has also included consideration of following issues:

- Exposures associated with the discharge of shallow groundwater into the proposed compensatory flood storage area to be constructed on the southern portion of Block 1. This is discussed further in **Section 3.3.3** where COPC in shallow groundwater have been identified for further assessment.
- Exposures associated with intrusive activities undertaken on the site to repair or replace subsurface services. It is expected that such works would be required to be undertaken in accordance with a site-specific management plan, however as groundwater is shallow, particularly on Block 1, there is the potential for direct contact with groundwater during such works. The shallow groundwater concentrations identified and considered for the compensatory flood storage area presented in **Section 3.3.3** are considered relevant to the assessment of issues that may be associated with works that directly intersect the shallow groundwater.

3.5 Soil

Soil data was collected by Woodward-Clyde (now URS) during the Stage 2 Survey in 1994 (reported in 1996a) and by HLA in 2005. Additional soil data was collected by URS in 2006 as part of further contaminant delineation works undertaken on the site. Based on the available data contaminated soil has been identified on Block 1 and to a lesser extent on Block 2. Contaminants identified include:

- Metals, in particular mercury and lead located in a few sampling locations on Block 1 (south western portion), mercury in soil at one sampling location on Block 2 and lead on one stockpile on Block 2;
- Petroleum hydrocarbons in the south western portion of Block 1;
- Volatile and semi-volatile CHCs identified in the south western portion of Block 1 and two sampling locations on Block 2;
- Volatile and semi-volatile CHCs identified in some stockpiles located on Block 1 and one stockpile located on Block 2; and
- Asbestos identified in two stockpiles on Block 1.

The available data, presented in **Appendix A**, have been reviewed with the aim of identifying COPC for further evaluation within the HHRA. This review has involved the following:

- Comparison of the maximum reported concentration of each analyte detected against human health risk based screening criteria outlined in **Section 3.2.1** to identify COPC;

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- Further assessment of issues related to vapour intrusion associated with localised areas of contamination; and
- Consideration of issues associated with the presence of asbestos in soil.

The above has been presented for both Block 1 and Block 2.

3.5.1 Identification of COPC in Soil

Tables 3-3 and 3-4 present a listing of the maximum concentrations of chemicals detected in soil on the site associated with Block 2 (Stage 1, Table 3-2) and Block 1 (Stages 2 and 3, Table 3-4) of the property based on all available data collected in 1994, 2005 and 2006 and comparison with the adopted screening levels. Only chemicals detected above the laboratory limit of reporting (LOR) in at least one sample have been considered as potential COPC. The sample locations are shown in Figure 4.

Table 3-3 Review of Soil Data and Identification of Chemicals of Potential Concern - Southlands Block 2 (Stage 1)

Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comment
arsenic	41	500 ¹	
cadmium	3	100 ¹	
chromium	114	60 ¹	Maximum detected in stockpile sample SP49, concentrations in stockpiles greater than soil samples
copper	436	5000 ¹	
lead	2220	1500 ¹	Maximum detected in stockpile sample SP50, concentrations in stockpiles greater than soil samples
nickel	101	3000 ¹	
zinc	2960	35000 ¹	
mercury	1233	75 ¹	Maximum reported at TP50, no other exceedances of criteria reported
TPH and MAH			
TPH C ₆ -C ₉	Nd	65 ²	TPH not assessed by Woodward Clyde. Exceedances of TPH identified in TP131_1.00
TPH C ₁₀ -C ₁₄	Nd	1000 ²	
TPH C ₁₅ -C ₂₈	250		
TPH C ₂₉ -C ₃₆	370		
Volatile Organic Compounds			
1,1-dichloroethene	0.07	413 ³	* Refer to notes below
1,2-dichloroethene	2.9	0.6	*** Detected at one location only, TP131_2.00
tetrachloroethene	0.5	1.3 ³	** Refer to notes below
chloroethane	0.03	18000 ³	* Refer to notes below
Polynuclear Aromatic Hydrocarbons			
naphthalene	5.2	190 ³	** Refer to notes below
phenanthrene	2.5	29126 (S)	Limited data available, assume pyrene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
acenaphthylene	0.9	29219 ³	
anthracene	1.1	10000 ³	
fluoranthene	5.2	22000 ³	
pyrene	6.1	29126 ³	
benzo(a)anthracene	3.6	2.1 ³	Not identified as COPC as total PAH meet NEPM HIL

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Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comment
chrysene	3.2	210.9 ³	
benzo(b)&(k)fluoranthene	7.7	2.1 ³	Based on lower value for group, not identified as COPC as total PAH meet NEPM HIL and screening value used is conservative.
benzo(a) pyrene	4	5 ¹	
indeno(1,2,3-c,d)pyrene	2.4	2.1 ³	Not identified as COPC as total PAH meet NEPM HIL
benzo(g,h,i)perylene	2.8	210.9 (S)	Limited data available, assume chrysene as surrogate – consistent with TEFs
PAHs (Sum of total)	24.4	100 ¹	
Phthalates			
di-n-butyl phthalate	0.5	62560 ³	
butyl benzyl phthalate	1.9	100000 ³	
bis(2-ethylhexyl) phthalate	10.4	123.1 ³	
Chlorinated Hydrocarbons			
hexachloroethane (HCE)#	5.2	120 ³	
hexachlorobutadiene (HCBd)#	7.7	22 ³	
pentachlorobenzene	0.12	490 ³	
hexachlorobenzene (HCB)#	8.8	1.1 ³	Maximum reported from TP50 with other exceedance noted at SS037
1,3,5-trichlorobenzene	1.9	215 (S)	* Refer to note below. No data available, adopt data for 1,2,4-trichlorobenzene as surrogate
1,2,4,5-tetrachlorobenzene	0.8	180 ³	
1,3-dichlorobenzene	0.028	600 ³	* Refer to notes below
1,4-dichlorobenzene	0.039	7.9 ³	* Refer to notes below
1,2-dichlorobenzene	0.016	600 ³	* Refer to notes below
1,2,4-trichlorobenzene	0.11	215 ³	* Refer to notes below
tetrachlorobenzene	0.006	180 (S)	No data available, adopt data for 1,2,4,5-tetrachlorobenzene as surrogate
Others			
n-nitrosodiphenyl & diphenylamine	8.4	15400 ³	Criteria for diphenylamine
bis(2-chloroethyl)ether	2.1	0.55 ³	Maximum and only exceedance of criteria at TP52

1 – Soil criteria derived from NEPM HIL for commercial industrial land use

2 – NSW EPA Service Station Guidelines (1994)

3 – Region IX PRG for industrial soils (2004)

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

- HCB, HCE and HCBd are not considered to be volatile chemicals and as such consideration of vapour intrusion issues has not been included in the screening level assessment for these chemicals. It is noted that while these chemicals have been identified in emissions to air at other locations within the BIP such as the CPWE where source concentrations are substantially higher, emissions to air from the low concentrations found in groundwater are not expected to be of significance. No air data collected from Southlands has reported any of these compounds above the limit of reporting (refer to **Section 3.8**).

Shaded rows are chemicals where maximum concentration reported is greater than the screening criteria, however the following should also be noted:

* Chemical detected in soil with a maximum concentration less than the adopted criteria noted in above table and less than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

** Chemical detected in soil with a maximum concentration less than the adopted criteria noted in above table and greater than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

*** Chemical detected in soil with a maximum concentration greater than the adopted criteria noted in above table and identified as a COPC and greater than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

No additional COPC have been identified based on consideration of potential cumulative effects of mixtures.

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Table 3-4 Review of Soil Data and Identification of Chemicals of Potential Concern - Southlands Block 1 (Stages 2 and potentially Stage 3)

Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comments
arsenic	62	500 ¹	
cadmium	11	100 ¹	
chromium	1500	60 ¹	Maximum reported at TP31 with the only other exceedances reported at SS058, SS059 and SS051
copper	1380	5000 ¹	
lead	7670	1500 ¹	Maximum reported at TP127 with the only other exceedance reported at TP31
nickel	373	3000 ¹	
zinc	4200	35000 ¹	
mercury	176	75 ¹	Maximum reported at TP06, with five other sample locations report concentrations in excess of the adopted criteria
TPH and MAH			
TPH C ₆ -C ₉	12100	65 ²	TPH not assessed by Woodward Clyde. Exceedances of TPH and other MAHs reported below identified within the south western corner of Block 1 in the vicinity of TP1, TP81 and TP82 and in the area of HA02
TPH C ₁₀ -C ₁₄	2440	1000 ²	
TPH C ₁₅ -C ₂₈	2870		
TPH C ₂₉ -C ₃₆	2720		
benzene	31.3	1 ²	BTEX was assessed by Woodward-Clyde, exceedances follow similar distribution noted for TPH in later investigations
toluene	4.8	130 ²	
ethylbenzene	141	50 ²	
m- & p-xylene	35	25 ²	
o-xylene	6.4		
isopropylbenzene	14.4	2000 ³	* Refer to notes below
n-propylbenzene	70.2	240 ³	**Refer to notes below
1,3,5-trimethylbenzene	1.5	70 ³	**Refer to notes below
sec-butylbenzene	3.1	220 ³	**Refer to notes below
1,2,4-trimethylbenzene	20	170 ³	**Refer to notes below
Volatile Organic Compounds			
vinyl chloride	53	0.75 ³	***Maximum reported at TP37, with other detections reported at TP29, SP056 and SP057
1,1-dichloroethene	2	413 ³	*Refer to notes below
trans-1,2-dichloroethene	106	234 ³	**Refer to notes below
1,1-dichloroethane	22.9	1738 ³	**Refer to notes below
cis-1,2-dichloroethene	165	146.3 ³	***Maximum reported at TP29
1,2-dichloroethane (EDC)	4.5	0.6 ³	***Maximum reported at TP29
trichloroethene (TCE)	95.1	0.11 ³	***Maximum reported at TP105
1,1,2-trichloroethane	1	1.6 ³	**Maximum and only exceedance of criteria in stockpile sample SP30
tetrachloroethene	90.8	1.3 ³	***Maximum reported at TP105
1,1,1,2-tetrachloroethane	10.2	0.93 ³	***Maximum reported at TP105
chloroform	3.9	0.47 ³	***Maximum reported at HA01
chloromethane	0.05	160 ³	*Refer to notes below

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Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comments
Polynuclear Aromatic Hydrocarbons			
naphthalene	28.3	190 ³	**Refer to notes below
2-methylnaphthalene	51.9	190 (S)	** No data available, assume naphthalene as surrogate
acenaphthylene	2.1	29219 (S)	Limited data available, assume acenaphthene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
acenaphthene	1.2	29219 ³	
phenanthrene	9.8	29126 (S)	Limited data available, assume pyrene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
anthracene	3.2	10000 ³	
fluoranthene	21.8	22000 ³	
pyrene	23.8	29126 ³	
benzo(a)anthracene	12	2.1 ³	
chrysene	10.4	210.9 ³	
benzo(b)&(k)fluoranthene	11.2	2.1 ³	Based on criteria for group
benzo(a) pyrene	6.6	5 ¹	
indeno(1,2,3-c,d)pyrene	2.8	2.1 ³	
dibenz(a,h)anthracene	1	0.21 ³	
benzo(g,h,i)perylene	3.2	210.9 (S)	Limited data available, assume chrysene as surrogate – consistent with TEFs
PAHs (Sum of total)	106.4	100 ¹	Total PAH and individual PAHs exceed criteria at one location (TP16)
Phthalates			
di-n-butyl phthalate	1.8	62560 ³	
butyl benzyl phthalate	2	100000 ³	
bis(2-ethylhexyl) phthalate	43.1	123.1 ³	
Chlorinated Hydrocarbons			
hexachloroethane (HCE)#	7.8	120 ³	
hexachlorobutadiene (HCBd)#	326	22 ³	Maximum reported at HA02 with exceedance of criteria identified at HA01
pentachlorobenzene	7.3	490 ³	
hexachlorobenzene (HCB)#	1170	1.1 ³	Maximum reported at HA02 with exceedance of criteria identified at HA01, HA03, SS049, HA006
1,3,5-trichlorobenzene	15	215 (S)	**Refer to notes below No data available, adopt data for 1,2,4-trichlorobenzene as surrogate
1,2,4,5-tetrachlorobenzene	35.6	180 ³	
1,3-dichlorobenzene	0.007	600 ³	*Refer to notes below
1,4-dichlorobenzene	0.017	7.9 ³	*Refer to notes below
1,2-dichlorobenzene	0.013	600 ³	*Refer to notes below
1,2,4-trichlorobenzene	0.094	215 ³	*Refer to notes below
tetrachlorobenzene	0.5	180 (S)	No data available, adopt data for 1,2,4,5-tetrachlorobenzene as surrogate

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Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comments
Others			
n-nitrosodiphenyl & diphenylamine	268	15400 ³	Criteria for diphenylamine
dibenzofuran	1.5	1563 ³	
carbazole	2.3	86	

1 – Soil criteria derived from NEPM HIL for commercial industrial land use

2 – NSW EPA Service Station Guidelines (1994)

3 – Region IX PRG for industrial soils (2004)

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

- HCB, HCE and HCBd are not considered to be volatile chemicals and as such consideration of vapour intrusion issues has not been included in the screening level assessment for these chemicals. It is noted that while these chemicals have been identified in emissions to air at other locations within the BIP such as the CPWE where source concentrations are substantially higher, emissions to air from the low concentrations found in groundwater are not expected to be of significance. No air data collected from Southlands has reported any of these compounds above the limit of reporting (refer to **Section 3.8**).

Shaded rows are chemicals where maximum concentration reported is greater than the screening criteria, however the following should also be noted.

* Chemical detected in soil with a maximum concentration less than the adopted criteria noted in above table and less than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

** Chemical detected in soil with a maximum concentration less than the adopted criteria noted in above table and greater than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

*** Chemical detected in soil with a maximum concentration greater than the adopted criteria noted in above table and identified as a COPC and greater than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

No additional COPC have been identified based on consideration of potential cumulative effects of mixtures.

The screening of the data has made no distinction between contamination at the surface (defined to be soil at depths 0 to 1m¹⁰) or at depth (defined as depths greater than 1m) as it has been assumed that development of the site may result in the movement of soil across the site with the potential for soil at depth to be placed on the surface during earth works.

3.5.2 Consideration of Vapour Intrusion from Soil Contamination

The identification of COPC in soils presented in **Section 3.5.1** was based on comparison of the maximum concentration reported with screening level guidelines. In relation to volatile chemicals, the following needs to be considered:

- NEPM HILs are currently only available for non-volatile or semi-volatile chemicals.
- The guidelines adopted for the purpose of screening volatile petroleum hydrocarbons (TPH and BTEX) are the NSW EPA Service Station Guidelines (1994). While the basis for the derivation of these guidelines is not clear, the values are relevant for sensitive land uses (i.e. residential). On this basis, for the assessment of a commercial/industrial area, the guidelines have been considered relevant to the protection of all exposures including vapour intrusion.
- The Region IX PRGs for industrial soil have been derived on the basis of exposure via ingestion, dermal contact, inhalation of particulates (i.e. dust) and inhalation of vapours outdoors, with target risks values of 10⁻⁶ for carcinogenic effects and HI of 1 for non-carcinogenic effects. The PRGs do

¹⁰ Surface soil has been defined as 0 to 1m compared to a typical definition of 0-0.5m depth as per ANZECC (1992) however, as this site is to be redeveloped, the works may involve excavation and mixing of soil within at the top 1m of the site.

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not consider vapour intrusion into buildings and potential effects on indoor air quality. As a consequence the PRGs for volatile chemicals (as defined by the PRG documentation) have been reduced by a safety factor of 100 fold to provide a value appropriate for screening soil concentrations for vapour intrusion into buildings located directly above the contamination. A 100 fold safety factor has been selected based on vapour migration modelling using infinite source models such as the Johnson & Ettinger (USEPA, 2003) and ASTM (2002) model that show indoor air concentrations to be typically 10 to 100 times higher than outdoor air concentrations¹¹.

The volatile chemicals detected in soil have been further screened against the modified PRG as noted in **Tables 3-3 and 3-4**. **Table 3-5** presents a summary of the volatile chemicals detected in soil that have been further screened against the modified criteria (where relevant). The final identification of additional volatile COPC has also considered the following:

- The identification of volatile COPC is relevant to the vapour intrusion pathway which can be assessed using soil gas and flux emissions data or modelled on the basis of soil concentrations. Soil gas and flux emissions data have been collected from a number of areas on Block 1 and Block 2 focussing on areas overlying shallow groundwater contamination rather than areas of soil contamination. Nonetheless, some vapour sampling locations coincided with areas subsequently found to have elevated soil concentrations. The vapour values therefore reflect both soil and groundwater contamination and provide the most appropriate basis for assessing vapour intrusion. Where the location of the soil gas and flux emissions data coincided with the elevated soil concentrations include soil locations TP01, TP18, TP19, TP81 and TP82 (as highlighted in **Table 3-5**). Hence elevated volatile COPC identified in soils at these locations have been assessed (vapour intrusion) on the basis of the soil gas and/or flux emissions data collected. Other volatile COPC identified in soil (where soil gas and flux emission data is not available) require assessment of vapour intrusion based on modelling from measured soil concentrations. The volatile COPC that require modelling are highlighted in **Table 3-5**.
- The available soil data have been further reviewed with respect to the depth of sampling. Soil data collected within the saturated zone is considered to be associated with the presence of impacted groundwater and as such vapour intrusion from this zone is assessed on the basis of groundwater contamination. Soil contamination within the unsaturated zone, is however, of possible significance with respect to the assessment of vapour intrusion. As most of the volatile chemicals identified on the site are located within Block 1, further screening of the soil data has been undertaken in this area. The depth of the unsaturated zone fluctuates within Block 1 as a consequence of changing groundwater elevation, hence the lower depth of the shallow aquifer, namely 1.5m, has been assumed to be representative of the depth of the unsaturated zone for the purpose of identifying volatile COPC. This is a conservative assumption.

Table 3-5 presents a summary of the volatile chemicals detected in the unsaturated zone of Blocks 1 and 2, the adopted screening criteria (including the modified value where relevant), locations where the screening criteria is exceeded and whether the locations coincide with measured soil gas or flux emissions data. The final volatile COPC that require modelling of potential indoor air concentrations based on soil concentrations (where no soil gas or flux emissions data is available) are then highlighted.

¹¹ Modeling of vapour migration presented in the Consolidated HHRA (URS, 2005a) for commercial areas located above the Main Plumes estimated indoor air concentrations to be approximately 4 times greater than outdoor air concentrations. Hence the use of a 100 fold factor for the purpose of screening the soil data to identify chemicals that may warrant further consideration with respect to vapour intrusion issues is considered adequate and conservative.

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Table 3-5 Summary of Volatile Chemicals of Potential Concern Identified in Soil - Southlands Block 2 (Stage 1) and Block 1 (Stages 1 and 2)

Volatile Chemicals in Soils	Modified Screening Criteria (mg/kg)	Identified as COPC and Maximum Concentration Reported (mg/kg)		Locations that Exceed Modified Criteria and other Comments
		Block 2	Block 1	
TPH C ₆ -C ₉	65 ²		12100	TPH not assessed by Woodward Clyde. Exceedances of TPH and other MAHs reported below identified within the south western corner of Block 1 in the vicinity of TP1, TP81 and TP82 and therefore assessed on basis of soil gas and flux data collected at these locations.
TPH C ₁₀ -C ₁₄	1000 ² for C ₁₀ -C ₃₆		2440	
benzene	1 ²		31.3	BTEX was assessed by Woodward-Clyde, exceedances follow similar distribution noted for TPH in the vicinity of TP1, TP81 and TP82 and therefore assessed on basis of soil gas and flux data collected at these locations.
ethylbenzene	50 ²		141	
m- & p-xylene	25 ²		35	
o-xylene			6.4	
n-propylbenzene	2.4 ³		70.2	Detected above modified criteria at TP82 and TP1 only and therefore assessed on basis of soil gas and flux data collected at these locations.
1,3,5-trimethylbenzene	0.7 ³		1.5	Detected above modified criteria at TP82 and TP1 only and therefore assessed on basis of soil gas and flux data collected at these locations.
sec-butylbenzene	2.2 ³		3.1	Detected above modified criteria at TP82 only and therefore assessed on basis of soil gas and flux data collected at this location.
1,2,4-trimethylbenzene	1.7 ³		20	Detected above modified criteria at TP82 and TP1 only and therefore assessed on basis of soil gas and flux data collected at these locations.
vinyl chloride	0.0075 ³		53	Maximum reported at TP37 Detected above modified criteria at TP15, TP29 and TP37 and stockpile samples SP056 and SP057
trans-1,2-dichloroethene	2.3 ³		106	Detected above modified criteria at TP15, TP29, TP37, HA01, HA02, TP107, TP110 and TP116
1,1-dichloroethane	17 ³		22.9	Detected above modified criteria at TP29 only
cis-1,2-dichloroethene	1.5 ³		165	Maximum reported at TP29 Detected above modified criteria at TP15, TP27, TP29, TP37, HA01, HA02, TP105, TP106, TP107, TP111, TP116, TP118, TP120, TP126, HA06, HA07 and stockpile SP22.
1,2-dichloroethane (EDC)	0.006 ³	2.9	4.5	Block 2: Only detection reported at TP131 Block 1: Maximum reported at TP29. Detected above modified criteria at TP29, TP37, TP45, TP105 and TP107
trichloroethene (TCE)	0.001 ³		95.1	Maximum reported at TP105 Detected above modified criteria at TP12, TP15, TP01, TP02, TP19, TP29, TP37, TP45, HA01, HA02, TP105, TP106, TP107, TP110, TP111, TP114, TP118, TP120, TP124, TP125, HA05, HA06 and HA07 and stockpile samples SP28 and SP30
1,1,2-trichloroethane	0.016 ³		1	Detected above modified criteria at TP45 and HA07 and in stockpile sample SP30
tetrachloroethene (PCE)	0.013 ³		90.8	Maximum reported at TP105 Detected above modified criteria at TP12, TP15, TP01, TP02, TP24, TP29, TP45, TP105, TP106, TP107, TP110, TP111, HA01 and HA07 and stockpile samples SP2, SP5, SP27, SP28, SP29 and SP30
1,1,2,2-tetrachloroethane	0.0093 ³		10.2	Maximum reported at TP105 Detected above modified criteria at TP29, TP105, TP106 and TP114
chloroform	0.0047 ³		3.9	Maximum reported at HA01

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Volatile Chemicals in Soils	Modified Screening Criteria (mg/kg)	Identified as COPC and Maximum Concentration Reported (mg/kg)		Locations that Exceed Modified Criteria and other Comments
		Block 2	Block 1	
				Detected above modified criteria at TP15, TP19, TP24, TP45, HA01 and TP105
naphthalene	1.9 ³		28.3	Detected above modified criteria at TP01, TP18, TP81 and TP82 and therefore assessed on basis of soil gas and flux data collected at these locations.
2-methylnaphthalene	1.9 (S)		51.9	Detected above modified criteria at TP01, TP18, TP81 and TP82 and therefore assessed on basis of soil gas and flux data collected at these locations.
1,3,5-trichlorobenzene	2.2 (S)		15	Detected above modified criteria at HA02 only

Notes:

2 – NSW EPA Service Station Guidelines (1994).

3 – Region IX PRG for industrial soils (2004) as modified to account for potential vapour intrusion issues.

(S) - criteria based on surrogate compound (no criteria available for chemical detected).

Shaded rows are the COPC identified for the assessment of vapour intrusion.

TP01 – locations that coincide with soil gas or flux emissions data, hence the assessment of inhalation exposures for these locations undertaken on the basis of measured soil gas or flux data.

No additional COPC have been identified based on consideration of potential cumulative effects of mixtures.

3.5.3 Asbestos in Soils

Fibrous cement sheeting fragments containing asbestos (chrysotile) have been reported in Block 2 (HLA, 2005). Fibrous cement fragments containing asbestos were also observed in a number of stockpiles. Asbestos fibres (chrysotile and amosite) were also reported in soil samples collected from TP42, TP46 and SP15 within Block 2. Hence asbestos has been included as a COPC.

3.5.4 Summary of Issues and COPC identified in Soils

On the basis of the above the following COPC have been identified in soils at the site. It should be noted that distinction between Blocks 1 and 2 are made solely on the basis of the available investigations and data. The contaminants listed should be considered to have the potential to be present on any either of the Blocks in locations not covered by the existing investigations. Site development and management should be cognisant of these issues and make appropriate contingency plans in the development and remediation management plan. For example, ACM has not been observed to date on Block 1, however the possibility of finding ACM anywhere on Southlands should be covered by contingency plans.

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Block 1	Block 2
chromium lead; mercury TPH benzene ethylbenzene m-, p- and o-xylenes vinyl chloride cis-1,2-dichloroethene 1,2-dichloroethane (EDC) trichloroethene (TCE) 1,1,2-trichloroethane tetrachloroethene (PCE) 1,1,1,2-tetrachloroethane chloroform PAHs, namely benzo(a)pyrene, benzo(a)anthracene, benzo(b&k)fluoranthene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene hexachlorobutadiene (HCB) hexachlorobenzene (HCB)	chromium lead mercury bis(2-chloroethyl)ether hexachlorobenzene (HCB) asbestos
<p><u>COPC identified on basis of vapour intrusion (to be modelled from soil concentrations):</u></p> vinyl chloride cis-1,2-dichloroethene 1,1-dichloroethane trans-1,2-dichloroethene 1,2-dichloroethane (EDC) trichloroethene (TCE) 1,1,2-trichloroethane tetrachloroethene (PCE) 1,1,1,2-tetrachloroethane chloroform 1,3,5-trichlorobenzene	<p><u>COPC identified on basis of vapour intrusion (to be modelled from soil concentrations):</u></p> 1,2-dichloroethane (EDC)

3.6 Pond Sediment

As discussed in **Section 1.3**, the existing ponds on Block 1 are to be filled in as part of the proposed development. While this eliminates the potential for exposure to water within the ponds, the sediment data requires further consideration. Sediment data has been collected from the permanent and ephemeral ponds (and drains). The higher concentrations reported are from the ephemeral ponds that could potentially be excavated and moved across the site, along with soil. Hence the available sediment data has been reviewed against the guidelines presented in **Section 3.2.1** with consideration of vapour intrusion issues outlined in **Section 3.5.2**. It is noted that no soil gas or flux emissions data collected from Block 1 coincided with any sediment sample locations.

Available data on sediment quality from the pond areas has been obtained from the following:

- Pond sediment data collected from a number of the ponds by Woodward-Clyde as part of the Stage 2 Survey (1996a); and
- Pond sediment data collected by HLA in April 2005 from six locations where surface water samples were also collected. Sediment samples were collected from two depths at each location.

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Table 3-6 presents a summary of the sediment data available (refer to Figure 5 for locations).

Table 3-6 Review of Pond Sediment Data and Identification of Chemicals of Potential Concern - Southlands Block 1 (Stages 1 and 2)

Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comment
arsenic	58	500 ¹	
cadmium	2	100 ¹	
chromium	188	60 ¹	Maximum reported at SED06, 0.5-0.6 m, only one other exceedance noted at locations SD023 (0-0.15m) of 159 mg/kg. All other concentrations less than adopted criteria
copper	369	5000 ¹	
lead	285	1500 ¹	
nickel	49	3000 ¹	
zinc	2040	35000 ¹	
mercury	45.1	75 ¹	
TPH and MAH			
TPH C ₆ -C ₉	113	65 ²	Maximum concentrations reported by Woodward Clyde (TPH not reported by HLA) at locations SD018 and SD019 which is in the same ephemeral drain as SED06
TPH C ₁₀ -C ₁₄	498	1000 ²	
TPH C ₁₅ -C ₂₈	3460		
TPH C ₂₉ -C ₃₆	0		
benzene	0.6	1 ²	
toluene		130 ²	
ethylbenzene	1.7	50 ²	
m- & p-xylene	1.5	25 ²	
o-xylene	1.0		
1,2,4-trimethylbenzene	0.8	170 ³	*Refer to notes below
Volatile Organic Compounds			
vinyl chloride	384	0.75 ³	***Maximum reported at location SED06 at depth 0.5-0.6m. Shallow sample (0.2-0.3m) reported other exceedance of 285 mg/kg
1,1-dichloroethene	0.5	413 ³	*Refer to notes below
trans-1,2-dichloroethene	13.4	234 ³	**Refer to notes below
1,1-dichloroethane	1.4	1738 ³	*Refer to notes below
cis-1,2-dichloroethene	302	146.3 ³	***Maximum reported at SED06, 0.2-0.3 m
1,2-dichloroethane	29.8	0.603 ³	***Maximum reported at SED06, 0.2-0.3 m
trichloroethene (TCE)	1050	0.11 ³	***Maximum reported at SED06, 0.5-0.6 m
1,1,2-trichloroethane	115	1.6 ³	***Maximum reported at SED06, 0.2-0.3 m
tetrachloroethene	7130	3.4 ³	***Maximum reported at SED06, 0.5-0.6 m
1,1,1,2-tetrachloroethane	42.7	0.93 ³	***Maximum reported at SED06, 0.2-0.3 m
hexachlorobutadiene	112	22 ³	***Maximum reported at SED06, 0.2-0.3 m
chloroform	213	0.47 ³	***Maximum reported at SED06, 0.2-0.3 m
chloromethane	0.05	160 ³	*Refer to notes below
carbon tetrachloride	0.07	0.55 ³	**Refer to notes below
Semivolatile Organic Compounds			
naphthalene	0.7	190 ³	*Refer to notes below
2-methylnaphthalene	1.2	190 (S)	*Refer to notes below , no data available, assume naphthalene as surrogate
acenaphthene	0.9	29219 ³	
phenanthrene	8.3	29126 (S)	Limited data available, assume pyrene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
anthracene	1.3	10000 ³	

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Chemicals Detected (COPC highlighted in shaded rows)	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comment
fluoranthene	0.9	22000 ³	
pyrene	2.9	29126 ³	
benzo(a)anthracene	0.8	2.1 ³	
chrysene	0.8	210.9 ³	
di-n-butyl phthalate	2.3	62560 ³	
bis(2-ethylhexyl) phthalate	22.4	123.1 ³	
hexachloroethane (HCE)	1.5	120 ³	
pentachlorobenzene	0.01	490 ³	
hexachlorobenzene (HCB)	0.21	1.1 ³	
1,3,5-trichlorobenzene	0.06	215 (S)	*Refer to notes below, no data available, adopt data for 1,2,4-trichlorobenzene as surrogate
1,2,4-trichlorobenzene	0.81	215 ³	*Refer to notes below
1,2,4,5-tetrachlorobenzene	0.6	180 ³	
1,3-dichlorobenzene	0.36	600 ³	*Refer to notes below
1,4-dichlorobenzene	1.1	7.9 ³	**Refer to notes below
1,2-dichlorobenzene	0.62	600 ³	*Refer to notes below
tetrachlorobenzene	0.55	180 (S)	No data available, adopt data for 1,2,4,5-tetrachlorobenzene as surrogate
Phenolic Compounds			
3- & 4-methylphenol	0.5	3078 ³	Minimum guideline available for 3- or 4-methylphenol
2,4-dichlorophenol	7.1	1847 ³	
2,4,6-trichlorophenol	0.6	62 ³	

1 – Soil criteria derived from NEPM HIL for commercial industrial land use

2 – NSW EPA Service Station Guidelines (1994)

3 – Region IX PRG for industrial soils (2004)

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

Shaded rows are chemicals where maximum concentration reported is greater than the adopted criteria, however also note the following

- * Chemical detected in soil with a maximum concentration less than the adopted criteria noted in above table and less than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)
- ** Chemical detected in soil with a maximum concentration less than the adopted criteria noted in above table and greater than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)
- *** Chemical detected in soil with a maximum concentration greater than the adopted criteria noted in above table and identified as a COPC and greater than the modified criteria relevant to the consideration of vapour intrusion issues (refer to **Section 3.5.2**)

No additional COPC have been identified based on consideration of potential cumulative effects of mixtures.

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On the basis of the above the following COPC have been identified in sediments on Block 1:

Block 1 – Pond Sediment
chromium
TPH
vinyl chloride
cis-1,2-dichloroethene
1,2-dichloroethane (EDC)
trichloroethene (TCE)
1,1,2-trichloroethane
tetrachloroethene (PCE)
1,1,2,2-tetrachloroethane
chloroform
hexachlorobutadiene (HCBD)
<u>COPC identified on basis of vapour intrusion (to be modelled from sediment concentration):</u>
TPH
vinyl chloride
cis-1,2-dichloroethene
trans-1,2-dichloroethene
1,2-dichloroethane (EDC)
trichloroethene (TCE)
1,1,2-trichloroethane
tetrachloroethene (PCE)
1,1,2,2-tetrachloroethane
chloroform
carbon tetrachloride
1,4-dichlorobenzene

3.7 Soil Gas and Flux Emission Rates

Flux emissions and some soil gas data have been collected from the Southlands property by URS as part of a number of sampling programs conducted since 1995. THE prime objective for sampling has been to quantify risks to human health for the groundwater program associated with vapour migration from the shallow groundwater plumes and include the following (refer to **Figure 6** for locations):

- The 1995 air emissions sampling was undertaken as part of the Stage 2 Health Risk Assessment (and reported in Appendix D of the Stage 2 Health Risk Assessment Report, Woodward-Clyde 1996b). Both flux emissions and soil gas data were collected. Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05) and AS6 (AS06) within Block 2;
- The 1996 air emissions sampling program was carried out prior to the commencement of the Stage 3 monitoring rounds. The data from this monitoring is presented in the Air Emissions Report, Woodward-Clyde 1997. Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05) and AS6 (AS06) within Block 2;
- The 1998 air emissions sampling round was completed as part of the Stage 3 monitoring program (reported in the progress report WCIE-233, March 1999). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor), AS03 (to the north of Block 1 in the Nant St tank farm) and AS5 (AS05) and AS6 (AS06) within Block 2;
- The 1999 air emissions sampling round was completed as part of the Stage 3 monitoring program (reported in the progress report WCIE-298, December 1999). Samples collected that are relevant to

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the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor), AS3 (AS03) (to the north of Block 1 in the Nant St tank farm) and AS5 (AS05), AS6 (AS06) and AS13 (AS13) within Block 2;

- The 2001 and 2002 air emissions sampling round was completed as part of the Stage 3 monitoring program (reported in the Air Emissions Sampling Program July 2001 Report, URS 2001 and Air Emissions Sampling Program November 2002 Report, URS 2002). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05), AS6 (AS06) and AS13 (AS13) within Block 2;
- The 2004, 2005 and 2006 air emissions sampling rounds were completed as part of the ongoing monitoring programs (reported in Air Emissions Sampling Program March 2004 Report, URS 2004a and Air Emissions Sampling Program July 2005 Report, URS 2006a and URS, 2007c). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05), AS6 (AS06), AS13 (AS13) and AS15 (AS15) within Block 2;
- In 2005 and 2006 sampling of soil gas was undertaken from one location on the southern portion of Southlands (Block 2) at location AS86 (SG01) (reported in Soil Gas Sampling - Southlands Report, URS 2006b); and
- In 2006 sampling of flux emissions and soil gas was undertaken from a limited number of locations on Southlands (limited due to the presence of shallow groundwater). Flux emissions data were collected from locations AS65 (FH02), AS66 (FH03), AS67 (FH04), AS68 (FH05) (located on Block 1) and AS69 (FH07) and AS70 (FH08) (located on Block 2). Soil gas data were collected from locations AS71 (SG09) and AS72 (SG10) (located on Block 2) (URS, 2006b).

Tables 3-7 and 3-8 present a summary of the chemicals reported in flux emissions data relevant to the assessment of Block 2 (**Table 3-7**) and Block 1 (**Table 3-8**) of Southlands. **Table 3-9** presents a summary of the soil gas data relevant to the assessment of Southlands.

As there are no screening criteria are relevant to flux emission rates, COPC have been identified as those chemicals detected in at least one sample.

Soil gas concentrations have been screened against the ambient air criteria. This is a conservative basis for identifying COPC as the concentrations present within the soil gas would not be inhaled directly. Actual concentrations in indoor or outdoor air will be at least 10 to 1,000 times lower¹² than in soil gas reported in the subsurface above a suspected source directly beneath a building or an open area due to diffusion (through soil and concrete), potential degradation, mixing and dispersion outdoors (significant) or within the building. As the screening criteria adopted for identifying COPC in soil gas is so conservative, concentrations that are close to, but less than the adopted criteria, have not been considered as COPC even if the chemical has the potential for cumulative effects with other COPC identified.

¹² USEPA (December, 2004/Revised February 2005). *Guidance for the Evaluation and Mitigation of Subsurface Vapor Intrusion to Indoor Air*. USEPA, Department of Toxic Substances Control, California Environmental Protection Agency. This document presents a default/generic attenuation factor (concentration indoors/concentration in soil gas) of 0.01 (for a residence with a basement) and 0.001 (for a commercial building).

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Table 3-7 Flux Emission Rates - Relevant to the Assessment of Southlands Block 2 (Stage 1)

		Chemical Detected – Flux Emission Rate (µg/min/m ²)																					
		vinyl chloride	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	1,1,1,2-tetrachloroethane	trichlorofluoromethane	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	1,1-dichloroethane	benzene	toluene	ethylbenzene	xylenes	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	p-isopropyltoluene	naphthalene
AS5	May '95	nd	nd	1.9	nd	nd	nd	nd	nd	nd													
	Oct '96	nd	0.22	nd	nd	nd	nd	nd	nd	nd													
	Dec '98	nd	0.039	nd	0.013	0.038	0.012	nd	0.017	nd													
	Aug '99	nd	nd	0.018	0.015	0.44	0.063	0.013	0.054	nd													
	Jul '01	nd	nd	nd	nd	nd	0.048	nd	nd	nd													
	Mar '04	nd	nd	nd	nd	nd	nd	nd	0.047	nd													
	Jul '05	nd	0.26	nd	nd	nd	nd	nd	nd	nd													
	Nov '06	nd	1.24	nd	nd	nd	nd	nd	nd	nd													
AS6	May '95	nd	nd	0.6	nd	nd	nd	nd	nd	nd													
	Oct '96	0.057	0.26	nd	nd	nd	0.022	nd	0.019	nd													
	Dec '98	nd	nd	nd	0.055	0.057	0.01	nd	nd	nd													
	Aug '99	nd	0.02	nd	nd	0.063	nd	nd	0.017	nd													
	Jul '01	nd	nd	nd	0.016	nd	0.016	nd	nd	nd													
	Nov '02	nd	0.044*	nd	nd	nd	nd	nd	nd	nd													
	Mar '04	nd	nd	nd	nd	1.44	nd	0.027	0.622	nd													
	Jul '05	nd	1.6	nd	nd	nd	nd	nd	nd	nd													
	Nov '06	nd	nd	nd	nd	0.15	nd	nd	0.05	nd													
AS13	Aug '99	nd	nd	nd	nd	0.22	0.013	nd	nd	nd													
	Jul '00	nd	0.072	nd	0.024	0.1	0.048	nd	0.016	nd													
	Nov '02	nd	0.022*	nd	nd	nd	nd	nd	nd	nd													

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		Chemical Detected – Flux Emission Rate (µg/min/m ²)																					
		vinyl chloride	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	1,1,2,2-tetrachloroethane	trichlorofluoromethane	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	1,1-dichloroethane	benzene	toluene	ethylbenzene	xylene	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	p-isopropyltoluene	naphthalene
	Mar '04	nd	nd	nd	nd	nd	nd	nd	nd	nd													
	Jul '05	nd	0.48	nd	nd	nd	nd	nd	nd	nd													
	Nov '06	nd	7.7	nd	nd	nd	nd	nd	0.06	nd													
AS15	Mar '04	nd	nd	nd	nd	nd	nd	nd	0.047	nd													
	Jul '05	nd	0.26	nd	nd	nd	nd	nd	nd	nd													
	Nov '06	nd	2.12	nd	nd	0.03	nd	nd	7.72	0.04													
AS69	Aug '06	nd	nd	nd	nd	0.044	0.038	nd	nd	nd	nd	nd	nd	nd	nd	0.085	0.95	0.091	0.44	0.038	0.19	0.016	0.057
AS70	Aug '06	nd	nd	nd	nd	0.69	0.069	nd	nd	nd	0.55	nd	nd	0.12	0.27	0.15	0.22	0.032	0.18	nd	0.092	nd	0.92
Max		0.057	7.7	1.9	0.055	1.4	0.069	0.027	7.72	0.04	0.55	nd	nd	0.12	0.27	0.15	0.95	0.091	0.44	0.038	0.19	0.016	0.92

* Emission rate of methylene chloride (dichloromethane) which is reported is at a level, which is below the emission rate of methylene chloride detected in the field blank.

< 0.2 Values marked with "<" are those reported to be less than the analytical limit of reporting.

0.06 Values in bold were detected during analysis.

Blank cells indicate that analytes were not reported in the sampling program.

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Table 3-8 Flux Emission Rates - Relevant to the Assessment of Southlands Block 1 (Stages 1 and 2)

		Chemical Detected – Flux Emission Rate (µg/min/m ²)																					
Location	Sample Date	vinyl chloride	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	1,1,1,2-tetrachloroethane	trichlorofluoromethane	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	1,1-dichloroethane	benzene	toluene	ethylbenzene	xylenes	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	p-isopropyltoluene	naphthalene
AS9**	May '95	nd	10.5	2.5	13	nd	0.5	nd	0.1	nd													
	Oct '96	2.38	0.73	0.089	nd	1.37	0.64	0.041	0.14	nd													
	Dec '98	nd	nd	nd	nd	nd	0.4	nd	0.079	nd													
	Aug '99	nd	nd	0.011	nd	0.34	0.34	0.026	0.068	0.38													
	Jul '01	nd	0.018*	nd	nd	nd	0.01	nd	0.014	0.024													
	Nov '02	nd	0.1*	nd	nd	nd	5	0.19	39	nd													
	Mar '04	nd	nd	nd	nd	nd	0.11	nd	0.25	nd													
	Jul '05	nd	0.296	nd	nd	0.12	nd	nd	nd	nd													
AS3	Dec '98	0.021	nd	nd	0.016	0.14	0.014	nd	0.018	nd													
	Aug '99	nd	nd	nd	nd	0.2	0.99	nd	0.012	nd													
AS65	Aug '06	nd	nd	nd	nd	0.113	0.25	nd	0.094	0.035	nd	0.18	0.63	nd	nd	0.047	0.44	0.025	0.13	nd	0.069	nd	0.047
AS66	Aug '06	nd	nd	nd	nd	1.38	0.055	nd	0.046	nd	nd	nd	nd	nd	nd	0.097	0.13	nd	0.12	nd	0.088	nd	3.2
AS67	Aug '06	nd	nd	0.066	0.022	0.082	0.44	nd	0.16	nd	nd	nd	nd	nd	nd	0.047	0.38	0.016	0.085	nd	0.047	nd	0.06
AS68	Aug '06	nd	nd	nd	nd	2.8	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.037	0.11	nd	0.069	nd	nd	nd	1.7
Max		2.38	10.5	2.5	13	2.8	5	0.19	39	0.38		0.18	0.63	nd	nd	0.097	0.44	0.025	0.13	nd	0.088	nd	3.2

* Emission rate of methylene chloride (dichloromethane) which is reported is at a level, which is below the emission rate of methylene chloride detected in the field blank.

** AS09 was not sampled in November 2006 due to access problems

Blank cells indicate that analytes were not reported in the sampling program

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Table 3-9 Soil Gas Concentrations - Relevant to the Assessment of Southlands

Location	Sample Depth (m)	Sample Date	Chemical Detected – Soil Gas Concentration (µg/m ³)									
			vinyl chloride	dichloromethane	chloroform	1,2-dichloroethane (EDC)	trichloroethene (TCE)	tetrachloroethene (PCE)	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	
Block 1												
AS9	0.6	May '95	200	5.1	160	nd	91	4.9				
Block 2												
AS5	0.6	May '95	nd	nd	9.2	nd	nd	nd				
AS6	0.6	May '95	nd	nd	20	nd	nd	nd				
AS86	1.1	Dec '05	205	32	5.9	343	46	195	282	602	852	
AS86	1.1	Apr '06	nd	nd	nd	26	nd	nd	nd	nd	nd	nd
AS72	0.47	Aug '06	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Max Block 2			205	32	20	343	46	195	282	602	852	
Screening Level			1 ²	4.1 ³	140 ²	0.36 ²	2.3 ²	200 ²	73 ³	37 ³	200 ²	

- 1 Air Investigation Level as per NEPM 2004, based on annual average
 - 2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.
 - 3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.
 - 4 Chronic Reference Exposure Level established by OEHLA
 - A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group
 - B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group
- Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs
 Blank cells indicate that analytes were not reported in the sampling program

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Table 3-9 (continued) Soil Gas Concentrations - Relevant to the Assessment of Southlands

Location	Sample Depth (m)	Sample Date	Chemical Detected – Soil Gas Concentration (µg/m ³)											
			benzene	toluene	ethylbenzene	xylenes	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	acetone	hexane	cyclohexane	4-ethyltoluene	ethanol	heptane
Block 1														
AS9	0.6	May '95												
Block 2														
AS5	0.6	May '95												
AS6	0.6	May '95												
AS86	1.1	Dec '05												
AS86	1.1	Apr '06	15	300	24	290	49	150	780	54	1500	120	61	310
AS72	0.47	Aug '06	4.3	5.4	1.3	9.0	1.3	nd	nd	nd	nd	nd	nd	nd
Max Block 2			15	300	24	290	49	150	780	54	1500	120	61	310
Screening Level			9.6¹	377¹	22000²	870¹	6.2³	6.2³	3300³	209³	2605³	377^B	100000⁴	209^A

- 1 Air Investigation Level as per NEPM 2004, based on annual average
 - 2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.
 - 3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.
 - 4 Chronic Reference Exposure Level established by OEHHA
 - A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group
 - B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group
- Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs

Blank cells indicate that analytes were not reported in the sampling program

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On the basis of the flux emissions and soil gas data collected, the following COPC have been identified have been identified on Block 1 and Block 2.

Block 1	Block 2
vinyl chloride	vinyl chloride
dichloromethane	dichloromethane
chloroform	chloroform
carbon tetrachloride	carbon tetrachloride
1,2-dichloroethane (EDC)	1,2-dichloroethane (EDC)
trichloroethene (TCE)	trichloroethene (TCE)
1,1,2-trichloroethane	1,1,2-trichloroethane
tetrachloroethane (PCE)	tetrachloroethane (PCE)
1,1,1,2-tetrachloroethane	1,1-dichloroethene
<i>cis</i> - and <i>trans</i> -1,2-dichloroethene	1,1-dichloroethane
benzene	<i>cis</i> - and <i>trans</i> -1,2-dichloroethene
toluene	trichlorofluoromethane (freon 11)
ethylbenzene	benzene
xylenes	toluene
1,2,4-trimethylbenzene	ethylbenzene
naphthalene	xylenes
	1,2,4- and 1,3,5-trimethylbenzene
	TPH C6-C9 aromatics (including isopropyltoluene)
	TPH C6-C9 aliphatics (including heptane)
	naphthalene

3.8 Ambient Air Adjacent to Open Drains

Air samples have been collected at locations adjacent¹³ to Springvale Drain and Floodvale Drain to provide a measure of ambient air concentrations associated with emissions to air from the surface water within the drains. Summa canisters have been used to collect ambient air over a continuous 8 hour period on the following occasions (refer to URS 2007b):

- 1st August 2006 - 2 samples were collected adjacent to Springvale Drain (AS73 and AS74) (formerly SPAM1 and SPAM2) and 2 samples were collected adjacent to Floodvale Drain (AS75 and AS76) (formerly FDAM1 and FD2);
- 19th September 2006 - 1 sample (AS87) was collected adjacent to Springvale Drain near the location where the highest concentration was reported in August 2006;
- 29th September 2006 – 1 sample from location AS87;
- 5th October 2006 – 1 sample from location AS87;
- In 2007 sampling was undertaken adjacent to Springvale Drain following full commissioning of the GTP. Samples were collected from AS91 (11th May, 13th June and 13th July 2007), AS92 (11th May, 13th June and 13th July 2007) and AS93 (13th June and 13th July 2007) within Southlands.

¹³ Samples collected adjacent to the drain included samples collected just above the surface water as well as samples collected within 5m of the drain.

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The following presents a summary of the chemicals detected in ambient air samples collected on the Southlands property. The data presented in the following table presents only those chemicals detected (refer to laboratory reports for all chemicals and limits of reporting) and the maximum reported from the primary or duplicate samples (where collected).

The measured ambient air concentrations have been screened against the adopted ambient air guidelines listed in **Section 3.2.3** to identify COPC.

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Table 3-10 Ambient Air Concentrations Adjacent to Springvale Drain and Floodvale Drains

Location	Sample Date	Chemical Detected – Concentration (µg/m ³)																
		freon 12	vinyl chloride	chlormethane	bromomethane	freon 11	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	tetrachloroethene (PCE)	cis-1,2-dichloroethene	1,1-dichloroethane	carbon disulfide	1,4-dioxane	1,3-butadiene	styrene
Floodvale Drain																		
AS75	Aug '06	2.8	0.44	1.2	0.71	1.4				7								
AS76	Aug '06	2.4		1.5		1.4	2.4			3.3								
Max		2.8	0.44^C	1.5	0.71	1.4	2.4			7								
Springvale Drain																		
AS74	Aug '06		99					59	53	1800	100	160	51					
AS73	Aug '06	2.6	4.4	1.4	1.4	1.7		2.4	3.8	96	4.1	1.8	3.0	96				
AS87	19 Sept '06		89					94	77	2400	180	160	80	6.2				
AS87	29 Sept '06	3.2	20	1.9				22	14	480	42	22	26					
AS87	Oct '06	3.1	1.4			1.7	1.2											
AS91**	May-Jul '07	2.9	2.2 (43)	1.7	0.66	1.4	1.5	1.2 (78)	(28)	15 (930)	7.3 (130)	3.9 (38)	11 (100)	(2.4)	(10)			
AS92*	May-Jul '07	2.6		1.8	0.66	1.4	1.2			7.4	1.5		1.5					
AS93*	Jun-Jul '07	2.9	5.7	1.5		1.5	1.6	7.9	6	72	12	3.4	23		5.3	1	0.42	3
Max		3.1	99	1.9	1.4	1.7	1.6	94	77	2400	180	160	80	96	5.3	1	0.42	3
Screening Level		210³	1²	95³	5.2³	730³	4.1¹	140²	6.1²	0.36²	2.3²	200^{2,C}	37³	521³	100²	0.61³	0.061³	260²

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Table 3-10(continued)

Air Concentrations Adjacent to Springvale Drain and Floodvale Drains

Location	Sample Date	Chemical Detected – Concentration (µg/m ³)														
		benzene	toluene	ethylbenzene	xylenes	4-ethyltoluene	1,2,4-trimethylbenzene	1,3,5-trimethylbenzene	acetone	2-butanone (MEK)	2-propanol	hexane	cyclohexane	heptane	4-methyl-2-pentanone	ethanol
Floodvale Drain																
AS75	Aug '06		3.7		2.1		0.99		6.4	5					5.5	
AS76	Aug '06	1.1	4.6	0.77	3.4		0.98		12						6.5	
Max		1.1	4.6	0.77	3.4		0.99		12						6.5	
Springvale Drain																
AS74	Aug '06															
AS73	Aug '06	1.2	4.2		3.0		0.75		12	2.6					5.5	
AS87	19 Sept '06	7.5	9.6						39	8.2						
AS87	29 Sept '06	1.7	3.4						8.2	3.2					5.3	
AS87	Oct '06	1.9	8.0	2.5	10.2	0.94	1.1		58	3.4	6.9	1.0	0.89	0.66	2.4	
AS91**	May-Jul '07	1.5	6.9	0.81	3.7				15	3.1	4.5	0.7	0.71		12	
AS92*	May-Jul '07	1.5	7.4	0.88	3.3				44	5.8	5.5	0.66			23	
AS93*	Jun-Jul '07	1.7	13	1.9	9.3	3.2	7.7	1.6	270	19	270	1.0		1.4	12	
Max		7.5	13	2.5	10.2	3.2	7.7		270	19	270	1.0	0.89	1.4	2.4	
Screening Level		9.6¹	377¹	22000²	870¹	377^B	6.2³	6.2³	3300³	5110³	7000⁴	209³	2605³	209^A	3100³	100000⁴

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Notes for Table 3-10

- 1 Air Investigation Level as per NEPM 2004, based on annual average
- 2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1×10^{-6} is adopted.
- 3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1×10^{-6} is adopted.
- 4 Chronic Reference Exposure Level established by OEHHA
- A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group
- B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group
- C Maximum concentration reported in air close to the adopted guideline. Based on information available about the chemical, there is the potential for cumulative risks to be of concern as the health endpoints are similar to a range of other chlorinated compounds also identified as COPC (refer to toxicity summaries in **Appendix B**). On this basis these chemicals have been selected as a COPC for further assessment.
- * Maximum concentration reported from sampling undertaken in May, June and July 2007.
- ** Data reported for AS91 includes range of values reported from May to July 2007. Values in (brackets) are concentrations of chlorinated compounds reported from AS62 that are believed to have been mislabelled and should be from AS91.

Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs

Blank cells indicate that analytes were not reported in the sampling program

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On the basis of the above the following COPC have been identified for the assessment of exposures related to the emissions of volatiles from Floodvale and Springvale Drains:

Springvale Drain (relevant to the assessment of exposures in Block 1 and Block 2)	Floodvale Drain (relevant to the assessment of exposures in Block 2)
1,2-dichloroethane (EDC) trichloroethene (TCE) tetrachloroethene (PCE) vinyl chloride carbon tetrachloride <i>cis</i> -1,2-dichloroethene 1,4-dioxane 1,3-butadiene	vinyl chloride 1,2-dichloroethane (EDC)

As noted in **Section 3.3.1**, the calculation of risk presented in the PHHRA (URS, 2006c, refer to **Appendix H**) identified emissions to air from Springvale Drain as an issue requiring risk management or remediation as part of the development. As part of the process to evaluate the options available to address this issue, air data have been collected adjacent to Springvale Drain following full commissioning of the GTP. These data have been presented in the draft report “Environmental Monitoring at Springvale Drain – Orica Southlands” (URS, February 2008). Review of these data indicate that concentrations in air adjacent to the drain have decreased since commissioning of the GTP, however the concentrations have not been reduced sufficiently to conclude that risks to human health will be at an acceptable level as a consequence of the operation of the GTP alone. Hence additional risk management measures are required to support the development.

Further data collected from August 2007 to June 2008 (URS, 2008b) indicate concentrations in air adjacent to Springvale Drain within Southlands that are lower than presented in **Table 3-10**. Based on review of the data collected during this period it was concluded that provided the discharge of shallow groundwater into Springvale Drain is appropriately managed such that concentrations in surface water (and subsequently in ambient air) remain at levels reported since August 2007, long-term risks to workers who may occupy Southlands in the future are considered to be low and acceptable. The potential for peak exposures to occur (such as during rainfall events or when the GTP is offline, as reflected in data collected prior to August 2007) should be further considered to ensure that long-term average exposures remain low and acceptable.

The proposed approach to addressing potential discharge of shallow groundwater into Springvale Drain is the installation of a shallow groundwater extraction system along the eastern side of the drain (refer to the RAP for further detail). The containment line is proposed to be connected into the existing GTP and will aim to intersect shallow groundwater prior to discharge into the drain, thereby reducing the existing exposure pathway. Hence the data set available for air concentrations adjacent to the drain prior to and following operation of the GTP are not expected to be relevant to the long term risks for workers on the site following development and installation of the shallow groundwater extraction system.

Due to maintenance requirements the GTP needs to be taken offline during short periods. The period of time the GTP is offline will not affect containment of the groundwater plumes, however it has the potential to cause groundwater levels in the vicinity of the drain to rise and discharge directly into the drain where emissions to air of volatile chemicals from the drain may occur. The concentrations in air during the period of time when the GTP is offline is expected to be of relevance for this assessment and the above COPC as well as COPC identified in the review of groundwater (and surface water data) presented in **Section 3.3.1** require consideration with respect to this issue.

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3.9 Impacts from Other Sources

A number of other sources of emissions to air are present on the BIP such as the GTP. In addition there are other projects that have been approved or are in the process of seeking approval within the BIP. These include the HCB repackaging plant and the Car Park Waste Encapsulation (CPWE) remediation project. The most current and recent assessment of risks associated with emissions to air relating to these sources, and other sources within the BIP including background is presented in the Human Health Impact Assessment prepared for the CPWE Remediation Project (URS, 2007d). While this project has not been approved at the time of this assessment, the air dispersion modelling and assessment of risk presented is the most current and reflects the other sources on the BIP including background with the inclusion of the CPWE remediation project, which provides a conservative estimate of emissions should the remediation project not be approved.

The assessment of risks to human health associated with emissions to air from the other BIP sources and background indicated that the risks were low and acceptable for all off-site areas, including workers and food processing facilities located in the vicinity of Southlands. On this basis, no additional COPC have been identified, however the predicted concentrations in air in the vicinity of Southlands should be taken into account in the assessment of overall exposure to the COPC identified in air.

Key emissions to air derived from other sources on the BIP that are relevant to the COPC identified and considered for the proposed Southlands development:

- tetrachloroethene (PCE);
- trichloroethene (TCE);
- 1,2-dichloroethene (cis- and *trans* –isomers);
- 1,1-dichloroethene;
- vinyl chloride;
- 1,1,2-trichloroethane;
- 1,1-dichloroethane;
- 1,2-dichloroethane;
- chloroform;
- methylene chloride (dichloromethane);
- mercury;
- hexachlorobenzene (HCB);
- hexachloroethane (HCE); and
- hexachlorobutadiene (HCBD).

On the basis of air modelling undertaken by Pacific Air and Environment (PAE, 2007¹⁴) as part of the CPWE Remediation Assessment, the ground level concentrations listed in **Table 3-11** have been estimated for the above Southlands COPC at the closest discrete receptor (located at the Kelloggs site) to Southlands (modelled discrete receptors in areas surrounding the BIP, refer to PAE (2007) for further discussion).

¹⁴ Maximum ground level concentrations for all sources based on revised modelling provided by PAE on 15 November 2007.

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The concentrations presented in **Table 3-11** are relevant to emissions from other sources¹⁵ on the BIP and are based on an annual average. It is noted that a number of other industries are located in areas surrounding Southlands, some of which would also release a range of pollutants into the air. As these emissions are not known to URS, they cannot be specifically included in the HHRA assessment. Possible exposures from other industrial sources (i.e. other than those identified on the BIP) are addressed for many COPC by published estimates of background intakes (from all sources) as outlined in **Section 4** (and the toxicity summaries presented in **Appendix B**) as well as the assessment of ambient air data collected along Springvale Drain that will include chemicals from sources other than the drain should they be present.

¹⁵ Sources include emissions from Orica (operation of the proposed CPWE remediation project, GTP, HCB Waste Repackaging Plant Stacks on Stores H and J, HCl Burner Vent Stack, Weak Gas Vent Stack and Old Chlorine Plant), Qenos (Two Coal Boiler Stacks, Gas Boiler Stack, Five Furnace Stacks, Two Ground Furnace Stacks, Elevated Flare Stack and Ground Flare) and Huntsman (Hot Oil Furnace)

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Table 3-11 Concentrations of COPC derived from Other Sources on the BIP (including operation of proposed CPWE remediation)

COPC	Ambient Air Concentration Relevant to Southlands (µg/m³)
tetrachloroethene (PCE)	0.41
trichloroethene (TCE)	0.0026
<i>cis</i> -1,2-dichloroethene	0.000061
<i>trans</i> -1,2-dichloroethene	0.0000074
1,1-dichloroethene	0.00011
vinyl chloride	0.0011
1,1,2-trichloroethane	0.000014
1,1-dichloroethane	0.0000055
1,2-dichloroethane (EDC)	0.00084
chloroform	0.00054
dichloromethane	0.000046
mercury	0.0016
hexachlorobenzene (HCB)	0.0000061
hexachlorobutadiene (HCBd)	0.0010
hexachloroethane (HCE)	0.016

The above concentrations have been included in the assessment of exposures for all users of the site.

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Identification of Issues and Data Evaluation

3.10 Summary of COPC Identified

Table 3-10 presents a summary of the COPC identified in Blocks 1 and 2 that are relevant to assessment of risks to human health associated with the proposed development of Southlands.

Table 3-12 Summary of COPC Identified

	COPC Identified									
	Block 2 (Stage 1)				Block 1 (Stages 2 and 3)					Other BIP Sources
	Soil (Direct contact)	Soil (Vapour intrusion)	Soil Gas and Flux Emissions (Vapour intrusion and migration to outdoors)	Air Concentrations adjacent to Drains	Soil and Sediment (Direct contact)	Soil and Sediment (Vapour intrusion)	Soil Gas and Flux Emissions (Vapour intrusion and migration to outdoors)	Shallow Groundwater (assessment of Flood Storage Area and intrusive works)	Air Concentrations adjacent to Drains (including shallow groundwater discharge when GTP offline)	
asbestos	•									
chromium	•				•					
lead					•					
mercury	•				•					•
TPH			•		•	•				
benzene			•		•	•	•			
toluene			•				•			
ethylbenzene			•		•	•	•			
xylenes			•		•	•	•			
1,2,4- and 1,3,5-trimethylbenzene			•				•			
1,3,5-trichlorobenzene						•				
1,4-dichlorobenzene						•				
vinyl chloride			•	•	•	•	•	•	•	•
1,1-dichloroethane			•			•				•
1,1-dichloroethene			•				•		•	•
cis-1,2-dichloroethane			•	•	•	•	•	•	•	•
trans-1,2-dichloroethane			•			•	•	•	•	•
1,2-dichloroethane (EDC)		•	•	•	•	•	•	•	•	•
trichloroethene (TCE)			•	•	•	•	•	•	•	•
1,1,2-trichloroethane			•	•	•	•	•	•	•	•
tetrachloroethene (PCE)			•	•	•	•	•	•	•	•
1,1,2,2-tetrachloroethane					•	•	•			
carbon tetrachloride			•	•		•	•	•	•	
chloroform			•	•	•	•	•	•	•	•
dichloromethane			•				•	•	•	•
hexachlorobenzene (HCB)	•				•					•
hexachlorobutadiene (HCBd)					•		•	•	•	•
hexachloroethane (HCE)								•	•	•
PAHs (carcinogenic)					•					
naphthalene			•				•			
bis(2-chloroethyl)ether	•									
trichlorofluoromethane			•							
1,4-dioxane				•					•	
1,3-butadiene				•					•	

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3.11 Suitability of Data for Risk Assessment

Each of the investigations relevant to the data evaluated has completed assessments of accuracy and precision using quality control data collected in the field and at the laboratory. No further evaluation of accuracy and precision has been undertaken by URS in completion of the HHRA. The data is considered to be suitable for use in the assessment of risks to human health for the following reasons:

- The accuracy and precision are acceptable for the purposes of the investigations as indicated by the data evaluations presented in the relevant reports.
- The amount of data collected is generally considered to be sufficient to allow a conservative estimate of potential concentrations of COPC within the relevant exposure media. There are some limitations associated with the data collected. These limitations are discussed further within the discussion presented in **Sections 5.5 and 6**.

On this basis the available soil, sediment, groundwater, surface water and air data provide an appropriate basis for identifying COPC and an assessment of the expected range of concentrations.

No specific data gaps have been identified, however risk assessment and subsequent risk management are iterative processes that should focus on key uncertainties identified in the HHRA in developing requirements for additional investigations to assess risks or develop remediation requirements and risk mitigation strategies. Refer to **Sections 5.5 and 6** for further discussion on these areas.

Section 4

Toxicity Assessment

4.1 General

As outlined in **Section 1.5**, the quantification of potential risk associated with exposure to COPC identified that are relevant to the assessment of exposure on Southlands requires the assessment of non-threshold and/or threshold effects. Toxicity values relevant to the quantification of non-threshold or threshold effects have been identified following guidance from enHealth (2004) and NEPC (1999) in accordance with toxicological data sources outlined in **Section 1.5.3**.

Detailed toxicity summaries of all the COPCs are presented in **Appendix B**. These present a summary of the key toxicological aspects of the COPC identified, key routes of exposure and health effects, carcinogenicity and relevant toxicity values adopted for the quantification of inhalation, oral and dermal exposure to the COPC identified. **Table 4-1** presents a summary of the toxicological data used in the HHRA.

The final approach adopted in this HHRA has also considered relevant guidance for the assessment on inhalation exposures within a workplace (**Section 4.2**) and mixtures (**Section 4.3**).

It is noted that the toxicity summaries and values presented in Table 5-1 are associated with long-term or chronic exposures (exposures that occur every day over a lifetime). This is relevant to the assessment of exposures identified for the site workers with the exception of short duration exposures that may occur during the period of down time a result of the GTP and resultant higher emissions from Springvale Drain of the volatile COPC. If the proposed risk mitigation measure is implemented such that exposures during the operation of the GTP are negligible (refer to **Section 5**), then the only exposures that require assessment are those associated with the shorter duration events during the maintenance of the GTP. These events do not occur every day over a working life as assumed in the chronic health risk assessment. They may, however, occur for periods of time longer than would be usually defined as acute (e.g. an hour or a day). They therefore need to be considered as representative of intermediate duration exposures which, by definition from the ATSDR¹⁶, involves exposures that may be continuous for 14 to 364 days per event (which may be during any one year period). On this basis further review of the toxicity values relevant to the assessment of intermediate duration exposures has been undertaken and is presented in **Section 4.4**.

The toxicological data presented is considered to be appropriate for the assessment of risks to human health associated with the potential exposure to the COPC. It is accepted that toxicological data has uncertainties (as outlined in **Section 6** of this report). However, the approaches adopted by the different regulatory bodies in determining the relevant toxicological values are considered to be conservative and likely to overestimate the risks.

¹⁶ ATSDR – Agency for Toxic Substances & Disease Registry, definitions available from web-site at: <http://www.atsdr.cdc.gov/toxpro2.html>

Section 4

Toxicity Assessment

Table 4-1 Summary of Toxicity Data Relevant to COPC Identified

Chemical	Non-Cancer Toxicity Endpoint	Animal Carcinogen and IARC Classification	Genotoxic	Oral Slope Factor (mg/kg/day) ⁻¹	Oral TDI (mg/kg/day)	Inhalation Unit Risk (µg/m ³) ⁻¹	Inhalation TC (or equivalent) (mg/m ³)	TWA ⁽⁶⁾ (mg/m ³)	Potential for background intake
hexachlorobenzene (HCB)	Liver	Yes, Group 2B	No	T	0.00016 ⁽¹⁾	T	O	0.002 ⁽⁷⁾	Negligible
hexachlorobutadiene (HCBd)	Kidney	Yes, Group 3	Equivocal	T	0.0002 ⁽³⁾	T	O	0.21	Negligible
hexachloroethane (HCE)	Kidney	Yes, Group 2B	No	T	0.001 ⁽⁴⁾	T	O	9.7	Negligible
1,2-dichloroethane (EDC)	Liver, kidney	Yes, Group 2B	Yes	0.012 ^{(1),(3)}	NT	(0.5 to 2.8)x10 ⁻⁶ ⁽²⁾ 2.8x10 ⁻⁶ proposed	NT	40	NA
vinyl chloride (VC)	Liver	Yes, Group 1	Yes	1.15 adults 2.3 lifetime ⁽¹⁾	NT	4.4x10 ⁻⁶ adulthood 8.8x10 ⁻⁶ lifetime ⁽⁴⁾	NT	13	NA
1,1-dichloroethene	CNS, liver	Limited data, Group 3	No	T	0.046 ⁽¹⁾	T	0.2 ⁽²⁾	20	Negligible
1,1-dichloroethane	CNS, kidney	Limited data, NA	No	T	0.1 ^{(4)*}	T	0.5 ^{(4)*}	412	Negligible
dichloromethane (DCM)	Liver, kidney, CNS	Yes, Group 2B	No	T	0.0012 ⁽³⁾	T	1 ⁽⁵⁾	174	Yes (20%)
chloroform (CFM)	Liver, kidney, CNS	Yes, Group 2B	No	T	0.013 ⁽¹⁾	4.2x10 ⁻⁷ ^(2*)	0.14 ⁽²⁾	10	Yes (50%)
carbon tetrachloride (CTC)	Liver, kidney	Yes, Group 2B	No	T	0.00142 ⁽¹⁾	T	0.0061 ⁽²⁾	0.63	Yes (65%)
trichloroethene (TCE)	CNS, liver	Yes, Group 2A	Equivocal	T	0.00146 ⁽¹⁾	4.3x10 ⁻⁷ ⁽²⁾	NT	54	Yes, low
tetrachloroethene (PCE)	Liver, kidney, CNS	Yes, Group 2A	No	T	0.014 ⁽³⁾	T	0.2 ⁽²⁾	335	Yes (34%)
cis-1,2-dichloroethene	Liver	Insufficient data, NA	Equivocal	T	0.01 ^{(4)*}	T	O	793	Negligible
trans-1,2-dichloroethene	Liver	Insufficient data, NA	Equivocal	T	0.017 ⁽¹⁾	T	O	793	Negligible
1,1,2-trichloroethane (1,1,2-TCA)	Liver, immune	Yes, Group 3	No	T	0.004 ⁽⁴⁾	T	O	55	Negligible
1,1,1,2-tetrachloroethane (1,1,1,2,2-TeCa)	Liver	Equivocal, Group 3	Equivocal	T	0.04 ⁽⁵⁾	(0.6 to 3)x10 ⁻⁶ ⁽²⁾ 3x10 ⁻⁶ proposed	NT	6.9	Negligible
benzene	Immune	Yes, Group 1	Yes	0.035 ⁽¹⁾	NT	6x10 ⁻⁶ ⁽²⁾	NT	3.2	NA
toluene	Kidney	No, Group 3	No	T	0.22 ^{(1),(3)}	T	0.26 ⁽²⁾	191	Yes (10%)
ethylbenzene	kidney, liver	Possible?, Group 2B	No	T	0.097 ^{(1),(3)}	T	22 ⁽²⁾	434	Yes, low
xylenes	CNS	No, Group 3	No	T	0.179 ^{(1),(3)}	T	0.87 ⁽²⁾	350	Yes (2%)
PAHs – benzo(a)pyrene		Yes, Group 2A	Yes	0.5 ⁽¹⁾	NT	8.7x10 ⁻² ⁽²⁾	NT	NA	NA

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Chemical	Non-Cancer Toxicity Endpoint	Animal Carcinogen and IARC Classification	Genotoxic	Oral Slope Factor (mg/kg/day) ⁻¹	Oral TDI (mg/kg/day)	Inhalation Unit Risk (µg/m ³) ⁻¹	Inhalation TC (or equivalent) (mg/m ³)	TWA ⁽⁶⁾ (mg/m ³)	Potential for background intake
equivalent									
1,4-dichlorobenzene	Liver, Kidney	Yes, Group 2B	No	T	0.0107 ⁽³⁾	T	1 ⁽²⁾	150	Yes, low
1,2,4- and 1,3,5-trimethylbenzene	CNS, respiratory	Insufficient data, NA	Insufficient	T	0.05 ^{(4)**}	T	0.006 ^{(4)**}	123	Yes (50%)
1,3,5-trichlorobenzene	Liver, respiratory	No, NA	No	T	0.0077 ⁽³⁾	T	0.0017 ⁽⁹⁾	37 peak limit	Yes (5%)
naphthalene	Haemic, respiratory	Possible, Group 2B	No	T	0.02 ⁽⁴⁾	T	0.003 ⁽⁴⁾	52	Yes, (5%)
TPH C6-C9 Aliphatic	CNS	No, NA	No	T	5 ⁽⁸⁾	T	0.7 ^{(4),(8)}	72	Yes (10%)
TPH C10-C14 Aromatic	CNS	No, NA	No	T	0.04 ⁽⁸⁾	T	0.2 ⁽⁸⁾	350	Yes (10%)
TPH C10-C14 Aliphatic	CNS	No, NA	No	T	0.1 ⁽⁸⁾	T	1 ⁽⁸⁾	350	Yes (10%)
TPH C15+ Aromatic	CNS	No, NA	No	T	0.03 ⁽⁸⁾	T	0 ⁽⁸⁾	NA	Yes (10%)
TPH C15+ Aliphatic	CNS	No, NA	No	T	2 ⁽⁸⁾	T	0 ⁽⁸⁾	NA	Yes (10%)
bis(2-chloroethyl)ether	Liver	Possible, Group 3	Insufficient	1.1 ⁽⁴⁾	NT	3.3x10 ⁻⁴ ⁽⁴⁾	NT	29	NA
trichlorofluoromethane	CNS, respiratory, liver	No, NA	No	T	0.3 ⁽⁴⁾	T	0.7 ^{(4)*}	5620 peak limit	negligible
1,4-dioxane	Liver	Possible, Group 2B	Equivocal	T	0.016 ⁽¹⁾	7.7x10 ⁻⁶ ^{(4)**}	3 ^{(4)**}	36	negligible
1,3-butadiene	Reproductive	Yes via inhalation, Group 1	Likely via inhalation	I	NT	1.7x10 ⁻⁴ ^{(4)**}	0.02 ^{(4)**}	22	Yes (5%)
mercury (Hg)	Elemental: CNS Inorganic: Kidney Methyl: CNS	No, NA Equivocal, NA Yes, Group 2B	--- No No	T	0.00071 ⁽¹⁾ for total mercury and 0.00023 for methylmercury ⁽¹⁾	T	0.0002 ⁽²⁾ for elemental mercury and 0.001 ⁽²⁾ total mercury (inorganic)	Elemental:0.025 Divalent Inorganic:0.025 Monovalent Inorganic 0.1 Alkyl: 0.01	Yes (15% for methyl mercury and 24% for inorganic mercury)
chromium (conservatively assumed to be Cr(VI))	Increased chromium tissue levels	Yes for inhalation of chromium VI, Group 1	Yes for inhalation of chromium VI	T	0.003 ⁽⁴⁾ for chromium IV	0.04 ⁽²⁾	NT	0.5 for chromium III and 0.05 for chromium IV	Yes (30%)
lead	Neurological, haematological	Probable, Group 2B	No	T	0.00357 ⁽³⁾	T	0.0005 ⁽²⁾	0.15	Yes (48%)

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Notes for Table 4-1

- (1) Derived from WHO Drinking Water Guidelines (2004)
(1*) Derived from WHO guidelines using inhalation data
(2) Derived from WHO Air Quality Guidelines (2000, 2000b or CICAD 58 (2004) for chloroform, CICAD 30 for 1,3-butadiene and CICAD 68 for PCE (2006)). Where a range is presented, the most conservative value (higher unit risk and lower TDI) has been adopted.
(2*) Noted to be a conservative approach as threshold may be appropriate
(3) Derived from NHMRC Australian Drinking Water Guidelines (2004)
(4) Derived by USEPA (IRIS evaluations)
(4)* Derived from HEAST as references on ORNL/RAIS database, peer-reviewed source
(4)* * Derived from USEPA OEHHA or NCEA, peer-reviewed source
(5) Derived by ATSDR (chronic exposures)
(6) Occupational data available from NOHSC except where noted, TWA values based on 8-hour average
(7) Occupational data available from ACGIH, TWA value based on 8-hour average
(8) Derived from TPHCWG (1999) evaluation on the basis of surrogates for TPH groups
(9) Review available from Health Canada, peer reviewed source
O Inhalation exposure evaluated using oral data as no relevant chronic inhalation data available
T Threshold approach adopted, hence no oral slope factor or inhalation unit risk considered relevant
NT Non-threshold approach adopted
NA Not available or not applicable (for background intakes for chemicals assessed on basis of non-threshold approach)

Section 5

Toxicity Assessment

4.2 Occupational Inhalation Exposures

General

Worksafe Australia (Australian Safety and Compensation Council [ASCC], formerly known as the National Occupational Health and Safety Commission [NOHSC]) has published a guidance on the assessment of inhalation exposures in the workplace, in particular NOHSC *Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment* [NOHSC:1003(1995)] and subsequent amendments.

Following this guidance the assessment of exposures within the workplace is undertaken on the basis of threshold values, referred to as Time Weighted Average (TWA) inhalation exposure standards. For chemicals classified by ASCC as carcinogenic, a carcinogenic risk is also evaluated to ensure exposures by workers to carcinogens have been assessed. The assessment of threshold TWA and carcinogenic risk has also considered exposure to multiple chemicals assuming an additive effect.

The threshold exposure standards (TWAs) are standards that represent airborne concentrations of individual chemical substances, which, based on current knowledge, “*should neither impair the health of, nor cause undue discomfort to, nearly all workers*” (ibid). The exposure standards are expressed as TWA values, based on exposure to an airborne substance over an eight-hour working day, for a five-day working week, over an entire working life.

Approach for On-Site Workers

Use of the above approach for the assessment of occupational exposures, while recommended in the NEPM, is generally accepted as being appropriate only under some circumstances. Situations where the approach is accepted are in the evaluation of exposure in an occupational environment for workers who work with or handle the COPC identified (where there is the opportunity for ongoing monitoring, medical surveillance and management of exposures) or those who undertake works using suitable workplace exposure standards for intrusive or confined space works.

Hence, the evaluation of workers on the Southlands site following completion of development using the above approach would be considered appropriate if the site were used to store or handle products containing the COPC. However, it is less clear as to the applicability of the approach for commercial/industrial use of a site where the use and handling of the COPC identified is unknown or not expected. Such workers may not be aware of the presence of residual impacts on the site and as such may not be aware of any requirements to minimise exposure or undertake monitoring. On this basis the assessment of inhalation exposures by workers on the site has been undertaken using the more sensitive toxicity values that are protective of all members of the population (as above) including young children.

It is noted, however that if the relevant workplace authority were to undertake an assessment of exposures in the workplace (regardless of the origin of the chemicals), the approach outlined in the ASCC guidance would be utilised.

Hence, for the purpose of comparison, and to address ASCC requirements in the workplace, the assessment of inhalation exposures by workers on the site has also been undertaken using the occupational guidance (TWAs and carcinogenic assessment). This comparison is presented in the calculations presented in **Appendix D**.

Approach for Intrusive Workers

The basis for the assessment of inhalation exposures by workers undertaking intrusive activities is not clear. While it is noted that workers undertaking intrusive works in the area may not be aware of the presence of contamination in groundwater, the nature of their exposure suggests that the use of occupational exposure standards is relevant. In particular the following is of note with respect to the use of occupational guidance for the assessment of inhalation exposures by intrusive workers:

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- Following current guidance and advice from ASCC and Workcover NSW exposures to carcinogenic chemicals are assessed on the basis of a non-threshold carcinogenic assessment to ensure that any long-term effects associated with short-duration intrusive works are assessed;
- The TWAs are threshold values protective of chronic exposures (i.e., 8 hours per day 5 days per week for a working lifetime). While the approach to the derivation of the TWAs differs from that of the sensitive threshold values, they are relevant to threshold effects where the potential for any effects (be it odour, irritation or other effect) are only relevant for the period of exposure. Acute exposure levels are greater than TWAs; thus, if the TWAs are met, then acute risks in relation to chemical exposure will also be acceptable.
- Intrusive works occur infrequently with exposure frequency and durations much less than that assumed when setting a TWA. Thus use of TWAs in the QRA is a very conservative approach for intrusive workers. It is not considered relevant to use chronic lifetime threshold values protective of all populations for the purpose of assessing such short duration exposures.
- Intrusive workers are expected to be fit and relatively healthy for which the TWAs are based. The workers would not be identified as a sensitive group (children, elderly or infirm) where use of TWAs is not appropriate.

A summary of the occupational inhalation exposure standards relevant for the COPC identified are also presented in **Table 4-1**.

4.3 Mixtures

The assessment of mixtures is an area where clear guidance has yet to be provided in Australia or internationally. EnHealth (2004) notes that where data are available on the interaction of chemicals, the data should be considered in the risk assessment. However, how this is to be addressed is not presented in the guidance. Some guidance is available from the NHMRC in the derivation of the Australian Drinking Water Guidelines (NHMRC, 2004), where the assessment focuses on two categories based on health effects:

- Chemicals where the effects are observed above a certain threshold dose; and
- Chemicals that do not appear to have a threshold, namely genotoxic carcinogens.

The approach adopted provides for assessment of chemicals on the basis of either a threshold or non-threshold approach. The total risk is calculated summing the individual non-threshold carcinogenic risk or threshold hazard quotients to obtain a total non-threshold risk and total threshold hazard index. With respect to interactions between chemicals the use of large margins of safety within the values adopted for the quantification of toxicity is considered sufficient to account for potential interactions with other chemicals. This approach assumes additivity irrespective of whether the toxic endpoints are the same or different. This approach is taken in lieu of specific information or guidance on actual additive effects. The result is conservative when it is also considered that the toxicity values incorporate a range of safety factors. This differs from an approach that calculates risks to human health based on end-points. Further discussion on the complex issue of assessing mixtures is presented in the toxicity summaries in **Appendix B**.

With respect to BTEX, ATSDR (2004) has undertaken a review of the interaction of these chemicals. In summary, the review identified that exposures to BTEX are best assessed using an individual chemical approach that considers shared neurological effects (where an additive approach is appropriate) and unique haematologic/carcinogenic effects (where the carcinogenic assessment of benzene alone is adequate). Application of this approach, however requires that the threshold values adopted for the assessment of BTEX are relevant to the protection of neurological effects. This has been reviewed and presented in the summary in **Appendix B** for BTEX mixtures. Essentially the assessment requires further consideration of benzene threshold effects to address overall neurotoxicity effects from BTEX. The values adopted for benzene are:

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- Oral RfD = 0.004 mg/kg/day available from IRIS (USEPA) based on decreased lymphocyte count; and
- Inhalation RfC = 0.03 mg/m³ available from IRIS (USEPA) based on decreased lymphocyte count.
- Background intake of benzene of 10% considered relevant to the area of the site.

No other data is available regarding specific interactions of other COPC identified that would not be adequately addressed on the basis of additivity of the HI or cancer risk using the toxicity values identified in this Section. It is noted that the assessment of a range of chlorinated compounds presented in this report have similar toxicological endpoints with a number demonstrating similar metabolic pathways that are considered to be associated with the toxicological effects observed (rather than the parent compound itself). For these compounds the consideration of cumulative exposure on the basis of additivity is considered appropriate.

4.4 Intermediate Duration Toxicity Values

As noted in **Section 4.1**, the assessment of potential inhalation exposures to workers on Southlands (associated with emissions to air from volatile COPC identified in Springvale Drain) requires the consideration of chronic exposures (addressed above) as well as intermediate duration exposures. On this basis toxicity values have been identified for the assessment of short or intermediate (i.e. between acute and chronic) duration events. These have been selected as follows:

- The quantification of non-threshold carcinogenic risk has been undertaken using the non-threshold values presented in **Table 4-1**. The assessment of non-threshold carcinogenic risk does not change with exposure type/duration as it is assumed that any exposure (even a single event) may result in the increased risk of contracting cancer over a lifetime (even after exposure has stopped). In relation to the COPC identified for the assessment of air emissions from Springvale Drain this is relevant to the assessment of vinyl chloride, EDC, TCE and chloroform.
- The quantification of threshold, or non-carcinogenic, effects has been undertaken using intermediate duration threshold values (referred to as Minimal Risk Levels, or MRLs) available from the ATSDR¹⁷. These are derived by the ATSDR to be protective of exposures by all members of the population for inhalation exposures to the COPC for an intermediate duration of time.

Table 4-2 presents a summary of the toxicity values adopted for the quantification on intermediate duration exposures (refer to **Appendix F** for calculations relevant to these exposures).

¹⁷ MRLs available from ATSDR at: <http://www.atsdr.cdc.gov/mrls/>

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Table 4-2 Summary of Toxicity Data for Intermediate Duration Inhalation Exposures

COPC	Non-Threshold Carcinogenic Inhalation Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	Threshold Non-Carcinogenic Tolerable Concentration (or MRL*) (mg/m^3)
carbon tetrachloride		0.19 ^H
chloroform	4.2x10 ⁻⁷ (WHO)	0.24 ^H
methylene chloride		1.04 ^H
1,1,2-trichloroethane		(0.04 mg/kg/day)** ^H
1,2-dichloroethane (EDC)	2.8x10 ⁻⁶ (WHO)	2.43 ^{H,C}
tetrachloroethene (PCE)		0.20 ^{N,C} (WHO)
trichloroethene (TCE)	4.3x10 ⁻⁷ (WHO)	0.54 ^N
1,1-dichloroethene		0.079 ^H
cis-1,2-dichloroethene		1.19 ^{Ha}
trans-1,2-dichloroethene		0.79 ^H
vinyl chloride	8.8x10 ⁻⁶ (USEPA)	0.077 ^H
hexachlorobutadiene (HCBd)		(0.0002 mg/kg/day)** ^R
hexachloroethane (HCE)		58.1 ^N

Notes:

* All values presented are MRL's presented by the ATSDR, unless otherwise noted. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. The inhalation value is equivalent to a tolerable concentration in air.

** No inhalation specific value available, hence oral value relevant to intermediate duration exposure has been adopted for the purpose of this assessment.

Threshold non carcinogenic values based on critical end points: H = hepatic effects, Ha = haematological effects, R = renal effects, N = neurological effects, C = value based on chronic effects as no intermediate duration value available.

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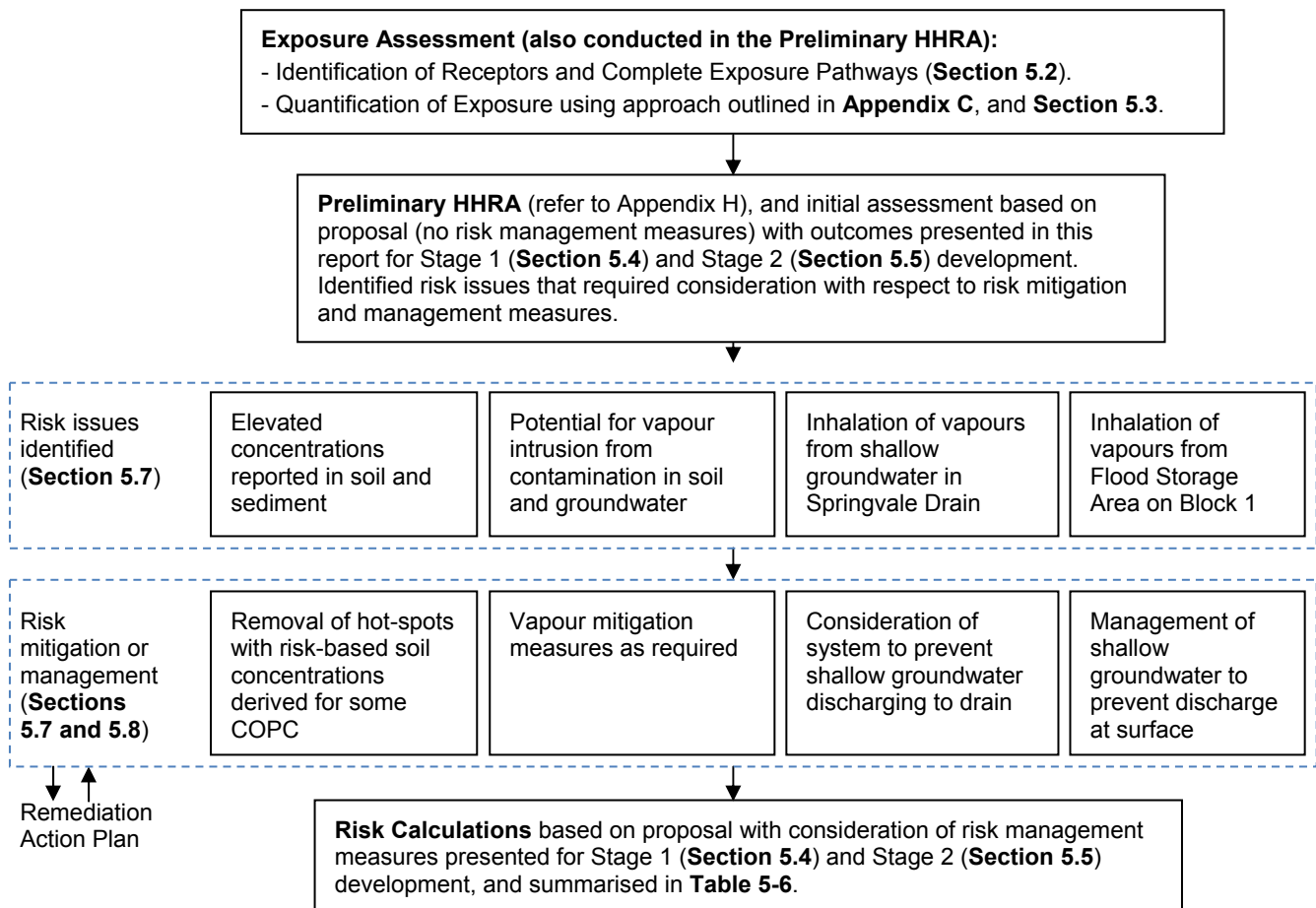
5.1 General

This section identifies the human populations (receptors) who may be exposed to the COPC identified, outlines the mechanisms (pathways) by which these populations may be exposed and provides a quantitative estimate of exposure, chemical intake and risk. The overall approach is outlined in **Sections 1.5.3 and 1.5.4** with further detail presented in this Section.

It is noted that the quantification of exposure and risk associated with the proposed development at Southlands is complex and has included a preliminary assessment and a number of staged reviews of risk management issues. A preliminary HHRA (included in Appendix H for reference) provided a review of risk issues assuming no risk management measures were implemented. The aim of the assessment was to identify the key drivers of risk at the site and the areas where risk management measures are to be implemented.

The outcome of the preliminary assessment is incorporated in the exposure scenarios considered and presented in this report. In addition, where risk management measures were identified, these were incorporated within the RAP. The proposed risk management measures were, in some cases, further considered and assessed to establish, as least in theory, the likely outcome of these measures on the calculated risk. These are presented in the following sections.

The following flow diagram presents an overview of the process adopted for the quantification of exposure and risk at this site.



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5.2 Identification of Complete Exposure Pathways

The identification of receptors and complete exposure pathways relevant to the proposed development and use of Southlands has considered the following:

- The site is zoned for commercial/industrial use and the proposed development is consistent with this zoning. In addition the proposed development does not allow for more sensitive uses of the site (such as childcare or healthcare facilities) hence the potential uses of Southlands are limited to commercial/industrial purposes.
- Groundwater will not allowed to be extracted for use on the site as Southlands is located within the Groundwater Extraction Exclusion Area (GEEA) and any potential future use of groundwater should be excluded under the long term environmental management plan.
- Groundwater extraction wells and associated pipe-work associated with the primary containment lines for the GTP are located along the McPherson Street boundary of the property. These are to remain with the pipe-work to be redirected (once designs are completed). Orica is to retain ownership of the easements used for this infrastructure. It is also understood that other easements are to be designated on the Southlands property for the purpose of ongoing trials as needed. While the current proposal has provided an indication of the location of these easements, the presence of these easements and others that may be required in the future have not been accounted for in this assessment.
- As the site is proposed to be predominantly sealed and landscaped following development the **long term generation of dust is not considered significant**. The generation of dusts during remediation and/or development should, however be managed to minimise dust emissions from the area and be addressed as part of the environmental management of the works. Following development, large open areas (such as the flood storage areas, should be seeded or compacted to prevent dust generation.
- The proposed development of the site involves the filling in of the existing ponds on Block 1, hence exposure associated with water in the existing ponds is considered **incomplete**. The sediment within and adjacent to the ponds may be removed, dried out and moved across the site during the development and as such contaminants in the sediment have been treated as soils in the HHRA.
- Stormwater runoff from the site is to be managed within compensatory flood storage areas and, as the site is expected to be predominantly paved, the runoff is not expected to come into contact with soil on the site after development. Hence the potential for stormwater runoff from the site to be affected by the presence of COPC in soil is considered negligible and hence **no significant exposure pathways** have been identified. Stormwater during development construction works would be addressed as part of the environmental management of the works.
- Shallow groundwater has, in the past, discharged to the ponds on Block 1. It is understood from the Flood Management Study (Connell Wagner, 2007) that the works associated with the placement of compensatory flood storage areas on Block 1 will involve excavation beneath a level where shallow groundwater could seep into and discharge directly to the compensatory flood storage areas. The proposed compensatory flood storage area is to be fenced off to restrict access. In addition the basin is expected to be constructed with subsurface drainage and crushed concrete (or similar) to ensure that groundwater near the surface of the pond is drawn away in the subsurface drainage system thereby limiting the potential for discharge. The drainage water (shallow groundwater) captured via this system is proposed to be directed to the GTP in a closed system. Hence shallow groundwater is not expected to be present at the ground surface with the only complete exposure pathway identified being the inhalation of volatile COPC that may migrate from the subsurface to outdoor air when the compensatory flood storage area is dry. However, should the plans change during development such that shallow groundwater is required to discharge into a compensatory flood storage area or similar structure, then re-evaluation of the risks will be required to ensure the proposed changes do not present unacceptable risks to human health (refer to [refer to **Appendix H**]

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for assessment of issues associated with the discharge of shallow groundwater into ponds or compensatory flood storage areas, URS 2006c).

- To address potential risk issues identified (PHHRA, URS 2006c) with respect to the inhalation of volatile COPC from Springvale Drain, a shallow groundwater extraction system is proposed to be installed adjacent to Springvale Drain to intercept shallow groundwater thereby reducing discharge into the drain. These wells are expected to be connected to the GTP and when operational, shallow groundwater is not expected to discharge into Springvale Drain. Hence the inhalation of volatile COPC associated with shallow groundwater discharge adjacent to Springvale Drain is considered **incomplete** while the GTP is operating (provided that the system operates as predicted). However when the GTP is offline there is the potential for shallow groundwater to rise to a level where it discharges into Springvale Drain, resulting in short term emissions to air of volatile COPC. Issues associated with these short-duration emissions require further assessment. It is understood that Orica is in the process of designing and seeking approval for a backup system (such as the Temporary Aquifer Storage and Recovery (Tasar) system or other appropriate system). Further details are provided in the RAP.
- The flood study (Connell Wagner 2007) indicated that the surface elevation needed to be increased prior to the development of Block 2 (Stage 1) to meet the design flood criteria. Raising the surface level is expected to be achieved through the limited placement of materials excavated from the southern portion of Block 1 onto Block 2, importation and placement of validated filled and construction of a concrete slab across the majority of the finished surface. On this basis direct contact with soil left in place on Block 2 is considered **incomplete**. During the Stage 1 development the southern portion of Block 1 will not include use of any fill materials (rather materials are expected to be excavated) and hence direct contact with soil in this area is considered relevant. However during the Stage 2 development, as with Stage 1 fill materials are expected to be placed on the southern portion of Block 1 to raise the surface elevation to meet the design flood criteria prior to the construction of buildings, hence direct contact with soil left in place in this area is also considered **incomplete**. Access to the northern portion of Block 1 is to be restricted.

Based on the landuse of the site and the nature and extent of contamination identified on the site, the following exposure pathways are considered to be complete and warrant further assessment.

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Table 5-1 Summary of Key Exposure Pathways

Receptor	Exposure Pathway	Activity
Adult workers – within Stage 1 (Block 2 and part of Block 1)	Inhalation of vapours of volatile chemicals in shallow groundwater and soil, emissions from the compensatory flood storage area, emissions from Floodvale and Springvale Drains (when proposed system is installed and the GTP and backup system is not operating) and emissions from other sources on the BIP. Direct contact (incidental ingestion and dermal contact) with contaminated soil and sediment in ephemeral ponds (following placement in areas where clean covering fill is not present after development)	Working on the site following redevelopment as well as gardening and non-intrusive maintenance work (such as maintenance of GTP aboveground infrastructure)
Adult workers – within Stage 2 (Block 1)	Inhalation of vapours of volatile chemicals in shallow groundwater and soil, emissions from Springvale Drain and emissions from other sources on the BIP.	Working on the site following redevelopment as well as gardening and non-intrusive maintenance work (such as maintenance of GTP aboveground infrastructure)
Adult workers in all areas of the site undertaking intrusive works	Inhalation of vapours of volatile chemicals in shallow groundwater and soil. Exposures also associated with emissions from the compensatory flood storage area, emissions from Floodvale and Springvale Drains (when GTP and backup system is not operating) and emissions from other sources on the BIP – however these will be lower than for long-term workers due to limited time spent on site and do not warrant separate assessment. Direct contact (incidental ingestion and dermal contact) with contaminated soil/sediment and shallow groundwater	Intrusive works undertaken to repair or replace subsurface services. Such works need to be completed in accordance with the long term environmental management plan.

Exposures by visitors would be lower than long term workers due to the shorter duration of time spent on site. Hence quantification of risks for visitors is not required. The proposed road adjacent to Springvale Drain is to be a private road and hence will not be accessed by the general public except on an occasional basis. More sensitive uses allowable under the Industrial 4(a) zoning such as a childcare facility has not been considered in this assessment. As such, only adult commercial and industrial workers are relevant to the assessment. Should alternate allowable uses be proposed, then the risks would need to be re-evaluated prior to approval.

Exposures by intrusive workers on the site (including intrusive works associated with the remediation works and maintenance of GTP infrastructure) have been included to provide information on the level of risk required to be addressed in the long term environmental management plan. Exposures by workers involved in the maintenance of Springvale and Floodvale Drains have not been specifically assessed. Rather it has been assumed that such works will be managed as an occupational health and safety matter under a site specific management plan that includes health and safety requirements relevant to the specific tasks to be undertaken and taking into account the nature of the contamination. Such a plan will also be required for the site following redevelopment and may include consideration of confined spaces as well as contact with water and inhalation of vapours when working in an open drain or any other space where shallow groundwater is present.

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5.3 Quantification of Exposure

5.3.1 General

Exposures associated with the key exposure pathways identified (refer to **Table 5-1**) have been quantified following the methodology presented in **Appendix C**. This appendix provides the key equations adopted to estimate chemical intake from each complete exposure pathway assessed, including inhalation of volatiles, ingestion and dermal contact with soils, sediments and shallow groundwater.

The quantification of exposure requires the quantification of chemical intake, which can then be compared against relevant toxicological values (refer to **Section 4**) to estimate a level of risk for each stage of the proposed development. As noted in **Section 1.5.3**, the following steps have been followed to estimate chemical intake:

- Identification of **exposure parameters** for each of the identified exposure pathways and receptors. These are values that describe the physical and behavioural parameters relevant to the potentially exposed population and the pathway of exposure. Some examples include ingestion rate (e.g. amount of soil or dust ingested at work each day), inhalation rate (volume of air inhaled during different activities), exposure frequency (i.e. hours per day or days per year), exposure duration (e.g. number of years at work) and body weight. Where available, exposure parameters have been obtained from Australian sources (enHealth 2002, CSMS, 1991, 1993, 1996 and 1998, and NEPC 1999). These are specific for each exposure pathway assessed and are presented in **Sections 5.4 and 5.5**.
- Calculation of **intake factors**. An intake factor is calculated using the exposure parameters defined above and provides a site specific and receptor specific value which, when multiplied by the concentration of each COPC, provides an estimate of the daily chemical intake of the COPCs for each receptor and pathway. Due to the number of exposure pathways and scenarios presented, the calculated intake factor is not presented in the report, however the calculated intake factors are presented in the risk calculations presented in **Appendices D, E, F and G** (where relevant).
- Estimation of the **chemical concentration** in each medium relevant to the receptor groups and exposure pathways. This involves the estimation of potential concentrations in air and other media such as soil and groundwater (where relevant to the assessment of a complete exposure pathway). The approach adopted in this assessment is summarised in **Section 5.3.2**; and
- Calculation of the **daily chemical intake** using the intake factor and the chemical concentration. Due to the number of exposure pathways and scenarios presented, the calculated chemical intake is not presented in the report, however the calculated intake factors are presented in the risk calculations presented in **Appendices D, E, F and G** (where relevant).

5.3.2 Exposure Concentrations

The exposure concentrations adopted for the assessment of the Stage 1 and Stage 2 development are summarised for each exposure pathway and scenario addressed and presented in **Sections 5.4 and 5.5**.

Soils

The Stage 1 development involves the placement of fill over the majority of Block 2, thence the potential for direct contact with site related soils in Block 2 is negligible. Block 1 will remain accessible.

Prior to any development soils from both Block 1 and Block 2 are expected to be moved across the site during remediation and other earthwork activities. Hence the potential concentration of COPC in soils in areas that are accessible (Block 1 or portions of Block 2) following the Stage 1 development is unknown (assuming no remediation of soils), hence the concentrations adopted for the purpose of this assessment

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are the maximum concentrations of COPC identified in soils on either Block 1 or Block 2. The maximum concentrations in these areas are presented in **Tables 3-3 and 3-4**.

Sediments

As discussed in **Section 3.6**, the existing ponds on Block 1 are to be filled in as part of the proposed development. While this is expected to eliminate the potential for exposure to water within the existing ponds, sediments from the ephemeral (including drains) and permanent ponds may be moved across the site during earthworks. Hence the concentrations considered in this assessment (assuming no remediation) are the maximum concentrations of COPC reported in sediments presented in **Table 3-6**.

Groundwater

Inhalation exposures associated with the presence of COPC in groundwater have been assessed on the basis of measured flux emissions or soil gas, as discussed below. The only complete pathway identified for direct contact with shallow groundwater is works undertaken by intrusive workers on the site, where excavations extend into the shallow groundwater. Concentrations of COPC considered relevant for the assessment of potential exposures to shallow groundwater are the maximum concentrations reported in **Table 3-2**. These concentrations have also been used to assess potential risks associated with the flood storage area proposed on Block 1 as part of the Stage 1 development.

Air

Concentrations of COPC have been considered in air for a range of sources and scenarios. The following provides a summary of the air concentrations considered in the quantification of exposure from the emission sources identified.

Emissions derived from Other Sources on the BIP

Potential sources of emissions to air derived from other sources identified on the BIP have been discussed in **Section 3.9**. The concentrations of all COPC relevant to the assessment of exposures on Southlands derived from other sources are presented in **Table 3-11**.

Vapour Migration from Subsurface Sources

Potential concentrations in indoor and outdoor air have been estimated using a vapour model. The models adopted for this assessment, where the proposed buildings are slab-on-grade only, have been selected based on the nature of the data considered. Details on the models utilised are provided in **Appendix C**, however the following provides additional detail on the models adopted and data utilised for key scenarios assessed.

Air concentrations have been estimated based on measured soil gas and flux emissions data collected above impacted shallow groundwater and some soil source areas. Where this data is available, the maximum soil gas concentration (assumed to be at 0.5m, beneath proposed fill materials) reported in **Table 3-9** has been used to estimate an emission rate from the ground surface. The estimated emission rate based on the soil gas data has been compared with the maximum measured flux emission rate for each block (presented in **Tables 3-7 and 3-8**). The maximum emission rate estimated from soil gas data or measured has then been used in the relevant vapour model to estimate an indoor or outdoor air concentration. For this approach the vapour model described by Farmer (1980) and DEH (2000) has been used to estimate indoor air concentrations. The outdoor air model described by USEPA (1996 and 2001) has been used. For the assessment of emissions from Block 1 and Block 2 the assessment has been presented based on both maximum and average soil gas and flux emission rates.

For locations on the site above volatile COPC identified in soil and sediment where no soil gas or flux emissions data is available (refer to **Sections 3.5.2 and 3.6**), potential air concentrations indoors and outdoors have been estimated based on the maximum reported concentration in soil (refer to **Table 3-5**) or sediment (refer to **Table 3-6**) and a vapour model. For this approach the vapour model described by ASTM (2002) has been used to estimate indoor air concentrations (assuming the source is at 0.5m,

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beneath proposed fill materials) as well as outdoor air concentrations above a surface source. Due to limitations in the data, air concentrations derived from maximum soil or sediment concentrations have been assessed. No average calculation is presented.

Emissions to Air from Floodvale Drain

Exposures associated with emissions to air derived from Floodvale Drain have been quantified using the maximum measured air concentrations reported adjacent to the drain. The maximum concentrations reported for the COPC identified are presented in **Table 3-10**. The assessment has assumed the maximum concentrations are relevant to exposures anywhere on the site, which is conservative as it does not account for further dispersion from the source area.

Emissions to Air from Springvale Drain

Exposures associated with emissions to air derived from Springvale Drain have been quantified using the maximum measured air concentrations reported adjacent to the drain. More data is available for Springvale Drain that address different conditions. Higher concentrations were reported in air adjacent to the drain prior to the operation of the GTP (to end of 2006), with lower concentrations reported following commissioning and operation of the GTP (from 2007). Emissions to air from the drain are associated with the discharge of shallow groundwater into the drain. The potential for discharge of shallow groundwater into the drain has been observed to be affected by the operation of the GTP, hence the quantification of exposure has considered potential concentrations when the GTP is operating and when it is not operating. Data available from adjacent to Springvale Drain is presented in **Table 3-10**. This data has been used to consider the following scenarios:

- Average and maximum concentrations reported prior to the operation of the GTP (to end of 2006), considered relevant to potential exposures should risk management measure not be implemented or operating (including the GTP being off-line); and
- Maximum concentration reported following operation of the GTP (from January 2007 to August 2007 only).

The assessment has assumed the concentrations considered are relevant to exposures anywhere on the site, which is conservative as it does not account for further dispersion from the source area.

Emissions to Air from Flood Storage Area

The proposed flood storage area on the southern portion of Block 1 as part of the Stage 1 development has the potential to intersect shallow groundwater. An assessment of the potential for this to occur and potential concentrations in air associated with this scenario is presented in **Appendix E**. The appendix also presents an assessment of the proposed mitigation measure.

In summary, air concentrations, however have been estimated based on a vapour model. The models adopted are as follows (refer to **Appendices C and E** for details):

- Estimation of air concentrations above area where shallow groundwater discharges to the surface based on a model of emissions from water with quiescent flow as described by the USEPA (1994) and shallow groundwater concentrations presented in **Table 3-2**.
- Estimation of air concentrations above the area where mitigation measures are implemented and no groundwater discharges to the surface, but may remain, on average, 0.5m below the surface, is based on the outdoor air vapour migration model (from groundwater source) to outdoor air as described by the USEPA (1996). Shallow groundwater concentrations considered are presented in **Table 3-2**.

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Concentration in Excavation into Groundwater

The concentration in air that may be present in an excavation that extends to the shallow groundwater has been estimated based on the shallow groundwater concentrations presented in **Table 3-2** and an attenuation factor as presented in **Appendix C**.

5.4 Quantification of Chemical Intake and Risk – Stage 1 Development

The quantification of risk associated with exposures relevant to the Stage 1 development has considered the proposed development as outlined in **Section 1.3** and adopted the methodology presented within the Consolidated HHRA (URS, 2005a). **Appendix C** outlines the equations used in the quantification of exposure and risk that are consistent with the overall methodology presented in **Section 1.5**.

The assessment of potential exposure (also refer to **Section 5.3**) and risk that may be associated with the Stage 1 development has been undertaken, as outlined in **Section 5.1**, based on a range of scenarios that address potential exposure and risk with and without the consideration of mitigation and management measures outlined in the RAP.

The following has been considered in the assessment of potential exposure and risk as part of the Stage 1 development:

- The development of Stage 1 will principally involve construction of commercial buildings on Block 2. No buildings will be constructed on Block 1. The southern portion of Block 1 (Stage 2) is expected to be landscaped and may be accessible; however the northern portion of Block 1 (proposed Stage 3) will be inaccessible with secure fencing.
- A physical barrier (fill and concrete surface) will be placed over most of Block 2 prior to development to meet the design flood criteria. Some areas will remain (provided the residual soil concentrations meet requires outlined in the RAP) without concrete surface covering or fill as they are to be used for open space or landscaping areas. No physical barrier is proposed for Block 1 during the Stage 1 development.
- Soil may be moved across the site, predominantly from Block 1 to Block 2 during earthworks associated with the development.
- Ponds on Block 1 are to be filled in as part of Stage 1.
- The compensatory flood storage area on Block 1 will be designed such that shallow groundwater will be drained, minimising the potential for surface seepage¹⁸. The calculation of risk has been undertaken assuming the effective operation of this system and for the scenario where the drainage system is not present (or not operational).
- A shallow groundwater extraction system is proposed to be installed along Springvale Drain intersecting shallow groundwater prior to discharge into the drain. This will be connected into the GTP¹⁷. While this system is proposed, the calculation of risk has been undertaken based on a range of scenarios. These include the GTP not operating, the GTP operating without the shallow groundwater extraction system and the GTP operating with the shallow groundwater extraction system installed and controlling discharge to the drain.

Based on these assumptions, a summary of the exposure parameters adopted for the quantification of chemical intake for the Stage 1 development and a summary of the concentrations assumed for calculation of risks are presented in **Table 5-2** for long-term commercial workers, **Table 5-3** for gardeners

¹⁸ The effectiveness of the shallow groundwater extraction system needs to be proven. If the system proposed does not meeting the proposed objectives then risks to workers on the site will need to be revised.

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and non-intrusive maintenance workers and **Table 5-4** for intrusive workers. Exposure parameters representative of a RME scenario have been selected for each of the receptor groups and exposure pathways. Where available, exposure data has been obtained from Australian sources (CSMS, 1991, 1993, 1996 and 1998, ANZECC 1992, NEPC 1999 and enHealth 2002 and 2003).

The calculated risks to human health associated with these exposures are also presented in these tables. Details of the risk calculations are presented in **Appendix D** and as noted in the Tables. The calculations relate to all COPC identified with the exception of asbestos. Issues associated with the presence of asbestos in soil for the site are addressed in **Section 5.6**.

Risk levels considered representative of acceptable risk (i.e target risk levels) are:

- non-threshold risk (sum over all pathways) = 1×10^{-5} (i.e. 1 in 100,000); and
- threshold hazard index (HI) (sum over all pathways) = 1.

Calculated risks for workers at the site that exceed the risk target values are highlighted in the summary tables. The following provides an overview of the exposure pathways and scenarios considered for the proposed Stage 1 development with reference to the relevant tables and exposure scenarios presented.

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Exposure Pathway Quantified and Scenario Considered for Each Receptor – Stage 1

Commercial Workers (Table 5-2)

- Inhalation indoors and outdoors of COPC from sources on BIP (**Scenario 1**)
- Inhalation indoors and outdoors of volatiles from groundwater and some soil sources (from measured soil gas and flux emissions data) (**Scenario 2**)
- Inhalation indoors and outdoors of volatiles from remaining soil and sediment hot-spots (**Scenario 3**)
- Inhalation indoors and outdoors of volatiles from Floodvale Drain (**Scenario 4**)
- Inhalation indoors and outdoors of volatiles from Springvale Drain
 - Risk issues were identified in Preliminary HHRA (Appendix H)
 - RAP has considered the implementation of a mitigation system
 - Calculation of risk has considered:
 - proposed system not operating (**Scenario 5**); and
 - proposed system operational (to be verified) (**Scenario 6**)
- Inhalation outdoors of volatiles from proposed Flood Storage Area
 - Calculation of risk based on discharge of shallow groundwater into area (**Scenario 7**) identified the potential for unacceptable risk
 - RAP has considered the implementation of a mitigation system
 - Calculation of risk based on the proposed mitigation system (drainage) operating (to be verified) (**Scenario 8**)
- Ingestion and dermal contact with soils and sediments that may be exposed at the surface (**Scenario 9**)

Gardeners and Maintenance Workers (non-intrusive works) (Table 5-3)

- Inhalation outdoors of COPC from sources on BIP (**Scenario 1**)
- Inhalation outdoors of volatiles from groundwater and some soil sources (from measured soil gas and flux emissions data) (**Scenario 2**)
- Inhalation outdoors of volatiles from remaining soil and sediment hot-spots (**Scenario 3**)
- Inhalation of volatiles from Floodvale and Springvale Drains
 - Risk issues were identified in Preliminary HHRA (**Appendix H**)
 - RAP has considered the implementation of a mitigation system
 - Calculation of risk has considered:
 - proposed system not operating (**Scenario 4**); and
 - proposed system operational (to be verified) (**Scenario 5**)
- Inhalation outdoors of volatiles from proposed Flood Storage Area (not presented as this scenario is the same as for commercial worker as this was assessed for an outdoor worker)
- Ingestion and dermal contact with soils and sediments that may be exposed at the surface (**Scenario 6**)

Intrusive Workers (intrusive activities only) (Table 5-4)

- Inhalation outdoors of volatiles from groundwater (assuming excavations extend into shallow groundwater) and soil sources (**Scenario 1**)
- Ingestion and dermal contact with soils and sediments during excavation (**Scenario 2**)
- Ingestion and dermal contact with shallow groundwater during excavation works (**Scenario 3**)

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Table 5-2 Summary of Exposure Pathways, Assumptions and Calculated Risks – Commercial Workers for Stage 1

Scenario/ Exposure Pathways		Chemical Concentrations	Exposure Parameters	Calculated Risk	
				Non-Threshold Risk	Threshold HI
			<p>Parameters relevant for all pathways</p> <p>Body weight of 70 kg. Exposure for 10 hours per day for 240 days/year (5 days per week for 48 weeks) for 30 years</p>		
1	Inhalation of COPC in ambient air from emission sources within the BIP	Modelled ground level concentrations based on emissions from the BIP that include the GTP, HCB repackaging, proposed CPWE remediation and other sources on BIP (refer to Table 3-11)	Inhalation of 1.17 m ³ air per hour indoors (9 hours) and 2.2 m ³ air per hour outdoors (1 hour).	1.5x10 ⁻⁹	0.0047
2	Inhalation of volatile chemicals derived from shallow groundwater beneath Southlands	<p>Measured soil gas and flux emission data (maximum and average) collected in areas above shallow groundwater plumes relevant to the proposed development on Block 2 (construction of buildings and outdoors) and Block 1 (outdoors only), refer to Section 5.3.2.</p> <p>Modelled air concentrations indoors (based on average and maximum measured data for Block 2) and outdoors (based on maximum estimated from maximum data from Block 1 or Block 2) are presented in Appendix D (refer to methodology adopted in Appendix C).</p>	<p>Inhalation of 1.17 m³ air per hour indoors on Block 2 (9 hours)</p> <p>Inhalation of 2.2 m³ air per hour outdoors or Block 2 or Block 1 (1 hour)</p>	<p>3.4x10⁻⁷ (average)</p> <p>8.9x10⁻⁷ (maximum)</p> <p>1.6x10⁻⁷</p>	<p>0.095 (average)</p> <p>0.19 (maximum)</p> <p>0.10</p>
3	Inhalation of volatile chemicals identified in soils/sediments that may be relocated across the site	<p>Modelled air concentrations based on maximum concentration reported in soil or sediment hot-spots (refer to Sections 3.5.2, 3.6 and 5.3.2) (Appendix D). Modelling has focused on:</p> <p>- Vapour intrusion into buildings on Block 2 assuming movement of soil from Block 1</p>	Inhalation of 1.17 m ³ air per hour indoors (9 hours)	<p>5.2x10⁻⁴ (soil)</p> <p>3.8x10⁻³</p>	<p>6 (soil)</p> <p>12</p>

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Scenario/ Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
	- Volatile emissions to outdoor air on Block 1 →	Inhalation of 2.2 m ³ air per hour outdoors (1 hour)	(sediment) 1.2x10 ⁻⁷ (soil) → 8.9x10 ⁻⁷ (sediment)	(sediment) 0.010 (soil) 0.065 (sediment)

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Table 5-2 (Continued) Summary of Exposure Assumptions and Calculated Risks – Commercial Workers for Stage 1

Scenario/ Exposure Pathways		Chemical Concentrations	Exposure Parameters	Calculated Risk	
4	Inhalation of vapours derived from Floodvale Drain	Measured air concentrations (maximum) adjacent to Floodvale Drain (Table 3-10)	Inhalation of 1.27 m ³ air per hour indoors (9 hrs) /outdoors (1 hr) (total 10 hours)	<u>Non-Threshold Risk</u> → 3.9x10 ⁻⁶	<u>Threshold HI</u> NA as no threshold COPC reported
Inhalation of vapours from Springvale Drain (range of scenarios presented)					
5	GTP and backup systems not operating at all or where GTP is operating and shallow groundwater discharge is not controlled.	Measured air concentrations (average and maximum before GTP operating [prior to 2007] and maximum reported after full commissioning of the GTP, refer to Table 3-10) adjacent to Springvale Drain.	Inhalation of 1.27 m ³ air per hour indoors (9 hrs) /outdoors (1 hr) (total 10 hours)	→ 6.4x10 ⁻⁴ (average) 1.3x10 ⁻³ (maximum) 5.3x10 ⁻⁴ (post GTP)	8.0 (average) 17 (maximum) 6.8 (post GTP)
			Inhalation of 2.2 m ³ air per hour outdoors (4 hours) in work areas directly adjacent to the open drains (where no buildings are constructed)	→ 4.4x10 ⁻⁴ (average) 9.0x10 ⁻⁴ (maximum) 3.6x10 ⁻⁴ (post GTP)	5.5 (average) 11 (maximum) 4.7 (post GTP)
6	GTP and or backup systems operating and proposed shallow groundwater extraction system installed and operating	Concentrations to be verified by monitoring – assumed to be below the analytical limit of reporting if system operates as proposed Concentrations that may occur if the GTP is offline have not been addressed separately, however an assessment has been undertaken to identify the maximum number of days in which the GTP can be offline before inhalation exposures approach risk values of concern. This is presented in Appendix F.		Negligible (but to be confirmed)	Negligible (but to be confirmed)

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Table 5-2 (Continued) Summary of Exposure Assumptions and Calculated Risks – Commercial Workers for Stage 1

Scenario/ Exposure Pathways		Chemical Concentrations	Exposure Parameters	Calculated Risk	
Inhalation of vapours derived from Flood Storage Area (range of scenarios presented)*					
7	No drainage system installed →	Modelled concentration in air above shallow groundwater that is assumed to be present as surface water in the compensatory flood storage area. Air concentrations estimated using volatilisation model for estimating emissions from water with queiscent flow (USEPA 1994). Assumed diameter of basin of 170m, depth of surface flow of 1cm and a flow rate of 0.015 m ³ /s. Concentrations in surface water discharge as in Table 3-2 . Concentrations in air estimated for workers 10m from the basin.	→ Inhalation of 2.2 m ³ air per hour outdoors (8 hours) in work areas directly adjacent to the basin. No buildings located next to basin hence outdoor exposures assessed. Assume groundwater discharges to basin 18% of the total work time (i.e. 18% of time spent at work), refer to Appendix E for further detail.	3.9x10 ⁻⁵	2.1
8	Drainage system effectively operating – no discharge of shallow groundwater to compensatory flood storage area →	While no shallow groundwater discharges into basin, it remains just below surface of the compensatory flood storage area. Assumed shallow groundwater with concentrations equal to those presented in Table 3-2 remain 30cm below ground surface. Concentrations in breathing zone estimated using an outdoor air model (USEPA, 1996 and 2001) assuming overlying soils are similar to fill and not saturated for 82% of the year.	→ Inhalation of 2.2 m ³ air per hour outdoors (8 hours) in work areas directly adjacent to the basin. No buildings located next to basin hence outdoor exposures assessed.	1.2x10 ⁻⁶	0.086

* Exposures relevant to all workers in outdoor areas on the site (such as maintenance and intrusive workers)

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Table 5-2 (Continued) Summary of Exposure Assumptions and Calculated Risks – Commercial Workers for Stage 1

Scenario/ Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk
Direct Contact Exposures			
<p>9 Ingestion and dermal contact with chemicals in soil and sediment remaining exposed as part of Stage 1</p>	<p>Soil and sediment concentrations from available data assuming soil and sediment from the ephemeral ponds may be moved across the site. Maximum soil concentration from all available data from Block 1 and Block 2, refer to Tables 3-3 and 3-4. Maximum sediment concentration from Table 3-6.</p>	<p>Exposure on Block 2: Incomplete due to placement of clean fill. Exposure on southern portion of Block 1 based on soil or sediment. Ingestion of 25 mg of soil or sediment per day by adults. Once ingested it is assumed that 100% is absorbed into the body. Dermal: When outdoors it is assumed that the hands and forearms get dirty each day (2300 cm² of skin). Once dirty it is assumed that 0.51 mg of soil or sediment adheres to each cm² of skin. Assume an adult will wash at the end of each day resulting in up to 12 hours of the day dirty. Maximum risk calculated using either Hawley or USEPA methodology.</p>	<p>→ 7.2x10⁻⁶ (soil) 4.4x10⁻⁵ (sediment) 3.8 (soil) 0.54 (sediment)</p> <p>→ 3.7x10⁻⁶ (soil) 1.9x10⁻⁵ (sediment) 0.91 (soil) 0.19 (sediment)</p>

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Table 5-3 Summary of Exposure Assumptions and Calculated Risks – Gardeners/Maintenance Workers

Exposure Pathways		Chemical Concentrations	Exposure Parameters	Calculated Risk	
			Parameters relevant for all pathways Body weight of 70 kg. Exposure for 10 hours per day (i.e. long work day) once every 2 weeks (gardening) over 30 years	Non-Threshold Risk	Threshold HI
1	Inhalation of COPC in ambient air from emission sources within the BIP	Modelled ground level concentrations based on emissions from the BIP that include GTP, HCB repackaging, proposed CPWE remediation and other sources on BIP (refer to Table 3-11)	2.2 m ³ air per hour outdoors (assuming all 10 hours is spent outdoors).	2.9x10 ⁻¹⁰	0.00088
2	Inhalation of vapours from volatile chemicals in shallow groundwater	Measured soil gas and flux emission data (maximum from Block 1 and Block 2) collected in areas above shallow groundwater plumes, outdoor exposure only. Air concentrations modelled from measured data as presented in Appendix D .	2.2 m ³ air per hour outdoors (assuming all 10 hours is spent outdoors).	1.7x10 ⁻⁷	0.11
3	Inhalation of volatile chemicals identified in soils or sediments that may be moved across site	Modelled air concentrations based on maximum concentration reported in soil or sediment hot-spots (Appendix D). Modelling of air concentrations has focused on: - Volatile emissions to outdoor air on Block 1 or Block 2 assuming soil or sediment movement	Inhalation of 2.2 m ³ air per hour outdoors (10 hours)	9.7x10 ⁻⁷	0.076
4	Inhalation of volatile emissions derived from Springvale and Floodvale drains	Measured air concentrations (maximum) adjacent to Springvale and Floodvale Drains when GTP not operating.	Inhalation of 2.2 m ³ air per hour outdoors in work areas directly adjacent to the open drains	2.4x10⁻⁴	3.1
5	Inhalation of volatile emissions from drain when proposed shallow groundwater extraction system operating	Concentrations to be verified by monitoring – assumed to be below the analytical limit of reporting if system operates as proposed		negligible	negligible

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Table 5-3 (Continued) Summary of Exposure Assumptions and Calculated Risks – Gardeners/Maintenance Workers

Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
			Non-Threshold Risk	Threshold HI
6 Ingestion and dermal contact with chemicals in soil and sediment remaining exposed as part of Stage 1	Soil and sediment concentrations from available data assuming soil and sediment from the ephemeral ponds may be moved across the site. Maximum concentration from all available data from Block 1 and Block 2. Maximum concentrations in soil presented in Tables 3-3 and 3-4 . Maximum concentrations in sediment presented in Table 3-6 .	Exposure on Block 2: Incomplete due to placement of clean fill. Exposure on southern portion of Block 1 based on soil or sediments:		
		Ingestion of 25 mg of soil or sediment per day by adults. Once ingested it is assumed that 100% is absorbed into the body.	7.8x10 ⁻⁷ (soil) 7.4x10 ⁻⁷ (sediment)	0.42 (soil) 0.045 (sediment)
		Dermal : When outdoors it is assumed that the hands, forearms and lower legs get dirty each day (4580 cm ² of skin). Once dirty it is assumed that 0.51 mg of soil or sediment adheres to each cm ² of skin. Assume an adult will wash at the end of each day with 12 hours of the day dirty. Maximum risk calculated using either Hawley or USEPA methodology.	8.4x10 ⁻⁷ (soil) 2.4x10 ⁻⁶ (sediment)	0.21 (soil) 0.038 (sediment)

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Table 5-4 Summary of Exposure Assumptions and Calculated Risks – Intrusive Workers

	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
				Non-Threshold Risk	Threshold HI
			<p>Parameters relevant for all pathways Body weight of 70 kg. Exposure for 8 hours per day (i.e. long work day) for up to 30 days over a one year period</p>		
1	Inhalation of vapours during intrusive works*	<p>In excavations, air concentrations modelled from shallow groundwater concentrations presented in Table 3-2 assuming water seeps into any excavation constructed. As groundwater is shallow it is assumed that exposures in excavations would not involve significant emissions from residual soil contamination in the unsaturated zone (i.e. emissions would be dominated by the presence of shallow groundwater).</p> <p>Outdoor air concentrations (not in excavation) as modelled for gardeners (from soil and groundwater) based in maximum emissions and soil concentrations (as relevant), refer to Section 5.3.2 and Appendix D for further detail.</p>	<p>2.2 m³ air per hour outdoors (assuming 4 hours each day spent in or at top of excavation and 6 hours spent outdoors onsite).</p>	4.0x10 ⁻⁶	6.2

* Other inhalation exposures associated with emissions from Springvale Drain, compensatory flood storage area and other sources on the BIP are as presented for the outdoor maintenance worker and have not been presented separately for intrusive works. Only exposures specifically different for workers involved in intrusive works have been presented.

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Table 5-4 (Continued) Summary of Exposure Assumptions and Calculated Risks – Intrusive Workers

	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
				Non-Threshold Risk	Threshold HI
2	Ingestion and dermal contact with chemicals in soil and sediment remaining exposed as part of Stage 1	Soil and sediment concentrations from available data assuming soil and sediment from the ephemeral ponds may be moved across the site. Maximum concentration from all available data (soil or sediment) from Blocks 1 and 2. Maximum concentrations in soil presented in Tables 3-3 and 3-4 . Maximum concentrations in sediment presented in Table 3-6 .	<p>Exposure on Block 2: Intrusive works must be managed if they affect the integrity of the barrier. Exposure on southern portion of Block 1 based on soil or sediments:</p> <p>→ Ingestion of 25 mg of soil or sediment per day by adults. Once ingested it is assumed that 100% is absorbed into the body.</p> <p>→ Dermal: When outdoors it is assumed that the hands and lower legs get dirty each day (3300 cm² of skin). Once dirty it is assumed that 0.51 mg of soil or sediment adheres to each cm² of skin. Assume an adult will wash at the end of each day with 12 hours of the day dirty. Maximum risk calculated using either Hawley or USEPA methodology.</p>	<p>1.9x10⁻⁷</p> <p>1.1x10⁻⁷</p>	<p>0.52</p> <p>0.18</p>
3	Ingestion and dermal contact with chemicals in shallow groundwater that seeps into excavations	Shallow groundwater concentrations presented in Table 3-2 assumed to be representative of concentrations that may seep into a shallow excavation anywhere on the Southlands site.	<p>→ Ingestion of 5 ml of groundwater (1 teaspoon) each day during excavation/intrusive works. Once ingested it is assumed that 100% is absorbed into the body.</p> <p>→ Dermal: When undertaking intrusive works it is assumed that not all of the workday is spent in the excavation in direct contact with water. Hence it is assumed that 2 hours each day is spent in an excavation where the hands and lower legs get wet each time (3300 cm² of skin).</p>	<p>6.7x10⁻⁷</p> <p>9.9x10⁻⁶</p>	<p>0.039</p> <p>1.2</p>

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5.5 Quantification of Chemical Intake and Risk – Stage 2 Development

The quantification of risk associated with exposures that may be relevant to the development of Stage 2 has considered the proposed development as outlined in **Section 1.3** and adopted the methodology presented within the Consolidated HHRA (URS, 2005a). **Appendix C** outlines the equations used in the quantification of exposure and risk that is consistent with the overall methodology presented in **Section 1.5**.

The assessment of potential exposure (also refer to **Section 5.3**) and risk that may be associated with the Stage 2 development has been undertaken, as outlined in **Section 5.1**, based on a range of scenarios that address potential exposure and risk with and without the consideration of mitigation and management measures outlined in the RAP.

The following has been considered in the assessment of potential exposure and risk as part of the Stage 2 development:

- The development of Stage 2 will principally involve construction of commercial buildings on the southern portion of Block 1. No buildings will be constructed on the northern portion of Block 1 (proposed Stage 3) which will remain inaccessible with secure fencing.
- A physical barrier (fill materials and concrete slab) will be placed over the southern portion of Block 1 prior to development to meet the design flood criteria. Hence no direct contact with soils will be possible by workers and visitors in the accessible areas of the site.
- Flood management requires the installation of alternate flood storage areas (to replace the compensatory flood storage areas constructed on Block 1 as part of the Stage 1 development). It is expected that the alternate flood storage areas would be unlikely to intersect shallow groundwater or that they will be designed to control the discharge of shallow groundwater into these areas.
- It is expected that the bulk of the significant soil movement will have occurred as part of the Stage 1 development. The assessment of potential exposures associated with the Stage 2 development has assumed that contaminated soil relocated during Stage 1 remains in place.
- It is expected that the proposed risk management measure, namely a shallow groundwater extraction system installed along Springvale Drain intersecting shallow groundwater prior to discharge into the drain, will have been implemented as part of the Stage 1 development. The implementation and operation of such a system (or similar) will be relevant to the ongoing use of the Stage 1 development as well as the proposed Stage 2 development. Also the development is such that no buildings or work areas are to be located within 20m of Springvale Drain as part of the proposed Stage 2 development.

As the development of Stage 2 is to follow on from Stage 1, exposures and risks identified for the Stage 1 development remain relevant (refer to **Tables 5-2, 5-3 and 5-4**). The only change to exposure and risk associated with the Stage 2 development is the construction of buildings on Block 1 and the reduction of exposure (becomes incomplete) associated with the removal of the compensatory flood storage area and placement of clean fill across the southern portion of Block 1. This is particularly relevant to exposures by long-term workers on the site; hence risks to these workers have been presented for the Stage 2 development. Other exposures and risks remain essentially unchanged from those presented in **Table 5-3 and 5-4**. **Table 5-5** presents a summary of the exposure parameters adopted for the quantification of chemical intake associated with the Stage 2 development, a description of the concentrations adopted in the assessment and calculated risks. **Table 5-5** presents calculations undertaken for long-term commercial workers on the southern portion of Block 1 and it is noted that a number of the exposures and risks presented are the same as presented for the Stage 1 development as the exposure scenarios remain unchanged.

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Exposure parameters that are considered representative of RME have been selected for the receptor groups and exposure pathways identified. Where available, exposure data has been obtained from Australian sources (CSMS, 1991, 1993, 1996 and 1998, ANZECC 1992, NEPC 1999 and enHealth 2002 and 2003).

Details of the risk calculations undertaken are presented in **Appendix D** and as noted in the tables. The calculations presented relate to all COPC identified with the exception of asbestos. Issues associated with the presence of asbestos in soils on the site are addressed in further detail in **Section 5.6**.

Risk levels considered representative of acceptable risk (i.e target risk levels) are:

- non-threshold risk (sum over all pathways) = 1×10^{-5} (i.e. 1 in 100,000); and
- threshold hazard index (HI) (sum over all pathways) = 1.

Calculated risks for workers at the site that exceed the risk target values are highlighted in the summary tables. The following provides an overview of the exposure pathways and scenarios considered for the proposed Stage 1 development with reference to the relevant tables and exposure scenarios presented.

Exposure Pathway Quantified and Scenario Considered for Each Receptor – Stage 2

Commercial Workers (Table 5-5)

- Inhalation indoors and outdoors of COPC from sources on BIP (**Scenario 1**)
- Inhalation indoors and outdoors of volatiles from groundwater and some soil sources (from measured soil gas and flux emissions data) (**Scenario 2**)
- Inhalation indoors and outdoors of volatiles from remaining soil and sediment hot-spots (**Scenario 3**)
- Inhalation indoors and outdoors of volatiles from Springvale Drain
 - Risk issues were identified in Preliminary HHRA (Appendix H)
 - RAP has considered the implementation of a mitigation system
 - Calculation of risk has considered:
 - proposed system not operating (**Scenario 4**); and
 - proposed system operational (to be verified) (**Scenario 5**)

Gardeners and Maintenance Workers (non-intrusive works) (same as presented in Table 5-3)

Intrusive Workers (intrusive activities only) (same as presented in Table 5-4)

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Table 5-5 Summary of Exposure Assumptions and Calculated Risks – Commercial Workers for Stage 2

Exposure Pathways		Chemical Concentrations	Exposure Parameters	Calculated Risk	
			<p>Parameters relevant for all pathways Body weight of 70 kg. Exposure for 10 hours per day for 240 days/year (5 days per week for 48 weeks) for 30 years</p>	Non-Threshold Risk	Threshold HI
1	Inhalation of COPC in ambient air derived from sources within the BIP	Modelled ground level concentrations based on emissions from the BIP that include GTP, HCB repackaging, proposed CPWE remediation and other sources on BIP		Inhalation of 1.17 m ³ air per hour indoors (9 hours) and 2.2 m ³ air per hour outdoors (1 hour).	1.5x10 ⁻⁹
2	Inhalation of volatile chemicals derived from groundwater	Measured soil gas and flux emission data (maximum and average) collected in areas above shallow groundwater plumes relevant to the assessment proposed development on the southern portion of Block 1 (construction of buildings and outdoors), refer to Section 5.3.2 . Modelled air concentrations indoors (based on average and maximum measured data for Block 1) and outdoors (based on maximum estimated from maximum data from Block 1 or Block 2) are presented in Appendix D .	Inhalation of 1.17 m ³ air per hour indoors on Block 2 (9 hours) Inhalation of 2.2 m ³ air per hour outdoors on Block 2 or Block 1 (1 hour)	1.3x10 ⁻⁶ (average) 4.2x10 ⁻⁶ (maximum)	0.89 (average) 2.7 (maximum)
3	Inhalation of volatile chemicals identified in soil/sediment that may be moved across site	Modelled air concentrations based on maximum concentration in soil or sediment hot-spots (refer to Sections 3.5.2, 3.6 and 5.3.2) (Appendix D). Modelling has focused on: - Vapour intrusion into buildings on Block 1 (same as modelled for Stage 1 as maximum soil concentrations across the whole site have been used) - Volatile emissions to outdoor air on Block 1	Inhalation of 1.17 m ³ air per hour indoors (9 hours) Inhalation of 2.2 m ³ air per hour outdoors (1 hour)	5.2x10 ⁻⁴ (soil) 3.8x10 ⁻³ (sediment)	6 (soil) 12 (sediment)
				1.2x10 ⁻⁷ (soil) 8.9x10 ⁻⁷ (sediment)	0.010 (soil) 0.065 (sediment)

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Table 5-5 (Continued) Summary of Exposure Assumptions and Calculated Risks – Commercial Workers for Stage 2

Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk		
			Non-Threshold Risk	Threshold HI	
Inhalation of vapours derived from Springvale Drain (range of scenarios presented)					
4	GTP and backup systems not operating at all or where GTP is operating and shallow groundwater discharge is not controlled	Measured air concentrations (average and maximum before GTP operating [prior to 2007] and maximum reported after full commissioning of the GTP, refer to Table 3-10) adjacent to Springvale Drain (same as for Stage 1 development).	→ Inhalation of 1.27 m ³ air per hour indoors (9 hrs) /outdoors (1 hr) (total 10 hours) →	6.4x10⁻⁴ (average) 1.3x10⁻³ (maximum) 5.3x10⁻⁴ (post GTP)	8.0 (average) 17 (maximum) 6.8 (post GTP)
			→ Inhalation of 2.2 m ³ air per hour outdoors (4 hours) in work areas directly adjacent to the open drains (where no buildings are constructed)	4.4x10⁻⁴ (average) 9.0x10⁻⁴ (maximum) 3.6x10⁻⁴ (post GTP)	5.5 (average) 11 (maximum) 4.7 (post GTP)
5	GTP and backup systems operating and proposed shallow groundwater extraction system installed and operating	Concentrations to be verified by monitoring – assumed to be below the analytical limit of reporting if system operates as proposed. Concentrations that may occur if the GTP is offline have not been addressed separately, however an assessment has been undertaken to identify the maximum number of days in which the GTP can be offline before inhalation exposures approach risk values of concern. This is presented in Appendix F .		Negligible (but to be confirmed)	Negligible (but to be confirmed)

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5.6 Characterisation of Risks to Human Health – Stage 1 and Stage 2 Developments

Based on the calculated risks relevant to the potential for exposure to COPC identified on the Southlands Site associated with the proposed Stage 1 and Stage 2 development presented in **Tables 5-2 to 5-5** the following presents an overview of the risks including issues that warrant consideration of risk mitigation measures within the RAP.

5.6.1 Soil/Sediment – Direct Contact

Potential risks to workers who may have regular access to the portions of Block 1 that have no barrier as part of the Stage 1 development associated with direct exposure (ingestion and dermal contact) to maximum concentrations of COPC in soil and sediment (assuming no remediation occurs and maximum soil concentrations may be moved to any location across the site¹⁹) would be considered **unacceptable**.

Risks to human health associated with the presence of asbestos in soils cannot be assessed in a meaningful way. Current industry practice requires the removal or containment of ACM such that the potential for exposure is minimised. Hence, it would be considered unacceptable if the development of the site did not include appropriate management measures to address the presence of ACM in soils.

It is noted that as Block 1 is not proposed to be covered with barriers as part of the Stage 1 development (it is to be landscaped with the inclusion of a compensatory flood storage area) there is the potential for workers who have regular access to this area to be inhale asbestos fibres should they be mobilised in dusts generated from the area. The generation of asbestos fibres in this area can be minimised via the use and maintenance of good ground cover and limiting regular access to the area.

It is noted that if access to the southern portion of Block 1 during Stage 1 was to be limited (as is currently proposed), then risks to human health will be lower and closer to the risks calculated for a gardener (where total non-threshold risk and threshold HI are less than the target risk levels and are considered **acceptable**, refer to **Table 5-3**).

The surface level of the southern portion of Block 1 is expected to be raised (with the use of fill materials and hardstand) for the purpose of flood management prior to the Stage 2 development. The placement of this layer (provided it meets a minimum depth requirement of 0.5m of clean fill as noted in the RAP) will address exposures associated with the presence of asbestos fibres in soil in this area as well as the potential for direct contact with COPC in soil or sediment. It is noted that the northern portion of Block 1 is not to be covered with barriers and will be fenced (inaccessible), hence measures should be taken to ensure that adequate ground cover is maintained to minimise the potential for dust generation where asbestos fibres may be mobilised to air.

In addition, it is noted that the surface level of Block 2 (Stage 1) is to be raised (with the use of fill materials and a concrete slab) to address flood management requirements. Hence, direct contact with COPC identified in soil or sediment on the site following Stage 1 and Stage 2 development will not be relevant. However, in the event that the fill materials/barriers were not placed or was breached during intrusive or construction works (and not managed appropriately to reinstate the barrier), then the assessment presented for the southern portion of Block 1 during Stage 1 is considered to be a conservative basis for assessment of potential exposures on Block 2 or Block 1 following development (as the assessment presented has considered the maximum soil concentration from either Block 1 or

¹⁹ This assumption has been included as the Stage 1 development is expected to result in the movement of soils across the whole site as part of earthworks required for flood management as the proposed development does not want to consider restrictions on the movement of soils across the site. Hence elevated concentrations in soils identified on Block 1 may be moved to Block 2 and placed beneath a building. While it is considered unlikely that the soil hot-spots identified will be spread over a large area (with no mixing with other soils), actual concentrations cannot be estimated and the assumption included in this assessment that concentrations remain unchanged is considered conservative.

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Block 2). This assumes that soil and sediment at the highest concentration measured in the investigations are moved from Block 1 to Block 2 during earthworks.

Irrespective of the need to limit access to the southern portion of Block 1 during Stage 1, review of the risk calculations associated with direct contact with soil and sediments by a worker who may have regular access to soil associated with the Stage 1 development indicates the following:

- The non-threshold risk is dominated by the presence of vinyl chloride (85% of total), with calculated risks for all other non-threshold COPC less than 1×10^{-6} and therefore considered to be negligible; and
- The threshold HI is dominated by HCB (52%) followed by lead (20%), mercury (11%), HCB (11%) and chromium (3%) in soil and in sediment by PCE (33%), TCE (31%) and HCB (24%). It is noted that the maximum concentration of most of these chemicals occurs in only one location and not indicative of widespread contamination. Calculated HI associated with other COPC are less than 0.01 and are therefore considered to be negligible.

Whilst the assumption that relocation of contaminated soil and sediment results in wide spread contamination unlikely, in order to remove the associated uncertainty the assumed “hot-spots” identified in the investigations to date could be remediated prior to the proposed development. Vapour migration and intrusion issues will also need to be considered in the review of risks to human health and identification of hot-spots where remediation may be warranted.

5.6.2 Vapour Migration from Subsurface Sources

Groundwater

Potential risks to workers associated with inhalation of volatile COPC that may be present in emissions to air from underlying shallow groundwater and/or some soil impacts, assuming Stage 1 development of Block 2 (with limited consideration of the placement of clean fill) and access to the southern portion of Block 1 (with no fill) are considered to be below target risk values and **acceptable**.

Potential risks to workers associated with inhalation of volatile COPC that may be present in emissions to air from the underlying shallow groundwater and/or some soil impacts, assuming Stage 2 development of the southern portion of Block 1 (without placement of clean fill) are less than the non-threshold target risk however greater than the threshold target HI for vapour intrusion only and are considered to be **unacceptable**.

On the basis of the above, vapour mitigation measures are recommended to be included in the building designs for the Stage 2 development. Such measures could include vapour barriers (such as a clay barrier or liner), sub slab depressurisation or building pressurisation. The final vapour mitigation measure adopted should be identified once building designs are identified to ensure that the approach adopted is adequate, practical and can be maintained.

There are some limitations associated with the data utilised for the above assessment. In particular the following should be noted:

- A number of soil gas and flux emissions sample locations coincide with locations where volatile COPC have been identified in soil, in particular the presence of petroleum related chemicals in the vicinity of TP81 and TP82 (refer to **Table 3-3**). While the data used is considered representative of volatile contributions from both groundwater and soil, the data collected from the locations that coincide with soil contamination is limited to one round of data only. A number of other locations have sufficient rounds of data to enable an assessment of variability; however this is not the case for these locations. In order to reduce the uncertainty the locations where petroleum related impacts have been identified could be remediated or additional investigations undertaken.
- The data collected from the Southlands site has been obtained from open ground. In addition most of the samples have been collected using an emissions flux hood (due to the shallow depth of groundwater limiting the ability to collect soil gas data). The diffusion of oxygen into the upper layers

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of the soil profile may result in increased degradation of some (but not all) volatile chemicals near the surface than under a large building slab where oxygen diffusion may be limited. To address this uncertainty, additional data is currently being collected at nearby commercial properties (where large slabs are present) to assess the potential for higher concentrations of volatile chemicals to be present beneath the slab compared to concentrations that measured at or near to the surface from open ground. The outcome of this assessment will be considered with respect to the potential need to include vapour mitigation measures into all building designs on Southlands.

Soil

Further consideration of vapour migration and intrusion issues that may be associated with the presence of volatile COPC in soil and sediment have been separately assessed as most of these areas have not been subject to soil gas and flux emissions sampling. The assessment undertaken has used a conservative vapour transport model to provide an estimate of potential concentrations outdoors and indoors (assuming that soil and sediment from both Block 1 and Block 2 may be moved anywhere across the site during earthworks). The assessment undertaken has indicated risks to human health associated with inhalation of volatile COPCs that may migrate into buildings constructed over soil with these assumed concentrations to be **unacceptable**. The volatile COPC that most significantly contribute to the total risk (with vapour intrusion being the most significant risk) are:

- non-threshold risk associated with soil is dominated by vinyl chloride (97% of total) and TCE (2%), with calculated risks for all other non-threshold COPC less than 1×10^{-5} ;
- threshold HI associated with soil is dominated by cis- and trans-1,2-dichloroethene (83%) and PCE (11%); and
- non-threshold risk and threshold HI associated with sediments is dominated by vinyl chloride, TCE, EDC, chloroform, 1,1,2-trichloroethane, cis- and trans-1,2-dichloroethene and PCE.

On the basis of the assessment undertaken for soil and sediment relevant to the Stage 1 and 2 development, the localised areas of elevated concentrations of volatile COPC have been identified that warrant further consideration within the RAP. This may include remediation or further investigation (i.e. soil gas and flux emission sampling).

5.6.3 Emissions to Air from Springvale Drain

Potential risks to workers associated with inhalation of volatile COPC that may be present in ambient air as a result of vapour emissions from Springvale Drain while the GTP is not operating at all (chronic exposures) or while the GTP is operating and shallow groundwater discharges to the drain following rainfall events (and no shallow groundwater extraction system (or similar) has been installed) are above the target risk values and therefore considered **unacceptable**. While a conservative approach has been adopted in the assessment of risks to workers in areas adjacent to Springvale Drain, the risks are sufficiently elevated (before and after commissioning the GTP, including following rainfall events) that additional risk management measures would, on the basis of the measurements made to date, warrant implementation as part of the development of the currently vacant Southlands property.

In relation to the operation of the proposed shallow groundwater extraction system, at the time of preparation of the risk assessment, it is not known how many times the GTP would be offline in a year. Review of surface water and air data collected to June 2008 (URS, 2008b) indicates that air concentrations adjacent to Springvale Drain from August 2007 to June 2008 are lower than previously reported prior to and just after commissioning of the GTP. While the GTP is operating as indicated by the data collected during this period, long-term risks to future workers on any development within Southlands have reduced to a level that is considered acceptable. The potential for peak exposures to occur during rainfall events, or when the GTP is off-line should be further considered to ensure that long-term average exposures remain low and acceptable.

To assist in the ongoing development of the proposed system along Springvale Drain an assessment of potential risks associated with peak inhalation exposures during short periods of time when the GTP is

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offline has been undertaken. The assessment identified the maximum number of days the GTP could be offline each year or over a working life before risks to workers are considered unacceptable assuming that shallow groundwater discharge into the drain occurs every time the GTP is offline. The assessment undertaken is presented in **Appendix F** and for the proposed development the following is noted:

- The assessment has is based on the current design where all buildings and work areas (including outdoor work areas) are at least 20m from the drain. The assessment, has considered that reasonable maximum exposures to workers may occur in the zone from 20 m to 50 m from the drain²⁰ for risks associated with emissions from the drain.
- With respect to overall exposure, consideration has been included for contributions from the compensatory flood storage area (designed to prevent shallow groundwater discharge) and emissions from groundwater to indoor (average) and outdoor air. Consideration of inhalation exposures associated with emissions to air from groundwater has been undertaken for the Stage 1 development assuming that no vapour mitigation measures are implemented for the buildings to minimise vapour intrusion. However, it is expected that the Stage 2 development will incorporate vapour mitigation measures to reduce risks to values at least as low as those calculated for Stage 1. In addition the contribution from direct contact and inhalation associated with soil/sediment has not been considered as it is assumed that remediation will take place prior to development. Risks associated with emissions to air from Floodvale drain have not been considered as these exposures will not coincide with the maximum exposures associated with Springvale Drain as the two drains are located on opposite sides of Block 2. Contributions from other sources on the BIP are essentially negligible. Hence cumulative issues result in a reduction in the target risk levels to 8×10^{-6} (non-threshold) and 0.7 (threshold HI).
- The assessment presented in **Appendix F**, indicates that the maximum number of days (based on average calculations) where the GTP can be offline before cumulative risks to workers approach unacceptable values are, for exposures 20-50m from drain, an average of 1060 days over 30 years and maximum of 650 days over 30 years (with no more than 270 days during any one year). These estimates should be considered by Orica as part of the overall design and management of the system and backup systems to be installed to manage issues associated with groundwater discharge into Springvale Drain.

The effectiveness of the proposed management system for Springvale Drain needs to be verified by the collection of appropriate data with risks to human health revised if required.

The assessment of exposure adjacent to Floodvale Drain has not identified risks that would be considered unacceptable. It is noted that the assessment is based on one round of sampling only. However review of surface water concentrations reported on a regular basis during the Quarterly Groundwater Monitoring Program (URS, 2007e) indicates that concentrations of chlorinated compounds in Floodvale Drain are lower than reported in Springvale Drain. In addition, since the operation of the GTP, concentrations of many chlorinated compounds have not been reported above the analytical LOR in Floodvale Drain. Hence the potential for elevated concentrations of volatile chlorinated compounds in Floodvale Drain, and associated emissions to air is considered low.

²⁰ The region 20m to 50m from the drain was identified as representative of a work area where workers may spend a significant portion of each day. A buffer zone of 20m is incorporated into the current development with areas between the drain and 20m used for flood storage, landscaping or other areas that are not (or could not be used as) work areas. The closest buildings and storage areas are 20m from the drain and any workers in such areas or buildings would be expected to work within a general area or bay that may be up to 30m wide. Hence it is considered unrealistic to assume a worker would spend all day exactly 20m from the drain. It is more reasonable to consider exposures in work areas within the 20-50m distance from the drain.

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5.6.4 Emissions to Air from Compensatory Flood Storage Area

The proposed compensatory flood storage area on the southern end of Block 2 as part of the Stage 1 development has been assessed with respect to potential vapour issues. The assessment (refer to **Appendix E** for more detail) has identified that outdoor inhalation exposures associated with shallow groundwater discharge into the compensatory flood storage area are potentially **unacceptable** (particularly with respect to emissions of EDC, vinyl chloride and carbon tetrachloride). However, if the proposed drainage system is implemented and shallow groundwater does not discharge at the surface of the compensatory flood storage area, then risks to workers in the vicinity of the area are low and **acceptable**. The effectiveness of the drainage system proposed needs to be verified with the collection of appropriate data, and risks to workers re-evaluated if needed.

5.6.5 Cumulative Exposures

The assessment of risks presented in **Tables 5-2 to 5-5** present a range of exposure scenarios and risk outcomes, some of which require risk management measures to be implemented prior to development of the site (refer to summary presented in **Section 5.7**). With respect to the consideration of total or cumulative risks, particularly to long-term workers on the site, not all exposure scenarios presented will be relevant. These relate to risks calculated for issues that are proposed to be addressed through remediation (soils) or management measures (emissions from the compensatory flood storage area, emissions to air from Springvale Drain and vapour intrusion into buildings). Hence total or cumulative risks to human health that are relevant to the proposed development (including remediation and management measures) can be calculated. **Table 5-6** presents a summary of total risks for long-term commercial workers assessed on the site.

Table 5-6 Summary of Total Risks – Commercial Workers for Stage 1 and Stage 2

Exposure Pathways	Calculated Risk	
	Non-Threshold Risk	Threshold HI
Inhalation of COPC in ambient air from emission sources within the BIP	1.5×10^{-9}	0.0047
Inhalation of volatile chemicals derived from shallow groundwater beneath Southlands (risk posed on Block 2 considered conservative estimate of risks posed on Block 1 after vapour mitigation measures are adopted)	5.5×10^{-7} (average) 1.0×10^{-6} (maximum)	0.19 (average) 0.29 (maximum)
Inhalation of vapours derived from Floodvale Drain (relevant to work areas on western side of Block 2)	3.9×10^{-6}	NA as no threshold COPC reported
Exposures when GTP operating and proposed shallow groundwater extraction system installed and operating	Negligible (needs to be confirmed)	
Exposures where drainage system effectively operating – no discharge of shallow groundwater to compensatory flood storage area	1.2×10^{-6}	0.086
Inhalation of volatile chemicals identified in soils/sediments that may be relocated across the site	Negligible following remediation	
Ingestion and dermal contact with chemicals in soil and sediment remaining exposed as part of Stage 1	Negligible following placement of barriers on site and restriction of access to Block 2 during Stage 1	
Total risk (after remediation and risk management measures)	6×10^{-6} (average)	0.3 (average)

On the basis of the above total risks to commercial workers on the site following proposed development (and implementation of remediation and management measures as identified) are considered low and acceptable.

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5.6.6 Exposures During Intrusive Works

The calculated risks for workers who may undertake intrusive works on the site following development indicate a potential for unacceptable exposures to groundwater via dermal contact and inhalation of volatile chemicals where shallow groundwater enters the excavation (refer to **Table 5-3**). These exposures need to be appropriately managed on the site within the long term environmental management plan and job specific occupational health and safety plans as required. In addition intrusive works that result in breaching of the barriers placed on the site needs to be appropriately managed, in relation to the presence of contaminants including asbestos fibres in soil beneath the barrier and disposal of soil excavated from beneath the barrier and reinstatement of the barrier.

5.7 Summary of Risk Issues

The following table presents a summary of the risk issues associated with the proposed Stage 1 and Stage 2 developments of the Southlands property. The table presents the risk issues identified and where mitigation measures within the RAP or other aspects of the proposed development are warranted.

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Table 5-7 Summary of Risk Issues

Issue	Issues Identified	Risk Mitigation Measures
All Aspects of the Proposed Development		
Intrusive workers in all areas including the maintenance of GTP infrastructure	Potential for elevated exposures to chemicals in shallow groundwater and inhalation of volatile chlorinated chemicals (particularly within confined spaces)	All works to be undertaken under a long term environmental management plan [EMP] that ensures development of job specific safe work practices including addressing any issues associated with works that breach the areas covered with barriers.
Workers involved in maintenance of Springvale and Floodvale Drains	Potential for elevated exposures to chemicals in surface water and inhalation of volatile emissions	All works to be undertaken under a long term environmental management plan that ensures development of job specific safe work practices.
Presence of asbestos fibres in soil	Potential for generation of dusts containing asbestos fibres.	Placement of appropriate barrier. Where no physical barrier is present a sound ground cover should be maintained to minimise generation of dusts. These areas need to be considered in a long term environmental management plan [EMP].
Inhalation of volatile chlorinated chemicals associated with emissions to air from Springvale Drain	Potential for significant exposures by all users of the site on basis of measurements completed to date.	Surface water concentrations within the drain would need to be lowered such that air concentrations are reduced by 100 to 1000 fold. This may require a reduction in the discharge of shallow groundwater into Springvale Drain. Proposed mitigation measure involves a shallow groundwater extraction system adjacent to Springvale Drain to intercept groundwater prior to discharge into the drain, with groundwater directed to the GTP. Exposures are expected to be lower once system is installed, however the effectiveness requires verification. Risk issues when the GTP is offline need to be reviewed in the final design and management of such a system.
Stage 1 Development (also relevant to Stage 2 development)		
Elevated concentrations identified in soil and sediment in the ephemeral ponds – that are moved across the site during earthworks	Potential for elevated exposure by workers in area not covered with barriers, including gardening activities (ingestion, dermal contact and vapour intrusion into buildings)	Remediation of assumed hot-spots to a level protective of all exposures including vapour intrusion (refer to Section 5.8). Remediation to derived risk-based concentrations or other guidelines that can be justified on the basis of risks to human health. Further sampling could be undertaken to better define extent of area affected by elevated concentrations
Inhalation of volatile chemicals in outdoor air (emissions from soil and groundwater)	No issues identified for outdoor air if the compensatory flood storage area is appropriately designed to prevent groundwater discharge to the surface.	Effectiveness of the proposed drainage system for the compensatory flood storage areas on Block 1 requires verification.
Inhalation of volatile chemicals in outdoor air and within buildings constructed on Block 2 (emissions from some subsurface soil and groundwater)	No issues identified (subject to findings of sub slab investigations).	Review of vapour intrusion issues that may arise as a result of the consideration of issues associated with the presence of a large slab.
Stage 2 Development		
Inhalation of volatile chemicals within buildings constructed on the southern portion of Block 1 (emissions from some subsurface soil and groundwater)	Potential for elevated exposure to volatile chlorinated chemicals, present in shallow groundwater, that may migrate into buildings (including buildings associated with GTP infrastructure)	If buildings are constructed in the area vapour mitigation measures should be incorporated into the building design. Such measures may include passive barriers, sub-slab depressurisation or building pressurisation.

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5.8 Further Consideration of Soil Issues

The HHRA has identified a number of issues associated with the presence of COPC in soil and sediment on the site for consideration within the RAP to mitigate exposure. Exposure to asbestos fibres and non-volatile COPC can be managed through limiting access, maintenance of good ground cover or placement of an appropriate barriers using clean fill. In addition inhalation exposures to volatile COPC identified in soil and sediment can be managed by the inclusion of vapour mitigation measures in all buildings to be constructed on the site. However, remediation of localised areas of elevated concentrations in soil, assumed on the basis of investigations to date, prior to the excavation and earthworks being undertaken could minimise the movement of soil across the site and thereby mitigate exposure after development.

The COPC that contribute most significantly to the total risk can be identified on the basis of direct contact exposures and vapour intrusion. These are outlined in **Section 5.6** and listed in **Table 5-7**. While a number of the assumed hot-spots could be remediated on the basis of the soil screening criteria (as the extent of contamination appears to be limited), site-specific risk based soil concentrations (RBSC) could also be calculated and used as the basis for identification and validation of remediation areas.

To assist in the consideration of site validation, preliminary RBSC have been calculated (refer to **Appendix G**). The preliminary RBSC are have been derived using the methodology adopted for the quantification of exposure and risk by a back calculation process. RBSC have been derived for each key chemical such that the total risks to workers (most sensitive receptor on-site) associated with ingestion, dermal contact (assuming limited placement of barriers) and vapour intrusion²¹ is equal to a non-threshold risk of 4×10^{-6} and/or a target HI of 0.7. These total risk targets have been selected such that the total risk calculated for commercial workers (presented in **Table 5-6**) plus the contribution from residual soil (assuming remediation to the levels presented) is equal to the overall target risk values adopted as indicative of acceptable risks to human health (ie total non-threshold risk of 1×10^{-5} and threshold HI).

The RBSC are preliminary and may be adjusted to address issues raised as part of the planning and approval process or changes to site development.

Table 5-8 presents a summary of the key COPC identified, the screening level criteria where relevant and preliminary RBSC.

²¹ RBSC have been derived to be protective of vapour intrusion issues. This approach is considered to be adequately protective of exposures outdoors (where air concentrations are lower) and on adjacent commercial premises (where concentrations are lower due to further mixing and dispersion off-site).

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Table 5-8 Preliminary Remediation Criteria for Key COPC

Key COPC Identified in Risk Calculations	Adopted Screening Criteria (mg/kg)	Preliminary RBSC (mg/kg)	Locations where adopted criteria (screening or RBSC) exceeded
mercury	75 (HIL "F")	Not derived	Block 1: TP098, TP094, TP01, TP02, TP06 Block 2: TP50
chromium	500 (HIL "F" for chromium VI)	Not derived	Block 1: TP31 Block 2: No exceedances
lead	1500 (HIL "F")	Not derived	Block 1: TP31, TP127 Block 2: TP50
hexachlorobenzene (HCB)	1.1 (US Region IX PRG)	Not derived	Block 1: HA006, SS049, TP01, TP02, TP029, HA01, HA02, HA03 Block 2: SS037, TP50
hexachlorobutadiene (HCBd)	22 (US Region IX PRG)	Not derived	Block 1: HA01, HA02 Block 2: No exceedances
vinyl chloride		0.3	Block 1 soils: TP15, TP29, TP37 Block 1 sediments: SD018, SD020, SD021, SD023, SED05, SED06 Block 2: No exceedances Stockpiles: SP056, SP057
1,1-dichloroethane		45	Block 1 and Block 2 soils: no exceedances Block 1 Sediments: SED06
trans-1,2-dichloroethene		5	Block 1 soils: TP15, TP29, TP37, HA001, HA02, HA006, TP106 Block 1 sediments: SD021, SED06 Block 2: No exceedances
cis-1,2-dichloroethene		4	Block 1 soils: TP15, TP27, TP29, TP37, HA01, HA001, HA002, TP105, TP107, TP111, TP116, TP118, TP120, TP126, HA006 Block 1 sediments: SD021, SD023, SED05, SED06 Block 2: No exceedances Stockpiles: SP28
1,2-dichloroethane (EDC)		4	Block 1 soils: TP29, TP37 Block 1 sediments: SED06 Block 2: TP131
trichloroethene (TCE)		12	Block 1 soils: TP15, TP29, HA01, TP105, TP107, HA006 Block 1 sediments: SD020, SED06, SD023 Block 2: No exceedances
1,1,2-trichloroethane		9	Block 1 and Block 2 soils: No exceedances Block 1 sediments: SD020, SED06
tetrachloroethene (PCE)		12	Block 1 soils: TP01, TP02, TP29, HA01 Block 1 sediments: SD020, SED06, SD023 Block 2: No exceedances
1,1,1,2-tetrachloroethane		20	Block 1 and Block 2 soils: No exceedances Block 1 sediments: SED06
chloroform		8	Block 1 and Block 2 soils: No exceedances Block 1 sediments: SD020, SD023, SED06
carbon tetrachloride		0.2	Block 1 and Block 2 soils: No exceedances Block 1 sediments: SD023, SED06
1,3,5-trichlorobenzene		15	No exceedances
TPH*			
C ₆ -C ₉ (as aliphatic)		100	Block 1: TP81, TP82, TP01
C ₁₀ -C ₁₄		2000	Block 1: TP82
C ₁₅ ⁺		5000	No exceedances

* Preliminary RBSC have been derived for TPH, not because TPH has been identified as a key COPC on the basis of the risk calculations, but because the adopted screening criteria that may be used for the purpose of remediation is considered too conservative. Refer to **Appendix G** for details on the approach adopted to deriving RBSC for TPH.

Section 6

Uncertainties

6.1 General

In general, the uncertainties and limitations of human health risk assessment can be classified into the following categories:

- Sampling and analysis;
- Receptor exposure assessment; and
- Toxicological assessment.

The risk assessment process following enHealth, ANZECC/NHMRC and USEPA guidance documents provides a systematic means for organising, analysing and presenting information on the nature and magnitude of risks to public health posed by chemical exposures. Despite the advanced state of the current risk assessment methodology, uncertainties and limitations are inherent in the risk assessment process. This section discusses the uncertainties and limitations associated with this risk assessment.

6.2 Sampling and Analysis

The data collected for soil, sediment, surface water, groundwater and air from the Southlands site has been based on the knowledge of the site hydrogeological conditions (both on and off site) and former site history and activities. In general, the analytical analysis has also been selected based on a knowledge of the site history and hence has focussed on chemicals which were known to have been formerly used at the site. The air data collected from the site has been analysed for the full suite of analytes relevant to the analytical method. There is the potential for chemicals to be present on the site which have not been characterised based on omission from site history records. The assessment of risk presented in this report has used maximum concentrations reported for many exposure pathways to be conservative. Where relevant, and sufficient data is available, average concentrations have also been used for the purpose of comparison.

A limitation of the soil gas and flux emissions data has been noted in the assessment. In particular this relates to the use of data collected from open ground to assess the potential for vapour intrusion into buildings constructed in large concrete slabs that have the potential to limit oxygen penetration into the subsurface and hence result in a greater potential for vapour intrusion to occur. While this is recognised as a limitation of the current data set, additional data is being collected to further investigate this issue. In addition the approach adopted in estimating indoor air concentrations from this data is considered conservative and is likely to result in an overestimate of actual concentrations.

6.3 Exposure Assessment

Risk assessments require the adoption of several assumptions in order to assess potential human exposure. This risk assessment includes assumptions about general characteristics and patterns of human exposure relevant to the development proposed for the Southlands site. The assumptions used are conservative and developed to provide an estimate of reasonable maximum exposures rather than the actual exposures. This approach tends to overestimate the risks. The assessment has been completed using upper bound concentrations reported within soil, sediments, surface water, groundwater and air (as relevant). This assumption may therefore overestimate the potential for exposure to the COPC in the areas assessed.

Given the uncertainty in the estimate of intake via dermal exposure (refer to **Appendix C** for details on the methodologies relevant), intake has been calculated using both methods described, however URS considers that the method of Hawley (1985), SEDISOIL (1996) and CSOIL (2001) is the most appropriate basis for assessment of risks. The Hawley method allows for determination of exposure time (ie time to washing and removing contaminants from the skin surface), the USEPA method has no consideration of exposure time. Experimental studies used to define dermal absorption fraction (ABS_d) values are associated with dermal application over 24 hours (i.e. the event is considered to be a 24 hour day by default). Due to the lack of information about the rate and relationship of absorption of chemicals through

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the skin over shorter exposure periods, the USEPA methodology does not recommend adjusting the ABSd to account for exposures over times less than 24 hours, rather it recommends adjusting exposure frequency and exposure duration to reflect site conditions. Dermal exposures have been calculated using both approaches (refer to **Appendix C** for detail) and the calculated risks are presented in **Appendix D**. It should be noted that comparison between the two approaches results in calculated risks which are slightly higher or lower for individual chemicals with the exception of PAHS and aromatic TPH C₁₀-C₃₆ using the USEPA approach which uses chemical specific absorption factors (based on a 24 hour exposure) which results in a more conservative calculated risk. It should be noted that PAHs are not a significant COPC at the Southlands site. In general the calculated risks presented by both approaches (with the exception of PAHs) is consistent and the outcomes and conclusions of the risk assessment are also consistent with the adoption of either approach.

The air exchange rate (2 exchanges per hour derived from a minimum ventilation rate of 15 L/s/occupant) used in the quantification of potential vapour intrusion issues is derived from the minimum air exchange rate presented in AS1668.2 1991. Advice from NOHSC (available on their web-site) indicates that the Building Code of Australia (BCA) currently references and uses guidance from AS1668.2-1991 and does not support the use AS1668.2-2002. As noted in the NOHSC advice (also available from BCA) that with respect to minimum ventilation rates, AS1668.2-2002 allows ventilation rates below 10 L/s/occupant and "advice from health authorities" suggests that this is inconsistent with international practice and as a consequence the BCA does not support the use of the new ventilation rates. The AS1668.2-1991 remains valid and used as the relevant building code until the issue is resolved. Hence ventilation rates in AS1668.2-2002 are not currently adopted in Australia and have not been considered in the air exchange rate used in the assessment of commercial use. A lower air exchange rate would result in proportionally higher indoor air concentrations; hence the final design of buildings on the site should consider the importance of the ventilation rate. Should a lower ventilation rate be used, then risks to workers associated with vapour intrusion should be revised, or buildings designed such that vapour intrusion is mitigated regardless of the ventilation rate.

The assessment of potential vapour migration issues has been based on data collected to the end of 2007. It is noted that further monitoring should be undertaken and reviewed to determine if the development of any part of Southlands results in changes to the subsurface conditions (in particular the potential presence of a freshwater lens limiting vapour migration from shallow groundwater) that may subsequently change the potential for vapour migration on and off the site. This area of uncertainty can be addressed on the site through the consideration of vapour mitigation measures on all buildings to be constructed on Block 1 and Block 2.

A number of other uncertainties exist particularly in relation to the effectiveness of proposed risk mitigation measures. These are noted and discussed in the report, where relevant. Should the measures not prove to be as effective as anticipated in this report, then resultant risks may prove to be unacceptable.

6.4 Toxicological Assessment

In general, the available scientific information is insufficient to provide a thorough understanding of all of the potential toxic properties of chemicals to which humans may be exposed. It is necessary, therefore, to extrapolate these properties from data obtained under other conditions of exposure and involving experimental laboratory animals.

This may introduce two types of uncertainties into the risk assessment, as follows:

- 1) Those related to extrapolating from one species to another; and
- 2) Those related to extrapolating from the high exposure doses, usually used in experimental animal studies, to the lower doses usually estimated for human exposure situations.

The majority of the toxicological knowledge of chemicals comes from experiments with laboratory animals, although there may be interspecies differences in chemical absorption, metabolism, excretion and toxic response. There may also be uncertainties concerning the relevance of animal studies using

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exposure routes that differ from human exposure routes. In addition, the frequent necessity to extrapolate results of short term or subchronic animal studies to humans exposed over a lifetime has inherent uncertainty.

In order to adjust for these uncertainties, ADIs and RfDs incorporate safety factors that may vary from 10 to 1000.

The USEPA assumes that humans are as sensitive to carcinogens as the most sensitive animal species. The policy decision, while designed to minimise the potential for underestimating risk, introduces the potential to overestimate carcinogenic risk. Conversely, it also does not allow for the possibility that humans may be more sensitive than the most sensitive animal species. The model used by the USEPA to determine slope factors is a linearised multistage model, which provides a conservative estimate of cancer risk at low doses and is likely to overestimate the actual slope factor. The result is that the use of slope factors has the general effect of overestimating the incremental cancer risks.

It is assumed in the approach adopted by the USEPA that a genotoxic mechanism applies, however, most carcinogens do not actually cause cancer by this mechanism. Non-genotoxic carcinogens have not been assessed on the basis of a non-threshold value in this assessment in accordance with Australian and WHO guidance.

The approach for evaluating risks to mixtures of chemicals assumes dose additively and does not account for potential synergism, antagonism or differences in target organ specificity and mechanism of action. Where data is available regarding the interaction of mixtures of some COPC (such as BTEX), these issues have been addressed in the assessment presented. In general, the additive approach has the effect of overestimating the risks. However it is noted that the assessment of a range of chlorinated compounds presented in this report have similar toxicological endpoints with a number demonstrating similar metabolic pathways that are considered to be associated with the toxicological effects observed (rather than the parent compound itself). For these compounds the consideration of cumulative exposure on the basis of additivity is considered appropriate.

For the consideration of a larger range of compounds where toxicological mechanisms and end points are not similar, the consideration of cumulative exposures on the basis of additivity is considered conservative.

Section 7

Conclusions

An assessment of risks to human health that may be associated with the proposed Stage 1 and 2 development of the Southlands property has been undertaken. The assessment has reviewed existing data to identify key issues that warrant detailed assessment within the HHRA. The HHRA presented has therefore been undertaken as a site-specific assessment that considers key aspects of the proposed development, flood management requirements and risk management measures. These include the filling in of existing ponds on Block 1, construction of compensatory flood storage areas on the southern portion of Block 1, the placement of fill on Blocks 1 and 2 prior to construction of any buildings to address flood issues and the restriction of access to the northern portion of Block 1 (proposed Stage 3 development). With respect to risk management measures the assessment has considered proposed measures designed to intersect shallow groundwater prior to discharge into Springvale Drain (with groundwater directed back to the GTP) and the drainage system proposed to control shallow groundwater in the area of the compensatory flood storage area on Block 1. The performance of these measures will need to be monitored through the collection of appropriate data for some time after commissioning. Should any of these assumptions change, or the proposed risk management measures do not achieve the stated objectives, then the assessment of risks to human health presented in the report will need to be reviewed and potentially revised.

Review of data available for Southlands indicates the presence of contamination in groundwater, soil, sediment, surface water and air. On the basis of the available data, risks to human health associated with the proposed development have been quantified in accordance with the scope and methodology outlined in **Section 1**. The assessment has identified a number of risk issues that warrant further consideration within the RAP particularly in relation to the following issues:

- Emissions to air from Springvale Drain associated with the discharge of shallow groundwater into the open drain. A risk management system has been proposed to intersect shallow groundwater prior to discharge into the drain. The proposed system involves a shallow groundwater extraction system adjacent to Springvale Drain to intercept groundwater prior to discharge into the drain, with groundwater directed to the GTP (refer to the RAP for details). Exposures are expected to be lower once the system is installed, however the effectiveness requires verification through the collection of appropriate data for some time after commissioning in a variety of conditions. In addition, risk issues when the GTP is offline need to be reviewed in the final design and management of such a system;
- Emissions to air from the compensatory flood storage area proposed for Block 1 associated with discharge of shallow groundwater into the area and the design and management of the proposed drainage system to prevent shallow groundwater discharge at the surface (refer to RAP for details). The effectiveness of the drainage system requires verification through the collection of appropriate data for some time after commissioning;
- Management of residual asbestos fibres in soils through the placement and management of clean fill;
- Management of residual soil contamination (using preliminary risk based criteria derived in the HHRA to assist in the removal of residual contamination);
- Mitigation of vapour intrusion into commercial buildings (on Block 1 in particular, and possibly Block 2); and
- Requirement for a long term environmental management plan that addresses intrusive works across the site and access to open drains and the northern portion of Block 1.

Section 8

Limitations

URS Australia Pty Ltd (URS) has prepared this Report in accordance with the usual care and thoroughness of the consulting profession for the use of Orica Australia Pty Ltd and Goodman Pty Ltd (The Proponent) and only those third parties who have been authorised in writing by URS to rely on the report. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report. It is prepared in accordance with the scope of work and for the purpose outlined in the Proposal dated 17 May 2006 and subsequent revised URS Proposal dated 7 June 2006.

All information in this Report is provided strictly in accordance with and subject to the following limitations and recommendations:

- a) This Report should be read in full and no excerpts are to be taken as representative of the findings. No responsibility is accepted by URS for use of any part of this Report in any other context.
- b) This Report is based solely on the scope of work agreed between URS and Orica Australia Pty Ltd (Orica) and Goodman and described in **Section 1.8** ("Objectives and Scope of Works") of this Report.
- c) This Report has been prepared for the sole benefit of Orica Australia Pty Ltd (Orica) and Goodman Pty Limited (The Proponent). Neither the whole nor any part of this Report may be used or relied upon by any party other than Orica Australia Pty Ltd (Orica) and Goodman Pty Limited. It is acknowledged that this Report may be used by the following parties for the purpose of assessing and approving activities described in this Report: where this Report is to be submitted to an accredited site auditor under the Contaminated Land Management Act 1997, that auditor; and where this Report is to be submitted to a public or regulatory authority pursuant to a requirement under applicable planning or environmental controls, that authority.
- d) This Report is dated October 2008 and is based on the conditions encountered during limited site investigations conducted, and information reviewed, from August 2006 to August 2008. URS accepts no responsibility for any events arising from any changes in site conditions or in the information reviewed that have occurred after the completion of the site investigations.
- e) Where this Report indicates that information has been provided to URS by third parties, URS has made no independent verification of this information except as expressly stated in the Report.
- f) Except as specifically stated above, URS makes no warranty, statement or representation of any kind concerning the suitability of the site for any purpose or the permissibility of any use, development or re-development of the site.
- g) URS makes no determination or recommendation regarding a decision to provide or not to provide financing with respect to the site.
- h) Investigations undertaken in respect of this Report are constrained by the particular site conditions, such as the location of services and vegetation. As a result, not all relevant site features and contamination may have been identified in this Report.
- i) Except as otherwise specifically stated in this Report, URS makes no warranty or representation as to the presence or otherwise of asbestos and/or asbestos containing materials ("ACM") on the site. If fill has been imported on to the site at any time, or if any buildings constructed prior to 1970 have been demolished on the site or materials from such buildings disposed of on the site, the site may contain asbestos or ACM. Even if asbestos was tested for and those test results did not reveal the presence of asbestos at specific points of sampling, asbestos may still be present at the site if fill has been imported at any time, or if any buildings constructed prior to 1970 have been demolished on the site or materials from such buildings disposed of on the site.
- j) Subsurface conditions can vary across a particular site and cannot be exhaustively defined by the investigations described in this Report. It is unlikely therefore that the results and estimations expressed in this Report will represent conditions at any location removed from the specific points of sampling.

Section 9

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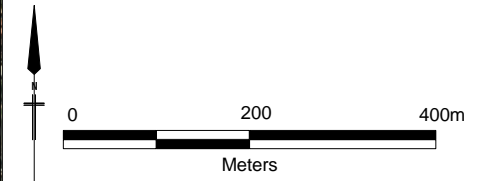
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Figures



- Legend:
- INVESTIGATION AREA
 - SPRINGVALE DRAIN
 - FLOODVALE DRAIN



Datum: GDA94, Projection: UTM, Grid: MGA Zone 56
 Map compiled using SKM Aerial Imagery, January 2005,
 Mapinfo StreetPro (and CadastralPlus) © 2005 and PSMA
 Australia Ltd.

Drawn: AJW | Approved: FINAL | Date: 21.02.08

Job No: **43217543** | File: 43217543.002.wor

Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
SITE LOCATION PLAN

Figure: 1



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LEGEND

- DT Inground Detention Tank
- AW Awning
- HS Hardstand

Development Area Schedule

STAGE 1	
Total Site Area	98,150 sqm
less Detention Basins	1,820 sqm
less Access Road	3,440 sqm
Dev. Site Area	92,890 sqm
Total Warehouse	43,000 sqm
Total Office (2 & 3 levels)	4,000 sqm
Total Floor Area	47,000 sqm
Awning	1,840 sqm
Future Parking Deck	4,655 sqm
Footprint (incl awning & parking deck)	50,935 sqm
Site Cover	55 %
FSR	51 %
Landscape Area - 23%	20,950 sqm
Carparking on grade	442
Future Carparking (2 lev)	300
Minor Earthworks Area	45,210 sqm

Drawn: AJW	Approved: FINAL	Date: 21.02.08
Job No: 43217543	File: 43217543.003.wor	
Client ORICA AUSTRALIA PTY LIMITED/ GOODMAN PTY LIMITED		
Project SOUTHLANDS REMEDIATION AND DEVELOPMENT PROJECT		
Title STAGE 1 SITE PLAN		



Southlands Remediation & Development Project
McPherson Street, Botany

STAGE 1 Site Plan

1:1000 @ A1	SRD DA006 (B)
1:2000 @ A3	
23 Jan 08	VK

Part 3A Project Application

Figure: 2



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LEGEND

DT Inground Detention Tank

Refer Plans SRD DA006 & SRD DA011

Subject to Future Project Approval



SOUTHLANDS AREA SCHEDULE

Lot 1 DP 254382	0.285 Ha
Lot 1 DP 528680	9.530 Ha
Lot 1 DP 85542	6.130 Ha
Lot 11 DP 109505	2.343 Ha
TOTAL SITE AREA	18.288 Ha
Springvale Drain	0.289 Ha
Narr Street	0.347 Ha

MASTERPLAN DEVELOPMENT AREA SCHEDULE

Total Site Area	18,288 Ha
Access Road	0.743 Ha
Detention Basins	1.049 Ha
TOTAL DEV. AREA	16,496 Ha
Total Warehouse	72,200 sqm
Total Office	6,750 sqm
Cafe/ Amenities	240 sqm
Total Floor Area	79,190 sqm
Total Awning	2,666 sqm
Possible Multi-deck Parking	4,665 sqm

FLOOR AREA 79,190 SQM
FOOTPRINT (incl. awning, parking deck) 83,355 SQM
FSR 0.48:1
SITE COVER 50.5%
LANDSCAPE AREA 41,360 SQM (25%)

Carparking Provided -on grade 820
 Possible Future Carparking (2 lev) 300
Total Carparking 1120

STAGE 1

TOTAL SITE AREA	98,150 sqm
DEV. SITE AREA	82,890 sqm
Total Warehouse	43,000 sqm
Total Office	4,000 sqm
Total Facility	47,000 sqm

Carparking Provided -on grade 440
 Possible Future Carparking (2 lev) 300
Total Carparking 740

STAGE 2

TOTAL SITE AREA	48,300 sqm
DEV. SITE AREA	40,310 sqm
Total Warehouse	14,850 sqm
Total Office	1,400 sqm
Cafe/ Amenities	240 sqm
Total Facility	16,490 sqm

Carparking Provided -on grade 280

STAGE 3

TOTAL SITE AREA	38,430 sqm
DEV. SITE AREA	31,760 sqm
Total Warehouse	14,350 sqm
Total Office	1,400 sqm
Total Facility	15,750 sqm

Carparking Provided -on grade 120

Drawn: AJW Approved: FINAL Date: 21.02.08

Job No: 43217543 File: 43217543.004.wor

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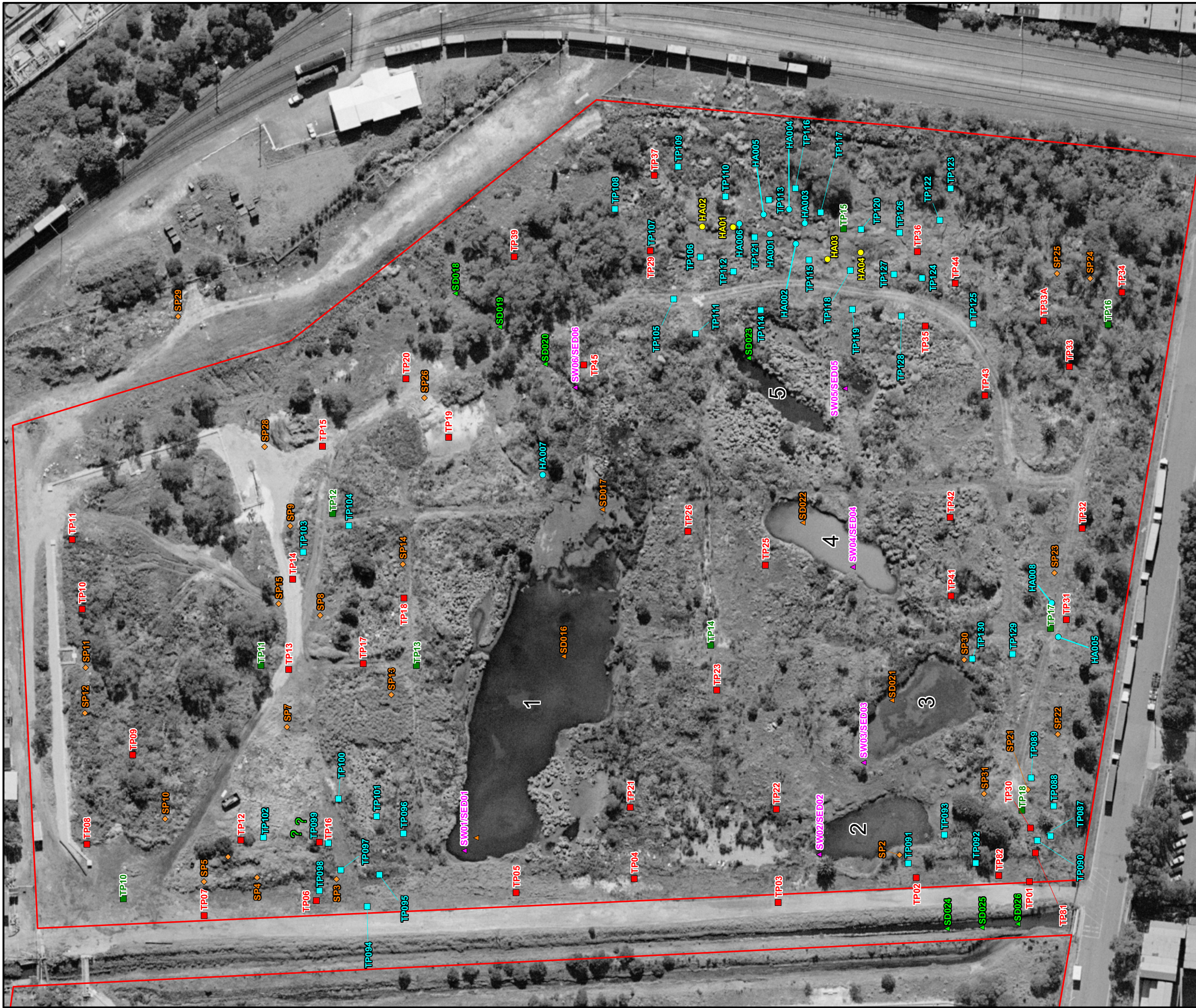
Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT


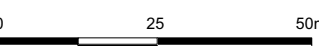

Title
 STAGE 2 SITE PLAN

Figure: 3

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Legend:		
— ORICA PROPERTY BOUNDARY		
URS DATA		
■	TEST PIT LOCATIONS	
●	HAND AUGER LOCATIONS	
HLA DATA		
▲	SEDIMENT SAMPLE LOCATIONS	
◆	STOCKPILE SAMPLE LOCATION	
●	HAND AUGER LOCATIONS	
■	TEST PIT LOCATION	
WOODWARD CLYDE DATA		
■	TEST PIT LOCATIONS	
●	HAND AUGER LOCATIONS	
 NORTH  Scale		
Source: SKM Ausimage, 2001, 2005 Datum: GDA94, Projection: UTM, Grid: MGA Zone 56		
Drawn: BH	Approved: GB	Date: 21/02/2008
Job No: 43217543	File No: 43217543.005.wor	
Client ORICA AUSTRALIA PTY LIMITED/ GOODMAN PTY LIMITED		
Project SOUTHLANDS REMEDIATION AND DEVELOPMENT PROJECT		
Title SOIL INVESTIGATION LOCATIONS - BLOCK 1		
Figure: 4		
		



- Legend:
- ORICA PROPERTY BOUNDARY
 - AMBIENT AIR (SUMMA CANISTER) SAMPLES
 - SOIL GAS SAMPLES
 - FLUX EMISSIONS SAMPLING LOCATION



Source: SKM Imagery, 2001, 2005
 Datum: GDA94, Projection: UTM, Grid: MGA Zone 56

Drawn: AW Approved: DRAFT Date: 09.10.06

Job No: 43217543 File: 43217543-006.wor

Client
 ORICA AUSTRALIA PTY LIMITED/
 GOODMAN PTY LIMITED

Project
 SOUTHLANDS REMEDIATION AND
 DEVELOPMENT PROJECT

Title
 AIR SAMPLING LOCATION
 PLAN - SOUTHLANDS

Figure: 6



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Appendix A

Summary of Soil and Groundwater Data

Appendix A

Soil and Groundwater Data

A.1 Introduction

This appendix presents a summary of the soil data considered in the HHRA undertaken for the assessment of Blocks 1 and 2 on Southlands. In addition groundwater data identified and considered relevant to the assessment of shallow groundwater on Block 1 is presented.

A.2 Soil Data

Soil data was collected by Woodward-Clyde (now URS) during the Stage 2 Survey in 1994 (reported in 1996) and by HLA in 2005. Additional soil data was collected by URS in 2006 as part of further contaminant delineation works undertaken on the site. These data are presented in this appendix for Block 1 and Block 2. The locations of the soil samples are presented in Figures 4 and 5.

A.2.1 Shallow Groundwater

The groundwater data identified as relevant to the current concentrations in shallow groundwater (used in the assessment of the flood storage area and Springvale Drain) have been obtained from the following reports:

- URS (2007b) Orica Botany Environmental Survey, Stage 4 – Remediation. Groundwater Treatment Plant (GTP) Quarterly Groundwater and Surface Water Monitoring Report - September 2007. 30 November 2007.
- URS (2007a) Interim Report on Environmental Monitoring at Springvale Drain. September 2007.
- URS (2006) Southern Plumes Source Area Delineation Investigation. February 2006.

The data, and review of the data, is included in this appendix. In addition the location of the groundwater monitoring wells, and maximum concentrations in the shallow groundwater (1-8m bgs) for key COPC are included in the figures attached (Figures 1.1-1.8).

Appendix A

Soil and Groundwater Data

A.3 Soil Data

**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	SS040	SS041	SS042	SS043	SS044	SS045	SS046	SS047	SS048	SS049	SS050	SS051	SS052	SS053	SS054	SS055	SS056	SS057	SS058	SS059	SS060	SS061
			TP10 0.1-0.3	TP10 0.8-1.0	TP11 0.1-0.3	TP11 0.5-0.7	TP12 0-0.2	TP12 0.3-0.5	TRENCH A 0.3-0.5	TRENCH A 1.1-1.3	TRENCH A 1.4-1.5	TRENCH A 0.3-0.5	TRENCH A 0.7-0.9	TRENCH A 0-0.35	TP13 0.1-0.3	TP13 1.0-1.3	TP14 0-0.15	TP14 0.5-0.55	TP15 0.1-0.3	TP15 0.7-0.8	TP16 0-0.15	TP16 0.9-1.0	TP17 0.1-0.3	TP17 0.7-0.9
Arsenic	mg/kg	500																						
Cadmium	mg/kg	100																						
Chromium	mg/kg	60	11.6	28.3	5.7	26.3	9.6	35.2	5.3	8.1	17.2	36.3	3.2	76.1	1.8	2.5	6.2	12.7	7.3	12.4	60.6	67.9	8.4	11.5
Copper	mg/kg	5000																						
Lead	mg/kg	1500																						
Nickel	mg/kg	3000																						
Zinc	mg/kg	35000																						
Mercury	mg/kg	75	2.9	1.7	<0.1	13.5	<0.1	1.7	<0.1	1.7	1.9	70.5	0.2	15.3	<0.1	<0.1	<0.1	0.5	0.2	0.1	1	2.7	<0.1	<0.1
Oil & Grease	mg/kg		-	-	-	-	-	-	-	-	-	18500	330	7960	-	-	700	3880	-	-	-	-	-	-
Total Organic Matter	mg/kg		-	-	-	-	-	-	-	-	-	138000	161000	118000	-	-	14400	68600	-	-	-	-	-	-
Formaldehyde	mg/kg		<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<2	<2	<10	<10	<10	<10	<10	<10	<10	<10
Total Phenols	mg/kg											2.5	<1	<1					<1	7				
Cation Exchange Capacity	mg/kg											14.5	18	18.6			2.7							
Ammonia-N	mg/kg											8.3	4				0.6	1.9						
Total Kjeldahl Nitrogen	mg/kg											832	1050	884			74.4	482						
P-Ortho	mg/kg											5.3	<0.7				1.7	1.9						
P-Total	mg/kg											29		19			6.3	12						

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
1 = Cr VI trigger value adopted as most conservative
-- = not analysed

**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	SS062		SS063																
			TP18	TP18	TP01 1.4-1.5	TP02 0.8-0.9	TP06 1.2-1.3	TP31 3.4-3.5	SP1	SP2	SP3	SP4	SP5	SP6	SP7	SP8	SP9	SP10	SP11	SP12	SP13
			0.2-0.4	0.5-0.7	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	11/04/2005	11/04/2005
Arsenic	mg/kg	500			61	18	6	26	6	7	9	<5	<5	8	<5	15	<5	6	7	<5	<5
Cadmium	mg/kg	100			<1	<1	4	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Chromium	mg/kg	60	4.8	20.7	15	9	12	1500	18	22	17	12	10	16	6	19	17	15	13	18	20
Copper	mg/kg	5000			135	38	157	84	53	64	86	47	33	87	10	112	41	89	57	86	67
Lead	mg/kg	1500			370	175	189	4890					51		10		26				
Nickel	mg/kg	3000			27	8	14	44	27	26		12	13	22	7	26		12	13	21	12
Zinc	mg/kg	35000			330	118	492	221	206	243	289	197	100	686	25	656	137	286	597	552	836
Mercury	mg/kg	75	<0.1	4.9	94.6	75.6	176	19.2						1.5	<0.1	<0.1	0.3			1	
Oil & Grease	mg/kg		-	-																	
Total Organic Matter	mg/kg		-	-																	
Formaldehyde	mg/kg		<10	<10																	
Total Phenols	mg/kg																				
Cation Exchange Capacity	mg/kg																				
Ammonia-N	mg/kg																				
Total Kjeldahl Nitrogen	mg/kg																				
P-Ortho	mg/kg																				
P-Total	mg/kg																				

Notes:
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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	SP14	SP15	SP21	SP22	SP23	SP24	SP25	SP26	SP27	SP28	SP29	SP30	SP31
			11/04/2005	11/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005
Arsenic	mg/kg	500	<5	9	<5	<5	<5	5	<5	<5	5	<5	<5	<5	<5
Cadmium	mg/kg	100	<1	1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Chromium	mg/kg	60	3	23	10	8	6	9	7	55	21	16	8	8	4
Copper	mg/kg	5000	22	156	14	36	28	29	12	86	76	22	28	19	16
Lead	mg/kg	1500	22		30	72	56	95	60			60	17	33	65
Nickel	mg/kg	3000	7	11	15	22	14	20	5	27	36	4	37	12	6
Zinc	mg/kg	35000	91	1420	75	207	172	121	55	172	214	76	53	84	50
Mercury	mg/kg	75	3.2	1	0.4	2	0.7	0.7	0.3			1.2	3		0.2
Oil & Grease	mg/kg														
Total Organic Matter	mg/kg														
Formaldehyde	mg/kg														
Total Phenols	mg/kg														
Cation Exchange Capacity	mg/kg														
Ammonia-N	mg/kg														
Total Kjeldahl Nitrogen	mg/kg														
P-Ortho	mg/kg														
P-Total	mg/kg														

Notes:
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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP105_1.00	TP105_2.10	TP106_1.00	TP106_1.70	TP107_1.00	TP107_2.10	QC109	TP108_1.00	TP108_2.10	TP109_1.20	TP109_2.00	TP110_0.90	TP110_1.90	TP111_0.90	TP111_2.10	TP112_1.40	TP112_2.20	TP113_0.80
			20/10/2006	20/10/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	3/11/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006	2/11/2006	2/11/2006	20/10/2006	20/10/2006	1/11/2006	1/11/2006
Arsenic	mg/kg	500	-	-	7	7	13	6	62	<5	10	8	<5	7	36	-	-	<5	<5	12
Cadmium	mg/kg	100	-	-	<1	<1	<1	<1	1	1	<1	<1	<1	<1	<1	-	-	2	2	<1
Chromium	mg/kg	60	-	-	12	8	12	18	49	22	8	22	<2	21	11	-	-	<2	<2	23
Copper	mg/kg	5000	-	-	256	105	45	160	179	240	48	202	<5	95	55	-	-	<5	<5	133
Lead	mg/kg	1500	-	-	97	94	1050	423	256	294	62	240	<5	123	48	-	-	<5	<5	172
Nickel	mg/kg	3000	-	-	12	10	15	12	369	18	20	22	<2	30	20	-	-	18	9	28
Zinc	mg/kg	35000	-	-	267	94	546	284	1400	967	324	233	10	534	143	-	-	669	238	228
Mercury	mg/kg	75	-	-	0.9	0.5	0.9	1.1	6.3	1.2	1.1	4.5	0.6	1.3	0.2	-	-	<0.1	<0.1	1.6
Oil & Grease	mg/kg																			
Total Organic Matter	mg/kg																			
Formaldehyde	mg/kg																			
Total Phenols	mg/kg																			
Cation Exchange Capacity	mg/kg																			
Ammonia-N	mg/kg																			
Total Kjeldahl Nitrogen	mg/kg																			
P-Ortho	mg/kg																			
P-Total	mg/kg																			

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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP113_1.70	QC106	TP114_1.00	TP114_2.00	TP115_1.00	TP116_0.80	TP116_1.90	TP116_2.40	TP117_1.00	TP117_2.00	TP118_0.90	TP118_1.00	TP118_1.40	TP118_1.80	TP119_1.00	TP119_1.70	TP120_0.90	TP120_1.40
			1/11/2006	1/11/2006	20/10/2006	20/10/2006	1/11/2006	2/11/2006	2/11/2006	1/11/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	2/11/2006	2/11/2006
Arsenic	mg/kg	500	19	<5	-	-	<5	9	17	<5	24	<5	34	11	30	<5	<5	<5	9	24
Cadmium	mg/kg	100	<1	<1	-	-	<1	3	6	<1	2	<1	<1	7	1	<1	<1	<1	9	<1
Chromium	mg/kg	60	9	<2	-	-	<2	23	40	<2	14	<2	58	7	10	<2	<2	<2	132	6
Copper	mg/kg	5000	11	<5	-	-	<5	274	452	<5	82	<5	192	71	24	<5	<5	<5	703	25
Lead	mg/kg	1500	19	<5	-	-	<5	290	277	<5	103	<5	165	18	16	<5	<5	<5	464	77
Nickel	mg/kg	3000	5	2	-	-	3	28	64	<2	101	<2	196	265	73	<2	<2	<2	78	64
Zinc	mg/kg	35000	20	158	-	-	228	810	1010	<5	4200	13	766	2640	760	<5	<5	36	1210	443
Mercury	mg/kg	75	0.3	<0.1	-	-	<0.1	10.3	6.7	0.2	0.8	<0.1	5	1.9	0.4	<0.1	<0.1	<0.1	35.3	0.6
Oil & Grease	mg/kg																			
Total Organic Matter	mg/kg																			
Formaldehyde	mg/kg																			
Total Phenols	mg/kg																			
Cation Exchange Capacity	mg/kg																			
Ammonia-N	mg/kg																			
Total Kjeldahl Nitrogen	mg/kg																			
P-Ortho	mg/kg																			
P-Total	mg/kg																			

Notes:
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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP120_1.80	TP121_0.90	TP122_0.80	TP122_1.90	TP123_0.90	TP123_1.50	TP123_2.10	TP124_1.00	TP124_1.90	TP125_1.00	TP125_1.60	TP126_0.70	QC107	QC202	TP126_1.30	TP126_1.90	TP127_1.10	TP127_2.20
			2/11/2006	1/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	3/11/2006	3/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006
Arsenic	mg/kg	500	<5	23	19	6	37	6	<5	<5	<5	8	<5	19	10	9	12	17	11	6
Cadmium	mg/kg	100	<1	4	8	<1	9	<1	<1	<1	1	<1	4	10	6.9	11	<1	2	<1	
Chromium	mg/kg	60	<2	66	68	2	234	36	3	11	3	18	<2	56	8	9	9	6	26	5
Copper	mg/kg	5000	<5	438	640	<5	1380	6	<5	57	12	194	<5	298	489	210	522	<5	209	<5
Lead	mg/kg	1500	<5	525	343	<5	338	<5	<5	91	10	250	<5	498	138	89	157	<5	7670	<5
Nickel	mg/kg	3000	<2	51	260	<2	373	42	2	43	18	22	4	64	241	180	248	15	90	13
Zinc	mg/kg	35000	7	279	3680	30	1370	232	13	143	26	288	11	835	1130	910	1210	62	649	62
Mercury	mg/kg	75	<0.1	2.5	21.9	<0.1	10.3	2.6	<0.1	1.1	0.3	2.1	0.4	2.6	0.4	0.28	1	<0.1	1.1	<0.1
Oil & Grease	mg/kg																			
Total Organic Matter	mg/kg																			
Formaldehyde	mg/kg																			
Total Phenols	mg/kg																			
Cation Exchange Capacity	mg/kg																			
Ammonia-N	mg/kg																			
Total Kjeldahl Nitrogen	mg/kg																			
P-Ortho	mg/kg																			
P-Total	mg/kg																			

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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP127_2.30	TP128_0.80	QC108	TP128_1.70	HA001	HA002	HA003	HA004	HA005	HA006	TP087_1.00	TP087_1.90	TP087_2.70	TP088_2.10	TP088_3.00	TP089_1.00	TP089_2.70	TP090_1.80
			2/11/2006	2/11/2006	2/11/2006	2/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	18/10/2006	18/10/2006
Arsenic	mg/kg	500	<5	<5	<5	<5	<5	7	10	<5	<5	<5	-	-	<5	6	-	<5	-	<5
Cadmium	mg/kg	100	<1	<1	<1	<1	<1	2	<1	<1	4	<1	-	-	<1	<1	-	<1	-	<1
Chromium	mg/kg	60	<2	5	<2	<2	6	15	16	8	8	4	-	-	3	9	-	10	-	7
Copper	mg/kg	5000	<5	13	<5	<5	143	326	87	44	121	17	-	-	<5	20	-	34	-	22
Lead	mg/kg	1500	<5	11	<5	<5	93	597	178	62	336	16	-	-	<5	51	-	39	-	71
Nickel	mg/kg	3000	4	6	<2	<2	4	23	17	8	25	7	-	-	2	16	-	18	-	12
Zinc	mg/kg	35000	20	19	7	9	52	347	439	122	665	58	-	-	<5	103	-	87	-	120
Mercury	mg/kg	75	0.2	<0.1	<0.1	<0.1	0.4	2.4	2.2	1.1	1	0.3	-	-	<0.1	24	-	2.9	-	5.1
Oil & Grease	mg/kg																			
Total Organic Matter	mg/kg																			
Formaldehyde	mg/kg																			
Total Phenols	mg/kg																			
Cation Exchange Capacity	mg/kg																			
Ammonia-N	mg/kg																			
Total Kjeldahl Nitrogen	mg/kg																			
P-Ortho	mg/kg																			
P-Total	mg/kg																			

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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP090_2.80	TP091_1.00	TP091_1.50	TP091_2.40	TP092_1.00	TP092_2.10	QC102	TP093_0.90	TP093_1.70	TP094_0.90	TP094_2.00	TP095_1.00	TP095_1.90	TP096_0.90	TP097_0.90	TP097_2.90	TP098_1.00	TP098_1.90
			18/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006	20/10/2006
Arsenic	mg/kg	500	-	<5	-	-	-	-	-	-	-	5	<5	8	<5	<5	5	11	9	<5
Cadmium	mg/kg	100	-	<1	-	-	-	-	-	-	-	1	<1	2	<1	<1	2	2	2	<1
Chromium	mg/kg	60	-	19	-	-	-	-	-	-	-	37	10	38	<2	3	30	46	46	2
Copper	mg/kg	5000	-	50	-	-	-	-	-	-	-	491	19	268	<5	42	220	176	311	8
Lead	mg/kg	1500	-	181	-	-	-	-	-	-	-	465	98	224	<5	106	877	1490	1130	18
Nickel	mg/kg	3000	-	20	-	-	-	-	-	-	-	24	3	28	<2	6	29	30	32	2
Zinc	mg/kg	35000	-	256	-	-	-	-	-	-	-	408	87	552	6	211	833	710	734	34
Mercury	mg/kg	75	-	52.9	-	-	-	-	-	-	-	-	4.3	35.5	<0.1	0.5	24.2	22.9	-	3.1
Oil & Grease	mg/kg																			
Total Organic Matter	mg/kg																			
Formaldehyde	mg/kg																			
Total Phenols	mg/kg																			
Cation Exchange Capacity	mg/kg																			
Ammonia-N	mg/kg																			
Total Kjeldahl Nitrogen	mg/kg																			
P-Ortho	mg/kg																			
P-Total	mg/kg																			

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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP098_2.40	TP099_1.00	TP099_1.90	TP100_1.80	TP100_2.50	QC104	TP101_1.00	TP101_1.80	TP102_1.00	TP102_2.00	QC103	QC201	TP103_0.80	TP103_1.80	TP103_2.90	QC105	TP104_0.70	TP104_1.90
			20/10/2006	20/10/2006	20/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006	20/10/2006	20/10/2006
Arsenic	mg/kg	500	<5	<5	<5	<5	<5	<5	<5	8	<5	<5	<5	2	-	-	-	-	-	-
Cadmium	mg/kg	100	<1	<1	<1	<1	<1	<1	<1	3	<1	<1	<1	<0.1	-	-	-	-	-	-
Chromium	mg/kg	60	3	11	<2	7	3	4	7	22	8	<2	<2	2	-	-	-	-	-	-
Copper	mg/kg	5000	<5	60	<5	46	<5	<5	34	158	28	<5	<5	5	-	-	-	-	-	-
Lead	mg/kg	1500	9	87	<5	62	<5	<5	215	245	216	<5	<5	4	-	-	-	-	-	-
Nickel	mg/kg	3000	<2	9	<2	5	<2	<2	3	24	6	<2	<2	1	-	-	-	-	-	-
Zinc	mg/kg	35000	20	172	7	118	<5	<5	112	664	158	5	5	13	-	-	-	-	-	-
Mercury	mg/kg	75	1	6.5	0.1	1.1	<0.1	<0.1	0.4	5.5	2.7	0.1	<0.1	0.48	-	-	-	-	-	-
Oil & Grease	mg/kg																			
Total Organic Matter	mg/kg																			
Formaldehyde	mg/kg																			
Total Phenols	mg/kg																			
Cation Exchange Capacity	mg/kg																			
Ammonia-N	mg/kg																			
Total Kjeldahl Nitrogen	mg/kg																			
P-Ortho	mg/kg																			
P-Total	mg/kg																			

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**Southlands Soil Data - Metals
Block 1**

Analyte	Units	NEPM-F	TP129_1.50	TP129_3.00	QC100	QC200	TP130_1.00	QC101	HA007	QC001	HA008	HA009
			18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	8/11/2006	8/11/2006	8/11/2006
Arsenic	mg/kg	500	12	-	-	-	<5	-	<5	<5	9	<5
Cadmium	mg/kg	100	2	-	-	-	<1	-	<1	<1	2	<1
Chromium	mg/kg	60	49	-	-	-	5	-	10	13	30	9
Copper	mg/kg	5000	249	-	-	-	59	-	66	79	856	34
Lead	mg/kg	1500	375	-	-	-	98	-	63	99	1300	70
Nickel	mg/kg	3000	54	-	-	-	17	-	4	5	26	14
Zinc	mg/kg	35000	667	-	-	-	166	-	100	108	646	216
Mercury	mg/kg	75	7	-	-	-	1.8	-	0.4	0.3	6.4	0.5
Oil & Grease	mg/kg											
Total Organic Matter	mg/kg											
Formaldehyde	mg/kg											
Total Phenols	mg/kg											
Cation Exchange Capacity	mg/kg											
Ammonia-N	mg/kg											
Total Kjeldahl Nitrogen	mg/kg											
P-Ortho	mg/kg											
P-Total	mg/kg											

Notes:
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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	SS040	SS041	SS042	SS043	SS044	SS045	SS046	SS047	SS048	SS049	SS050	SS051	SS052	SS053	SS054	SS055	SS056	SS057	SS058	
			TP10_0.1-0.3	TP10_0.8-1.0	TP11_0.1-0.3	TP11_0.5-0.7	TP12	TP12	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TP13	TP13	TP14	TP14	TP15	TP15	TP15	TP16
Total Petroleum Hydrocarbons								0-0.2	0.3-0.5	0.3-0.5	1.1-1.3	1.4-1.5	0.3-0.5	0.7-0.9	0-0.35	0.1-0.3	1.0-1.3	0-0.15	0.5-0.55	0.1-0.3	0.7-0.8	0-0.15
C6-C9Fraction	mg/kg	65																				
C10-C14Fraction	mg/kg	1000																				
C15-C28Fraction	mg/kg	1000																				
C29-C36Fraction	mg/kg	1000																				
Monocyclic Aromatic Hydrocarbons																						
Benzene	mg/kg	1	<0.01	<0.01	<0.01	<0.01	< 0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	0.12	<0.01
Toluene	mg/kg	1.4	<0.01	<0.01	<0.01	<0.01	< 0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	0.09	<0.01
Ethylbenzene	mg/kg	3.1	<0.01	<0.01	<0.01	<0.01	< 0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04	<0.01
meta- & para-Xylene	mg/kg	14	<0.01	<0.01	<0.01	<0.01	< 0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04	<0.01
ortho-Xylene	mg/kg	14	<0.01	<0.01	<0.01	<0.01	< 0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04	<0.01
Isopropylbenzene	mg/kg	20																				
n-Propylbenzene	mg/kg	2.4																				
1,3,5-Trimethylbenzene	mg/kg	0.7																				
sec-Butylbenzene	mg/kg	2.2																				
1,2,4-Trimethylbenzene	mg/kg	1.7																				
n-Butylbenzene	mg/kg	2.4																				
p-Isopropyltoluene	mg/kg																					
Styrene	mg/kg	17																				
tert-Butylbenzene	mg/kg	3.9																				

Notes:
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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	SS059	SS060	SS061	SS062	SS063											HA02
			TP16	TP17	TP17	TP18	TP18	TP01_1.4-1.5	TP14_0.6-0.7	TP20_1.9-2.0	TP26_0.8-0.9	TP29_0.3-0.4	TP30_1.3-1.4	TP37_1.2-1.3	TP42_2.5-2.6	TP81_1.2-1.3	TP82_1.4-1.5	
			0.9-1.0	0.1-0.3	0.7-0.9	0.2-0.4	0.5-0.7	7/04/2005	8/04/2005	7/04/2005	8/04/2005	7/04/2005	7/04/2005	7/04/2005	8/04/2005	7/04/2005	7/04/2005	8/04/2005
Total Petroleum Hydrocarbons																		
C6-C9Fraction	mg/kg	65					2520	<2	<2	<2	36	5	42	<2	1870	12100	<5	
C10-C14Fraction	mg/kg	1000					<50	70	<50	<50	<50	<50	<50	<50	1370	2440	230	
C15-C28Fraction	mg/kg	1000					1050	1820	220	170	310	250	360	310	1540	1200	2630	
C29-C36Fraction	mg/kg	1000					1910	2720	210	280	430	460	470	520	340	300	1690	
Monocyclic Aromatic Hydrocarbons																		
Benzene	mg/kg	1	<0.01	<0.01	<0.01	<0.01	31.3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	11.6	10.3	<0.5	
Toluene	mg/kg	1.4	<0.01	<0.01	<0.01	<0.01	1.2	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	4.8	<0.5	
Ethylbenzene	mg/kg	3.1	<0.01	<0.01	<0.01	<0.01	15.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.7	141	<0.5	
meta-¶-Xylene	mg/kg	14	<0.01	<0.01	<0.01	<0.01	8.4	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	1.1	35	<1.0	
ortho-Xylene	mg/kg	14	<0.01	<0.01	<0.01	<0.01	1.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	6.3	<0.5	
Isopropylbenzene	mg/kg	20					1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	7.4	14.4	<0.5	
n-Propylbenzene	mg/kg	2.4					3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	27.8	70.2	<0.5	
1,3,5-Trimethylbenzene	mg/kg	0.7					0.8	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	1.5	<0.5	
sec-Butylbenzene	mg/kg	2.2					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	3.1	<0.5	
1,2,4-Trimethylbenzene	mg/kg	1.7					5.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	20	<0.5	
n-Butylbenzene	mg/kg	2.4																
p-Isopropyltoluene	mg/kg																	
Styrene	mg/kg	17																
tert-Butylbenzene	mg/kg	3.9																

Notes:
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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	HA03	TP105_1.00	TP105_2.10	TP106_1.00	TP106_1.70	TP107_1.00	TP107_2.10	QC109	TP108_1.00	TP108_2.10	TP109_1.20	TP109_2.00	TP110_0.90	TP110_1.90	TP111_0.90	TP111_2.10	TP112_1.40
			8/04/2005	20/10/2006	20/10/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	3/11/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006	2/11/2006	2/11/2006	20/10/2006	20/10/2006
Total Petroleum Hydrocarbons																			
C6-C9Fraction	mg/kg	65	<2	102	-	15	20	-	-	4	<2	-	-	-	-	<5	9	-	23
C10-C14Fraction	mg/kg	1000	<50	<50	-	<50	<50	-	-	<50	<50	-	-	-	-	<120	<50	-	<50
C15-C28Fraction	mg/kg	1000	140	<100	-	<100	230	-	-	340	<100	-	-	-	-	900	<100	-	<100
C29-C36Fraction	mg/kg	1000	200	<100	-	<100	350	-	-	510	<100	-	-	-	-	1740	<100	-	240
Monocyclic Aromatic Hydrocarbons																			
Benzene	mg/kg	1	<0.5	<0.2	-	<0.2	<0.2	<0.5	<0.5	<0.5	<0.2	-	-	-	<0.5	<0.5	<0.2	-	<0.2
Toluene	mg/kg	1.4	<0.5	<0.2	-	<0.2	<0.2	-	-	<0.2	<0.2	-	-	-	-	<0.5	<0.2	-	<0.2
Ethylbenzene	mg/kg	3.1	<0.5	<0.2	-	<0.2	<0.2	-	-	<0.2	<0.2	-	-	-	-	<0.5	<0.2	-	<0.2
meta-¶-Xylene	mg/kg	14	<0.5	<0.2	-	<0.2	<0.2	-	-	<0.2	<0.2	-	-	-	-	<0.5	<0.2	-	<0.2
ortho-Xylene	mg/kg	14	<0.5	<0.2	-	<0.2	<0.2	-	-	<0.2	<0.2	-	-	-	-	<0.5	<0.2	-	<0.2
Isopropylbenzene	mg/kg	20	<0.5	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
n-Propylbenzene	mg/kg	2.4	<0.5	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
1,3,5-Trimethylbenzene	mg/kg	0.7	<0.5	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
sec-Butylbenzene	mg/kg	2.2	<0.5	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
1,2,4-Trimethylbenzene	mg/kg	1.7	<0.5	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
n-Butylbenzene	mg/kg	2.4	-	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
p-Isopropyltoluene	mg/kg		-	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
Styrene	mg/kg	17	-	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-
tert-Butylbenzene	mg/kg	3.9	-	-	-	<0.5	-	<0.5	<0.5	<0.5	-	-	-	-	<0.5	<0.5	-	-	-

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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	TP112_2.20	TP113_0.80	TP113_1.70	QC106	TP114_1.00	TP114_2.00	TP115_1.00	TP116_0.80	TP116_1.90	TP116_2.40	TP117_1.00	TP117_2.00	TP118_0.90	TP118_1.00	TP118_1.40	TP118_1.80	TP119_1.00
			1/11/2006	1/11/2006	1/11/2006	1/11/2006	20/10/2006	20/10/2006	1/11/2006	2/11/2006	2/11/2006	2/11/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006
Total Petroleum Hydrocarbons																			
C6-C9Fraction	mg/kg	65	10	-	-	-	-	<2	-	-	-	-	<2	-	4	-	-	-	<2
C10-C14Fraction	mg/kg	1000	<50	-	-	-	-	<50	-	-	-	-	<60	-	<50	-	-	-	<50
C15-C28Fraction	mg/kg	1000	<100	-	-	-	-	<100	-	-	-	-	1110	-	470	-	-	-	<100
C29-C36Fraction	mg/kg	1000	<100	-	-	-	-	<100	-	-	-	-	1420	-	700	-	-	-	<100
Monocyclic Aromatic Hydrocarbons			ND	-	-	-	-	ND	-	-	-	-	2530	-	1170	-	-	-	ND
Benzene	mg/kg	1	<0.2	-	-	-	-	<0.2	-	<0.5	<0.5	<0.5	<0.5	-	<0.2	<0.5	<0.5	<0.5	<0.2
Toluene	mg/kg	1.4	<0.2	-	-	-	-	<0.2	-	-	-	-	<0.2	-	<0.2	-	-	-	<0.2
Ethylbenzene	mg/kg	3.1	<0.2	-	-	-	-	<0.2	-	-	-	-	<0.2	-	<0.2	-	-	-	<0.2
meta-¶-Xylene	mg/kg	14	<0.2	-	-	-	-	<0.2	-	-	-	-	<0.2	-	<0.2	-	-	-	<0.2
ortho-Xylene	mg/kg	14	<0.2	-	-	-	-	<0.2	-	-	-	-	<0.2	-	<0.2	-	-	-	<0.2
Isopropylbenzene	mg/kg	20	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
n-Propylbenzene	mg/kg	2.4	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trimethylbenzene	mg/kg	0.7	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
sec-Butylbenzene	mg/kg	2.2	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4-Trimethylbenzene	mg/kg	1.7	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
n-Butylbenzene	mg/kg	2.4	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
p-Isopropyltoluene	mg/kg		-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
Styrene	mg/kg	17	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5
tert-Butylbenzene	mg/kg	3.9	-	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5

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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	TP119_1.70	TP120_0.90	TP120_1.40	TP120_1.80	TP121_0.90	TP122_0.80	TP122_1.90	TP123_0.90	TP123_1.50	TP123_2.10	TP124_1.00	TP124_1.90	TP125_1.00	TP125_1.60	TP126_0.70	QC107	QC202
			3/11/2006	2/11/2006	2/11/2006	2/11/2006	1/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	3/11/2006	3/11/2006	2/11/2006	2/11/2006
Total Petroleum Hydrocarbons																			
C6-C9Fraction	mg/kg	65	-	-	27	<2	8	-	-	<2	<5	-	-	-	-	<2	8	<2	<2
C10-C14Fraction	mg/kg	1000	-	-	<120	<50	<50	-	-	<50	<120	-	-	-	-	<50	<50	<50	<50
C15-C28Fraction	mg/kg	1000	-	-	690	<100	1890	-	-	970	740	-	-	-	-	<100	220	390	180
C29-C36Fraction	mg/kg	1000	-	-	1420	<100	1880	-	-	2000	1700	-	-	-	-	<100	280	780	530
Monocyclic Aromatic Hydrocarbons																			
Benzene	mg/kg	1	<0.5	<0.5	<0.5	<0.5	<0.2	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	500	1170
Toluene	mg/kg	1.4	-	-	<0.5	<0.2	<0.2	-	-	<0.2	<0.5	-	-	-	-	<0.2	<0.2	<0.2	-
Ethylbenzene	mg/kg	3.1	-	-	<0.5	<0.2	<0.2	-	-	<0.2	<0.5	-	-	-	-	<0.2	<0.2	<0.2	-
meta-para-Xylene	mg/kg	14	-	-	<0.5	<0.2	<0.2	-	-	<0.2	<0.5	-	-	-	-	<0.2	<0.2	<0.2	-
ortho-Xylene	mg/kg	14	-	-	<0.5	<0.2	<0.2	-	-	<0.2	<0.5	-	-	-	-	<0.2	<0.2	<0.2	-
Isopropylbenzene	mg/kg	20	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
n-Propylbenzene	mg/kg	2.4	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trimethylbenzene	mg/kg	0.7	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
sec-Butylbenzene	mg/kg	2.2	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4-Trimethylbenzene	mg/kg	1.7	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
n-Butylbenzene	mg/kg	2.4	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
p-Isopropyltoluene	mg/kg		<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Styrene	mg/kg	17	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
tert-Butylbenzene	mg/kg	3.9	<0.5	<0.5	<0.5	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5

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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	TP126_1.30	TP126_1.90	TP127_1.10	TP127_2.20	TP127_2.30	TP128_0.80	QC108	TP128_1.70	HA001	HA002	HA003	HA004	HA005	HA006	TP087_1.00	TP087_1.90	TP087_2.70	TP088_2.10
			2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	2/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	19/10/2006	19/10/2006	19/10/2006
Total Petroleum Hydrocarbons																				
C6-C9Fraction	mg/kg	65	<2	-	-	-	-	-	-	-	-	-	-	-	-	-	<2	<2	137	9
C10-C14Fraction	mg/kg	1000	<50	-	-	-	-	-	-	-	-	-	-	-	-	-	<50	<50	<50	<50
C15-C28Fraction	mg/kg	1000	280	-	-	-	-	-	-	-	-	-	-	-	-	-	<100	<100	<100	<100
C29-C36Fraction	mg/kg	1000	640	-	-	-	-	-	-	-	-	-	-	-	-	-	<100	<100	<100	<100
Monocyclic Aromatic Hydrocarbons																				
Benzene	mg/kg	1	<0.2	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.2	<0.2	<0.2	<0.2
Toluene	mg/kg	1.4	<0.2	-	-	-	-	-	-	-	-	-	-	-	-	-	<0.2	<0.2	<0.2	0.3
Ethylbenzene	mg/kg	3.1	<0.2	-	-	-	-	-	-	-	-	-	-	-	-	-	<0.2	<0.2	1	<0.2
meta-¶-Xylene	mg/kg	14	<0.2	-	-	-	-	-	-	-	-	-	-	-	-	-	<0.2	<0.2	<0.2	0.7
ortho-Xylene	mg/kg	14	<0.2	-	-	-	-	-	-	-	-	-	-	-	-	-	<0.2	<0.2	<0.2	0.2
Isopropylbenzene	mg/kg	20	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
n-Propylbenzene	mg/kg	2.4	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trimethylbenzene	mg/kg	0.7	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
sec-Butylbenzene	mg/kg	2.2	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4-Trimethylbenzene	mg/kg	1.7	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
n-Butylbenzene	mg/kg	2.4	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
p-Isopropyltoluene	mg/kg		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Styrene	mg/kg	17	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
tert-Butylbenzene	mg/kg	3.9	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5

Notes:
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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	TP088_3.00	TP089_1.00	TP089_2.70	TP090_1.80	TP090_2.80	TP091_1.00	TP091_1.50	TP091_2.40	TP092_1.00	TP092_2.10	QC102	TP093_0.90	TP093_1.70	TP103_0.80	TP103_1.80	TP103_2.90	QC105
			19/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006	20/10/2006
Total Petroleum Hydrocarbons																			
C6-C9Fraction	mg/kg	65	<2	10	-	3	-	<2	<5	<2	<2	7	7	<2	-	<2	<2	<2	<2
C10-C14Fraction	mg/kg	1000	<50	660	-	<50	-	<50	<120	<50	<50	<50	<50	<50	-	<50	<50	<50	<50
C15-C28Fraction	mg/kg	1000	<100	2870	-	<100	-	<100	<250	<100	<100	<100	<100	<100	-	<100	<100	<100	<100
C29-C36Fraction	mg/kg	1000	<100	<100	-	<100	-	<100	<250	<100	<100	<100	<100	<100	-	<100	<100	<100	<100
Monocyclic Aromatic Hydrocarbons			ND	3530	-	ND	-	ND	ND	ND	ND	ND	ND	ND	-	ND	ND	ND	ND
Benzene	mg/kg	1	<0.2	<0.2	-	<0.2	-	<0.2	<0.5	<0.2	<0.2	<0.2	<0.2	<0.2	-	<0.2	<0.2	<0.2	<0.2
Toluene	mg/kg	1.4	<0.2	<0.2	-	<0.2	-	<0.2	<0.5	<0.2	<0.2	<0.2	<0.2	<0.2	-	<0.2	<0.2	<0.2	<0.2
Ethylbenzene	mg/kg	3.1	<0.2	<0.2	-	<0.2	-	<0.2	<0.5	<0.2	<0.2	<0.2	<0.2	<0.2	-	<0.2	<0.2	<0.2	<0.2
meta-¶-Xylene	mg/kg	14	<0.2	<0.2	-	<0.2	-	<0.2	<0.5	<0.2	<0.2	<0.2	<0.2	<0.2	-	<0.2	<0.2	<0.2	<0.2
ortho-Xylene	mg/kg	14	<0.2	<0.2	-	<0.2	-	<0.2	<0.5	<0.2	<0.2	<0.2	<0.2	<0.2	-	<0.2	<0.2	<0.2	<0.2
Isopropylbenzene	mg/kg	20																	
n-Propylbenzene	mg/kg	2.4																	
1,3,5-Trimethylbenzene	mg/kg	0.7																	
sec-Butylbenzene	mg/kg	2.2																	
1,2,4-Trimethylbenzene	mg/kg	1.7																	
n-Butylbenzene	mg/kg	2.4																	
p-Isopropyltoluene	mg/kg																		
Styrene	mg/kg	17																	
tert-Butylbenzene	mg/kg	3.9																	

Notes:
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**Southlands Soil Data - TPH and MAH
Block 1**

Analyte	Units	NSW_EPA_1994/ USEPA Region 9	TP104_0.70	TP104_1.90	TP129_1.50	TP129_3.00	QC100	QC200	TP130_1.00	QC101	HA007	QC001	HA008	HA009
			20/10/2006	20/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	8/11/2006	8/11/2006	8/11/2006
Total Petroleum Hydrocarbons														
C6-C9Fraction	mg/kg	65	<2	<2	<2	-	-	-	<2	-	-	-	-	-
C10-C14Fraction	mg/kg	1000	<50	<50	<50	-	-	-	<50	-	-	-	-	-
C15-C28Fraction	mg/kg	1000	<100	<100	670	-	-	-	<100	-	-	-	-	-
C29-C36Fraction	mg/kg	1000	<100	<100	940	-	-	-	<100	-	-	-	-	-
Monocyclic Aromatic Hydrocarbons			ND	ND	1610	-	-	-	ND	-	-	-	-	-
Benzene	mg/kg	1	<0.2	<0.2	<0.2	-	-	-	<0.2	-	<0.5	<0.5	-	-
Toluene	mg/kg	1.4	<0.2	<0.2	<0.2	-	-	-	<0.2	-	-	-	-	-
Ethylbenzene	mg/kg	3.1	<0.2	<0.2	<0.2	-	-	-	<0.2	-	-	-	-	-
meta-¶-Xylene	mg/kg	14	<0.2	<0.2	<0.2	-	-	-	<0.2	-	-	-	-	-
ortho-Xylene	mg/kg	14	<0.2	<0.2	<0.2	-	-	-	<0.2	-	-	-	-	-
Isopropylbenzene	mg/kg	20												
n-Propylbenzene	mg/kg	2.4												
1,3,5-Trimethylbenzene	mg/kg	0.7												
sec-Butylbenzene	mg/kg	2.2												
1,2,4-Trimethylbenzene	mg/kg	1.7												
n-Butylbenzene	mg/kg	2.4												
p-Isopropyltoluene	mg/kg													
Styrene	mg/kg	17												
tert-Butylbenzene	mg/kg	3.9												

Notes:
 -- = criteria not available
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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX	SS040	SS041	SS042	SS043	SS044	SS045	SS046	SS047	SS048	SS049
		(Industrial Land) mg/kg	TP10_0.1-0.3	TP10_0.8-1.0	TP11_0.1-0.3	TP11_0.5-0.7	TP12	TP12	TRENCH A	TRENCH A	TRENCH A	TRENCH A
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
1,1-Dichloroethene	mg/kg	4.1	<0.02	<0.02	<0.02	<0.02	<0.02	0.02	<0.02	<0.02	<0.02	<0.02
trans-1,2-Dichloroethene	mg/kg	2.3	<0.02	<0.02	<0.02	<0.02	<0.02	0.10	<0.02	<0.02	<0.02	<0.02
1,1-Dichloroethane	mg/kg	17										
cis-1,2-Dichloroethane	mg/kg	1.5	<0.02	<0.02	<0.02	<0.02	<0.02	0.10	<0.02	<0.02	<0.02	<0.02
1,2-Dichloroethane	mg/kg	0.006										
Trichloroethene	mg/kg	0.0011	<0.15	<0.15	<0.15	<0.15	0.20	0.20	<0.15	<0.15	<0.15	<0.15
1,1,2-Trichloroethane	mg/kg	0.016										
Tetrachloroethene	mg/kg	0.013	<0.15	<0.15	<0.15	<0.15	0.30	<0.15	<0.15	<0.15	<0.15	<0.15
1,1,2,2-Tetrachloroethane	mg/kg	0.0093										
Hexachlorobutadiene	mg/kg	0.22										
Trihalomethanes												
Chloroform	mg/kg	0.12	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4
Bromochloromethane												
Bromodichloromethane												
Bromoform												
Dibromochloromethane												
1,1,1,2-Tetrachloroethane												
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Dichloromethane	mg/kg	0.21	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chloromethane	mg/kg	1.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
1,1,2-Trichloroethane	mg/kg	0.016	<0.01	<0.01	<0.01	<0.01	0.04	<0.01	<0.01	<0.01	<0.01	<0.01
1,2-Dichloroethane	mg/kg	0.006	<0.01	<0.01	<0.01	<0.01	<0.01	0.04	<0.01	<0.01	<0.01	<0.01
Chloroethane	mg/kg	0.065	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Miscellaneous												
Ethene	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclopentane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclohexane	mg/kg	1.4	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Carbon Disulphide- Disulphide	mg/kg		<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4
1,1-Dichloropropylene												
1,2,3-Trichloropropane												
1,2-Dibromo-3-chloropropane												
1,3-Dichloropropane												
Bromobenzene												
Bromomethane												
Carbon Tetrachloride												
cis-1,4-Dichloro-2-butene												
Dibromomethane												
Dichlorodifluoromethane												
Pentachloroethane												
Tetrachloroethane												
trans-1,3-dichloropropene												
trans-1,4-Dichloro-2-butene												
Trichlorofluoromethane												
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene												
1,2,4-Trichlorobenzene												
1,2-Dichlorobenzene												
1,3-Dichlorobenzene												
1,4-Dichlorobenzene												
2-Chlorotoluene												
4-Chlorotoluene												
Chlorobenzene												
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene												
1,3-Dichlorobenzene												
1,4-Dichlorobenzene												
Hexachlorobenzene												
Hexachlorobutadiene												
Hexachlorocyclopentadiene												
Hexachloroethane												
Hexachloropropylene												
Pentachlorobenzene												
Fumigants												
1,2-Dibromomethane												
1,2-Dichloropropane												
2,2-Dichloropropane												
cis-1,3-Dichloropropylene												
trans-1,3-Dichloropropylene												
2-Butanone (MEK)												
2-Hexanone (MBK)												
2-Propanone												
4-Methyl-2-pentanone												
Vinyl Acetate												
Iodomethane												
Haloothers												
4-Bromophenyl phenyl ether												
4-Chlorophenyl phenyl ether												
Bis(2-chloroethoxy) methane												
Bis(2-chloroethyl) ether												

Notes:

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- Bold/ Box = concentrations above criteria
- = not analysed

- * : Results determined using Methanol Extraction Method and are corrected for blank.
- # : Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for both methods and therefore reported 'Neat' even though originally outside calibration.
- ## : Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
- ^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because of inhomogeneity).

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX	SS050	SS051	SS052	SS053	SS054	SS055	SS056	SS057
		(Industrial Land) mg/kg	TRENCH A 0.7-0.9	TRENCH A 0-0.35	TP13 0.1-0.3	TP13 1.0-1.3	TP14 0-0.15	TP14 0.5-0.55	TP15 0.1-0.3	TP15 0.7-0.8
Halogenated Aliphatic Compounds										
Vinylchloride	mg/kg	0.0075	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	0.11	3.5*
1,1-Dichloroethene	mg/kg	4.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	0.06	4.4*
trans-1,2-Dichloroethene	mg/kg	2.3	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	0.02	12*
1,1-Dichloroethane	mg/kg	17								
cis-1,2-Dichloroethene	mg/kg	1.5	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	21*
1,2-Dichloroethane	mg/kg	0.006								
Trichloroethene	mg/kg	0.0011	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	12*
1,1,2-Trichloroethane	mg/kg	0.016								
Tetrachloroethene	mg/kg	0.013	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	4.4*
1,1,2,2-Tetrachloroethane	mg/kg	0.0093								
Hexachlorobutadiene	mg/kg	0.22								
Trihalomethanes										
Chloroform	mg/kg	0.12	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	1.1*
Bromochloromethane										
Bromodichloromethane										
Bromoform										
Dibromochloromethane										
1,1,1,2-Tetrachloroethane										
Other Chloromethanes										
Carbon Tetrachloride	mg/kg	0.005	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04
Dichloromethane	mg/kg	0.21	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.4
Chloromethane	mg/kg	1.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	0.05
Chloroethanes										
1,1,1-Trichloroethane	mg/kg	12	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04
1,1,2-Trichloroethane	mg/kg	0.016	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04
1,2-Dichloroethane	mg/kg	0.006	<0.01	<0.01	<0.02	<0.02	<0.01	<0.01	<0.01	<0.04
Chloroethane	mg/kg	0.065	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.04
Miscellaneous										
Ethane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclopentane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclohexane	mg/kg	1.4	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Carbon Disulphide- Disulphide	mg/kg		<0.4	<0.4	-	-	<0.4	<0.4	<0.4	<0.4
1,1-Dichloropropylene										
1,2,3-Trichloropropane										
1,2-Dibromo-3-chloropropane										
1,3-Dichloropropane										
Bromobenzene										
Bromomethane										
Carbon Tetrachloride										
cis-1,4-Dichloro-2-butene										
Dibromomethane										
Dichlorodifluoromethane										
Pentachloroethane										
Tetrachloroethane										
trans-1,3-dichloropropene										
trans-1,4-Dichloro-2-butene										
Trichlorofluoromethane										
Chlorinated Aromatic Compounds										
1,2,3-Trichlorobenzene										
1,2,4-Trichlorobenzene										
1,2-Dichlorobenzene										
1,3-Dichlorobenzene										
1,4-Dichlorobenzene										
2-Chlorotoluene										
4-Chlorotoluene										
Chlorobenzene										
Chlorinated Hydrocarbons										
1,2-Dichlorobenzene										
1,3-Dichlorobenzene										
1,4-Dichlorobenzene										
Hexachlorobenzene										
Hexachlorobutadiene										
Hexachlorocyclopentadiene										
Hexachloroethane										
Hexachloropropylene										
Pentachlorobenzene										
Fumigants										
1,2-Dibromomethane										
1,2-Dichloropropane										
2,2-Dichloropropane										
cis-1,3-Dichloropropylene										
trans-1,3-Dichloropropylene										
2-Butanone (MEK)										
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Bis(2-chloroethyl) ether										

Notes:

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- ## : Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
- ^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX	SS058	SS059	SS060	SS061	SS062	SS063					
		(Industrial Land) mg/kg	TP16 0-0.15	TP16 0.9-1.0	TP17 0.1-0.3	TP17 0.7-0.9	TP18 0.2-0.4	TP18 0.5-0.7	TP01 1.4-1.5 7/04/2005	TP02 0.8-0.9 7/04/2005	TP15 1.6-1.7 8/04/2005	TP19 1.2-1.3 8/04/2005	TP24 1.4-1.5 8/04/2005
Halogenated Aliphatic Compounds													
Vinylchloride	mg/kg	0.0075	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<5	<5	<5	<5	<5
1,1-Dichloroethene	mg/kg	4.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethene	mg/kg	2.3	<0.02	<0.02	<0.02	0.02	<0.02	<0.02	<0.5	<0.5	<0.5	<0.5	<0.5
1,1-Dichloroethane	mg/kg	17							<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,2-Dichloroethane	mg/kg	1.5	<0.02	<0.02	<0.02	0.02	<0.02	<0.02	<0.5	<0.5	0.7	<0.5	<0.5
1,2-Dichloroethane	mg/kg	0.006							<0.5	<0.5	<0.5	<0.5	<0.5
Trichloroethene	mg/kg	0.0011	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	3.8	3.7	<0.5	1	<0.5
1,1,2-Trichloroethane	mg/kg	0.016							<0.5	<0.5	<0.5	<0.5	<0.5
Tetrachloroethene	mg/kg	0.013	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	12.1	22.2	<0.5	<0.5	1.1
1,1,2,2-Tetrachloroethane	mg/kg	0.0093							<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	0.22							<0.5	<0.5	<0.5	<0.5	<0.5
Trihalomethanes													
Chloroform	mg/kg	0.12	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.5	<0.5	<0.5	0.8	0.6
Bromochloromethane													
Bromodichloromethane													
Bromoform													
Dibromochloromethane													
1,1,1,2-Tetrachloroethane													
Other Chloromethanes													
Carbon Tetrachloride	mg/kg	0.005	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01					
Dichloromethane	mg/kg	0.21	<0.2	<0.2	<5	<2	<2	<2					
Chloromethane	mg/kg	1.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02					
Chloroethanes													
1,1,1-Trichloroethane	mg/kg	12	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01					
1,1,2-Trichloroethane	mg/kg	0.016	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01					
1,2-Dichloroethane	mg/kg	0.006	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01					
Chloroethane	mg/kg	0.065	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02					
Miscellaneous													
Ethene	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2					
Cyclopentane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2					
Cyclohexane	mg/kg	1.4	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2					
Carbon Disulphide- Disulphide	mg/kg		<0.4	<0.4	<0.4	<0.4	<0.4	<0.4					
1,1-Dichloropropylene													
1,2,3-Trichloropropane													
1,2-Dibromo-3-chloropropane													
1,3-Dichloropropane													
Bromobenzene													
Bromomethane													
Carbon Tetrachloride													
cis-1,4-Dichloro-2-butene													
Dibromomethane													
Dichlorodifluoromethane													
Pentachloroethane													
Tetrachloroethane													
trans-1,3-dichloropropene													
trans-1,4-Dichloro-2-butene													
Trichlorofluoromethane													
Chlorinated Aromatic Compounds													
1,2,3-Trichlorobenzene													
1,2,4-Trichlorobenzene													
1,2-Dichlorobenzene													
1,3-Dichlorobenzene													
1,4-Dichlorobenzene													
2-Chlorotoluene													
4-Chlorotoluene													
Chlorobenzene													
Chlorinated Hydrocarbons													
1,2-Dichlorobenzene													
1,3-Dichlorobenzene													
1,4-Dichlorobenzene													
Hexachlorobenzene													
Hexachlorobutadiene													
Hexachlorocyclopentadiene													
Hexachloroethane													
Hexachloropropylene													
Pentachlorobenzene													
Fumigants													
1,2-Dibromomethane													
1,2-Dichloropropane													
2,2-Dichloropropane													
cis-1,3-Dichloropropylene													
trans-1,3-Dichloropropylene													
2-Butanone (MEK)													
2-Hexanone (MBK)													
2-Propanone													
4-Methyl-2-pentanone													
Vinyl Acetate													
Iodomethane													
Haloothers													
4-Bromophenyl phenyl ether													
4-Chlorophenyl phenyl ether													
Bis(2-chloroethoxy) methane													
Bis(2-chloroethyl) ether													

Notes:

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- < = analyte not detected above laboratory practical quantitation limit (PQL)
- Bold/ Box = concentrations above criteria
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- # : Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP27 1.2-1.3	TP29 0.3-0.4	TP29 0.8-0.9	TP37 1.2-1.3	TP38 1.3-1.4	TP45 1.3-1.4	HA01	HA02	HA03
			7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	11/04/2005	8/04/2005	8/04/2005	8/04/2005
Halogenated Aliphatic Compounds											
Vinylchloride	mg/kg	0.0075	<5	<5	33	53	<5	<5	<5	<5	<5
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	2	1.2	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethene	mg/kg	2.3	0.9	7.8	106	27.8	<0.5	<0.5	3.3	6.4	<0.5
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	22.9	1.9	<0.5	<0.5	1.6	<0.5	<0.5
cis-1,2-Dichloroethane	mg/kg	1.5	5.7	12.2	165	69.8	0.6	1	36.7	1.6	<0.5
1,2-Dichloroethane	mg/kg	0.006	<0.5	0.6	4.5	4	<0.5	0.6	<0.5	<0.5	<0.5
Trichloroethene	mg/kg	0.0011	<0.5	17.6	3.6	2.8	<0.5	2.6	18.4	1.1	<0.5
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	0.8	<0.5	<0.5	<0.5
Tetrachloroethene	mg/kg	0.013	<0.5	20.5	0.8	<0.5	<0.5	1.6	21.1	<0.5	<0.5
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	5.4	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	1	58	283	4.7
Trihalomethanes											
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	1.9	3.9	<0.5	<0.5
Bromochloromethane											
Bromodichloromethane											
Bromoform											
Dibromochloromethane											
1,1,1,2-Tetrachloroethane											
Other Chloromethanes											
Carbon Tetrachloride	mg/kg	0.005									
Dichloromethane	mg/kg	0.21									
Chloromethane	mg/kg	1.6									
Chloroethanes											
1,1,1-Trichloroethane	mg/kg	12									
1,1,2-Trichloroethane	mg/kg	0.016									
1,2-Dichloroethane	mg/kg	0.006									
Chloroethane	mg/kg	0.065									
Miscellaneous											
Ethene	mg/kg										
Cyclopentane	mg/kg										
Cyclohexane	mg/kg	1.4									
Carbon Disulphide- Disulphide	mg/kg										
1,1-Dichloropropylene											
1,2,3-Trichloropropane											
1,2-Dibromo-3-chloropropane											
1,3-Dichloropropane											
Bromobenzene											
Bromomethane											
Carbon Tetrachloride											
cis-1,4-Dichloro-2-butene											
Dibromomethane											
Dichlorodifluoromethane											
Pentachloroethane											
Tetrachloroethane											
trans-1,3-dichloropropene											
trans-1,4-Dichloro-2-butene											
Trichlorofluoromethane											
Chlorinated Aromatic Compounds											
1,2,3-Trichlorobenzene											
1,2,4-Trichlorobenzene											
1,2-Dichlorobenzene											
1,3-Dichlorobenzene											
1,4-Dichlorobenzene											
2-Chlorotoluene											
4-Chlorotoluene											
Chlorobenzene											
Chlorinated Hydrocarbons											
1,2-Dichlorobenzene											
1,3-Dichlorobenzene											
1,4-Dichlorobenzene											
Hexachlorobenzene											
Hexachlorobutadiene											
Hexachlorocyclopentadiene											
Hexachloroethane											
Hexachloropropylene											
Pentachlorobenzene											
Fumigants											
1,2-Dibromomethane											
1,2-Dichloropropane											
2,2-Dichloropropane											
cis-1,3-Dichloropropylene											
trans-1,3-Dichloropropylene											
2-Butanone (MEK)											
2-Hexanone (MBK)											
2-Propanone											
4-Methyl-2-pentanone											
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4-Bromophenyl phenyl ether											
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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP105_1.0	TP105_2.1	TP106_1.0	TP106_1.7	TP107_1.0	TP107_2.1	QC109	TP108_1.0	TP108_2.1	TP109_1.2	
			0	0	0	0	0	0	0	0	0	0	0
			20/10/2006	20/10/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	1/11/2006	1/11/2006	1/11/2006
Halogenated Aliphatic Compounds													
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,2-Dichloroethene	mg/kg	2.3	2.1	<0.5	0.8	<0.5	1.1	19.7	0.8	<0.5	<0.5	<0.5	
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	<0.5	<0.5	0.9	<0.5	<0.5	<0.5	<0.5	
cis-1,2-Dichloroethene	mg/kg	1.5	41.6	<0.5	2.4	<0.5	4.8	82.2	6.2	<0.5	<0.5	<0.5	
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trihalomethanes													
Chloroform	mg/kg	0.12	1.3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromoform			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Other Chloromethanes													
Carbon Tetrachloride	mg/kg	0.005											
Dichloromethane	mg/kg	0.21											
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Chloroethanes													
1,1,1-Trichloroethane	mg/kg	12											
1,1,2-Trichloroethane	mg/kg	0.016	1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloroethane	mg/kg	0.006											
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Miscellaneous													
Ethane	mg/kg												
Cyclopentane	mg/kg												
Cyclohexane	mg/kg	1.4											
Carbon Disulphide- Disulphide	mg/kg		-	-	<0.5	-	-	-	-	-	-	-	
1,1-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromomethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dichlorodifluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Tetrachloroethane			-	<0.5	-	<0.5	<0.5	0.6	<0.5	<0.5	<0.5	<0.5	
trans-1,3-Dichloropropene			-	-	-	-	-	-	-	-	-	-	
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trichlorofluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Chlorinated Aromatic Compounds													
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Chlorinated Hydrocarbons													
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobenzene			<0.5	<0.5	<0.5	<0.5	<1	<1	<0.5	<0.5	<0.5	<0.5	
Hexachlorobutadiene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorocyclopentadiene			<0.5	<0.5	<0.5	<0.5	<2.5	<2.5	<0.5	<0.5	<0.5	<0.5	
Hexachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Pentachlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Fumigants													
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2-Butanone (MEK)			-	-	<5	-	<5	<5	<5	-	-	-	
2-Hexanone (MBK)			-	-	<5	-	<5	<5	<5	-	-	-	
2-Propanone			-	-	<5	-	<5	<5	<5	-	-	-	
4-Methyl-2-pentanone			-	-	<5	-	<5	<5	<5	-	-	-	
Vinyl Acetate			-	-	<5	-	<5	<5	<5	-	-	-	
Iodomethane													
Haloethers													
4-Bromophenyl phenyl ether			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
4-Chlorophenyl phenyl ether			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bis(2-chloroethoxy) methane			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bis(2-chloroethyl) ether			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	

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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP109_2.0	TP110_0.9	TP110_1.9	TP111_0.9	TP111_2.1	TP112_1.4	TP112_2.2	TP113_0.8	TP113_1.7	QC106	
			0	0	0	0	0	0	0	0	0	0	0
			1/11/2006	2/11/2006	2/11/2006	20/10/2006	20/10/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006	1/11/2006
Halogenated Aliphatic Compounds													
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	2.4	1.3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	0.7	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,2-Dichloroethane	mg/kg	1.5	<0.5	1.3	<0.5	13.8	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	0.8	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trihalomethanes													
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromochloromethane	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromodichloromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromoform	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dibromochloromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,1,2-Tetrachloroethane	-	-	-	-	-	-	-	-	-	-	-	-	
Other Chloromethanes													
Carbon Tetrachloride	mg/kg	0.005	-	-	-	-	-	-	-	-	-	-	
Dichloromethane	mg/kg	0.21	-	-	-	-	-	-	-	-	-	-	
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Chloroethanes													
1,1,1-Trichloroethane	mg/kg	12	-	-	-	-	-	-	-	-	-	-	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloroethane	mg/kg	0.006	-	-	-	-	-	-	-	-	-	-	
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Miscellaneous													
Ethene	mg/kg	-	-	-	-	-	-	-	-	-	-	-	
Cyclopentane	mg/kg	-	-	-	-	-	-	-	-	-	-	-	
Cyclohexane	mg/kg	1.4	-	-	-	-	-	-	-	-	-	-	
Carbon Disulphide- Disulphide	mg/kg	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2,3-Trichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dibromo-3-chloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromomethane	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Carbon Tetrachloride	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,4-Dichloro-2-butene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dibromomethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dichlorodifluoromethane	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Pentachloroethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Tetrachloroethane	<0.5	0.6	<0.5	0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,3-dichloropropene	-	-	-	-	-	-	-	-	-	-	-	-	
trans-1,4-Dichloro-2-butene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trichlorofluoromethane	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Chlorinated Aromatic Compounds													
1,2,3-Trichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2,4-Trichlorobenzene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2-Chlorotoluene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
4-Chlorotoluene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Chlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Chlorinated Hydrocarbons													
1,2-Dichlorobenzene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichlorobenzene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,4-Dichlorobenzene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobenzene	<0.5	<1	<1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobutadiene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorocyclopentadiene	<0.5	<2.5	<2.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachloroethane	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachloropropylene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Pentachlorobenzene	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Fumigants													
1,2-Dibromomethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2,2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2-Butanone (MEK)	-	<5	<5	-	-	-	-	-	-	-	-	-	
2-Hexanone (MBK)	-	<5	<5	-	-	-	-	-	-	-	-	-	
2-Propanone	-	-	-	-	-	-	-	-	-	-	-	-	
4-Methyl-2-pentanone	-	<5	<5	-	-	-	-	-	-	-	-	-	
Vinyl Acetate	-	<5	<5	-	-	-	-	-	-	-	-	-	
Iodomethane	-	-	-	-	-	-	-	-	-	-	-	-	
Haloothers													
4-Bromophenyl phenyl ether	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
4-Chlorophenyl phenyl ether	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bis(2-chloroethoxy) methane	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bis(2-chloroethyl) ether	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (POL)
Bold/ Box = concentrations above criteria
- = not analysed
* : Results determined using Methanol Extraction Method and are corrected for blank.
: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
: Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP114_1.0	TP114_2.0	TP115_1.0	TP116_0.8	TP116_1.9	TP116_2.4	TP117_1.0	TP117_2.0	TP118_0.9	TP118_1.0	
			0	0	0	0	0	0	0	0	0	0	0
			20/10/2006	20/10/2006	1/11/2006	2/11/2006	2/11/2006	2/11/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	
Halogenated Aliphatic Compounds													
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	9.5	<0.5	<0.5	<0.5	<0.5	0.7	<0.5	
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	0.6	<0.5	<0.5	<0.5	<0.5	<0.5	1.8	
cis-1,2-Dichloroethane	mg/kg	1.5	<0.5	<0.5	<0.5	8.8	<0.5	<0.5	<0.5	<0.5	5.3	<0.5	
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trichloroethene	mg/kg	0.0011	0.9	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	0.8	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trihalomethanes													
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromochloromethane													
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromoform			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,1,1,2-Tetrachloroethane													
Other Chloromethanes													
Carbon Tetrachloride	mg/kg	0.005											
Dichloromethane	mg/kg	0.21											
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Chloroethanes													
1,1,1-Trichloroethane	mg/kg	12											
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloroethane	mg/kg	0.006											
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Miscellaneous													
Ethene	mg/kg												
Cyclopentane	mg/kg												
Cyclohexane	mg/kg	1.4											
Carbon Disulphide- Disulphide	mg/kg		-	-	-	<0.5	<0.5	<0.5	<0.5	-	-	-	
1,1-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bromomethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Dichlorodifluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,3-dichloropropene													
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Trichlorofluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	
Chlorinated Aromatic Compounds													
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Chlorinated Hydrocarbons													
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorobenzene			<0.5	<0.5	<0.5	<1	<1	<1	<0.5	<0.5	<1	<1	
Hexachlorobutadiene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachlorocyclopentadiene			<0.5	<0.5	<0.5	<2.5	<2.5	<2.5	<0.5	<0.5	<2.5	<2.5	
Hexachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Hexachloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Pentachlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Fumigants													
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
2-Butanone (MEK)			-	-	-	<5	<5	<5	<5	-	<5	<5	
2-Hexanone (MBK)			-	-	-	<5	<5	<5	<5	-	<5	<5	
2-Propanone			-	-	-	-	-	-	<5	-	-	-	
4-Methyl-2-pentanone			-	-	-	<5	<5	<5	-	<5	<5	<5	
Vinyl Acetate			-	-	-	<5	<5	<5	<5	-	<5	<5	
Iodomethane													
Haloethers													
4-Bromophenyl phenyl ether			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
4-Chlorophenyl phenyl ether			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bis(2-chloroethoxy) methane			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	
Bis(2-chloroethyl) ether			-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	

Notes:

-- = criteria not available

(mg/kg) = milligrams per kilogram

< = analyte not detected above laboratory practical quantitation limit (POL)

Bold/ Box = concentrations above criteria

- = not analysed

*: Results determined using Methanol Extraction Method and are corrected for blank.

#: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b

even though originally outside calibration.

##: Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.

^: 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP118_1.4	TP118_1.8	TP119_1.0	TP119_1.7	TP120_0.9	TP120_1.4	TP120_1.8	TP121_0.9	TP122_0.8	TP122_1.9
			0 3/11/2006	0 3/11/2006	0 3/11/2006	0 3/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 1/11/2006	0 2/11/2006	0 2/11/2006
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	<0.5	<0.5	0.7	<0.5	<0.5	<0.5	<0.5
1,1-Dichloroethane	mg/kg	17	0.9	<0.5	<0.5	<0.5	<0.5	0.5	<0.5	<0.5	<0.5	<0.5
cis-1,2-Dichloroethane	mg/kg	1.5	0.9	<0.5	<0.5	<0.5	<0.5	4.4	<0.5	<0.5	<0.5	<0.5
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Trihalomethanes												
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromoform			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005										
Dichloromethane	mg/kg	0.21										
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12										
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichloroethane	mg/kg	0.006										
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Miscellaneous												
Ethane	mg/kg											
Cyclopentane	mg/kg											
Cyclohexane	mg/kg	1.4										
Carbon Disulphide- Disulphide	mg/kg		-	-	-	-	<0.5	<0.5	<0.5	-	<0.5	<0.5
1,1-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromomethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Dichlorodifluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropene			-	-	-	-	-	-	-	-	-	-
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Trichlorofluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene			<1	<1	<1	<1	<1	<1	<1	<0.5	<1	<1
Hexachlorobutadiene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Hexachlorocyclopentadiene			<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<0.5	<2.5	<2.5
Hexachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Hexachloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Pentachlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Fumigants												
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Butanone (MEK)			<5	<5	<5	<5	<5	<5	<5	-	<5	<5
2-Hexanone (MBK)			<5	<5	<5	<5	<5	<5	<5	-	<5	<5
2-Propanone			-	-	-	-	-	-	-	-	-	-
4-Methyl-2-pentanone			<5	<5	<5	<5	<5	<5	<5	-	<5	<5
Vinyl Acetate			<5	<5	<5	<5	<5	<5	<5	-	<5	<5
Iodomethane												
Haloothers												
4-Bromophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
4-Chlorophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Bis(2-chloroethoxy) methane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5
Bis(2-chloroethyl) ether			<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
Bold/ Box = concentrations above criteria
- = not analysed

* : Results determined using Methanol Extraction Method and are corrected for blank.
: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
: Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP123_0.9	TP123_1.5	TP123_2.1	TP124_1.0	TP124_1.9	TP125_1.0	TP125_1.6	TP126_0.7	QC107	QC202
			0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 3/11/2006	0 3/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	-
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	1.5	<0.5	v
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.7	<0.5	-
cis-1,2-Dichloroethane	mg/kg	1.5	<0.5	<0.5	<0.5	1.2	<0.5	0.5	<0.5	4.4	<0.5	-
1,2-Dichloroethane	mg/kg	0.006	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Trihalomethanes												
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bromoform			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005										
Dichloromethane	mg/kg	0.21										
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	-
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12										
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,2-Dichloroethane	mg/kg	0.006										
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	-
Miscellaneous												
Ethene	mg/kg											
Cyclopentane	mg/kg											
Cyclohexane	mg/kg	1.4										
Carbon Disulphide- Disulphide	mg/kg		<0.5	<0.5	<0.5	<0.5	<0.5	-	-	<0.5	<0.5	<0.5
1,1-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bromomethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	-
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Dichlorodifluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	-
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
trans-1,3-Dichloropropane			-	-	-	-	-	-	-	-	-	-
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Trichlorofluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	-
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene			<1	<1	<1	<1	<1	<1	<1	<1	<1	<0.5
Hexachlorobutadiene			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorocyclopentadiene			<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2
Hexachloroethane			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachloropropylene			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Pentachlorobenzene			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Fumigants												
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
2-Butanone (MEK)			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
2-Hexanone (MBK)			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
2-Propanone			-	-	-	-	-	-	-	-	-	-
4-Methyl-2-pentanone			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Vinyl Acetate			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Iodomethane												
Haloethers												
4-Bromophenyl phenyl ether			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
4-Chlorophenyl phenyl ether			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bis(2-chloroethoxy) methane			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-
Bis(2-chloroethyl) ether			<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-

Notes:

- = criteria not available
- (mg/kg) = milligrams per kilogram
- < = analyte not detected above laboratory practical quantitation limit (POL)
- Bold/ Box = concentrations above criteria
- = not analysed

- *: Results determined using Methanol Extraction Method and are corrected for blank.
- #: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
- ##: Methanol extraction gave 0 - 30% recovery and therefore 'Neat' result reported.
- ^: 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP126_1.3	TP126_1.9	TP127_1.1	TP127_2.2	TP127_2.3	TP128_0.8	QC108	TP128_1.7	HA001	HA002
			0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 2/11/2006	0 8/11/2006
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	11.8	4.5
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.5	0.5
cis-1,2-Dichloroethane	mg/kg	1.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	14.2	8.4
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.5
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Trihalomethanes												
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromoform			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005										
Dichloromethane	mg/kg	0.21										
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12										
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichloroethane	mg/kg	0.006										
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Miscellaneous												
Ethene	mg/kg											
Cyclopentane	mg/kg											
Cyclohexane	mg/kg	1.4										
Carbon Disulfide- Disulphide	mg/kg		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,1-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromomethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Dichlorodifluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropane												
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Trichlorofluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene			<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Hexachlorobutadiene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorocyclopentadiene			<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5	<2.5
Hexachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Pentachlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Fumigants												
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Butanone (MEK)			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
2-Hexanone (MBK)			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
2-Propanone			-	-	-	-	-	-	-	-	-	-
4-Methyl-2-pentanone			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Vinyl Acetate			<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Iodomethane												
Haloothers												
4-Bromophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
4-Chlorophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bis(2-chloroethoxy) methane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bis(2-chloroethyl) ether			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5

Notes:

-- = criteria not available

(mg/kg) = milligrams per kilogram

< = analyte not detected above laboratory practical quantitation limit (POL)

Bold/ Box = concentrations above criteria

- = not analysed

* : Results determined using Methanol Extraction Method and are corrected for blank.

: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b

even though originally outside calibration.

: Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.

^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	HA003	HA004	HA005	HA006	TP087_1.0	TP087_1.9	TP087_2.7	TP088_2.1	TP088_3.0	TP089_1.0	
			8/11/2006	8/11/2006	8/11/2006	8/11/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	18/10/2006
			0	0	0	0	0	0	0	0	0	0	
Halogenated Aliphatic Compounds													
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	7.1	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	0.6	<0.5	<0.5	<0.5	<0.5	<0.5	-	
cis-1,2-Dichloroethane	mg/kg	1.5	<0.5	<0.5	1.4	22.4	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Trihalomethanes													
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Bromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Bromoform			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Other Chloromethanes													
Carbon Tetrachloride	mg/kg	0.005											
Dichloromethane	mg/kg	0.21											
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
Chloroethanes													
1,1,1-Trichloroethane	mg/kg	12					<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	0.8							
1,2-Dichloroethane	mg/kg	0.006					<0.5	<0.5	<0.5	<0.5	<0.5	-	
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
Miscellaneous													
Ethene	mg/kg												
Cyclopentane	mg/kg												
Cyclohexane	mg/kg	1.4											
Carbon Disulphide- Disulphide	mg/kg		<0.5	<0.5	<0.5	<0.5							
1,1-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
trans-1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Bromomethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Dichlorodifluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
trans-1,3-Dichloropropene			<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Trichlorofluoromethane			<5	<5	<5	<5	<5	<5	<5	<5	<5	-	
Chlorinated Aromatic Compounds													
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Chlorinated Hydrocarbons													
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Hexachlorobenzene			<1	<1	<1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Hexachlorobutadiene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Hexachlorocyclopentadiene			<2.5	<2.5	<2.5	<2.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Hexachloroethane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Hexachloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Pentachlorobenzene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
Fumigants													
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5							
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
2-Butanone (MEK)			<5	<5	<5	<5	<0.5	<0.5	<0.5	<0.5	<0.5	-	
2-Hexanone (MBK)			<5	<5	<5	<5							
2-Propanone			-	-	-	-							
4-Methyl-2-pentanone			<5	<5	<5	<5							
Vinyl Acetate			<5	<5	<5	<5							
Iodomethane							<0.5	<0.5	<0.5	<0.5	<0.5	-	
Haloethers													
4-Bromophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5							
4-Chlorophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5							
Bis(2-chloroethoxy) methane			<0.5	<0.5	<0.5	<0.5							
Bis(2-chloroethyl) ether			<0.5	<0.5	<0.5	<0.5							

Notes:

- = criteria not available
- (mg/kg) = milligrams per kilogram
- < = analyte not detected above laboratory practical quantitation limit (POL)
- Bold/ Box = concentrations above criteria
- = not analysed

- *: Results determined using Methanol Extraction Method and are corrected for blank.
- #: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
- #: Methanol extraction gave 0 - 30% recovery and therefore 'Neat' result reported.
- ^: 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP089_2.7	TP090_1.8	TP090_2.8	TP091_1.0	TP091_1.5	TP091_2.4	TP092_1.0	TP092_2.1	QC102	TP093_0.9	
			0	0	0	0	0	0	0	0	0		0
			18/10/2006	18/10/2006	18/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006
Halogenated Aliphatic Compounds													
Vinylchloride	mg/kg	0.0075	<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
1,1-Dichloroethene	mg/kg	4.1	<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
cis-1,2-Dichloroethene	mg/kg	1.5	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Trihalomethanes													
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bromochloromethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bromodichloromethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bromoform			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Dibromochloromethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Other Chloromethanes													
Carbon Tetrachloride	mg/kg	0.005	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Dichloromethane	mg/kg	0.21	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Chloromethane	mg/kg	1.6	<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
Chloroethanes													
1,1,1-Trichloroethane	mg/kg	12	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,2-Dichloroethane	mg/kg	0.006	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Chloroethane	mg/kg	0.065	<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
Miscellaneous													
Ethene	mg/kg		<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Cyclopentane	mg/kg		<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Cyclohexane	mg/kg	1.4	<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Carbon Disulphide- Disulphide	mg/kg		<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,1-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,3-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bromobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bromomethane			<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
Carbon Tetrachloride			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
cis-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Dibromomethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Dichlorodifluoromethane			<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
Pentachloroethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Tetrachloroethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
trans-1,3-Dichloropropene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
trans-1,4-Dichloro-2-butene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Trichlorofluoromethane			<5	<5	<5	<5	<12	<5	<5	<5	-	<5	
Chlorinated Aromatic Compounds													
1,2,3-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,2,4-Trichlorobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	<0.5	<0.5	
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
2-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
4-Chlorotoluene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Chlorobenzene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Chlorinated Hydrocarbons													
1,2-Dichlorobenzene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
1,3-Dichlorobenzene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
1,4-Dichlorobenzene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Hexachlorobenzene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Hexachlorobutadiene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Hexachlorocyclopentadiene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Hexachloroethane			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Hexachloropropylene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Pentachlorobenzene			-	<0.5	<0.5	-	<1.2	-	<0.5	<0.5	<0.5	-	
Fumigants													
1,2-Dibromomethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
1,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
2,2-Dichloropropane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
trans-1,3-Dichloropropylene			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
2-Butanone (MEK)			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
2-Hexanone (MEK)			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
2-Propanone			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
4-Methyl-2-pentanone			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Vinyl Acetate			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Iodomethane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Haloethers													
4-Bromophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
4-Chlorophenyl phenyl ether			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bis(2-chloroethoxy) methane			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	
Bis(2-chloroethyl) ether			<0.5	<0.5	<0.5	<0.5	<1.2	<0.5	<0.5	<0.5	-	<0.5	

Notes:
 -- = criteria not available
 (mg/kg) = milligrams per kilogram
 < = analyte not detected above laboratory practical quantitation limit (POL)
 Bold/ Box = concentrations above criteria
 - = not analysed

*: Results determined using Methanol Extraction Method and are corrected for blank.
 #: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
 #: Methanol extraction gave 0 - 30% recovery and therefore 'Neat' result reported.
 ^: 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP093_1.7	TP094_0.9	TP094_2.0	TP095_1.0	TP095_1.9	TP096_0.9	TP097_0.9	TP097_2.9	TP098_1.0	TP098_1.9
			0 19/10/2006	0 20/10/2006	0 20/10/2006	0 19/10/2006	0 19/10/2006	0 19/10/2006	0 20/10/2006	0 20/10/2006	0 20/10/2006	0 20/10/2006
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075	<5									
1,1-Dichloroethene	mg/kg	4.1										
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5									
1,1-Dichloroethane	mg/kg	17	<0.5									
cis-1,2-Dichloroethane	mg/kg	1.5										
1,2-Dichloroethane	mg/kg	0.006	<0.5									
Trichloroethane	mg/kg	0.0011	<0.5									
1,1,2-Trichloroethane	mg/kg	0.016	<0.5									
Tetrachloroethane	mg/kg	0.013										
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5									
Hexachlorobutadiene	mg/kg	0.22	<0.5									
Trihalomethanes												
Chloroform	mg/kg	0.12										
Bromochloromethane			<0.5									
Bromodichloromethane			<0.5									
Bromoform			<0.5									
Dibromochloromethane			<0.5									
1,1,1,2-Tetrachloroethane												
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005										
Dichloromethane	mg/kg	0.21										
Chloromethane	mg/kg	1.6	<5									
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12	<0.5									
1,1,2-Trichloroethane	mg/kg	0.016										
1,2-Dichloroethane	mg/kg	0.006	<0.5									
Chloroethane	mg/kg	0.065	<5									
Miscellaneous												
Ethene	mg/kg											
Cyclopentane	mg/kg											
Cyclohexane	mg/kg	1.4										
Carbon Disulphide- Disulphide	mg/kg											
1,1-Dichloropropylene			<0.5									
1,2,3-Trichloropropane			<0.5									
1,2-Dibromo-3-chloropropane			<0.5									
1,3-Dichloropropane			<0.5									
Bromobenzene			<0.5									
Bromomethane			<5									
Carbon Tetrachloride			<0.5									
cis-1,4-Dichloro-2-butene			<0.5									
Dibromomethane			<0.5									
Dichlorodifluoromethane			<5									
Pentachloroethane			<0.5									
Tetrachloroethane			<0.5									
trans-1,3-dichloropropene			<5									
trans-1,4-Dichloro-2-butene			<0.5									
Trichlorofluoromethane			<5									
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene			<0.5									
1,2,4-Trichlorobenzene			<0.5	<0.5						<0.5	<0.5	
1,2-Dichlorobenzene			<0.5									
1,3-Dichlorobenzene			<0.5									
1,4-Dichlorobenzene			<0.5									
2-Chlorotoluene			<0.5									
4-Chlorotoluene			<0.5									
Chlorobenzene			<0.5									
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene			<0.5	<0.5						<0.5	<0.5	
1,3-Dichlorobenzene			<0.5	<0.5						<0.5	<0.5	
1,4-Dichlorobenzene			<0.5	<0.5						<0.5	<0.5	
Hexachlorobenzene			<0.5	<0.5						<0.5	<0.5	
Hexachlorobutadiene			<0.5	<0.5						<0.5	<0.5	
Hexachlorocyclopentadiene			<0.5	<0.5						<0.5	<0.5	
Hexachloroethane			<0.5	<0.5						<0.5	<0.5	
Hexachloropropylene			<0.5									
Pentachlorobenzene			<0.5									
Fumigants												
1,2-Dibromomethane												
1,2-Dichloropropane			<0.5									
2,2-Dichloropropane			<0.5									
cis-1,3-Dichloropropylene			<0.5									
trans-1,3-Dichloropropylene			<0.5									
2-Butanone (MEK)			<0.5									
2-Hexanone (MBK)												
2-Propanone												
4-Methyl-2-pentanone												
Vinyl Acetate												
Iodomethane			<0.5									
Haloethers												
4-Bromophenyl phenyl ether												
4-Chlorophenyl phenyl ether												
Bis(2-chloroethoxy) methane												
Bis(2-chloroethyl) ether												

Notes:

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- (mg/kg) = milligrams per kilogram
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- Bold/ Box = concentrations above criteria
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- # : Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
- ## : Methanol extraction gave 0 - 30% recovery and therefore 'Neat' result reported.
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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	TP098_2.4	TP099_1.0	TP099_1.9	TP100_1.8	TP100_2.5	QC104	TP101_1.0	TP101_1.8	TP102_1.0	TP102_2.0
			0 20/10/2006	0 20/10/2006	0 20/10/2006	0 19/10/2006	0 19/10/2006	19/10/2006	0 19/10/2006	0 19/10/2006	0 19/10/2006	0 19/10/2006
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075										
1,1-Dichloroethene	mg/kg	4.1										
trans-1,2-Dichloroethene	mg/kg	2.3										
1,1-Dichloroethane	mg/kg	17										
cis-1,2-Dichloroethane	mg/kg	1.5										
1,2-Dichloroethane	mg/kg	0.006										
Trichloroethane	mg/kg	0.011										
1,1,2-Trichloroethane	mg/kg	0.016										
Tetrachloroethane	mg/kg	0.013										
1,1,2,2-Tetrachloroethane	mg/kg	0.0093										
Hexachlorobutadiene	mg/kg	0.22										
Trihalomethanes												
Chloroform	mg/kg	0.12										
Bromochloromethane												
Bromodichloromethane												
Bromoform												
Dibromochloromethane												
1,1,1,2-Tetrachloroethane												
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005										
Dichloromethane	mg/kg	0.21										
Chloromethane	mg/kg	1.6										
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12										
1,1,2-Trichloroethane	mg/kg	0.016										
1,2-Dichloroethane	mg/kg	0.006										
Chloroethane	mg/kg	0.065										
Miscellaneous												
Ethene	mg/kg											
Cyclopentane	mg/kg											
Cyclohexane	mg/kg	1.4										
Carbon Disulphide- Disulphide	mg/kg								0			
1,1-Dichloropropylene												
1,2,3-Trichloropropane												
1,2-Dibromo-3-chloropropane												
1,3-Dichloropropane												
Bromobenzene												
Bromomethane												
Carbon Tetrachloride												
cis-1,4-Dichloro-2-butene												
Dibromomethane												
Dichlorodifluoromethane												
Pentachloroethane												
Tetrachloroethane												
trans-1,3-dichloropropene												
trans-1,4-Dichloro-2-butene												
Trichlorofluoromethane												
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene			-	-	-	-	-	-	-	-	-	-
1,2,4-Trichlorobenzene			-	<0.5	-	-	-	-	-	-	-	-
1,2-Dichlorobenzene			-	-	-	-	-	-	-	-	-	-
1,3-Dichlorobenzene			-	-	-	-	-	-	-	-	-	-
1,4-Dichlorobenzene			-	-	-	-	-	-	-	-	-	-
2-Chlorotoluene			-	-	-	-	-	-	-	-	-	-
4-Chlorotoluene			-	-	-	-	-	-	-	-	-	-
Chlorobenzene			-	-	-	-	-	-	-	-	-	-
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene			-	<0.5	-	-	-	-	-	-	-	-
1,3-Dichlorobenzene			-	<0.5	-	-	-	-	-	-	-	-
1,4-Dichlorobenzene			-	<0.5	-	-	-	-	-	-	-	-
Hexachlorobenzene			-	<0.5	-	-	-	-	-	-	-	-
Hexachlorobutadiene			-	<0.5	-	-	-	-	-	-	-	-
Hexachlorocyclopentadiene			-	<0.5	-	-	-	-	-	-	-	-
Hexachloroethane			-	<0.5	-	-	-	-	-	-	-	-
Hexachloropropylene			-	<0.5	-	-	-	-	-	-	-	-
Pentachlorobenzene			-	<0.5	-	-	-	-	-	-	-	-
Fumigants												
1,2-Dibromomethane												
1,2-Dichloropropane												
2,2-Dichloropropane												
cis-1,3-Dichloropropylene												
trans-1,3-Dichloropropylene												
2-Butanone (MEK)												
2-Hexanone (MBK)												
2-Propanone												
4-Methyl-2-pentanone												
Vinyl Acetate												
Iodomethane												
Haloethers												
4-Bromophenyl phenyl ether												
4-Chlorophenyl phenyl ether												
Bis(2-chloroethoxy) methane												
Bis(2-chloroethyl) ether												

Notes:

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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX	QC103	QC201	TP103_0.8	TP103_1.8	TP103_2.9	QC105	TP104_0.7	TP104_1.9	TP129_1.5	TP129_3.0
		(Industrial Land) mg/kg	19/10/2006	19/10/2006	20/10/2006	20/10/2006	20/10/2006	20/10/2006	20/10/2006	20/10/2006	18/10/2006	18/10/2006
Halogenated Aliphatic Compounds												
Vinylchloride	mg/kg	0.0075			-	-	<5	<5	<5	-	-	<5
1,1-Dichloroethene	mg/kg	4.1			-	-	<0.5	<0.5	<0.5	-	-	<0.5
trans-1,2-Dichloroethene	mg/kg	2.3			-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,1-Dichloroethane	mg/kg	17			-	-	<0.5	<0.5	<0.5	-	-	<0.5
cis-1,2-Dichloroethane	mg/kg	1.5			-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2-Dichloroethane	mg/kg	0.006			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Trichloroethene	mg/kg	0.0011			-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,1,2-Trichloroethane	mg/kg	0.016			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Tetrachloroethene	mg/kg	0.013			-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,1,2,2-Tetrachloroethane	mg/kg	0.0093			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Hexachlorobutadiene	mg/kg	0.22			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Trihalomethanes												
Chloroform	mg/kg	0.12			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Bromochloromethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Bromodichloromethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Bromoform					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Dibromochloromethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,1,1,2-Tetrachloroethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Other Chloromethanes												
Carbon Tetrachloride	mg/kg	0.005			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Dichloromethane	mg/kg	0.21			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Chloromethane	mg/kg	1.6			-	-	<5	<5	<5	-	-	<5
Chloroethanes												
1,1,1-Trichloroethane	mg/kg	12			-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,1,2-Trichloroethane	mg/kg	0.016			-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2-Dichloroethane	mg/kg	0.006			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Chloroethane	mg/kg	0.065			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Miscellaneous												
Ethene	mg/kg				-	-	<0.5	<0.5	<0.5	-	-	<0.5
Cyclopentane	mg/kg				-	-	<0.5	<0.5	<0.5	-	-	<0.5
Cyclohexane	mg/kg	1.4			-	-	<0.5	<0.5	<0.5	-	-	<0.5
Carbon Disulphide- Disulphide	mg/kg				-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,1-Dichloropropylene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2,3-Trichloropropane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2-Dibromo-3-chloropropane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,3-Dichloropropane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Bromobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Bromomethane					-	-	<5	<5	<5	-	-	<5
Carbon Tetrachloride					-	-	<0.5	<0.5	<0.5	-	-	<0.5
cis-1,4-Dichloro-2-butene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Dibromomethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Dichlorodifluoromethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Pentachloroethane					-	-	<5	<5	<5	-	-	<5
Tetrachloroethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
trans-1,3-dichloropropane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
trans-1,4-Dichloro-2-butene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Trichlorofluoromethane					-	-	<5	<5	<5	-	-	<5
Chlorinated Aromatic Compounds												
1,2,3-Trichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2,4-Trichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2-Dichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,3-Dichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,4-Dichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
2-Chlorotoluene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
4-Chlorotoluene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Chlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Chlorinated Hydrocarbons												
1,2-Dichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,3-Dichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,4-Dichlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Hexachlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Hexachlorobutadiene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Hexachlorocyclopentadiene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Hexachloroethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Hexachloropropylene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Pentachlorobenzene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Fumigants												
1,2-Dibromomethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
1,2-Dichloropropane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
2,2-Dichloropropane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
cis-1,3-Dichloropropylene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
trans-1,3-Dichloropropylene					-	-	<0.5	<0.5	<0.5	-	-	<0.5
2-Butanone (MEK)					-	-	-	-	-	-	-	-
2-Hexanone (MBK)					-	-	-	-	-	-	-	-
2-Propanone					-	-	-	-	-	-	-	-
4-Methyl-2-pentanone					-	-	-	-	-	-	-	-
Vinyl Acetate					-	-	-	-	-	-	-	-
Iodomethane					-	-	<0.5	<0.5	<0.5	-	-	<0.5
Haloethers												
4-Bromophenyl phenyl ether					-	-	-	-	-	-	-	-
4-Chlorophenyl phenyl ether					-	-	-	-	-	-	-	-
Bis(2-chloroethoxy) methane					-	-	-	-	-	-	-	-
Bis(2-chloroethyl) ether					-	-	-	-	-	-	-	-

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Southlands Soil Data - VOC
Block 1

Analyte grouping/ Analyte	Units	USEPA- Region_IX (Industrial Land) mg/kg	QC100	QC200	TP130_1.0	QC101	HA007	QC001	HA008	HA009
			18/10/2006	18/10/2006	18/10/2006	18/10/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006
					0					
Halogenated Aliphatic Compounds										
Vinylchloride	mg/kg	0.0075	<5	<5	<5	-	<5	<5	-	-
1,1-Dichloroethene	mg/kg	4.1	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
trans-1,2-Dichloroethene	mg/kg	2.3	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,1-Dichloroethane	mg/kg	17	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
cis-1,2-Dichloroethane	mg/kg	1.5	<0.5	<0.5	<0.5	-	4	<0.5	-	-
1,2-Dichloroethane	mg/kg	0.008	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Trichloroethene	mg/kg	0.0011	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,1,2-Trichloroethane	mg/kg	0.016	<0.5	<0.5	<0.5	-	1	<0.5	-	-
Tetrachloroethene	mg/kg	0.013	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,1,2,2-Tetrachloroethane	mg/kg	0.0093	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Hexachlorobutadiene	mg/kg	0.22	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Trihalomethanes										
Chloroform	mg/kg	0.12	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Bromochloromethane							<0.5	<0.5	-	-
Bromodichloromethane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Bromoform			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Dibromochloromethane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,1,1,2-Tetrachloroethane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Other Chloromethanes										
Carbon Tetrachloride	mg/kg	0.005	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Dichloromethane	mg/kg	0.21								
Chloromethane	mg/kg	1.6	<5	<5	<5	-	<5	<5	-	-
Chloroethanes										
1,1,1-Trichloroethane	mg/kg	12	<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,1,2-Trichloroethane	mg/kg	0.016								
1,2-Dichloroethane	mg/kg	0.006								
Chloroethane	mg/kg	0.065	<5	<5	<5	-	<5	<5	-	-
Miscellaneous										
Ethene	mg/kg									
Cyclopentane	mg/kg									
Cyclohexane	mg/kg	1.4								
Carbon Disulphide- Disulphide	mg/kg									
1,1-Dichloropropylene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,2,3-Trichloropropane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,2-Dibromo-3-chloropropane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,3-Dichloropropane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Bromobenzene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Bromomethane			<5	<5	<5	-	<5	<5	-	-
Carbon Tetrachloride										
cis-1,4-Dichloro-2-butene			<0.5	-	<0.5	-	<0.5	<0.5	-	-
Dibromomethane										
Dichlorodifluoromethane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Pentachloroethane			<5	<5	<5	-	<5	<5	-	-
Tetrachloroethane			<0.5	-	<0.5	-	<0.5	<0.5	-	-
trans-1,3-Dichloropropene			-	-	-	-	-	-	-	-
trans-1,4-Dichloro-2-butene			<0.5	-	<0.5	-	<0.5	<0.5	-	-
Trichlorofluoromethane			<5	<5	<5	-	<5	<5	-	-
Chlorinated Aromatic Compounds										
1,2,3-Trichlorobenzene			<0.5	-	<0.5	-	<0.5	<0.5	-	-
1,2,4-Trichlorobenzene			<0.5	-	<0.5	<0.5	<0.5	<0.5	-	-
1,2-Dichlorobenzene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,3-Dichlorobenzene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,4-Dichlorobenzene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
2-Chlorotoluene			<0.5	-	<0.5	-	<0.5	<0.5	-	-
4-Chlorotoluene			<0.5	-	<0.5	-	<0.5	<0.5	-	-
Chlorobenzene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
Chlorinated Hydrocarbons										
1,2-Dichlorobenzene			<0.5	<0.5	-	<0.5	<0.5	<0.5	-	-
1,3-Dichlorobenzene			<0.5	<0.5	-	<0.5	<0.5	<0.5	-	-
1,4-Dichlorobenzene			<0.5	<0.5	-	<0.5	<0.5	<0.5	-	-
Hexachlorobenzene			<0.5	-	<0.5	<1	<1	<1	-	-
Hexachlorobutadiene			<0.5	-	<0.5	<0.5	<0.5	<0.5	-	-
Hexachlorocyclopentadiene			<0.5	-	<0.5	<2.5	<2.5	<2.5	-	-
Hexachloroethane			<0.5	-	<0.5	<0.5	<0.5	<0.5	-	-
Hexachloropropylene			<0.5	-	<0.5	<0.5	<0.5	<0.5	-	-
Pentachlorobenzene			<0.5	-	<0.5	<0.5	<0.5	<0.5	-	-
Fumigants										
1,2-Dibromomethane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
1,2-Dichloropropane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
2,2-Dichloropropane			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
cis-1,3-Dichloropropylene			<0.5	<0.5	<0.5	-	<0.5	<0.5	-	-
trans-1,3-Dichloropropylene			<0.5	-	<0.5	-	<0.5	<0.5	-	-
2-Butanone (MEK)			-	-	-	-	<5	<5	-	-
2-Hexanone (MBK)			-	-	-	-	<5	<5	-	-
2-Propanone			-	-	-	-	-	-	-	-
4-Methyl-2-pentanone			-	-	-	-	<5	<5	-	-
Vinyl Acetate			-	-	-	-	<5	<5	-	-
Iodomethane			<0.5	-	<0.5	-	<0.5	<0.5	-	-
Haloethers										
4-Bromophenyl phenyl ether			-	-	-	-	<0.5	<0.5	-	-
4-Chlorophenyl phenyl ether			-	-	-	-	<0.5	<0.5	-	-
Bis(2-chloroethoxy) methane			-	-	-	-	<0.5	<0.5	-	-
Bis(2-chloroethyl) ether			-	-	-	-	<0.5	<0.5	-	-

Notes:

- = criteria not available
- (mg/kg) = milligrams per kilogram
- < = analyte not detected above laboratory practical quantitation limit (POL)
- Bold/ Box = concentrations above criteria
- = not analysed

- * : Results determined using Methanol Extraction Method and are corrected for blank.
- # : Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for b even though originally outside calibration.
- ## : Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
- ^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	SS040	SS041	SS042	SS043	SS044	SS045	SS046	SS047	SS048	SS049	SS050	SS051	SS052	SS053	SS054	SS055	SS056	SS057	SS058	SS059
				TP10	TP10	TP11	TP11	TP12	TP12	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TP13	TP13	TP14	TP14	TP15	TP15	TP16	TP16	
Polynuclear Aromatic Hydrocarbons				0.1-0.3	0.8-1.0	0.1-0.3	0.5-0.7	0-0.2	0.3-0.5	0.3-0.5	1.1-1.3	1.4-1.5	0.3-0.5	0.7-0.9	0-0.35	0.1-0.3	1.0-1.3	0-0.15	0.5-0.55	0.1-0.3	0.7-0.8	0-0.15	0.9-1.0
Naphthalene	mg/kg	--	190																				
2-Methylnaphthalene	mg/kg	--	190																				
Acenaphthylene	mg/kg	--	--																				
Acenaphthene	mg/kg	--	29000																				
Phenanthrene	mg/kg	--	--																				
Anthracene	mg/kg	--	100000																				
Fluoranthene	mg/kg	--	22000																				
Pyrene	mg/kg	--	29000																				
Benz(a)anthracene	mg/kg	--	2.1																				
Chrysene	mg/kg	--	210																				
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1																				
Benzo(a)pyrene	mg/kg	5	--																				
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1																				
Dibenz(a,h)anthracene	mg/kg	--	0.21																				
Benzo(g,h,i)perylene	mg/kg	--	--																				
TotalPAH	mg/kg	100	--																				
Fluorene	mg/kg																						
N-2-Fluorenyl Acetamide	mg/kg																						
2-Chloronaphthalene	mg/kg																						
3-Methylanthrene	mg/kg																						
7,12-Dimethylbenz(a)anthracene	mg/kg																						
Phthalate Esters	mg/kg																						
Di-n-butylphthalate	mg/kg	--	820000																				
Butylbenzylphthalate	mg/kg	--	100,000																				
bis(2-ethylhexyl)phthalate	mg/kg	--	120																				
Diethyl phthalate	mg/kg																						
Dimethyl phthalate	mg/kg																						
Di-n-octylphthalate	mg/kg																						
Nitrosamines	mg/kg																						
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550																				
Methapyrilene	mg/kg																						
N-Nitrosodibutylamine	mg/kg																						
N-Nitrosodiethylamine	mg/kg																						
N-Nitrosodi-n-propylamine	mg/kg																						
N-Nitrosomethylethylamine	mg/kg																						
N-Nitrosomorpholine	mg/kg																						
N-Nitrosopiperidine	mg/kg																						
N-Nitrosopyrrolidine	mg/kg																						
Nitroaromatics and Ketones	mg/kg																						
Nitrobenzene	mg/kg	--	100																				
1,3,5-Trinitrobenzene	mg/kg																						
1-Naphthylamine	mg/kg																						
2,4-Dinitrotoluene	mg/kg																						
2,6-Dinitrotoluene	mg/kg																						
2-Picoline	mg/kg																						
4-Aminobiphenyl	mg/kg																						
4-Nitroquinoline-N-oxide	mg/kg																						
5-Nitro-o-toluidine	mg/kg																						
Acetophenone	mg/kg																						
Azobenzene	mg/kg																						
Chlorobenzilate	mg/kg																						
Dimethylaminoazobenzene	mg/kg																						
Isophorone	mg/kg																						
Pentachloronitrobenzene	mg/kg																						
Phenacetin	mg/kg																						
Pronamide	mg/kg																						
Haloethers	mg/kg																						
Bis(2-chloroethyl)ether	mg/kg	--	0.55																				
Chlorinated Hydrocarbons	mg/kg																						
Hexachloroethane	mg/kg	--	120	0.042	0.004	<0.003	<0.002	<0.003	<0.003	<0.003	0.016	0.028	7.8	0.029	0.15	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.013	<0.004
Hexachlorobutadiene	mg/kg	--	22	0.10	0.005	0.1500	<0.002	0.1300	0.007	<0.003	0.013	0.012	2.9	0.36	0.020	0.043	<0.004	<0.002	<0.005	<0.003	<0.009	0.033	<0.004
Pentachlorobenzene	mg/kg	--	490	0.003	<0.002	<0.003	<0.002	<0.003	<0.003	<0.003	<0.002	<0.003	0.22	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.050	<0.004
Hexachlorobenzene (HCB)	mg/kg	--	1.1	0.043	0.067	<0.003	<0.002	0.0050	0.008	<0.003	0.013	0.023	3.0	0.004	0.024	<0.002	<0.004	0.003	0.022	<0.003	<0.009	0.15	0.028
Anilines and Benzidines	mg/kg																						
Dibenzofuran	mg/kg	--	1600																				
Carbazole	mg/kg	--	86																				
2-Nitroaniline	mg/kg																						
3,3'-Dichlorobenzidine	mg/kg																						
3-Nitroaniline	mg/kg																						

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	SS040	SS041	SS042	SS043	SS044	SS045	SS046	SS047	SS048	SS049	SS050	SS051	SS052	SS053	SS054	SS055	SS056	SS057	SS058	SS059
				TP10	TP10	TP11	TP11	TP12	TP12	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TRENCH A	TP13	TP13	TP14	TP14	TP15	TP15	TP16	TP16	
4-Chloroaniline	mg/kg			0.1-0.3	0.8-1.0	0.1-0.3	0.5-0.7	0-0.2	0.3-0.5	0.3-0.5	1.1-1.3	1.4-1.5	0.3-0.5	0.7-0.9	0-0.35	0.1-0.3	1.0-1.3	0-0.15	0.5-0.55	0.1-0.3	0.7-0.8	0-0.15	0.9-1.0
4-Nitroaniline	mg/kg																						
Aniline	mg/kg																						
Miscellaneous Compounds	mg/kg																						
1,3,5-Trichlorobenzene	mg/kg	--	220	<0.003	<0.002	<0.003	<0.002	<0.003	<0.003	<0.003	<0.002	<0.003	0.039	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	<0.002	<0.004
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180																				
1,3-Dichlorobenzene	mg/kg	--	600	<0.003	<0.002	<0.003	<0.002	<0.003	<0.003	<0.003	<0.002	<0.003	0.007	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.006	<0.004
1,4-Dichlorobenzene	mg/kg	--	7.9	<0.003	<0.002	<0.003	<0.002	<0.003	0.006	<0.003	<0.002	<0.003	0.016	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.017	0.004
1,2-Dichlorobenzene	mg/kg	--	600	<0.003	<0.002	<0.003	<0.002	<0.003	0.003	<0.003	<0.002	<0.003	0.013	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.009	<0.004
1,2,4-Trichlorobenzene	mg/kg	--	220	<0.003	<0.002	<0.003	<0.002	<0.003	<0.003	<0.003	<0.002	<0.003	0.094	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.030	<0.004
Tetrachlorobenzene	mg/kg	--	180	<0.003	<0.002	<0.003	<0.002	<0.003	<0.003	<0.003	<0.002	<0.003	0.50	<0.003	<0.005	<0.002	<0.004	<0.002	<0.005	<0.003	<0.009	0.016	<0.004
Phenols	mg/kg																						
2-Chlorophenol	mg/kg	--											<1	<1	<1					<1	<1		
4-Chlorophenol	mg/kg	--											<1	<1	<1					<1	<1		
2,6-Dichlorophenol	mg/kg	--											<1	<1	<1					<1	<1		
2,4-Dichlorophenol	mg/kg	--											<1	<1	<1					<1	<1		
Pentachlorophenol	mg/kg	--											<1	<1	<1					<1	<1		
2,4,6-Trichlorophenol	mg/kg												<1	<1	<1					<1	<1		
2,4,5-Trichlorophenol	mg/kg																						
2,4-Dimethylphenol	mg/kg																						
2-Methylphenol	mg/kg																						
2-Nitrophenol	mg/kg																						
3- & 4-Methylphenol	mg/kg																						
4-Chloro-3-Methylphenol	mg/kg																						
Phenol	mg/kg																						
2,3,4,6-Tetrachlorophenol	mg/kg																						
4,4-DDD	mg/kg																						
4,4-DDE	mg/kg																						
4,4-DDT	mg/kg																						
a-BHC	mg/kg																						
Aldrin	mg/kg																						
b-BHC	mg/kg																						
d-BHC	mg/kg																						
Dieldrin	mg/kg																						
Endosulfan 1	mg/kg																						
Endosulfan 2	mg/kg																						
Endosulfan sulfate	mg/kg																						
Endrin	mg/kg																						
g-BHC	mg/kg																						
Heptachlor	mg/kg																						
Heptachlor epoxide	mg/kg																						
Organophosphorus Pesticides (OP)	mg/kg																						
Chlorfenvinphos	mg/kg																						
Chlorpyrifos	mg/kg																						
Chlorpyrifos-methyl	mg/kg																						
Diazinon	mg/kg																						
Dichlorvos	mg/kg																						
Dimethoate	mg/kg																						
Ethion	mg/kg																						
Fenthion	mg/kg																						
Malathion	mg/kg																						
Pirimphos-ethyl	mg/kg																						
Prothiofos	mg/kg																						
cis-Isosafrole	mg/kg																						
Diallyl	mg/kg																						
Methanesulfonate ethyl	mg/kg																						
Methanesulfonate methyl	mg/kg																						
Safrole	mg/kg																						
trans-Isosafrole	mg/kg																						

Notes:
 -- = criteria not available
 (mg/kg) = milligrams per kilogram
 < = analyte not detected above laboratory practical quantitation limit (PQL)
 - = not analysed

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	SS060	SS061	SS062	SS063	TP01_0.5-0.6	TP01_1.4-1.5	TP02_0.8-0.9	TP06_1.2-1.3	TP11_0.3-0.4	TP14_0.6-0.7	TP16_1.4-1.5	TP18_1.4-1.5	TP19_1.2-1.3	TP23_1.4-1.5	TP26_0.8-0.9	
				TP17	TP17	TP18	TP18												
Polynuclear Aromatic Hydrocarbons																			
Naphthalene	mg/kg	--	190					<0.5	3.3	<0.5	<0.5	<0.5	<0.5	<0.5	0.7	<0.5	<0.5	<0.5	<0.5
2-Methylnaphthalene	mg/kg	--	190					<0.5	2.8	<0.5	<0.5	<0.5	<0.5	<0.5	3	<0.5	<0.5	<0.5	<0.5
Acenaphthylene	mg/kg	--	--					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	2.1	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthene	mg/kg	--	29000					<0.5	0.9	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Phenanthrene	mg/kg	--	--					<0.5	2.2	1.3	<0.5	0.7	<0.5	9.8	0.9	<0.5	<0.5	<0.5	0.7
Anthracene	mg/kg	--	100000					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	3.2	<0.5	<0.5	<0.5	<0.5	<0.5
Fluoranthene	mg/kg	--	22000					<0.5	1.3	0.8	0.9	1.2	<0.5	21.8	<0.5	2	0.6	2	
Pyrene	mg/kg	--	29000					<0.5	1	0.7	1	1.3	0.6	23.8	<0.5	2.3	0.6	2.2	
Benz(a)anthracene	mg/kg	--	2.1					<0.5	<0.5	<0.5	0.5	0.7	<0.5	12	<0.5	1.1	<0.5	1	
Chrysene	mg/kg	--	210					<0.5	<0.5	<0.5	<0.5	0.6	<0.5	10.4	<0.5	1.1	<0.5	1.2	
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1					<1	<1	<1	<1	<1	<1	10	<1	2	<1	3.2	
Benzo(a)pyrene	mg/kg	5	--					<0.5	<0.5	<0.5	<0.5	0.6	<0.5	6.6	<0.5	0.9	<0.5	3.1	
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	2.8	<0.5	<0.5	<0.5	2.4	
Dibenz(a,h)anthracene	mg/kg	--	0.21					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	1	<0.5	<0.5	<0.5	0.6	
Benzo(g,h,i)perylene	mg/kg	--	--					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	2.9	<0.5	<0.5	<0.5	3.2	
TotalPAH	mg/kg	100	--					nc	11.5	2.8	2.4	5.1	0.6	106.4	4.6	9.4	1.2	19.6	
Fluorene	mg/kg																		
N-2-Fluorenyl Acetamide	mg/kg																		
2-Chloronaphthalene	mg/kg																		
3-Methylcholanthrene	mg/kg																		
7,12-Dimethylbenz(a)anthracene	mg/kg																		
Phthalate Esters																			
Di-n-butylphthalate	mg/kg	--	820000					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	1.1	<0.5	<0.5	<0.5	<0.5
Butylbenzylphthalate	mg/kg	--	100,000					2	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
bis(2-ethylhexyl)phthalate	mg/kg	--	120					<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Diethyl phthalate	mg/kg																		
Dimethyl phthalate	mg/kg																		
Di-n-octylphthalate	mg/kg																		
Nitrosamines																			
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550					<1.0	10.4	268	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Methapyriline	mg/kg																		
N-Nitrosodibutylamine	mg/kg																		
N-Nitrosodiethylamine	mg/kg																		
N-Nitrosodi-n-propylamine	mg/kg																		
N-Nitrosomethylethylamine	mg/kg																		
N-Nitrosomorpholine	mg/kg																		
N-Nitrosopiperidine	mg/kg																		
N-Nitrosopyrrolidine	mg/kg																		
Nitroaromatics and Ketones																			
Nitrobenzene	mg/kg	--	100					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trinitrobenzene	mg/kg																		
1-Naphthylamine	mg/kg																		
2,4-Dinitrotoluene	mg/kg																		
2,6-Dinitrotoluene	mg/kg																		
2-Picoline	mg/kg																		
4-Aminobiphenyl	mg/kg																		
4-Nitroquinoline-N-oxide	mg/kg																		
5-Nitro-o-toluidine	mg/kg																		
Acetophenone	mg/kg																		
Azobenzene	mg/kg																		
Chlorobenzilate	mg/kg																		
Dimethylaminoazobenzene	mg/kg																		
Isophorone	mg/kg																		
Pentachloronitrobenzene	mg/kg																		
Phenacetin	mg/kg																		
Pronamide	mg/kg																		
Haloothers																			
Bis(2-chloroethyl)ether	mg/kg	--	0.55					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chlorinated Hydrocarbons																			
Hexachloroethane	mg/kg	--	120	<0.002	<0.002	<0.002	0.49	<0.5	<0.5	1.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	--	22	<0.002	<0.002	<0.002	0.13	<0.5	1.1	2.4	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Pentachlorobenzene	mg/kg	--	490	<0.002	<0.002	<0.002	0.007	<0.5	<0.5	1.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene (HCB)	mg/kg	--	1.1	<0.002	<0.002	<0.002	0.077	<1.0	3.2	14.1	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anilines and Benzidines																			
Dibenzofuran	mg/kg	--	1600					<0.5	<0.5	1.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Carbazole	mg/kg	--	86					<0.5	<0.5	2.3	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Nitroaniline	mg/kg																		
3,3'-Dichlorobenzidine	mg/kg																		
3-Nitroaniline	mg/kg																		

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	SS060	SS061	SS062	SS063	TP01_0.5-0.6	TP01_1.4-1.5	TP02_0.8-0.9	TP06_1.2-1.3	TP11_0.3-0.4	TP14_0.6-0.7	TP16_1.4-1.5	TP18_1.4-1.5	TP19_1.2-1.3	TP23_1.4-1.5	TP26_0.8-0.9	
				TP17	TP17	TP18	TP18												
4-Chloroaniline	mg/kg																		
4-Nitroaniline	mg/kg																		
Aniline	mg/kg																		
Miscellaneous Compounds	mg/kg																		
1,3,5-Trichlorobenzene	mg/kg	--	220	<0.002	<0.002	<0.002	0.003	<0.5	0.8	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180					<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene	mg/kg	--	600	<0.002	<0.002	<0.002	<0.002												
1,4-Dichlorobenzene	mg/kg	--	7.9	<0.002	<0.002	<0.002	<0.002												
1,2-Dichlorobenzene	mg/kg	--	600	<0.002	<0.002	<0.002	<0.002												
1,2,4-Trichlorobenzene	mg/kg	--	220	<0.002	<0.002	<0.002	<0.002												
Tetrachlorobenzene	mg/kg	--	180	<0.002	<0.002	<0.002	<0.002												
Phenols	mg/kg																		
2-Chlorophenol	mg/kg	--		<1	<1														
4-Chlorophenol	mg/kg	--		<1	<1														
2,6-Dichlorophenol	mg/kg	--		<1	<1														
2,4-Dichlorophenol	mg/kg	--		<1	<1														
Pentachlorophenol	mg/kg	--		<1	<1														
2,4,6-Trichlorophenol	mg/kg			<1	<1														
2,4,5-Trichlorophenol	mg/kg																		
2,4-Dimethylphenol	mg/kg																		
2-Methylphenol	mg/kg																		
2-Nitrophenol	mg/kg																		
3- & 4-Methylphenol	mg/kg																		
4-Chloro-3-Methylphenol	mg/kg																		
Phenol	mg/kg																		
2,3,4,6-Tetrachlorophenol	mg/kg																		
4,4-DDD	mg/kg																		
4,4-DDE	mg/kg																		
4,4-DDT	mg/kg																		
a-BHC	mg/kg																		
Aldrin	mg/kg																		
b-BHC	mg/kg																		
d-BHC	mg/kg																		
Dieldrin	mg/kg																		
Endosulfan 1	mg/kg																		
Endosulfan 2	mg/kg																		
Endosulfan sulfate	mg/kg																		
Endrin	mg/kg																		
g-BHC	mg/kg																		
Heptachlor	mg/kg																		
Heptachlor epoxide	mg/kg																		
Organophosphorus Pesticides (OP)	mg/kg																		
Chlorfenvinphos	mg/kg																		
Chlorpyrifos	mg/kg																		
Chlorpyrifos-methyl	mg/kg																		
Diazinon	mg/kg																		
Dichlorvos	mg/kg																		
Dimethoate	mg/kg																		
Ethion	mg/kg																		
Fenthion	mg/kg																		
Malathion	mg/kg																		
Pirimphos-ethyl	mg/kg																		
Prothiofos	mg/kg																		
cis-Isosafrole	mg/kg																		
Diallate	mg/kg																		
Methanesulfonate ethyl	mg/kg																		
Methanesulfonate methyl	mg/kg																		
Safrole	mg/kg																		
trans-Isosafrole	mg/kg																		

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

**Southlands Soil Data - SVOC
Block 1**

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP29_0.3-0.4	TP29_0.8-0.9	TP32_0.4-0.5	TP38_1.3-1.4	TP39_2.1-2.2	TP45_1.3-1.4	TP81_1.2-1.3	TP82_1.4-1.5	HA01	HA02	HA03	TP105_1.00	TP105_2.10	TP106_1.00	TP106_1.70
				7/04/2005	7/04/2005	7/04/2005	7/04/2005	8/04/2005	11/04/2005	7/04/2005	7/04/2005	8/04/2005	8/04/2005	8/04/2005	20/10/2006	20/10/2006	1/11/2006	1/11/2006
Polynuclear Aromatic Hydrocarbons																		
Naphthalene	mg/kg	--	190	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	2.4	28.3	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
2-Methylnaphthalene	mg/kg	--	190	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	51.9	37.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthylene	mg/kg	--	--	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthene	mg/kg	--	29000	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	1.2	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Phenanthrene	mg/kg	--	--	<0.5	<0.6	0.8	<0.5	<0.5	<0.5	3.3	2.7	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Anthracene	mg/kg	--	100000	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Fluoranthene	mg/kg	--	22000	<0.5	<0.6	0.6	0.8	<0.5	<0.5	0.8	0.8	<0.5	<0.6	<0.5	<0.5	<0.5	0.9	<0.5
Pyrene	mg/kg	--	29000	<0.5	<0.6	0.6	0.8	<0.5	<0.5	0.8	0.5	<0.5	<0.6	<0.5	<0.5	<0.5	0.9	<0.5
Benz(a)anthracene	mg/kg	--	2.1	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Chrysene	mg/kg	--	210	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1	<1	<1	<1	0.6	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Benzo(a)pyrene	mg/kg	5	--	<0.5	<0.6	<0.5	0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	0.6	<0.5
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Dibenz(a,h)anthracene	mg/kg	--	0.21	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	-	-	-	-
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
TotalPAH	mg/kg	100	--	nc	nc	2	2.7	nc	nc	60.4	69.8	nc	nc	nc	ND	ND	2.4	ND
Fluorene	mg/kg														<0.5	<0.5	<0.5	<0.5
N-2-Fluorenyl Acetamide	mg/kg														<0.5	<0.5	<0.5	<0.5
2-Chloronaphthalene	mg/kg														<0.5	<0.5	<0.5	<0.5
3-Methylcholanthrene	mg/kg														<0.5	<0.5	<0.5	<0.5
7,12-Dimethylbenz(a)anthracene	mg/kg														<0.5	<0.5	<0.5	<0.5
Phthalate Esters																		
Di-n-butylphthalate	mg/kg	--	820000	1.8	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Butylbenzylphthalate	mg/kg	--	100,000	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
bis(2-ethylhexyl)phthalate	mg/kg	--	120	<5.0	<5.0	<5.0	<5.0	43.1	<5.0	<5.0	<5.0	<5.0	8.7	<5.0	<5	<5	<5	<5
Diethyl phthalate	mg/kg														<0.5	<0.5	<0.5	<0.5
Dimethyl phthalate	mg/kg														<0.5	<0.5	<0.5	<0.5
Di-n-octylphthalate	mg/kg														<0.5	<0.5	<0.5	<0.5
Nitrosamines																		
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1	<1	<1	<1
Methapyrilene	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosodibutylamine	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosodiethylamine	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosodi-n-propylamine	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosomethylethylamine	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosomorpholine	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosopiperidine	mg/kg														<0.5	<0.5	<0.5	<0.5
N-Nitrosopyrrolidine	mg/kg														<1	<1	<1	<1
Nitroaromatics and Ketones																		
Nitrobenzene	mg/kg	--	100	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trinitrobenzene	mg/kg														<0.5	<0.5	<0.5	<0.5
1-Naphthylamine	mg/kg														<0.5	<0.5	<0.5	<0.5
2,4-Dinitrotoluene	mg/kg														<1	<1	<1	<1
2,6-Dinitrotoluene	mg/kg														<1	<1	<1	<1
2-Picoline	mg/kg														<0.5	<0.5	<0.5	<0.5
4-Aminobiphenyl	mg/kg														<0.5	<0.5	<0.5	<0.5
4-Nitroquinoline-N-oxide	mg/kg														<0.5	<0.5	<0.5	<0.5
5-Nitro-o-toluidine	mg/kg														<0.5	<0.5	<0.5	<0.5
Acetophenone	mg/kg														<0.5	<0.5	<0.5	<0.5
Azobenzene	mg/kg														<1	<1	<1	<1
Chlorobenzilate	mg/kg														<0.5	<0.5	<0.5	<0.5
Dimethylaminoazobenzene	mg/kg														<0.5	<0.5	<0.5	<0.5
Isophorone	mg/kg														<0.5	<0.5	<0.5	<0.5
Pentachloronitrobenzene	mg/kg														<0.5	<0.5	<0.5	<0.5
Phenacetin	mg/kg														<0.5	<0.5	<0.5	<0.5
Pronamide	mg/kg														<0.5	<0.5	<0.5	<0.5
Haloethers																		
Bis(2-chloroethyl)ether	mg/kg	--	0.55	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5				
Chlorinated Hydrocarbons																		
Hexachloroethane	mg/kg	--	120	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5				
Hexachlorobutadiene	mg/kg	--	22	<0.5	<0.6	<0.5	<0.5	<0.5	0.7	<0.6	<0.5	69.2	326	0.7				
Pentachlorobenzene	mg/kg	--	490	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	7.3	<0.5				
Hexachlorobenzene (HCB)	mg/kg	--	1.1	3.4	1.2	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	410	1170	41.3				
Anilines and Benzidines																		
Dibenzofuran	mg/kg	--	1600	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	-	-	<0.5	<0.5
Carbazole	mg/kg	--	86	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.6	<0.5	-	-	<0.5	<0.5
2-Nitroaniline	mg/kg														-	-	<1	<1
3,3'-Dichlorobenzidine	mg/kg														-	-	<0.5	<0.5
3-Nitroaniline	mg/kg														-	-	<1	<1

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP29_0.3-0.4	TP29_0.8-0.9	TP32_0.4-0.5	TP38_1.3-1.4	TP39_2.1-2.2	TP45_1.3-1.4	TP81_1.2-1.3	TP82_1.4-1.5	HA01	HA02	HA03	TP105_1.00	TP105_2.10	TP106_1.00	TP106_1.70
				7/04/2005	7/04/2005	7/04/2005	7/04/2005	8/04/2005	11/04/2005	7/04/2005	7/04/2005	8/04/2005	8/04/2005	8/04/2005	20/10/2006	20/10/2006	1/11/2006	1/11/2006
4-Chloroaniline	mg/kg														-	-	<0.5	<0.5
4-Nitroaniline	mg/kg														-	-	<0.5	<0.5
Aniline	mg/kg														-	-	<0.5	<0.5
Miscellaneous Compounds	mg/kg																	
1,3,5-Trichlorobenzene	mg/kg	--	220	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	0.7	<0.5	15	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	35.6	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene	mg/kg	--	600															
1,4-Dichlorobenzene	mg/kg	--	7.9															
1,2-Dichlorobenzene	mg/kg	--	600															
1,2,4-Trichlorobenzene	mg/kg	--	220															
Tetrachlorobenzene	mg/kg	--	180															
Phenols	mg/kg																	
2-Chlorophenol	mg/kg	--													<0.5	<0.5	<0.5	<0.5
4-Chlorophenol	mg/kg	--																
2,6-Dichlorophenol	mg/kg	--													<0.5	<0.5	<0.5	<0.5
2,4-Dichlorophenol	mg/kg	--													<0.5	<0.5	<0.5	<0.5
Pentachlorophenol	mg/kg	--													<1	<1	<1	<1
2,4,6-Trichlorophenol	mg/kg														<0.5	<0.5	<0.5	<0.5
2,4,5-Trichlorophenol	mg/kg														<0.5	<0.5	<0.5	<0.5
2,4-Dimethylphenol	mg/kg														<0.5	<0.5	<0.5	<0.5
2-Methylphenol	mg/kg														<0.5	<0.5	<0.5	<0.5
2-Nitrophenol	mg/kg														<0.5	<0.5	<0.5	<0.5
3- & 4-Methylphenol	mg/kg														<0.5	<0.5	<0.5	<0.5
4-Chloro-3-Methylphenol	mg/kg														<0.5	<0.5	<0.5	<0.5
Phenol	mg/kg														<0.5	<0.5	<0.5	<0.5
2,3,4,6-Tetrachlorophenol	mg/kg														<0.5	<0.5	<0.5	<0.5
4,4-DDD	mg/kg														<0.5	<0.5	<0.5	<0.5
4,4-DDE	mg/kg														<0.5	<0.5	<0.5	<0.5
4,4-DDT	mg/kg														<1	<1	<1	<1
a-BHC	mg/kg														<0.5	<0.5	<0.5	<0.5
Aldrin	mg/kg														<0.5	<0.5	<0.5	<0.5
b-BHC	mg/kg														<0.5	<0.5	<0.5	<0.5
d-BHC	mg/kg														<0.5	<0.5	<0.5	<0.5
Dieldrin	mg/kg														<0.5	<0.5	<0.5	<0.5
Endosulfan 1	mg/kg														<0.5	<0.5	<0.5	<0.5
Endosulfan 2	mg/kg														<0.5	<0.5	<0.5	<0.5
Endosulfan sulfate	mg/kg														<0.5	<0.5	<0.5	<0.5
Endrin	mg/kg														<0.5	<0.5	<0.5	<0.5
g-BHC	mg/kg														<0.5	<0.5	<0.5	<0.5
Heptachlor	mg/kg														<0.5	<0.5	<0.5	<0.5
Heptachlor epoxide	mg/kg														<0.5	<0.5	<0.5	<0.5
Organophosphorus Pesticides (OP)	mg/kg																	
Chlorfenvinphos	mg/kg														<0.5	<0.5	<0.5	<0.5
Chlorpyrifos	mg/kg														<0.5	<0.5	<0.5	<0.5
Chlorpyrifos-methyl	mg/kg														<0.5	<0.5	<0.5	<0.5
Diazinon	mg/kg														<0.5	<0.5	<0.5	<0.5
Dichlorvos	mg/kg														<0.5	<0.5	<0.5	<0.5
Dimethoate	mg/kg														<0.5	<0.5	<0.5	<0.5
Ethion	mg/kg														<0.5	<0.5	<0.5	<0.5
Fenthion	mg/kg														<0.5	<0.5	<0.5	<0.5
Malathion	mg/kg														<0.5	<0.5	<0.5	<0.5
Pirimphos-ethyl	mg/kg														<0.5	<0.5	<0.5	<0.5
Prothiofos	mg/kg														<0.5	<0.5	<0.5	<0.5
cis-Isosafrole	mg/kg														<0.5	<0.5	<0.5	<0.5
Diallate	mg/kg														<0.5	<0.5	<0.5	<0.5
Methanesulfonate ethyl	mg/kg														<0.5	<0.5	<0.5	<0.5
Methanesulfonate methyl	mg/kg														<0.5	<0.5	<0.5	<0.5
Safrole	mg/kg														<0.5	<0.5	<0.5	<0.5
trans-Isosafrole	mg/kg														<0.5	<0.5	<0.5	<0.5

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP107_1.00 3/11/2006	TP107_2.10 3/11/2006	QC109 3/11/2006	TP108_1.00 1/11/2006	TP108_2.10 1/11/2006	TP109_1.20 1/11/2006	TP109_2.00 1/11/2006	TP110_0.90 2/11/2006	TP110_1.90 2/11/2006	TP111_0.90 20/10/2006	TP111_2.10 20/10/2006	TP112_1.40 1/11/2006	TP112_2.20 1/11/2006	TP113_0.80 1/11/2006	TP113_1.70 1/11/2006	QC106 1/11/2006	TP114_1.00 20/10/2006
Polynuclear Aromatic Hydrocarbons																				
Naphthalene	mg/kg	--	190	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
2-Methylnaphthalene	mg/kg	--	190	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Acenaphthylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Acenaphthene	mg/kg	--	29000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Phenanthrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	0.5	<0.5	<0.5	<0.5	-	<0.5
Anthracene	mg/kg	--	100000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Fluoranthene	mg/kg	--	22000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Pyrene	mg/kg	--	29000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Benz(a)anthracene	mg/kg	--	2.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Chrysene	mg/kg	--	210	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1	<1	<1	<1	<1	<1	<1	<1	-	-	<1	<1	<1	<1	<1	<1	-	<1
Benzo(a)pyrene	mg/kg	5	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Dibenz(a,h)anthracene	mg/kg	--	0.21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
TotalPAH	mg/kg	100	--	ND	ND	ND	ND	ND	ND	ND	-	-	ND	ND	0.5	ND	ND	ND	-	ND
Fluorene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-2-Fluorenyl Acetamide	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
2-Chloronaphthalene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
3-Methylcholanthrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
7,12-Dimethylbenz(a)anthracene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Phthalate Esters																				
Di-n-butylphthalate	mg/kg	--	820000	<0.5	<0.5	1	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Butylbenzylphthalate	mg/kg	--	100,000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
bis(2-ethylhexyl)phthalate	mg/kg	--	120	<5	<5	12.8	<5	<5	<5	<5	-	-	<5	<5	<5	<5	<5	<5	-	<5
Diethyl phthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Dimethyl phthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Di-n-octylphthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Nitrosamines																				
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550	<1	<1	<1	<1	<1	<1	<1	-	-	<1	<1	<1	<1	<1	<1	-	<1
Methapyrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosodibutylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosodiethylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosodi-n-propylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosomethylethylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosomorpholine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosopiperidine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
N-Nitrosopyrrolidine	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	-	-	<1	<1	<1	<1	<1	<1	-	<1
Nitroaromatics and Ketones																				
Nitrobenzene	mg/kg	--	100	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
1,3,5-Trinitrobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
1-Naphthylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
2,4-Dinitrotoluene	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	-	-	<1	<1	<1	<1	<1	<1	-	<1
2,6-Dinitrotoluene	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	-	-	<1	<1	<1	<1	<1	<1	-	<1
2-Picoline	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
4-Aminobiphenyl	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
4-Nitroquinoline-N-oxide	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
5-Nitro-o-toluidine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Acetophenone	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Azobenzene	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	-	-	<1	<1	<1	<1	<1	<1	-	<1
Chlorobenzilate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Dimethylaminoazobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Isophorone	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Pentachloronitrobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Phenacetin	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Pronamide	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5
Haloethers																				
Bis(2-chloroethyl)ether	mg/kg	--	0.55																	
Chlorinated Hydrocarbons																				
Hexachloroethane	mg/kg	--	120																	
Hexachlorobutadiene	mg/kg	--	22																	
Pentachlorobenzene	mg/kg	--	490																	
Hexachlorobenzene (HCB)	mg/kg	--	1.1																	
Anilines and Benzidines																				
Dibenzofuran	mg/kg																			

**Southlands Soil Data - SVOC
Block 1**

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP114_2.00	TP115_1.00	TP116_0.80	TP116_1.90	TP116_2.40	TP117_1.00	TP117_2.00	TP118_0.90	TP118_1.00	TP118_1.40	TP118_1.80	TP119_1.00	TP119_1.70	TP120_0.90	TP120_1.40	TP120_1.80	TP121_0.90	
				20/10/2006	1/11/2006	2/11/2006	2/11/2006	2/11/2006	1/11/2006	1/11/2006	1/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	2/11/2006	2/11/2006	2/11/2006
Polynuclear Aromatic Hydrocarbons																					
Naphthalene	mg/kg	--	190	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
2-Methylnaphthalene	mg/kg	--	190	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthene	mg/kg	--	29000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Phenanthrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Anthracene	mg/kg	--	100000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Fluoranthene	mg/kg	--	22000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Pyrene	mg/kg	--	29000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Benzo(a)anthracene	mg/kg	--	2.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Chrysene	mg/kg	--	210	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Benzo(a)pyrene	mg/kg	5	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Dibenz(a,h)anthracene	mg/kg	--	0.21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
TotalPAH	mg/kg	100	--	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-2-Fluorenyl Acetamide	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
2-Chloronaphthalene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
3-Methylcholanthrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
7,12-Dimethylbenz(a)anthracene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Phthalate Esters																					
Di-n-butylphthalate	mg/kg	--	820000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Butylbenzylphthalate	mg/kg	--	100,000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
bis(2-ethylhexyl)phthalate	mg/kg	--	120	<5	<5	<5	5.8	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Diethyl phthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Dimethyl phthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Di-n-octylphthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Nitrosamines																					
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Methapyrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosodibutylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosodiethylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosodi-n-propylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosomethylethylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosomorpholine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosopiperidine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
N-Nitrosopyrrolidine	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Nitroaromatics and Ketones																					
Nitrobenzene	mg/kg	--	100	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trinitrobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
1-Naphthylamine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
2,4-Dinitrotoluene	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
2,6-Dinitrotoluene	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
2-Picoline	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
4-Aminobiphenyl	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
4-Nitroquinoline-N-oxide	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
5-Nitro-o-toluidine	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Acetophenone	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Azobenzene	mg/kg	--	--	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Chlorobenzilate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Dimethylaminoazobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Isophorone	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.6	<0.5	<0.5	<0.5	<0.5	<0.5
Pentachlor																					

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP128_0.80	QC108	TP128_1.70	HA001	HA002	HA003	HA004	HA005	HA006	TP094_0.90	TP094_2.00	TP095_1.00	TP095_1.90	TP096_0.90	TP097_0.90	TP097_2.90	TP098_1.00	
				2/11/2006	2/11/2006	2/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	20/10/2006	20/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006
Polynuclear Aromatic Hydrocarbons																					
Naphthalene	mg/kg	--	190	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			<0.5	-	<0.5	-	<0.5	<0.5	-	<0.5	
2-Methylnaphthalene	mg/kg	--	190	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			-	-	-	-	-	-	-	-	
Acenaphthylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			1.2	-	<0.5	-	<0.5	<0.5	-	<0.5	
Acenaphthene	mg/kg	--	29000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			<0.5	-	<0.5	-	<0.5	<0.5	-	<0.5	
Phenanthrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			5.4	-	<0.5	-	<0.5	<0.5	-	<0.5	
Anthracene	mg/kg	--	100000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			1.6	-	<0.5	-	<0.5	<0.5	-	<0.5	
Fluoranthene	mg/kg	--	22000	<0.5	<0.5	<0.5	0.7	<0.5	<0.5	<0.5			10.7	-	<0.5	-	<0.5	<0.5	-	<0.5	
Pyrene	mg/kg	--	29000	<0.5	<0.5	<0.5	0.6	<0.5	<0.5	<0.5			11.1	-	<0.5	-	<0.5	<0.5	-	<0.5	
Benz(a)anthracene	mg/kg	--	2.1	<0.5	<0.5	<0.5	0.5	<0.5	<0.5	<0.5			5	-	<0.5	-	<0.5	<0.5	-	<0.5	
Chrysene	mg/kg	--	210	<0.5	<0.5	<0.5	0.5	<0.5	<0.5	<0.5			4.9	-	<0.5	-	<0.5	<0.5	-	<0.5	
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1	<1	<1	<1	<1	<1	<1	<1			11.2	-	<1	-	<1	<1	-	<1	
Benzo(a)pyrene	mg/kg	5	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			-	-	<0.5	-	<0.5	<0.5	-	<0.5	
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			2.6	-	<0.5	-	<0.5	<0.5	-	<0.5	
Dibenz(a,h)anthracene	mg/kg	--	0.21	-	-	-	-	-	-	-			0.7	-	<0.5	-	<0.5	<0.5	-	<0.5	
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			3.1	-	<0.5	-	<0.5	<0.5	-	<0.5	
TotalPAH	mg/kg	100	--	ND	ND	ND	2.3	ND	ND	ND			0	-	ND	-	ND	ND	-	ND	
Fluorene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			<0.5	-	<0.5	-	<0.5	<0.5	-	<0.5	
N-2-Fluorenyl Acetamide	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			-	-	-	-	-	-	-	-	
2-Chloronaphthalene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			-	-	-	-	-	-	-	-	
3-Methylcholanthrene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			-	-	-	-	-	-	-	-	
7,12-Dimethylbenz(a)anthracene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5			-	-	-	-	-	-	-	-	
Phthalate Esters																					
Di-n-butylphthalate	mg/kg	--	820000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Butylbenzylphthalate	mg/kg	--	100,000	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
bis(2-ethylhexyl)phthalate	mg/kg	--	120	<5	<5	<5	<5	<5	<5	<5											
Diethyl phthalate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Dimethyl phthalate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Di-n-octylphthalate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Nitrosamines																					
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550	<1	<1	<1	<1	<1	<1	<1											
Methapyrilene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosodibutylamine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosodiethylamine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosodi-n-propylamine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosomethylethylamine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosomorpholine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosopiperidine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
N-Nitrosopyrrolidine	mg/kg			<1	<1	<1	<1	<1	<1	<1											
Nitroaromatics and Ketones																					
Nitrobenzene	mg/kg	--	100	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
1,3,5-Trinitrobenzene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
1-Naphthylamine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
2,4-Dinitrotoluene	mg/kg			<1	<1	<1	<1	<1	<1	<1											
2,6-Dinitrotoluene	mg/kg			<1	<1	<1	<1	<1	<1	<1											
2-Picoline	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
4-Aminobiphenyl	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
4-Nitroquinoline-N-oxide	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
5-Nitro-o-toluidine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Acetophenone	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Azobenzene	mg/kg			<1	<1	<1	<1	<1	<1	<1											
Chlorobenzilate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Dimethylaminoazobenzene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Isophorone	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Pentachloronitrobenzene	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Phenacetin	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Pronamide	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
Haloethers																					
Bis(2-chloroethyl)ether	mg/kg	--	0.55																		
Chlorinated Hydrocarbons																					
Hexachloroethane	mg/kg	--	120																		
Hexachlorobutadiene	mg/kg	--	22																		
Pentachlorobenzene	mg/kg	--	490																		
Hexachlorobenzene (HCB)	mg/kg	--	1.1																		
Anilines and Benzidines																					
Dibenzofuran	mg/kg	--	1600	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5									
Carbazole	mg/kg	--	86	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5									
2-Nitroaniline	mg/kg			<1	<1	<1	<1	<1	<1	<1											
3,3'-Dichlorobenzidine	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5											
3-Nitroaniline	mg/kg			<1	<1	<1	<1	<1	<1	<1											

**Southlands Soil Data - SVOC
Block 1**

			USEPA Region IX (Industrial Land)	TP128_0.80	QC108	TP128_1.70	HA001	HA002	HA003	HA004	HA005	HA006	TP094_0.90	TP094_2.00	TP095_1.00	TP095_1.90	TP096_0.90	TP097_0.90	TP097_2.90	TP098_1.00
Analyte	Units	NEPM-F		2/11/2006	2/11/2006	2/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006	20/10/2006	20/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006	20/10/2006
4-Chloroaniline	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
4-Nitroaniline	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Aniline	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Miscellaneous Compounds	mg/kg																			
1,3,5-Trichlorobenzene	mg/kg	--	220	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
1,3-Dichlorobenzene	mg/kg	--	600																	
1,4-Dichlorobenzene	mg/kg	--	7.9																	
1,2-Dichlorobenzene	mg/kg	--	600																	
1,2,4-Trichlorobenzene	mg/kg	--	220																	
Tetrachlorobenzene	mg/kg	--	180																	
Phenols	mg/kg																			
2-Chlorophenol	mg/kg	--		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
4-Chlorophenol	mg/kg	--																		
2,6-Dichlorophenol	mg/kg	--		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
2,4-Dichlorophenol	mg/kg	--		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Pentachlorophenol	mg/kg	--		<1	<1	<1	<1	<1	<1	<1	<1	<1								
2,4,6-Trichlorophenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
2,4,5-Trichlorophenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
2,4-Dimethylphenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
2-Methylphenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
2-Nitrophenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
3- & 4-Methylphenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
4-Chloro-3-Methylphenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Phenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
2,3,4,6-Tetrachlorophenol	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
4,4-DDD	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
4,4-DDE	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
4,4-DDT	mg/kg			<1	<1	<1	<1	<1	<1	<1	<1	<1								
a-BHC	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Aldrin	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
b-BHC	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
d-BHC	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Dieldrin	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Endosulfan 1	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Endosulfan 2	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Endosulfan sulfate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Endrin	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
g-BHC	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Heptachlor	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Heptachlor epoxide	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Organophosphorus Pesticides (OP)	mg/kg																			
Chlorfenvinphos	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Chlorpyrifos	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Chlorpyrifos-methyl	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Diazinon	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Dichlorvos	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Dimethoate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Ethion	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Fenthion	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Malathion	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Pirimphos-ethyl	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Prothiofos	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
cis-Isosafrole	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Diallate	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Methanesulfonate ethyl	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Methanesulfonate methyl	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
Safrole	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								
trans-Isosafrole	mg/kg			<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5								

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

**Southlands Soil Data - SVOC
Block 1**

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP098_1.90	TP098_2.40	TP099_1.00	TP099_1.90	TP100_1.80	TP100_2.50	QC104	TP101_1.00	TP101_1.80	TP102_1.00	TP102_2.00	QC103	QC201	TP103_0.80	TP103_1.80	TP103_2.90	QC105
				20/10/2006	20/10/2006	20/10/2006	20/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006
Polynuclear Aromatic Hydrocarbons																				
Naphthalene	mg/kg	--	190	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
2-Methylnaphthalene	mg/kg	--	190	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Acenaphthylene	mg/kg	--	--	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Acenaphthene	mg/kg	--	29000	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Phenanthrene	mg/kg	--	--	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Anthracene	mg/kg	--	100000	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Fluoranthene	mg/kg	--	22000	-	-	0.5	-	<0.5	-	-	0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Pyrene	mg/kg	--	29000	-	-	0.5	-	<0.5	-	-	0.6	-	<0.5	-	-	-	<0.5	-	-	<0.5
Benz(a)anthracene	mg/kg	--	2.1	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Chrysene	mg/kg	--	210	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1	-	-	<1	-	<1	-	-	<1	-	<1	-	-	-	<1	-	-	<1
Benzo(a)pyrene	mg/kg	5	--	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Dibenz(a,h)anthracene	mg/kg	--	0.21	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
Benzo(g,h,i)perylene	mg/kg	--	--	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
TotalPAH	mg/kg	100	--	-	-	0	-	ND	-	-	0	-	ND	-	-	-	ND	-	-	ND
Fluorene	mg/kg	--	--	-	-	<0.5	-	<0.5	-	-	<0.5	-	<0.5	-	-	-	<0.5	-	-	<0.5
N-2-Fluorenyl Acetamide	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2-Chloronaphthalene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3-Methylcholanthrene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
7,12-Dimethylbenz(a)anthracene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Phthalate Esters																				
Di-n-butylphthalate	mg/kg	--	820000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Butylbenzylphthalate	mg/kg	--	100,000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
bis(2-ethylhexyl)phthalate	mg/kg	--	120	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Diethyl phthalate	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Dimethyl phthalate	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Di-n-octylphthalate	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Nitrosamines																				
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Methapyrene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosodibutylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosodiethylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosodi-n-propylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosomethylethylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosomorpholine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosopiperidine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
N-Nitrosopyrrolidine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Nitroaromatics and Ketones																				
Nitrobenzene	mg/kg	--	100	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
1,3,5-Trinitrobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
1-Naphthylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2,4-Dinitrotoluene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2,6-Dinitrotoluene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2-Picoline	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
4-Aminobiphenyl	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
4-Nitroquinoline-N-oxide	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
5-Nitro-o-toluidine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Acetophenone	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Azobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlorobenzilate	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Dimethylaminoazobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Isophorone	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pentachloronitrobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Phenacetin	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pronamide	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Haloethers																				
Bis(2-chloroethyl)ether	mg/kg	--	0.55	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlorinated Hydrocarbons																				
Hexachloroethane	mg/kg	--	120	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hexachlorobutadiene	mg/kg	--	22	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pentachlorobenzene	mg/kg	--	490	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hexachlorobenzene (HCB)	mg/kg	--	1.1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Anilines and Benzidines																				
Dibenzofuran	mg/kg	--	1600	-	-	-	-	-	-	-	0	0	-	-	-	-	-	-	-	-
Carbazole	mg/kg	--	86	-	-	-	-	-	-	-	1.5	1.5	-	-	-	-	-	-	-	-
2-Nitroaniline	mg/kg	--	--	-	-	-	-	-	-	-	2.3	2.3	-	-	-	-	-	-	-	-
3,3'-Dichlorobenzidine	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3-Nitroaniline	mg/kg	--	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

**Southlands Soil Data - SVOC
Block 1**

			USEPA Region IX (Industrial Land)	TP098_1.90	TP098_2.40	TP099_1.00	TP099_1.90	TP100_1.80	TP100_2.50	QC104	TP101_1.00	TP101_1.80	TP102_1.00	TP102_2.00	QC103	QC201	TP103_0.80	TP103_1.80	TP103_2.90	QC105
Analyte	Units	NEPM-F		20/10/2006	20/10/2006	20/10/2006	20/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	19/10/2006	20/10/2006	20/10/2006	20/10/2006	20/10/2006
4-Chloroaniline	mg/kg																-	-	-	-
4-Nitroaniline	mg/kg																-	-	-	-
Aniline	mg/kg																-	-	-	-
Miscellaneous Compounds	mg/kg																			
1,3,5-Trichlorobenzene	mg/kg	--	220														-	-	-	-
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180														-	-	-	-
1,3-Dichlorobenzene	mg/kg	--	600														-	-	-	-
1,4-Dichlorobenzene	mg/kg	--	7.9																	
1,2-Dichlorobenzene	mg/kg	--	600																	
1,2,4-Trichlorobenzene	mg/kg	--	220																	
Tetrachlorobenzene	mg/kg	--	180																	
Phenols	mg/kg																			
2-Chlorophenol	mg/kg	--															-	-	-	-
4-Chlorophenol	mg/kg	--															-	-	-	-
2,6-Dichlorophenol	mg/kg	--															-	-	-	-
2,4-Dichlorophenol	mg/kg	--															-	-	-	-
Pentachlorophenol	mg/kg	--															-	-	-	-
2,4,6-Trichlorophenol	mg/kg																-	-	-	-
2,4,5-Trichlorophenol	mg/kg																-	-	-	-
2,4-Dimethylphenol	mg/kg																-	-	-	-
2-Methylphenol	mg/kg																-	-	-	-
2-Nitrophenol	mg/kg																-	-	-	-
3- & 4-Methylphenol	mg/kg																-	-	-	-
4-Chloro-3-Methylphenol	mg/kg																-	-	-	-
Phenol	mg/kg																-	-	-	-
2,3,4,6-Tetrachlorophenol	mg/kg																-	-	-	-
4,4-DDD	mg/kg																-	-	-	-
4,4-DDE	mg/kg																-	-	-	-
4,4-DDT	mg/kg																-	-	-	-
a-BHC	mg/kg																-	-	-	-
Aldrin	mg/kg																-	-	-	-
b-BHC	mg/kg																-	-	-	-
d-BHC	mg/kg																-	-	-	-
Dieldrin	mg/kg																-	-	-	-
Endosulfan 1	mg/kg																-	-	-	-
Endosulfan 2	mg/kg																-	-	-	-
Endosulfan sulfate	mg/kg																-	-	-	-
Endrin	mg/kg																-	-	-	-
g-BHC	mg/kg																-	-	-	-
Heptachlor	mg/kg																-	-	-	-
Heptachlor epoxide	mg/kg																-	-	-	-
Organophosphorus Pesticides (OP)	mg/kg																			
Chlorfenvinphos	mg/kg																-	-	-	-
Chlorpyrifos	mg/kg																-	-	-	-
Chlorpyrifos-methyl	mg/kg																-	-	-	-
Diazinon	mg/kg																-	-	-	-
Dichlorvos	mg/kg																-	-	-	-
Dimethoate	mg/kg																-	-	-	-
Ethion	mg/kg																-	-	-	-
Fenthion	mg/kg																-	-	-	-
Malathion	mg/kg																-	-	-	-
Pirimphos-ethyl	mg/kg																-	-	-	-
Prothiofos	mg/kg																-	-	-	-
cis-Isosafrole	mg/kg																-	-	-	-
Diallyl	mg/kg																			
Methanesulfonate ethyl	mg/kg																			
Methanesulfonate methyl	mg/kg																			
Safrole	mg/kg																			
trans-Isosafrole	mg/kg																			

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

Southlands Soil Data - SVOC
Block 1

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP104_0.70	TP104_1.90	TP129_1.50	TP129_3.00	QC100	QC200	TP130_1.00	QC101	HA007	QC001	HA008	HA009
				20/10/2006	20/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006
Polynuclear Aromatic Hydrocarbons															
Naphthalene	mg/kg	--	190	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2-Methylnaphthalene	mg/kg	--	190	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Acenaphthylene	mg/kg	--	--	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Acenaphthene	mg/kg	--	29000	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Phenanthrene	mg/kg	--	--	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Anthracene	mg/kg	--	100000	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Fluoranthene	mg/kg	--	22000	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Pyrene	mg/kg	--	29000	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Benz(a)anthracene	mg/kg	--	2.1	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Chrysene	mg/kg	--	210	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	23.1	<1	-	-	-	-	-	-	-	<1	<1	-	-
Benzo(a)pyrene	mg/kg	5	--	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Indeno(1,2,3-cd)pyrene	mg/kg	--	2.1	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Dibenz(a,h)anthracene	mg/kg	--	0.21	<0.5	-	-	-	-	-	-	-	-	-	-	-
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
TotalPAH	mg/kg	100	--	ND	-	-	-	-	-	-	-	ND	ND	-	-
Fluorene	mg/kg	--	--	<0.5	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-2-Fluorenyl Acetamide	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2-Chloronaphthalene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
3-Methylanthracene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
7,12-Dimethylbenz(a)anthracene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Phthalate Esters															
Di-n-butylphthalate	mg/kg	--	820000	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Butylbenzylphthalate	mg/kg	--	100,000	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
bis(2-ethylhexyl)phthalate	mg/kg	--	120	-	-	-	-	-	-	-	-	<5	<5	-	-
Diethyl phthalate	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Dimethyl phthalate	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Di-n-octylphthalate	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Nitrosamines															
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	25550	-	-	-	-	-	-	-	-	<1	<1	-	-
Methapyrene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosodibutylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosodiethylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosodi-n-propylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosomethylethylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosomorpholine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosopiperidine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
N-Nitrosopyrrolidine	mg/kg	--	--	-	-	-	-	-	-	-	-	<1	<1	-	-
Nitroaromatics and Ketones															
Nitrobenzene	mg/kg	--	100	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
1,3,5-Trinitrobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
1-Naphthylamine	mg/kg	--	--	-	-	-	-	-	-	-	-	<1	<1	-	-
2,4-Dinitrotoluene	mg/kg	--	--	-	-	-	-	-	-	-	-	<1	<1	-	-
2,6-Dinitrotoluene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2-Picoline	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4-Aminobiphenyl	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4-Nitroquinoline-N-oxide	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
5-Nitro-o-toluidine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Acetophenone	mg/kg	--	--	-	-	-	-	-	-	-	-	<1	<1	-	-
Azobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Chlorobenzilate	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Dimethylaminoazobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Isophorone	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Pentachloronitrobenzene	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Phenacetin	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Pronamide	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Haloethers															
Bis(2-chloroethyl)ether	mg/kg	--	0.55	-	-	-	-	-	-	-	-	-	-	-	-
Chlorinated Hydrocarbons															
Hexachloroethane	mg/kg	--	120	-	-	-	-	-	-	-	-	-	-	-	-
Hexachlorobutadiene	mg/kg	--	22	-	-	-	-	-	-	-	-	-	-	-	-
Pentachlorobenzene	mg/kg	--	490	-	-	-	-	-	-	-	-	-	-	-	-
Hexachlorobenzene (HCB)	mg/kg	--	1.1	-	-	-	-	-	-	-	-	-	-	-	-
Anilines and Benzidines															
Dibenzofuran	mg/kg	--	1600	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Carbazole	mg/kg	--	86	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2-Nitroaniline	mg/kg	--	--	-	-	-	-	-	-	-	-	<1	<1	-	-
3,3'-Dichlorobenzidine	mg/kg	--	--	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
3-Nitroaniline	mg/kg	--	--	-	-	-	-	-	-	-	-	<1	<1	-	-

**Southlands Soil Data - SVOC
Block 1**

			USEPA Region IX (Industrial Land)	TP104_0.70	TP104_1.90	TP129_1.50	TP129_3.00	QC100	QC200	TP130_1.00	QC101	HA007	QC001	HA008	HA009
Analyte	Units	NEPM-F		20/10/2006	20/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	8/11/2006	8/11/2006	8/11/2006	8/11/2006
4-Chloroaniline	mg/kg			-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4-Nitroaniline	mg/kg			-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Aniline	mg/kg			-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Miscellaneous Compounds	mg/kg														
1,3,5-Trichlorobenzene	mg/kg	--	220	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180	-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
1,3-Dichlorobenzene	mg/kg	--	600	-	-	-	-	-	-	-	-				
1,4-Dichlorobenzene	mg/kg	--	7.9	-	-	-	-	-	-	-	-				
1,2-Dichlorobenzene	mg/kg	--	600	-	-	-	-	-	-	-	-				
1,2,4-Trichlorobenzene	mg/kg	--	220	-	-	-	-	-	-	-	-				
Tetrachlorobenzene	mg/kg	--	180	-	-	-	-	-	-	-	-				
Phenols	mg/kg														
2-Chlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4-Chlorophenol	mg/kg	--		-	-	-	-	-	-	-	-				
2,6-Dichlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2,4-Dichlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Pentachlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<1	<1	-	-
2,4,6-Trichlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2,4,5-Trichlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2,4-Dimethylphenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2-Methylphenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2-Nitrophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
3- & 4-Methylphenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4-Chloro-3-Methylphenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Phenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
2,3,4,6-Tetrachlorophenol	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4,4-DDD	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4,4-DDE	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
4,4-DDT	mg/kg	--		-	-	-	-	-	-	-	-	<1	<1	-	-
a-BHC	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Aldrin	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
b-BHC	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
d-BHC	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Dieldrin	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Endosulfan 1	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Endosulfan 2	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Endosulfan sulfate	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Endrin	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
g-BHC	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Heptachlor	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Heptachlor epoxide	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Organophosphorus Pesticides (OP)	mg/kg														
Chlorfenvinphos	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Chlorpyrifos	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Chlorpyrifos-methyl	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Diazinon	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Dichlorvos	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Dimethoate	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Ethion	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Fenthion	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Malathion	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Pirimphos-ethyl	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Prothiofos	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
cis-Isosafrole	mg/kg	--		-	-	-	-	-	-	-	-	<0.5	<0.5	-	-
Diallyl	mg/kg	--		-	-	-	-	-	-	-	-				
Methanesulfonate ethyl	mg/kg	--		-	-	-	-	-	-	-	-				
Methanesulfonate methyl	mg/kg	--		-	-	-	-	-	-	-	-				
Safrole	mg/kg	--		-	-	-	-	-	-	-	-				
trans-Isosafrole	mg/kg	--		-	-	-	-	-	-	-	-				

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

**Southlands Soil Data - Metals
Block 2**

Analyte	Units	NEPM-F	SS022	SS023	SS024	SS025	SS026	SS027	SS028	SS029	SS030	SS031	SS032	SS033	SS034	SS035	SS036	SS037	SS038	SS039	TP50_1.2-1.3	SP16	SP17	SP18	SP19
			TP1	TP1	TP2	TP2	TP3	TP3	TP4	TP4	TP5	TP5	TP6	TP6	TP7	TP7	TP8	TP8	TP9	TP9					
Arsenic	mg/kg	500																			41	7	<5	<5	<5
Cadmium	mg/kg	100																			2	<1	<1	<1	<1
Chromium	mg/kg	60	2.7	11.3	45.2	23.4	18.6	11.2	31.2	46.6	28.4	32.6	19.3	6.9	2.9	27.2	9.8	33.6	9.7	14.9	20	15	8	6	11
Copper	mg/kg	5000																			150	27	16	13	34
Lead	mg/kg	1500																			251	70	52	36	
Nickel	mg/kg	3000																			45	7	5	4	11
Zinc	mg/kg	35000																			917	125	58	71	254
Mercury	mg/kg	75	0.51	0.21	0.92	8.3	<0.1	0.1	5.6	0.28	0.13	8.7	0.45	<0.1	<0.1	0.20	0.31	66	<0.1	0.21	1233		0.4	0.2	0.1
pH			9.20	6.72	8.23	7.51	9.00	8.48	6.74	6.83	9.18	7.55	8.63	7.51	9.08	8.00	7.46	8.31	8.01	7.90					
Oil & Grease	mg/kg		-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	230	110			
Total Organic Matter	mg/kg		-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	18600	54000			
Formaldehyde	mg/kg		1.2	4	3.6	5.7	<2	82	8.3	3.7	<2	11	1.2	<2	<2	1.8	<2	3.9	<2	<2					
Total Phenols	mg/kg																								
Cation Exchange Capacity	mg/kg																						0.4	0.6	
Ammonia-N	mg/kg																						<10	5.9	
Total Kjeldahl Nitrogen	mg/kg																						30	217	
P-Ortho	mg/kg																						1.1	1.8	
P-Total	mg/kg																						2.2	4.1	

Notes:
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 1 = Cr VI trigger value adopted as most conservative
 - = not analysed

**Southlands Soil Data - Metals
Block 2**

Analyte	Units	NEPM-F	SP20	SP32	SP33	SP34	SP35	SP36	SP37	SP38	SP39	SP40	SP41	SP42	SP43	SP44	SP45	SP46	SP47
			13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005
Arsenic	mg/kg	500	<5	5	6	11	5	<5	<5	14	16	6	<5	7	10	10	5	6	6
Cadmium	mg/kg	100	1	<1	<1	<1	<1	<1	<1	<1	<1	2	<1	<1	<1	1	<1	<1	<1
Chromium	mg/kg	60	13	9	9	78	7	9	6	11	35	40	<2	50	39	26	9	12	10
Copper	mg/kg	5000	39	34	50	51	40	32	27	34	36	133	<5	132	78	74	81	37	26
Lead	mg/kg	1500											78					78	51
Nickel	mg/kg	3000	9	10	6	15	8	6	6	8	5	18	<2	21	15	13	11	6	4
Zinc	mg/kg	35000	188	283	209	299	189	279	239	178	102	549	46	2960	202	494	137	109	82
Mercury	mg/kg	75	0.6	0.7			0.3	0.3	0.2	1.4		0.6	<0.1	0.7	1	0.5	1.2		
pH																			
Oil & Grease	mg/kg																		
Total Organic Matter	mg/kg																		
Formaldehyde	mg/kg																		
Total Phenols	mg/kg																		
Cation Exchange Capacity	mg/kg																		
Ammonia-N	mg/kg																		
Total Kjeldahl Nitrogen	mg/kg																		
P-Ortho	mg/kg																		
P-Total	mg/kg																		

Notes:
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- = not analysed

**Southlands Soil Data - Metals
Block 2**

Analyte	Units	NEPM-F	SP48	SP49	SP50	TP084_1.00	TP084_2.50	TP085_1.20	TP085_2.50	TP086_1.50	TP086_2.20	TP131_1.00	TP131_2.00	TP132_1.10	TP132_1.60
			13/04/2005	13/04/2005	13/04/2005	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	3/11/2006	3/11/2006	3/11/2006
Arsenic	mg/kg	500	12	7	<5	<5	<5	<5	<5	<5	8	28	15	<5	8
Cadmium	mg/kg	100	2	1	<1	<1	<1	<1	<1	<1	3	2	2	<1	2
Chromium	mg/kg	60	84		28	8	<2	7	<2	3	20	50	27	12	37
Copper	mg/kg	5000	138	288	186	6	<5	10	<5	14	104	288	282	62	436
Lead	mg/kg	1500				16	<5	24	<5	28	146	628	287	141	626
Nickel	mg/kg	3000	19	39	28	<2	<2	2	<2	<2	52	101	70	17	45
Zinc	mg/kg	35000	559	632	321	14	9	13	<5	15	679	944	551	433	1030
Mercury	mg/kg	75	0.7	0.5	0.4	0.3	0.1	0.5	<0.1	0.2	5.3	35.8	7.1	0.2	3.2
pH															
Oil & Grease	mg/kg														
Total Organic Matter	mg/kg														
Formaldehyde	mg/kg														
Total Phenols	mg/kg														
Cation Exchange Capacity	mg/kg														
Ammonia-N	mg/kg														
Total Kjeldahl Nitrogen	mg/kg														
P-Ortho	mg/kg														
P-Total	mg/kg														

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
1 = Cr VI trigger value adopted as most conservative
- = not analysed

**Southlands Soil Data - TPH and MAH
Block 2**

Analyte	Units	NSW_EPA_1994	SS022	SS023	SS024	SS025	SS026	SS027	SS028	SS029	SS030	SS031	SS032
			TP1_0.2-0.4	TP1_0.7-0.8	TP2_0.2-0.4	TP2_0.7-0.8	TP3_0.1-0.3	TP3_0.4-0.5	TP4_0.2-0.4	TP4_1.2-1.4	TP5_0.1-0.3	TP5_1.5-1.7	TP6_0.2-0.4
Total Petroleum Hydrocarbons													
C6-C9Fraction	mg/kg	65											
C10-C14Fraction	mg/kg	1000											
C15-C28Fraction	mg/kg	1000											
C29-C36Fraction	mg/kg	1000											
Monocyclic Aromatic Hydrocarbons													
Benzene	mg/kg	1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Toluene	mg/kg	1.4	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Ethylbenzene	mg/kg	3.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
meta-para-Xylene	mg/kg	14	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
ortho-Xylene	mg/kg	14	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Isopropylbenzene	mg/kg	--											
n-Propylbenzene	mg/kg	--											
1,3,5-Trimethylbenzene	mg/kg	--											
sec-Butylbenzene	mg/kg	--											
1,2,4-Trimethylbenzene	mg/kg	--											
n-Butylbenzene													
n-Propylbenzene													
p-Isopropyltoluene													
Styrene													
tert-Butylbenzene													

Notes:
 -- = criteria not available
 < = analyte not detected above laboratory practical quantitation limit (PQL)
 Bold/ Box = concentrations above criteria
 Assessment criteria = ANZECC 2000
 1 = Cr VI trigger value adopted as most conservative
 - = not analysed
 * : Results determined using Methanol Extraction Method and are corrected for blank.

Southlands Soil Data - TPH and MAH
Block 2

Analyte	Units	NSW_EPA_1994	SS033	SS034	SS035	SS036	SS037	SS038	SS039	TP084_1.0	TP084_2.50	TP085_1.20	TP085_2.50	TP086_1.50	TP086_2.20	TP131_1.00	TP131_2.00	TP132_1.10	TP132_1.60	
			TP6_1.4-1.6	TP7_0.1-0.3	TP7_1.5-1.7	TP8_0.2-04	TP8_1.4-1.6	TP9_0.1-03	TP9_1.2-1.4	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	3/11/2006	3/11/2006	3/11/2006	3/11/2006	
Total Petroleum Hydrocarbons																				
C6-C9Fraction	mg/kg	65								-	-	-	-	-	-	<2	<2	<2	<2	
C10-C14Fraction	mg/kg	1000								-	-	-	-	-	-	<50	<50	<50	<50	
C15-C28Fraction	mg/kg	1000								-	-	-	-	-	-	250	160	<100	<100	
C29-C36Fraction	mg/kg	1000								-	-	-	-	-	-	370	290	<100	<100	
Monocyclic Aromatic Hydrocarbons																				
Benzene	mg/kg	1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	-	-	-	-	-	-	<0.2	<0.5	<0.5	<0.2	
Toluene	mg/kg	1.4	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	-	-	-	-	-	-	<0.2	<0.2	<0.2	<0.2	
Ethylbenzene	mg/kg	3.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	-	-	-	-	-	-	<0.2	<0.2	<0.2	<0.2	
meta-¶-Xylene	mg/kg	14	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	-	-	-	-	-	-	<0.2	<0.2	<0.2	<0.2	
ortho-Xylene	mg/kg	14	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	-	-	-	-	-	-	<0.2	<0.2	<0.2	<0.2	
Isopropylbenzene	mg/kg	--								-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
n-Propylbenzene	mg/kg	--								-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
1,3,5-Trimethylbenzene	mg/kg	--								-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
sec-Butylbenzene	mg/kg	--								-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
1,2,4-Trimethylbenzene	mg/kg	--								-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
n-Butylbenzene										-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
n-Propylbenzene										-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
p-Isopropyltoluene										-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
Styrene										-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	
tert-Butylbenzene										-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5	

Notes:
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Assessment criteria = ANZECC 2000
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**Southlands Soil Data - VOC
Block 2**

Analyte grouping/ Analyte	Units	USEPA- Region_IX	SS022	SS023	SS024	SS025	SS026	SS027	SS028	SS029	SS030
		(Industrial Land) mg/kg	TP1 0.2-0.4	TP1 0.7-0.8	TP2 0.2-0.4	TP2 0.7-0.8	TP3 0.1-0.3	TP3 0.4-0.5	TP4 0.2-0.4	TP4 1.2-1.4	TP5 0.1-0.3
Halogenated Aliphatic Compounds											
Vinylchloride	mg/kg	0.75									
1,1-Dichloroethene	mg/kg	410	<0.02	<0.02	<0.02	<0.02	<0.02	0.03	<0.02	0.07	<0.02
trans-1,2-Dichloroethene	mg/kg	230	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
1,1-Dichloroethane	mg/kg	1700									
cis-1,2-Dichloroethene	mg/kg	150	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
1,2-Dichloroethane	mg/kg	0.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Trichloroethene	mg/kg	0.11	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
1,1,2-Trichloroethane	mg/kg	1.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Tetrachloroethene	mg/kg	1.3	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
1,1,2,2-Tetrachloroethane	mg/kg	0.93									
Hexachlorobutadiene	mg/kg	22									
Chloroethene	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Trihalomethanes											
Chloroform	mg/kg	12	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4
Other Chloromethanes											
Carbon Tetrachloride	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Dichloromethane	mg/kg		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chloromethane	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Chloroethanes											
1,1,1-Trichloroethane	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Chloroethane	mg/kg		<0.02	0.03	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Miscellaneous											
Ethene	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclopentane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclohexane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Carbon Bisulphide	mg/kg		-	-	-	-	-	-	-	-	-
1,1,1,2-Tetrachloroethane											
1,1-Dichloropropylene											
1,2,3-Trichloropropane											
1,2-Dibromo-3-chloropropane											
1,3-Dichloropropane											
Bromobenzene											
Bromomethane											
cis-1,4-Dichloro-2-butene											
Dibromomethane											
Dichlorodifluoromethane											
Hexachlorobutadiene											
Iodomethane											
Pentachloroethane											
trans-1,3-dichloropropene											
trans-1,4-Dichloro-2-butene											
Trichlorofluoromethane											
Trihalomethanes											
Bromochloromethane											
Bromodichloromethane											
Bromoform											
Dibromochloromethane											
Fumigants											
1,2-Dibromomethane											
1,2-Dichloropropane											
2,2-Dichloropropane											
cis-1,3-Dichloropropylene											
trans-1,3-Dichloropropylene											
Haloethers											
4-Bromophenyl phenyl ether											
4-Chlorophenyl phenyl ether											
Bis(2-chloroethoxy) methane											
Bis(2-chloroethyl) ether											
Oxygenated Compounds											
2-Butanone (MEK)											
2-Hexanone (MBK)											
2-Propanone											
4-Methyl-2-pentanone											
Vinyl Acetate											
1,2,3-Trichlorobenzene											
1,2,4-Trichlorobenzene											
1,2-Dichlorobenzene											
1,3-Dichlorobenzene											
1,4-Dichlorobenzene											
2-Chlorotoluene											
4-Chlorotoluene											
Chlorobenzene											
Chlorinated Hydrocarbons											
1,2-Dichlorobenzene											
1,3-Dichlorobenzene											
1,4-Dichlorobenzene											
Hexachlorobenzene											
Hexachlorocyclopentadiene											
Hexachloroethane											
Hexachloropropylene											
Pentachlorobenzene											

Notes:
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 Assessment criteria = ANZECC 2000
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 # : Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for both methods and therefore reported 'Neat' even though originally outside calibration.
 ## : Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
 ^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error because of inhomogeneity).

Southlands Soil Data - VOC
Block 2

Analyte grouping/ Analyte	Units	USEPA- Region_IX	SS031	SS032	SS033	SS034	SS035	SS036	SS037	SS038	SS039
		(Industrial Land) mg/kg	TP5 1.5-1.7	TP6 0.2-0.4	TP6 1.4-1.6	TP7 0.1-0.3	TP7 1.5-1.7	TP8 0.2-0.4	TP8 1.4-1.6	TP9 0.1-0.3	TP9 1.2-1.4
Halogenated Aliphatic Compounds											
Vinylchloride	mg/kg	0.75									
1,1-Dichloroethene	mg/kg	410	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
trans-1,2-Dichloroethene	mg/kg	230	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
1,1-Dichloroethane	mg/kg	1700									
cis-1,2-Dichloroethene	mg/kg	150	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
1,2-Dichloroethane	mg/kg	0.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Trichloroethene	mg/kg	0.11	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
1,1,1-Trichloroethane	mg/kg	1.6	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Tetrachloroethene	mg/kg	1.3	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
1,1,1,2-Tetrachloroethane	mg/kg	0.93									
Hexachlorobutadiene	mg/kg	22									
Chloroethene	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Trihalomethanes											
Chloroform	mg/kg	12	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4
Other Chloromethanes											
Carbon Tetrachloride	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Dichloromethane	mg/kg		<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chloromethane	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Chloroethanes											
1,1,1-Trichloroethane	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Chloroethane	mg/kg		<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Miscellaneous											
Ethene	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclopentane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Cyclohexane	mg/kg		<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Carbon Bisulphide	mg/kg		-	-	-	-	-	-	-	-	-
1,1,1,2-Tetrachloroethane											
1,1-Dichloropropylene											
1,2,3-Trichloropropane											
1,2-Dibromo-3-chloropropane											
1,3-Dichloropropane											
Bromobenzene											
Bromomethane											
cis-1,4-Dichloro-2-butene											
Dibromomethane											
Dichlorodifluoromethane											
Hexachlorobutadiene											
Iodomethane											
Pentachloroethane											
trans-1,3-dichloropropene											
trans-1,4-Dichloro-2-butene											
Trichlorofluoromethane											
Trihalomethanes											
Bromochloromethane											
Bromodichloromethane											
Bromoform											
Dibromochloromethane											
Fumigants											
1,2-Dibromomethane											
1,2-Dichloropropane											
2,2-Dichloropropane											
cis-1,3-Dichloropropylene											
trans-1,3-Dichloropropylene											
Haloethers											
4-Bromophenyl phenyl ether											
4-Chlorophenyl phenyl ether											
Bis(2-chloroethoxy) methane											
Bis(2-chloroethyl) ether											
Oxygenated Compounds											
2-Butanone (MEK)											
2-Hexanone (MBK)											
2-Propanone											
4-Methyl-2-pentanone											
Vinyl Acetate											
1,2,3-Trichlorobenzene											
1,2,4-Trichlorobenzene											
1,2-Dichlorobenzene											
1,3-Dichlorobenzene											
1,4-Dichlorobenzene											
2-Chlorotoluene											
4-Chlorotoluene											
Chlorobenzene											
Chlorinated Hydrocarbons											
1,2-Dichlorobenzene											
1,3-Dichlorobenzene											
1,4-Dichlorobenzene											
Hexachlorobenzene											
Hexachlorocyclopentadiene											
Hexachloroethane											
Hexachloropropylene											
Pentachlorobenzene											

Notes:

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Southlands Soil Data - VOC
Block 2

Analyte grouping/ Analyte	Units	USEPA- Region IX		TP084_1.0	TP084_2.5	TP085_1.2	TP085_2.5	TP086_1.5	TP086_2.2	TP131_1.0	TP131_2.0
		(Industrial Land)	TP50_1.2-1.3	0	0	0	0	0	0	0	0
		mg/kg	12/04/2005	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	3/11/2006	3/11/2006
Halogenated Aliphatic Compounds											
Vinylchloride	mg/kg	0.75	<5	-	-	-	-	-	-	<5	<5
1,1-Dichloroethene	mg/kg	410	<0.5	-	-	-	-	-	-	<0.5	<0.5
trans-1,2-Dichloroethene	mg/kg	230	<0.5	-	-	-	-	-	-	<0.5	<0.5
1,1-Dichloroethane	mg/kg	1700	<0.5	-	-	-	-	-	-	<0.5	<0.5
cis-1,2-Dichloroethene	mg/kg	150	<0.5	-	-	-	-	-	-	<0.5	<0.5
1,2-Dichloroethane	mg/kg	0.6	<0.5	-	-	-	-	-	-	<0.5	<0.5
Trichloroethene	mg/kg	0.11	<0.5	-	-	-	-	-	-	<0.5	<0.5
1,1,2-Trichloroethane	mg/kg	1.6	<0.5	-	-	-	-	-	-	<0.5	<0.5
Tetrachloroethene	mg/kg	1.3	0.5	-	-	-	-	-	-	<0.5	<0.5
1,1,2,2-Tetrachloroethane	mg/kg	0.93	<0.5	-	-	-	-	-	-	<0.5	<0.5
Hexachlorobutadiene	mg/kg	22	<0.5	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chloroethene	mg/kg										
Trihalomethanes											
Chloroform	mg/kg	12	<0.5	-	-	-	-	-	-	<0.5	<0.5
Other Chloromethanes											
Carbon Tetrachloride	mg/kg			-	-	-	-	-	-	<0.5	<0.5
Dichloromethane	mg/kg			-	-	-	-	-	-	<5	<5
Chloroethanes	mg/kg			-	-	-	-	-	-	<5	<5
1,1,1- Trichloroethane	mg/kg			-	-	-	-	-	-	<0.5	<0.5
Chloroethane	mg/kg			-	-	-	-	-	-	<5	<5
Miscellaneous											
Ethene	mg/kg										
Cyclopentane	mg/kg										
Cyclohexane	mg/kg										
Carbon Bisulphide	mg/kg										
1,1,1,2-Tetrachloroethane				-	-	-	-	-	-	<0.5	<0.5
1,1-Dichloropropylene				-	-	-	-	-	-	<0.5	<0.5
1,2,3-Trichloropropane				-	-	-	-	-	-	<0.5	<0.5
1,2-Dibromo-3-chloropropane				-	-	-	-	-	-	<0.5	<0.5
1,3-Dichloropropane				-	-	-	-	-	-	<0.5	<0.5
Bromobenzene				-	-	-	-	-	-	<0.5	<0.5
Bromomethane				-	-	-	-	-	-	<5	<5
cis-1,4-Dichloro-2-butene				-	-	-	-	-	-	<0.5	<0.5
Dibromomethane				-	-	-	-	-	-	<0.5	<0.5
Dichlorodifluoromethane				-	-	-	-	-	-	<5	<5
Hexachlorobutadiene				-	-	-	-	-	-	<0.5	<0.5
Iodomethane				-	-	-	-	-	-	<0.5	<0.5
Pentachloroethane				-	-	-	-	-	-	<0.5	<0.5
trans-1,3-dichloropropene				-	-	-	-	-	-	-	-
trans-1,4-Dichloro-2-butene				-	-	-	-	-	-	<0.5	<0.5
Trichlorofluoromethane				-	-	-	-	-	-	<5	<5
Trihalomethanes											
Bromochloromethane				-	-	-	-	-	-	<0.5	<0.5
Bromodichloromethane				-	-	-	-	-	-	<0.5	<0.5
Bromoform				-	-	-	-	-	-	<0.5	<0.5
Dibromochloromethane				-	-	-	-	-	-	<0.5	<0.5
Fumigants											
1,2-Dibromomethane				-	-	-	-	-	-	<0.5	<0.5
1,2-Dichloropropane				-	-	-	-	-	-	<0.5	<0.5
2,2-Dichloropropane				-	-	-	-	-	-	<0.5	<0.5
cis-1,3-Dichloropropylene				-	-	-	-	-	-	<0.5	<0.5
trans-1,3-Dichloropropylene				-	-	-	-	-	-	<0.5	<0.5
Haloethers											
4-Bromophenyl phenyl ether				-	-	-	-	-	-	<0.5	<0.5
4-Chlorophenyl phenyl ether				-	-	-	-	-	-	<0.5	<0.5
Bis(2-chloroethoxy) methane				-	-	-	-	-	-	<0.5	<0.5
Bis(2-chloroethyl) ether				-	-	-	-	-	-	<0.5	<0.5
Oxygenated Compounds											
2-Butanone (MEK)				-	-	-	-	-	-	<5	<5
2-Hexanone (MBK)				-	-	-	-	-	-	<5	<5
2-Propanone				-	-	-	-	-	-	-	-
4-Methyl-2-pentanone				-	-	-	-	-	-	<5	<5
Vinyl Acetate				-	-	-	-	-	-	<5	<5
1,2,3-Trichlorobenzene				-	-	-	-	-	-	<0.5	<0.5
1,2,4-Trichlorobenzene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2-Dichlorobenzene				-	-	-	-	-	-	<0.5	<0.5
1,3-Dichlorobenzene				-	-	-	-	-	-	<0.5	<0.5
1,4-Dichlorobenzene				-	-	-	-	-	-	<0.5	<0.5
2-Chlorotoluene				-	-	-	-	-	-	<0.5	<0.5
4-Chlorotoluene				-	-	-	-	-	-	<0.5	<0.5
Chlorobenzene				-	-	-	-	-	-	<0.5	<0.5
Chlorinated Hydrocarbons											
1,2-Dichlorobenzene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,4-Dichlorobenzene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<1	<1
Hexachlorocyclopentadiene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<2.5	<2.5
Hexachloroethane				-	<0.5	<0.5	<0.5	<0.5	<0.5	0.6	<0.5
Hexachloropropylene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Pentachlorobenzene				-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5

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- = not analysed

* : Results determined using Methanol Extraction Method and are corrected for blank.

: Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for t even though originally outside calibration.

: Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.

^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error becaus

Southlands Soil Data - VOC
Block 2

Analyte grouping/ Analyte	Units	USEPA- Region IX		
		(Industrial Land) mg/kg	TP132_1.1 0 3/11/2006	TP132_1.6 0 3/11/2006
Halogenated Aliphatic Compounds				
Vinylchloride	mg/kg	0.75	<5	<5
1,1-Dichloroethene	mg/kg	410	<0.5	<0.5
trans-1,2-Dichloroethene	mg/kg	230	<0.5	<0.5
1,1-Dichloroethane	mg/kg	1700	<0.5	<0.5
cis-1,2-Dichloroethene	mg/kg	150	<0.5	<0.5
1,2-Dichloroethane	mg/kg	0.6	<0.5	<0.5
Trichloroethene	mg/kg	0.11	<0.5	<0.5
1,1,2-Trichloroethane	mg/kg	1.6	<0.5	<0.5
Tetrachloroethene	mg/kg	1.3	<0.5	<0.5
1,1,2,2-Tetrachloroethane	mg/kg	0.93	<0.5	<0.5
Hexachlorobutadiene	mg/kg	22	<0.5	<0.5
Chloroethene	mg/kg			
Trihalomethanes				
Chloroform	mg/kg	12	<0.5	<0.5
Other Chloromethanes				
Carbon Tetrachloride	mg/kg		<0.5	<0.5
Dichloromethane	mg/kg		<5	<5
Chloroethanes				
1,1,1- Trichloroethane	mg/kg		<0.5	<0.5
Chloroethane	mg/kg		<5	<5
Miscellaneous				
Ethene	mg/kg			
Cyclopentane	mg/kg			
Cyclohexane	mg/kg			
Carbon Bisulphide	mg/kg			
1,1,1,2-Tetrachloroethane			<0.5	<0.5
1,1-Dichloropropylene			<0.5	<0.5
1,2,3-Trichloropropane			<0.5	<0.5
1,2-Dibromo-3-chloropropane			<0.5	<0.5
1,3-Dichloropropane			<0.5	<0.5
Bromobenzene			<0.5	<0.5
Bromomethane			<5	<5
cis-1,4-Dichloro-2-butene			<0.5	<0.5
Dibromomethane			<0.5	<0.5
Dichlorodifluoromethane			<5	<5
Hexachlorobutadiene			<0.5	<0.5
Iodomethane			<0.5	<0.5
Pentachloroethane			<0.5	<0.5
trans-1,3-dichloropropene			-	-
trans-1,4-Dichloro-2-butene			<0.5	<0.5
Trichlorofluoromethane			<5	<5
Trihalomethanes				
Bromochloromethane			<0.5	<0.5
Bromodichloromethane			<0.5	<0.5
Bromoform			<0.5	<0.5
Dibromochloromethane			<0.5	<0.5
Fumigants				
1,2-Dibromomethane			<0.5	<0.5
1,2-Dichloropropane			<0.5	<0.5
2,2-Dichloropropane			<0.5	<0.5
cis-1,3-Dichloropropylene			<0.5	<0.5
trans-1,3-Dichloropropylene			<0.5	<0.5
Haloethers				
4-Bromophenyl phenyl ether			<0.5	<0.5
4-Chlorophenyl phenyl ether			<0.5	<0.5
Bis(2-chloroethoxy) methane			<0.5	<0.5
Bis(2-chloroethyl) ether			<0.5	<0.5
Oxygenated Compounds				
2-Butanone (MEK)			<5	<5
2-Hexanone (MBK)			<5	<5
2-Propanone			-	-
4-Methyl-2-pentanone			<5	<5
Vinyl Acetate			<5	<5
1,2,3-Trichlorobenzene			<0.5	<0.5
1,2,4-Trichlorobenzene			<0.5	<0.5
1,2-Dichlorobenzene			<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5
2-Chlorotoluene			<0.5	<0.5
4-Chlorotoluene			<0.5	<0.5
Chlorobenzene			<0.5	<0.5
Chlorinated Hydrocarbons				
1,2-Dichlorobenzene			<0.5	<0.5
1,3-Dichlorobenzene			<0.5	<0.5
1,4-Dichlorobenzene			<0.5	<0.5
Hexachlorobenzene			<1	<1
Hexachlorocyclopentadiene			<2.5	<2.5
Hexachloroethane			<0.5	<0.5
Hexachloropropylene			<0.5	<0.5
Pentachlorobenzene			<0.5	<0.5

Notes:

- = criteria not available
- (mg/kg) = milligrams per kilogram
- < = analyte not detected above laboratory practical quantitation limit (PQL)
- bold/ Box = concentrations above criteria
- Assessment criteria = ANZECC 2000
- 1 = Cr VI trigger value adopted as most conservative
- = not analysed

- * : Results determined using Methanol Extraction Method and are corrected for blank.
- # : Results were derived from 'Neat' analysis but confirmed by dilution (i.e. results similar for t even though originally outside calibration.
- ## : Methanol extraction gave 0 - 30 % recovery and therefore 'Neat' result reported.
- ^ : 'Neat' reported, though outside calibration, (smaller sample size gave larger error becaus

Southlands Soil Data - SVOC
Block 2

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	SS022	SS023	SS024	SS025	SS026	SS027	SS028	SS029	SS030	SS031	SS032	SS033	SS034	SS035	SS036	SS037	SS038	SS039	TP48_1.8-1.9	TP49_0.9-1.0	TP50_1.2-1.3	TP51_1.3-1.4	
				TP1	TP1	TP2	TP2	TP3	TP3	TP4	TP4	TP5	TP5	TP6	TP6	TP7	TP8	TP8	TP9	TP9						
Polynuclear Aromatic Hydrocarbons																										
Naphthalene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
2-Methylnaphthalene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Acenaphthylene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Acenaphthene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Phenanthrene	mg/kg	--	--																			<0.5	0.6	2.3	<0.5	
Anthracene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Fluoranthene	mg/kg	--	--																			<0.5	<0.5	<0.5	0.5	
Pyrene	mg/kg	--	--																			<0.5	<0.5	0.6	0.5	
Benz(a)anthracene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Chrysene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	--																			<1	<1	<1	<1	
Benzo(a)pyrene	mg/kg	5	--																			<0.5	<0.5	<0.5	<0.5	
Indeno(1,2,3-cd)pyrene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Dibenz(a,h)anthracene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
Benzo(g,h,i)perylene	mg/kg	--	--																			<0.5	<0.5	<0.5	<0.5	
TotalPAH	mg/kg	100	--																			nc	0.6	2.9	1	
2-Chloronaphthalene	mg/kg																									
3-Methylcholanthrene	mg/kg																									
7,12-Dimethylbenz(a)anthracene	mg/kg																									
Fluorene	mg/kg																									
N-2-Fluorenyl Acetamide	mg/kg																									
Phthalate Esters																										
Di-n-butylphthalate	mg/kg	--	--																				0.5	<0.5	<0.5	<0.5
Butylbenzylphthalate	mg/kg	--	100.000																			<0.5	<0.5	<0.5	<0.5	
bis(2-ethylhexyl)phthalate	mg/kg	--	120																			<5.0	<5.0	<5.0	<5.0	
Diethyl phthalate	mg/kg																									
Dimethyl phthalate	mg/kg																									
Di-n-octylphthalate	mg/kg																									
Nitrosamines																										
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	--																				<1.0	2.6	8.4	<1.0
Methapyrilene	mg/kg																									
N-Nitrosodibutylamine	mg/kg																									
N-Nitrosodiethylamine	mg/kg																									
N-Nitrosodi-n-propylamine	mg/kg																									
N-Nitrosomethylethylamine	mg/kg																									
N-Nitrosomorpholine	mg/kg																									
N-Nitrosopiperidine	mg/kg																									
N-Nitrosopyrrolidine	mg/kg																									
Nitroaromatics and Ketones																										
Nitrobenzene	mg/kg	--	100																				<0.5	<0.5	<0.5	<0.5
1,3,5-Trinitrobenzene	mg/kg																									
1-Naphthylamine	mg/kg																									
2,4-Dinitrotoluene	mg/kg																									
2,6-Dinitrotoluene	mg/kg																									
2-Picoline	mg/kg																									
4-Aminobiphenyl	mg/kg																									
4-Nitroquinoline-N-oxide	mg/kg																									
5-Nitro-o-toluidine	mg/kg																									
Acetophenone	mg/kg																									
Azobenzene	mg/kg																									
Chlorobenzilate	mg/kg																									
Dimethylaminoazobenzene	mg/kg																									
Isophorone	mg/kg																									
Pentachloronitrobenzene	mg/kg																									
Phenacetin	mg/kg																									
Pronamide	mg/kg																									
Haloethers																										
Bis(2-chloroethyl)ether	mg/kg	--	0.55																				<0.5	<0.5	<0.5	<0.5
Chlorinated Hydrocarbons																										
Hexachloroethane	mg/kg	--	120	0.002	<0.004	<0.005	0.097	<0.002	<0.002	0.007	<0.005	<0.002	0.097	<0.002	<0.003	<0.002	<0.002	0.43	<0.002	0.019		<0.5	<0.5	5.2	<0.5	
Hexachlorobutadiene	mg/kg	--	22	<0.002	<0.004	<0.005	0.19	<0.002	<0.002	<0.003	<0.005	<0.002	0.055	<0.002	<0.003	<0.002	<0.002	0.009	0.95	<0.002	0.11	<0.5	0.7	7.7	<0.5	
Pentachlorobenzene	mg/kg	--	490	<0.002	<0.004	<0.005	<0.005	<0.002	<0.002	<0.003	<0.005	<0.002	<0.003	<0.002	<0.003	<0.002	<0.002	<0.002	0.12	<0.002	0.003	<0.5	<0.5	<0.5	<0.5	
Hexachlorobenzene (HCB)	mg/kg	--	1.1	0.005	<0.004	<0.005	0.11	<0.002	0.003	0.005	<0.005	<0.002	0.053	<0.002	<0.003	<0.002	<0.002	0.013	1.7	<0.002	0.075	<1.0	<1.0	8.8	<1.0	
Anilines and Benzidines																										
Dibenzofuran	mg/kg	--	3100																			<0.5	<0.5	<0.5	<0.5	
Carbazole	mg/kg	--	86																			<0.5	<0.5	<0.5	<0.5	
2-Nitroaniline	mg/kg																									
3,3'-Dichlorobenzidine	mg/kg																									
3-Nitroaniline	mg/kg																									
4-Chloroaniline	mg/kg																									
4-Nitroaniline	mg/kg																									
Aniline	mg/kg																									

Southlands Soil Data - SVOC
Block 2

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	SS022	SS023	SS024	SS025	SS026	SS027	SS028	SS029	SS030	SS031	SS032	SS033	SS034	SS035	SS036	SS037	SS038	SS039	TP48_1.8-1.9 12/04/2005	TP49_0.9-1.0 12/04/2005	TP50_1.2-1.3 12/04/2005	TP51_1.3-1.4 13/04/2005		
				TP1	TP1	TP2	TP2	TP3	TP3	TP4	TP4	TP5	TP5	TP6	TP6	TP7	TP7	TP8	TP8	TP9	TP9						
Miscellaneous Compounds																											
1,3,5-Trichlorobenzene	mg/kg	--	--	<0.002	<0.004	<0.005	0.009	<0.002	<0.002	<0.003	<0.005	<0.002	0.004	<0.002	<0.003	<0.002	<0.002	<0.002	0.59	<0.002	0.003	<0.5	<0.5	1.9	<0.5		
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180																			<0.5	<0.5	0.8	<0.5		
1,3-Dichlorobenzene	mg/kg	--	600	<0.002	<0.004	<0.005	<0.005	<0.002	<0.002	<0.003	<0.005	<0.002	<0.003	<0.002	<0.003	<0.002	<0.002	<0.002	0.028	<0.002	<0.002						
1,4-Dichlorobenzene	mg/kg	--	7.9	0.023	<0.004	<0.005	<0.005	<0.002	<0.002	<0.003	<0.005	<0.002	<0.003	<0.002	<0.003	<0.002	<0.002	<0.002	0.039	<0.002	<0.002						
1,2-Dichlorobenzene	mg/kg	--	600	<0.002	<0.004	<0.005	<0.005	<0.002	<0.002	<0.003	<0.005	<0.002	<0.003	<0.002	<0.003	<0.002	<0.002	<0.002	<0.002	0.016	<0.002	<0.002					
1,2,4-Trichlorobenzene	mg/kg	--	220	<0.002	<0.004	<0.005	<0.005	<0.002	<0.002	<0.003	<0.005	<0.002	<0.003	<0.002	<0.003	<0.002	<0.002	<0.002	0.11	<0.002	<0.002						
Tetrachlorobenzene	mg/kg	--	180	<0.002	<0.004	<0.005	0.006	<0.002	<0.002	<0.003	<0.005	<0.002	<0.003	<0.002	<0.003	<0.002	<0.002	<0.002	<0.005	<0.002	<0.002						
Phenols																											
2-Chlorophenol	mg/kg	--																									
4-Chlorophenol	mg/kg	--																									
2,6-Dichlorophenol	mg/kg	--																									
2,4-Dichlorophenol	mg/kg	--																									
2,4,6-Trichlorophenol	mg/kg	--																									
Pentachlorophenol	mg/kg	--																									
2,3,4,6-Tetrachlorophenol	mg/kg	--																									
2,4,5-Trichlorophenol	mg/kg	--																									
2,4-Dimethylphenol	mg/kg	--																									
2-Methylphenol	mg/kg	--																									
2-Nitrophenol	mg/kg	--																									
3,4-Methylphenol	mg/kg	--																									
4-Chloro-3-Methylphenol	mg/kg	--																									
Phenol	mg/kg	--																									
4,4-DDD	mg/kg	--																									
4,4-DDE	mg/kg	--																									
4,4-DDT	mg/kg	--																									
a-BHC	mg/kg	--																									
Aldrin	mg/kg	--																									
b-BHC	mg/kg	--																									
d-BHC	mg/kg	--																									
Dieldrin	mg/kg	--																									
Endosulfan 1	mg/kg	--																									
Endosulfan 2	mg/kg	--																									
Endosulfan sulfate	mg/kg	--																									
Endrin	mg/kg	--																									
g-BHC	mg/kg	--																									
Heptachlor	mg/kg	--																									
Heptachlor epoxide	mg/kg	--																									
Organophosphorus Pesticides (OP)																											
Chlorfenvinphos	mg/kg	--																									
Chlorpyrifos	mg/kg	--																									
Chlorpyrifos-methyl	mg/kg	--																									
Diazinon	mg/kg	--																									
Dichlorvos	mg/kg	--																									
Dimethoate	mg/kg	--																									
Ethion	mg/kg	--																									
Fenitrothion	mg/kg	--																									
Malathion	mg/kg	--																									
Phosphor-ethyl	mg/kg	--																									
Prothiofos	mg/kg	--																									
Miscellaneous Compounds																											
cis-Isosafrole	mg/kg	--																									
Diallate	mg/kg	--																									
Methanesulfonate ethyl	mg/kg	--																									
Methanesulfonate methyl	mg/kg	--																									
Safrole	mg/kg	--																									
trans-Isosafrole	mg/kg	--																									
Naphthalene																											
Naphthalene	mg/kg	--																									

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

**Southlands Soil Data - SVOC
Block 2**

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP52 1.4-1.5 13/04/2005	TP53 0.7-0.8 13/04/2005	TP54 0.2-0.3 13/04/2005	TP55 0.7-0.8 13/04/2005	TP56 0.4-0.5 13/04/2005	TP58 1.9-2.0 13/04/2005	TP59 1.6-1.7 13/04/2005	TP60 0.3-0.4 13/04/2005	TP61 1.3-1.4 13/04/2005	TP65 1.5-1.6 13/04/2005	TP66 1.5-1.6 13/04/2005	TP67 2.2-2.3 13/04/2005	TP70 2.9-3.0 13/04/2005	TP73 1.8-1.9 12/04/2005
Polynuclear Aromatic Hydrocarbons																	
Naphthalene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	5.2	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Methylnaphthalene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Acenaphthene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Phenanthrene	mg/kg	--	--	0.7	0.7	1.2	<0.5	<0.5	<0.5	<0.5	1.3	0.6	0.8	0.6	0.6	0.5	1.2
Anthracene	mg/kg	--	--	<0.5	<0.5	0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Fluoranthene	mg/kg	--	--	1.3	0.5	3.4	2.1	<0.5	<0.5	0.7	1.4	1	1.2	0.8	<0.5	0.6	2
Pyrene	mg/kg	--	--	1	<0.5	4.1	2.6	<0.5	<0.5	0.7	2	1	1.4	0.8	<0.5	0.6	2.2
Benz(a)anthracene	mg/kg	--	--	0.8	<0.5	2.5	1.8	<0.5	<0.5	<0.5	1.1	0.6	0.8	<0.5	<0.5	<0.5	1.2
Chrysene	mg/kg	--	--	0.6	<0.5	2.5	1.4	<0.5	<0.5	<0.5	1	<0.5	0.6	<0.5	<0.5	<0.5	0.9
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	--	0.6	<1	4	2	<1	<1	<1	<1	<1	1	<1	<1	<1	2
Benzo(a)pyrene	mg/kg	5	--	0.6	<0.5	3.2	1.7	<0.5	<0.5	<0.5	0.7	0.5	0.7	<0.5	<0.5	<0.5	1.1
Indeno(1,2,3-cd)pyrene	mg/kg	--	--	<0.5	<0.5	1.4	0.7	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.6
Dibenz(a,h)anthracene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	<0.5	1.5	0.7	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.6
TotalPAH	mg/kg	100	--	5.6	1.2	24.4	13	nc	5.2	1.4	7.5	3.7	6.5	2.2	0.6	1.7	11.8
2-Chloronaphthalene	mg/kg																
3-Methylcholanthrene	mg/kg																
7,12-Dimethylbenz(a)anthracene	mg/kg																
Fluorene	mg/kg																
N-2-Fluorenyl Acetamide	mg/kg																
Phthalate Esters																	
Di-n-butylphthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Butylbenzylphthalate	mg/kg	--	100,000	<0.5	<0.5	<0.5	<0.5	1.9	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
bis(2-ethylhexyl)phthalate	mg/kg	--	120	<5.0	<5.0	<5.0	<5.0	<5.0	10.4	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Diethyl phthalate	mg/kg																
Dimethyl phthalate	mg/kg																
Di-n-octylphthalate	mg/kg																
Nitrosamines																	
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	--	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	6.4	<1.0	<1.0
Methapyrilene	mg/kg																
N-Nitrosodibutylamine	mg/kg																
N-Nitrosodiethylamine	mg/kg																
N-Nitrosodi-n-propylamine	mg/kg																
N-Nitrosomethylethylamine	mg/kg																
N-Nitrosomorpholine	mg/kg																
N-Nitrosopiperidine	mg/kg																
N-Nitrosopyrrolidine	mg/kg																
Nitroaromatics and Ketones																	
Nitrobenzene	mg/kg	--	100	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3,5-Trinitrobenzene	mg/kg																
1-Naphthylamine	mg/kg																
2,4-Dinitrotoluene	mg/kg																
2,6-Dinitrotoluene	mg/kg																
2-Picoline	mg/kg																
4-Aminobiphenyl	mg/kg																
4-Nitroquinoline-N-oxide	mg/kg																
5-Nitro-o-toluidine	mg/kg																
Acetophenone	mg/kg																
Azobenzene	mg/kg																
Chlorobenzilate	mg/kg																
Dimethylaminoazobenzene	mg/kg																
Isophorone	mg/kg																
Pentachloronitrobenzene	mg/kg																
Phenacetin	mg/kg																
Pronamide	mg/kg																
Haloethers																	
Bis(2-chloroethyl)ether	mg/kg	--	0.55	2.1	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Chlorinated Hydrocarbons																	
Hexachloroethane	mg/kg	--	120	0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	--	22	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.8	<0.5	<0.5
Pentachlorobenzene	mg/kg	--	490	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene (HCB)	mg/kg	--	1.1	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anilines and Benzidines																	
Dibenzofuran	mg/kg	--	3100	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Carbazole	mg/kg	--	86	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2-Nitroaniline	mg/kg																
3,3'-Dichlorobenzidine	mg/kg																
3-Nitroaniline	mg/kg																
4-Chloroaniline	mg/kg																
4-Nitroaniline	mg/kg																
Aniline	mg/kg																

Southlands Soil Data - SVOC
Block 2

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP52_1.4-1.5 13/04/2005	TP53_0.7-0.8 13/04/2005	TP54_0.2-0.3 13/04/2005	TP55_0.7-0.8 13/04/2005	TP56_0.4-0.5 13/04/2005	TP58_1.9-2.0 13/04/2005	TP59_1.6-1.7 13/04/2005	TP60_0.3-0.4 13/04/2005	TP61_1.3-1.4 13/04/2005	TP65_1.5-1.6 13/04/2005	TP66_1.5-1.6 13/04/2005	TP67_2.2-2.3 13/04/2005	TP70_2.9-3.0 13/04/2005	TP73_1.8-1.9 12/04/2005
Miscellaneous Compounds	mg/kg																
1,3,5-Trichlorobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene	mg/kg	--	600														
1,4-Dichlorobenzene	mg/kg	--	7.9														
1,2-Dichlorobenzene	mg/kg	--	600														
1,2,4-Trichlorobenzene	mg/kg	--	220														
Tetrachlorobenzene	mg/kg	--	180														
Phenols	mg/kg																
2-Chlorophenol	mg/kg	--															
4-Chlorophenol	mg/kg	--															
2,6-Dichlorophenol	mg/kg	--															
2,4-Dichlorophenol	mg/kg	--															
2,4,6-Trichlorophenol	mg/kg	--															
Pentachlorophenol	mg/kg	--															
2,3,4,6-Tetrachlorophenol	mg/kg																
2,4,5-Trichlorophenol	mg/kg																
2,4-Dimethylphenol	mg/kg																
2-Methylphenol	mg/kg																
2-Nitrophenol	mg/kg																
3,4-Methylphenol	mg/kg																
4-Chloro-3-Methylphenol	mg/kg																
Phenol	mg/kg																
4,4-DDD	mg/kg																
4,4-DDE	mg/kg																
4,4-DDT	mg/kg																
a-BHC	mg/kg																
Aldrin	mg/kg																
b-BHC	mg/kg																
d-BHC	mg/kg																
Dieldrin	mg/kg																
Endosulfan 1	mg/kg																
Endosulfan 2	mg/kg																
Endosulfan sulfate	mg/kg																
Endrin	mg/kg																
g-BHC	mg/kg																
Heptachlor	mg/kg																
Heptachlor epoxide	mg/kg																
Organophosphorus Pesticides (OP)	mg/kg																
Chlorfenvinphos	mg/kg																
Chlorpyrifos	mg/kg																
Chlorpyrifos-methyl	mg/kg																
Diazinon	mg/kg																
Dichlorvos	mg/kg																
Dimethoate	mg/kg																
Ethion	mg/kg																
Fenitrothion	mg/kg																
Malathion	mg/kg																
Pirimphos-ethyl	mg/kg																
Prothiofos	mg/kg																
Miscellaneous Compounds	mg/kg																
cis-Isosafrole	mg/kg																
Diallate	mg/kg																
Methanesulfonate ethyl	mg/kg																
Methanesulfonate methyl	mg/kg																
Safrole	mg/kg																
trans-Isosafrole	mg/kg																
Naphthalene	mg/kg																
Naphthalene	mg/kg																

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
- = not analysed

Southlands Soil Data - SVOC
Block 2

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP75_0.3-0.4	TP76_2.4-2.5	TP78_0.8-0.9	TP80_1.5-1.6	TP084_1.0	TP084_2.5	TP085_1.2	TP085_2.5	TP086_1.5	TP086_2.20	TP131_1.0	TP131_2.0	TP132_1.1	TP132_1.6
				12/04/2005	12/04/2005	13/04/2005	13/04/2005	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	18/10/2006	3/11/2006	3/11/2006	3/11/2006
Polynuclear Aromatic Hydrocarbons																	
Naphthalene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5	-	<0.5	-	<0.5	<0.5	<0.5	<0.5
2-Methylnaphthalene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Acenaphthylene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5	-	0.9	-	<0.5	<0.5	<0.5	<0.5
Acenaphthene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5	-	<0.5	-	<0.5	<0.5	<0.5	<0.5
Phenanthrene	mg/kg	--	--	<0.5	1.8	2.5	<0.5	0.7	-	<0.5	-	1.7	-	<0.5	<0.5	<0.5	<0.5
Anthracene	mg/kg	--	--	<0.5	<0.5	0.6	<0.5	<0.5	-	<0.5	-	1.1	-	<0.5	<0.5	<0.5	<0.5
Fluoranthene	mg/kg	--	--	0.6	2	1.5	0.5	0.9	-	1	-	5.2	-	<0.5	<0.5	<0.5	<0.5
Pyrene	mg/kg	--	--	0.6	1.9	2	0.6	1	-	1.2	-	6.1	-	<0.5	<0.5	<0.5	<0.5
Benz(a)anthracene	mg/kg	--	--	<0.5	1	0.8	<0.5	<0.5	-	0.7	-	3.6	-	<0.5	<0.5	<0.5	<0.5
Chrysene	mg/kg	--	--	<0.5	0.7	0.8	<0.5	<0.5	-	0.7	-	3.2	-	<0.5	<0.5	<0.5	<0.5
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	--	<1	1	<1	<1	<1	-	1.7	-	7.7	-	<1	<1	<1	<1
Benzo(a)pyrene	mg/kg	5	--	<0.5	0.9	0.6	<0.5	<0.5	-	1.1	-	4	-	<0.5	<0.5	<0.5	<0.5
Indeno(1,2,3-cd)pyrene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	-	0.7	-	2.4	-	<0.5	<0.5	<0.5	<0.5
Dibenz(a,h)anthracene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	<0.5	-	<0.5	-	0.8	-	-	-	-	-
Benzo(g,h,i)perylene	mg/kg	--	--	<0.5	0.6	<0.5	<0.5	<0.5	-	0.8	-	2.8	-	<0.5	<0.5	<0.5	<0.5
TotalPAH	mg/kg	100	--	1.2	9.9	8.8	1.1	0	-	0	-	0	-	ND	ND	ND	ND
2-Chloronaphthalene	mg/kg													<0.5	<0.5	<0.5	<0.5
3-Methylcholanthrene	mg/kg													<0.5	<0.5	<0.5	<0.5
7,12-Dimethylbenz(a)anthracene	mg/kg													<0.5	<0.5	<0.5	<0.5
Fluorene	mg/kg							<0.5	-	<0.5	-	<0.5	-	<0.5	<0.5	<0.5	<0.5
N-2-Fluorenyl Acetamide	mg/kg													<0.5	<0.5	<0.5	<0.5
Phthalate Esters																	
Di-n-butylphthalate	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Butylbenzylphthalate	mg/kg	--	100,000	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
bis(2-ethylhexyl)phthalate	mg/kg	--	120	<5.0	<5.0	<5.0	<5.0	-	-	-	-	-	-	<5	<5	<5	<5
Diethyl phthalate	mg/kg													<0.5	<0.5	<0.5	<0.5
Dimethyl phthalate	mg/kg													<0.5	<0.5	<0.5	<0.5
Di-n-octylphthalate	mg/kg													<0.5	<0.5	<0.5	<0.5
Nitrosamines																	
N-Nitrosodiphenyl&Diphenylamine	mg/kg	--	--	<1.0	<1.0	<1.0	<1.0	-	-	-	-	-	-	<1	<1	<1	<1
Methapyrilene	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosodibutylamine	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosodiethylamine	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosodi-n-propylamine	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosomethylethylamine	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosomorpholine	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosopiperidine	mg/kg													<0.5	<0.5	<0.5	<0.5
N-Nitrosopyrrolidine	mg/kg													<1	<1	<1	<1
Nitroaromatics and Ketones																	
Nitrobenzene	mg/kg	--	100	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
1,3,5-Trinitrobenzene	mg/kg													<0.5	<0.5	<0.5	<0.5
1-Naphthylamine	mg/kg													<0.5	<0.5	<0.5	<0.5
2,4-Dinitrotoluene	mg/kg													<1	<1	<1	<1
2,6-Dinitrotoluene	mg/kg													<1	<1	<1	<1
2-Picoline	mg/kg													<0.5	<0.5	<0.5	<0.5
4-Aminobiphenyl	mg/kg													<0.5	<0.5	<0.5	<0.5
4-Nitroquinoline-N-oxide	mg/kg													<0.5	<0.5	<0.5	<0.5
5-Nitro-o-toluidine	mg/kg													<0.5	<0.5	<0.5	<0.5
Acetophenone	mg/kg													<0.5	<0.5	<0.5	<0.5
Azobenzene	mg/kg													<1	<1	<1	<1
Chlorobenzilate	mg/kg													<0.5	<0.5	<0.5	<0.5
Dimethylaminoazobenzene	mg/kg													<0.5	<0.5	<0.5	<0.5
Isophorone	mg/kg													<0.5	<0.5	<0.5	<0.5
Pentachloronitrobenzene	mg/kg													<0.5	<0.5	<0.5	<0.5
Phenacetin	mg/kg													<0.5	<0.5	<0.5	<0.5
Pronamide	mg/kg													<0.5	<0.5	<0.5	<0.5
Haloethers																	
Bis(2-chloroethyl)ether	mg/kg	--	0.55	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Chlorinated Hydrocarbons																	
Hexachloroethane	mg/kg	--	120	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	--	22	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Pentachlorobenzene	mg/kg	--	490	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Hexachlorobenzene (HCB)	mg/kg	--	1.1	<1.0	<1.0	<1.0	<1.0	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Anilines and Benzidines																	
Dibenzofuran	mg/kg	--	3100	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Carbazole	mg/kg	--	86	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2-Nitroaniline	mg/kg													<1	<1	<1	<1
3,3'-Dichlorobenzidine	mg/kg													<0.5	<0.5	<0.5	<0.5
3-Nitroaniline	mg/kg													<1	<1	<1	<1
4-Chloroaniline	mg/kg													<0.5	<0.5	<0.5	<0.5
4-Nitroaniline	mg/kg													<0.5	<0.5	<0.5	<0.5
Aniline	mg/kg													<0.5	<0.5	<0.5	<0.5

Southlands Soil Data - SVOC
Block 2

Analyte	Units	NEPM-F	USEPA Region IX (Industrial Land)	TP75_0.3-0.4 12/04/2005	TP76_2.4-2.5 12/04/2005	TP78_0.8-0.9 13/04/2005	TP80_1.5-1.6 13/04/2005	TP084_1.0 0 18/10/2006	TP084_2.5 0 18/10/2006	TP085_1.2 0 18/10/2006	TP085_2.5 0 18/10/2006	TP086_1.5 0 18/10/2006	TP086_2.20 0 18/10/2006	TP131_1.0 0 3/11/2006	TP131_2.0 0 3/11/2006	TP132_1.1 0 3/11/2006	TP132_1.6 0 3/11/2006
Miscellaneous Compounds	mg/kg																
1,3,5-Trichlorobenzene	mg/kg	--	--	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
1,2,4,5-Tetrachlorobenzene	mg/kg	--	180	<0.5	<0.5	<0.5	<0.5	-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
1,3-Dichlorobenzene	mg/kg	--	600														
1,4-Dichlorobenzene	mg/kg	--	7.9														
1,2-Dichlorobenzene	mg/kg	--	600														
1,2,4-Trichlorobenzene	mg/kg	--	220														
Tetrachlorobenzene	mg/kg	--	180														
Phenols	mg/kg																
2-Chlorophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
4-Chlorophenol	mg/kg	--															
2,6-Dichlorophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2,4-Dichlorophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2,4,6-Trichlorophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Pentachlorophenol	mg/kg	--						-	-	-	-	-	-	<1	<1	<1	<1
2,3,4,6-Tetrachlorophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2,4,5-Trichlorophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2,4-Dimethylphenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2-Methylphenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
2-Nitrophenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
3,4-Methylphenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
4-Chloro-3-Methylphenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Phenol	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
4,4-DDD	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
4,4-DDE	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
4,4-DDT	mg/kg	--						-	-	-	-	-	-	<1	<1	<1	<1
a-BHC	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Aldrin	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
b-BHC	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
d-BHC	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Dieldrin	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Endosulfan 1	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Endosulfan 2	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Endosulfan sulfate	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Endrin	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
g-BHC	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Heptachlor	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Heptachlor epoxide	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Organophosphorus Pesticides (OP)	mg/kg																
Chlorfenvinphos	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Chlorpyrifos	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Chlorpyrifos-methyl	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Diazinon	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Dichlorvos	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Dimethoate	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Ethion	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Fenitrothion	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Malathion	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Phosphor-ethyl	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Prothiofos	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Miscellaneous Compounds	mg/kg																
cis-Isosafrole	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Diallate	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Methanesulfonate ethyl	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Methanesulfonate methyl	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Safrole	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
trans-Isosafrole	mg/kg	--						-	-	-	-	-	-	<0.5	<0.5	<0.5	<0.5
Naphthalene	mg/kg																
Naphthalene	mg/kg	--						-	-	-	-	-	-	<5	<5	<5	<5

Notes:
 -- = criteria not available
 (mg/kg) = milligrams per kilogram
 < = analyte not detected above laboratory practical quantitation limit (PQL)
 - = not analysed

Stockpile Sample Results- Metals
Orca Southlands Remediation and Development Project

Analyte	Units	NEPM-F	Waste Classification Guidelines			SP1	SP2	SP3	SP4	SP5	SP6	SP7	SP8	SP9	SP10	SP11	SP12
			Inert	Solid	Industrial	7/04/2005	7/04/2005	7/04/2005	7/04/2005	7/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	11/04/2005	11/04/2005
Arsenic	mg/kg	500	10	100	400	6	7	9	<5	<5	8	<5	15	<5	6	7	<5
Cadmium	mg/kg	100	2	20	80	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Chromium	mg/kg	60	10	100	400	18	22	17	12	10	16	6	19	17	15	13	18
Copper	mg/kg	5000				53	64	86	47	33	87	10	112	41	89	57	86
Lead	mg/kg	1500	10	100	400	159	162	271	130	51	332	10	430	26	103	151	148
Nickel	mg/kg	3000	4	40	160	27	26	48	12	13	22	7	26	46	12	13	21
Zinc	mg/kg	35000				206	243	289	197	100	686	25	656	137	286	597	552
Mercury	mg/kg	75	0.4	4	16	37.5	52	35.6	45.6	15.2	1.5	<0.1	<0.1	0.3	46.7	28.6	1

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (POL)
Bold/ Box = concentrations above criteria
Assessment criteria = Waste Classification Guidelines (DEC)
1 = Cr VI trigger value adopted as most conservative
- = not analysed

Stockpile Sample Results- Metals
Orica Southlands Remediation and Development Project

Analyte	Units	NEPM-F	Waste Classification Guidelines			SP13	SP14	SP15	SP16	SP17	SP18	SP19	SP20	SP21	SP22	SP23	SP24
			Inert	Solid	Industrial	11/04/2005	11/04/2005	11/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	7/04/2005	7/04/2005	7/04/2005
Arsenic	mg/kg	500	10	100	400	<5	<5	9	7	<5	<5	<5	<5	<5	<5	<5	5
Cadmium	mg/kg	100	2	20	80	<1	<1	1	<1	<1	<1	1	<1	<1	<1	<1	<1
Chromium	mg/kg	60	10	100	400	20	3	23	15	8	6	11	13	10	8	6	9
Copper	mg/kg	5000				67	22	156	27	16	13	34	39	14	36	28	29
Lead	mg/kg	1500	10	100	400	171	22	553	70	52	36	108	135	30	72	56	95
Nickel	mg/kg	3000	4	40	160	12	7	11	7	5	4	11	9	15	22	14	20
Zinc	mg/kg	35000				836	91	1420	125	58	71	254	188	75	207	172	121
Mercury	mg/kg	75	0.4	4	16	4.4	3.2	1	5.5	0.4	0.2	0.1	0.6	0.4	2	0.7	0.7

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (POL)
Bold/ Box = concentrations above criteria
Assessment criteria = Waste Classification Guidelines (DEC)
1 = Cr VI trigger value adopted as most conservative
- = not analysed

Stockpile Sample Results- Metals
Orca Southlands Remediation and Development Project

Analyte	Units	NEPM-F	Waste Classification Guidelines			SP25	SP26	SP27	SP28	SP29	SP30	SP31	SP32	SP33	SP34	SP35	SP36
			Inert	Solid	Industrial	7/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005
Arsenic	mg/kg	500	10	100	400	<5	<5	5	<5	<5	<5	5	6	11	5	<5	
Cadmium	mg/kg	100	2	20	80	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	
Chromium	mg/kg	60	10	100	400	7	55	21	16	8	4	9	9	78	7	9	
Copper	mg/kg	5000				12	86	76	22	28	19	16	34	50	51	40	32
Lead	mg/kg	1500	10	100	400	60	130	109	60	17	33	65	135	179	126	205	142
Nickel	mg/kg	3000	4	40	160	5	27	36	4	37	12	6	10	6	15	8	6
Zinc	mg/kg	35000				55	172	214	76	53	84	50	283	209	299	189	279
Mercury	mg/kg	75	0.4	4	16	0.3	18.3	47.7	1.2	3	66.2	0.2	0.7	4.7	25.2	0.3	0.3

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (POL)
Bold/ Box = concentrations above criteria
Assessment criteria = Waste Classification Guidelines (DEC)
1 = Cr VI trigger value adopted as most conservative
- = not analysed

Stockpile Sample Results- Metals
Orca Southlands Remediation and Development Project

Analyte	Units	NEPM-F	Waste Classification Guidelines			SP37	SP38	SP39	SP40	SP41	SP42	SP43	SP44	SP45	SP46	SP47	SP48
			Inert	Solid	Industrial	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005	13/04/2005
Arsenic	mg/kg	500	10	100	400	<5	14	16	6	<5	7	10	10	5	6	6	12
Cadmium	mg/kg	100	2	20	80	<1	<1	<1	2	<1	<1	1	<1	<1	<1	<1	2
Chromium	mg/kg	60	10	100	400	6	11	35	40	<2	50	39	26	9	12	10	84
Copper	mg/kg	5000				27	34	36	133	<5	132	78	74	81	37	26	138
Lead	mg/kg	1500	10	100	400	211	124	126	1400	78	603	172	350	114	78	51	532
Nickel	mg/kg	3000	4	40	160	6	8	5	18	<2	21	15	13	11	6	4	19
Zinc	mg/kg	35000				239	178	102	549	46	2960	202	494	137	109	82	559
Mercury	mg/kg	75	0.4	4	16	0.2	1.4	11.8	0.6	<0.1	0.7	1	0.5	1.2	20.2	18.6	0.7

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (POL)
Bold/ Box = concentrations above criteria
Assessment criteria = Waste Classification Guidelines (DEC)
1 = Cr VI trigger value adopted as most conservative
- = not analysed

Stockpile Sample Results- Metals
Orica Southlands Remediation and Development Project

Analyte	Units	NEPM-F	Waste Classification Guidelines			SP49	SP50
			Inert	Solid	Industrial	13/04/2005	13/04/2005
Arsenic	mg/kg	500	10	100	400	7	<5
Cadmium	mg/kg	100	2	20	80	1	<1
Chromium	mg/kg	60	10	100	400	114	28
Copper	mg/kg	5000				288	186
Lead	mg/kg	1500	10	100	400	774	2220
Nickel	mg/kg	3000	4	40	160	39	28
Zinc	mg/kg	35000				632	321
Mercury	mg/kg	75	0.4	4	16	0.5	0.4

Notes:

-- = criteria not available

(mg/kg) = milligrams per kilogram

< = analyte not detected above laboratory practical quantitation limit (PQL)

Bold/ Box = concentrations above criteria

Assessment criteria – Waste Classification Guidelines (DEC)

1 = Cr VI trigger value adopted as most conservative

- = not analysed

Stockpile Sample Results- TPH and VOC
 Orica Southlands Remediation and Development Project

Analyte	Units	USEPA Region IX (Industrial Land) mg/kg	NSW EPA 1994	Waste Classification Guidelines			SP2	SP5	SP22	SP27	SP28	SP29	SP30	SP34
				Inert (SCC)	Solid (SCC)	Industrial (SCC)	7/04/2005	7/04/2005	7/04/2005	8/04/2005	8/04/2005	8/04/2005	8/04/2005	13/04/2005
Total Petroleum Hydrocarbons														
C6-C9 Fraction	mg/kg	--	65	650	650	2600	-	-	-	-	-	-	-	<2
C10-C14 Fraction	mg/kg	--	1000				-	-	-	-	-	-	-	<50
C15-C28 Fraction	mg/kg	--	1000				-	-	-	-	-	-	-	<100
C29-C36 Fraction	mg/kg	--	1000				-	-	-	-	-	-	-	140
Sum C10-C36	mg/kg	--	1000	5000	10000	40000								140
Monocyclic Aromatic Hydrocarbons														
Benzene	mg/kg	--	1	18	18	72	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Toluene	mg/kg	--	1.4	518	518	2073	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethylbenzene	mg/kg	--	3.1	1080	1080	4320	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
meta- & para- Xylene	mg/kg	--	14				0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
ortho- Xylene	mg/kg	--	--				<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Total Xylene	mg/kg	--	14	1800	1800	7200								
Halogenated Aliphatic Compounds														
trans-1,2-Dichloroethene	mg/kg	230	--				<0.5	<0.5	1.1	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,2-Dichloroethene	mg/kg	150	--				<0.5	<0.5	6.2	<0.5	<0.5	<0.5	<0.5	<0.5
Trichloroethene	mg/kg	0.11	--				<0.5	<0.5	<0.5	<0.5	0.7	<0.5	0.5	<0.5
1,1,2-Trichloroethane	mg/kg	1.6	--				<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	2	<0.5
Tetrachloroethene	mg/kg	1.3	--				0.8	0.8	<0.5	1	0.6	0.6	1.1	<0.5
Hexachlorobutadiene	mg/kg	22	--				<0.5	<0.5	<0.5	0.6	0.6	10.2	<0.5	<0.5

Notes:

- = criteria not available
- (mg/kg) = milligrams per kilogram
- < = analyte not detected above laboratory practical quantitation limit (PQL)
- Bold/ Box = concentrations above criteria
- Assessment criteria = USEPA, Waste Classification Guidelines (DEC), NSW EPA 1994
- 1 = Cr VI trigger value adopted as most conservative
- = not analysed

Stockpile Sample Results- SVOC
Orica Southlands Remediation and Development Projec

Analyte grouping / Analyte	Units	NEPMA-F mg/kg	USEPA Region IX (Industrial Land) mg/kg	Waste Classification Guidelines			SP15 11/04/2005	SP20 13/04/2005	SP27 8/04/2005	SP33 13/04/2005	SP34 13/04/2005	SP36 13/04/2005	SP38 13/04/2005	SP40 13/04/2005	SP46 13/04/2005	SP48 13/04/2005	SP49 13/04/2005
				Inert	Solid	Industrial											
Polynuclear Aromatic Hydrocarbons																	
Acenaphthylene	mg/kg	--	--				<0.5	<0.5	<0.5	<0.5	0.7	<0.5	<0.5	1.6	<0.5	<0.5	0.7
Phenanthrene	mg/kg	--	--				0.6	<0.5	<0.5	0.7	0.6	0.6	<0.5	1.5	0.6	<0.5	<0.5
Anthracene	mg/kg	--	--				<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.5	<0.5	<0.5	<0.5
Fluoranthene	mg/kg	--	--				0.7	0.7	<0.5	1	0.9	1.8	<0.5	3	1.6	0.8	0.9
Pyrene	mg/kg	--	--				0.7	0.7	<0.5	1	1	2	0.5	3.1	1.8	0.8	0.9
Benzo(a)anthracene	mg/kg	--	--				<0.5	<0.5	<0.5	0.6	0.6	1	<0.5	1.8	0.9	0.5	0.5
Chrysene	mg/kg	--	--				<0.5	<0.5	<0.5	<0.5	0.5	1.1	<0.5	1.6	0.9	<0.5	<0.5
Benzo(b) & Benzo(k)fluoranthene	mg/kg	--	--				<1	<1	<1	<1	<1	2	<1	2	1	<1	<1
Benzo(a)pyrene	mg/kg	5	--	0.08	0.8	3.2	<0.5	<0.5	<0.5	0.6	0.5	1.1	<0.5	1.1	0.3	<0.5	<0.5
Indeno(1,2,3-cd)pyrene	mg/kg	--	--				<0.5	<0.5	<0.5	<0.5	0.6	<0.5	0.5	<0.5	<0.5	<0.5	<0.5
Benzo(g,h,i)perylene	mg/kg	--	--				<0.5	<0.5	<0.5	<0.5	0.6	<0.5	0.6	<0.5	<0.5	<0.5	<0.5
Total PAH	mg/kg	100	--	200*	200*	800*	2	1.4	3.9	4.8	10.8	0.5	17.3	7.6	2.1	3	
Phthalate Esters																	
Butylbenzylphthalate	mg/kg	--	100,000				<0.5	2.3	<0.5	0.9	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.8
Chlorinated Hydrocarbons																	
Hexachloroethane	mg/kg	--	120				<0.5	<0.5	<0.5	<0.5	0.6	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Hexachlorobutadiene	mg/kg	--	22				<0.5	<0.5	0.9	<0.5	1.9	<0.5	<0.5	<0.5	0.6	<0.5	<0.5
Hexachlorobenzene (HCB)	mg/kg	--	1.1				<1.0	<1.0	<1.0	<1.0	1.4	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

Notes:
-- = criteria not available
(mg/kg) = milligrams per kilogram
< = analyte not detected above laboratory practical quantitation limit (PQL)
Bold Box = concentrations above criteria
Assessment criteria = USEPA, Waste Classification Guidelines (DEC), NSW EPA 1994
1 = Cr VI trigger value adopted as most conservative
- = not analysed
SCC = Total Concentration
* = SCC values

Appendix A

Soil and Groundwater Data

A.4 Shallow Groundwater Data and Figures

Summary of Groundwater Data - Assessment of Shallow Groundwater Block 1 Southlands

Location	Sample Depth	Date	Analyte	Carbon Tetrachloride	Chloroform	Methylene chloride	1,1,2-Tetrachloroethane	1,1,1-Trichloroethane	1,1,2-Trichloroethane	1,1-Dichloroethane	1,2-Dichloroethane	Chloroethane	Tetrachloroethane	Trichloroethane	1,1-Dichloroethane	cis-1,2-Dichloroethane	trans-1,2-Dichloroethane	Vinyl chloride	Hexachlorobutadiene	1,2-Dichlorobenzene	1,3-Dichlorobenzene	1,4-Dichlorobenzene	Hexachlorobutadiene SV	Hexachloroethane	1,3,5-Trichlorobenzene	
				LOR	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.01	0.001	0.001	0.001	0.001	0.001	0.01	0.001	0.002	0.002	0.002	0.001	0.002
				Units	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	
BP104	2	BP104_2	8/11/2005		0.022	0.019	0.005	0.001	0.001	0.002	0.021	0.037	0.01	0.006	0.049	0.001	0.055	0.003	0.09		0.002	0.002	0.002	0.002	0.002	0.002
BP106	2	BP106_2	9/11/2005		0.02	0.044	0.1	0.02	0.354	0.02	0.757	0.31	0.2	0.02	0.058	0.27	26	6.27	34		0.002	0.002	0.002	0.002	0.002	0.002
BP95	3	BP95_3	27/09/2007		1.24	0.792	0.025	0.168	0.005	0.049	0.031	0.041	0.05	0.025	5.62	0.012	0.936	0.083	3.98		0.005	0.002	0.002	0.002	0.003	0.002
BP103	3	BP103_3	8/11/2005		0.001	0.011	0.005	0.001	0.001	0.001	0.002	0.111	0.01	0.001	0.023	0.001	0.006	0.001	0.02		0.002	0.002	0.002	0.002	0.002	0.002
BP105	3	BP105_3	9/11/2005		0.029	0.001	0.005	0.001	0.001	0.001	0.002	0.002	0.01	0.004	0.013	0.001	0.008	0.005	0.01		0.002	0.002	0.002	0.002	0.002	0.02
BP45	4	BP45_4	4/09/2007		0.005	0.028	0.025	0.005	0.005	0.005	0.014	2.97	0.05	0.012	0.086	0.01	0.021	0.005	0.46		0.005	0.002	0.002	0.002	0.002	0.002
BP46	4	BP46_4	4/09/2007		0.02	0.375	0.1	0.02	0.02	0.101	0.058	11.1	0.2	1.06	1.8	0.028	6.3	0.171	13.2		0.02	0.002	0.002	0.002	0.012	0.002
BP47	4	BP47_4	4/09/2007		0.001	0.001	0.005	0.001	0.001	0.001	0.001	0.001	0.01	0.001	0.001	0.001	0.001	0.01	0.001		0.002	0.002	0.002	0.002	0.002	0.002
BP48	4	BP48_4	4/09/2007		47.1	27	0.25	0.75	0.05	1.75	0.097	3.04	<0.5	71.1	7.4	0.082	2.19	0.078	18.5		0.05	0.002	0.002	0.003	0.003	0.154
BP49	4	BP49_4	4/09/2007		72	30.7	0.25	1.2	0.05	2.97	0.125	2.8	<0.5	66	17.1	0.076	12.2	0.432	19.7		0.05	0.004	0.002	0.006	0.048	0.24
BP104	4	BP104_4	8/11/2005		1.58	6.95	1	0.181	0.02	2.82	0.18	26.8	0.2	0.222	8.24	0.105	14.3	0.273	6.76		0.002	0.002	0.002	0.05	0.003	0.002
BP106	4	BP106_4	9/11/2005		0.005	0.018	0.025	0.005	0.075	0.005	0.664	0.202	0.05	0.006	0.011	0.026	2.62	1.7	20.5		0.002	0.002	0.002	0.002	0.002	0.002
BP103	5	BP103_5	8/11/2005		0.05	3.38	0.25	0.332	0.05	0.718	0.316	92.3	<0.5	1.16	4.77	0.326	6.61	0.093	8.62		0.002	0.002	0.004	0.009	0.002	0.002
BP105	5	BP105_5	9/11/2005		66.3	9.82	0.25	0.73	0.059	2.1	0.056	1.29	<0.5	33.2	10.6	0.05	1.56	0.08	0.62		0.002	0.002	0.002	0.508	0.617	0.002
BP03	6	BP03_6	4/09/2007		0.2	3.36	<1	0.2	0.2	0.361	0.619	212	<2	3.3	4.98	0.586	2.32	0.2	62.9		0.004	0.008	0.003	0.002	0.002	0.002
BP06	6	BP06_6	18/09/2007		0.02	0.219	0.1	0.02	0.02	0.02	0.283	18.6	0.2	0.02	0.921	0.199	0.308	0.055	11		0.02	-	-	-	-	-
BP23	6	BP23_6	19/09/2007		0.806	18.5	0.691	0.368	0.02	5.03	0.2	2.13	0.2	3.73	12.2	0.149	12	0.215	35.1		0.02	0.002	0.002	0.002	0.006	0.011
BP50	6	BP50_6	19/09/2007		0.001	0.004	0.005	0.001	0.001	0.001	0.001	0.003	0.01	0.001	0.001	0.001	0.06	0.004	0.08		0.002	0.002	0.002	0.002	0.002	0.002
BP51	6	BP51_6	19/09/2007		0.001	0.001	0.005	0.001	0.001	0.007	0.008	0.001	0.01	0.001	0.001	0.001	0.003	0.003	0.08		0.001	0.002	0.002	0.002	0.002	0.002
BP95	6	BP95_6	27/09/2007		43.4	22.6	0.146	0.923	0.064	2.87	0.207	1.35	0.2	35.6	12	0.052	6.36	0.248	12.9		0.02	0.002	0.002	0.002	0.004	0.109
BP104	6	BP104_6	8/11/2005		155	10.1	0.25	0.172	0.05	0.842	0.184	63.1	<0.5	61.7	1.55	1.09	0.3	0.054	4.4		0.002	0.002	0.002	0.21	0.362	0.002
BP106	6	BP106_6	9/11/2005		0.02	0.277	0.1	1.66	0.62	0.061	0.357	0.134	0.2	0.041	0.302	0.63	27.8	3.17	27.2		0.002	0.002	0.002	0.002	0.002	0.002
BP103	7	BP103_7	8/11/2005		0.005	1.8	0.025	0.035	0.005	0.097	0.148	2.24	0.05	0.026	1.69	0.124	0.476	0.063	1.36		0.002	0.002	0.002	0.028	0.002	0.002
BP105	7	BP105_7	9/11/2005		120	13.8	0.25	1.01	0.11	3.74	0.073	0.636	<0.5	83.4	19.4	0.05	2.15	0.167	0.74		0.005	0.003	0.01	0.085	0.822	0.002
BP02	8	BP02_8	3/09/2007		0.2	1.46	<1	0.2	0.2	0.461	0.901	585	<2	0.638	10.5	0.921	3.39	0.232	112		0.2	0.004	0.006	0.006	0.002	0.002
BP45	8	BP45_8	4/09/2007		0.005	0.468	0.025	0.048	0.005	0.08	0.058	3.38	0.05	0.06	0.4	0.058	4.32	0.065	4.01		0.07	0.002	0.003	0.002	0.075	0.002
BP46	8	BP46_8	4/09/2007		25.4	11.5	0.25	0.225	0.05	1.04	0.237	136	<0.5	54.3	3.91	0.304	1.09	0.118	44.7		0.226	0.002	0.004	0.002	0.338	0.075
BP47	8	BP47_8	4/09/2007		0.96	0.94	0.025	0.028	0.005	0.078	0.013	0.735	0.05	5.72	0.61	0.009	0.202	0.006	1.37		0.087	0.002	0.002	0.002	0.152	0.012
BP48	8	BP48_8	4/09/2007		113	33.1	0.25	2.32	0.05	6.58	0.193	3.97	<0.5	62.5	6.28	0.316	1.22	0.104	9.34		0.076	0.002	0.002	0.004	0.074	0.768
BP50	8	BP50_8	19/09/2007		0.005	0.691	0.027	0.317	0.012	0.292	0.056	0.211	0.05	0.223	0.352	0.041	7.2	0.384	4.45		0.045	0.002	0.002	0.002	0.049	0.002
BP104	8	BP104_8	8/11/2005		134	5.14	<1	0.2	0.2	0.338	0.376	451	<2	80.4	4.73	0.832	0.591	0.2	15.8		0.002	0.002	0.003	0.065	0.132	0.002
BP106	8	BP106_8	9/11/2005		0.02	1.88	0.1	1.28	0.02	5.34	0.034	0.39	0.2	11.4	1.92	0.02	2.62	0.059	0.94		0.002	0.002	0.004	0.191	0.002	0.002
WG68	I	WG68_I	19/09/2007		0.001	0.001	0.005	0.001	0.001	0.001	0.005	0.008	0.01	0.016	0.005	0.001	0.083	0.24	0.99		0.128	0.021	0.013	0.042	0.233	0.002
MWB03	I	MWB03_I	3/08/2007		0.005	0.006		0.005	0.005	0.005	0.009	0.01	0.05	0.005	0.005	0.005	0.365	0.064	1.49		0.005	0.005	0.005	0.005		
MWB10	I	MWB10_I	3/08/2007		0.1	0.381		1.25	0.1	0.192	0.1	0.157	<1	0.73	2.89	0.1	5.9	0.717	<1		0.1	0.1	0.1	0.1		
WG224	I	WG224_I	3/08/2007		83.8	20.9		1.63	<0.5	5.56	<0.5	2.72	<5	55	11.6	<0.5	5.83	<0.5	8.41		<0.5	<0.5	<0.5	<0.5		
WG225	I	WG225_I	3/08/2007		3.72	3.83		0.144	0.02	0.296	0.034	1.31	0.2	13.8	1.77	0.035	0.543	0.028	1.71		0.023	0.02	0.02	0.02		
WG226	I	WG226_I	3/08/2007		0.01	0.182		0.022	0.012	0.03	0.114	1.63	0.1	0.234	0.436	0.082	1.87	0.114	7.16		0.01	0.01	0.01	0.01		
MWB01	S	MWB01_S	18/09/2007		1.93	5.12	0.1	1.01	0.02	0.57	0.15	0.775	0.2	12.9	7.44	0.124	12.8	0.93	15.4		0.074	-	-	-	-	-
MWB02	S	MWB02_S	17/09/2007		0.001	0.001	0.005	0.001	0.001	0.001	0.001	0.001	0.01	0.001	0.001	0.001	0.001	0.001	0.01		0.001	-	-	-	-	-
MWB03	S	MWB03_S	17/09/2007		0.006	0.122	0.086	0.005	0.005	0.007	0.05	0.1	0.05	0.012	0.011	0.023	4.11	0.244	2.72		0.015	-	-	-	-	-
MWB04	S	MWB04_S	11/09/2007		37	7.33	0.276	0.968	0.02	1.68	0.072	1.28	0.2	16.7	4.54	0.168	14.7	0.202	4.62		0.02	-	-	-	-	-
MWB10	S	MWB10_S	14/09/2007		0.02	1.93	0.1	0.18	0.02	0.222	0.085	0.297	0.2	1.18	1.17	0.08	11.7	0.599	5.44		0.053	-	-	-	-	-
MWB12	S	MWB12_S	20/09/2007		0.001	0.001	0.005	0.001	0.001	0.001	0.001	0.001	0.01	0.005	0.001	0.001	0.001	0.001	0.01		0.001	-	-	-	-	-
WG41	S	WG41_S	19/09/2007		0.001	0.003	0.005	0.001	0.001	0.006	0.008	0.013	0.01	0.02	0.021	0.002	0.209	0.266	0.84		0.016	0.022	0.015	0.032	0.026	0.002
MWB03	S	MWB03_S	3/08/2007		0.1	0.228		0.1	0.1	0.1	0.138	<1	0.1	0.113	0.1	5.72	0.514	3.94		0.1	0.1	0.1	0.1			
MWB10	S	MWB10_S	3/08/2007		0.2	1.1		0.2	0.2	0.2	0.2	0.234	<2	0.503	0.6	0.2	9.46	0.575	4.45		0.2	0.2	0.2	0.2		



Legend:

- WG41S** Monitoring Well
- 585** Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW | Approved: DRAFT | Date: 17/01/2008

Job No: **43217741** | File: 43217741.001wor

Client
ORICA AUSTRALIA PTY LTD

Project
REVIEW OF SHALLOW GROUNDWATER CHLORINATED HYDROCARBON DATA BLOCK 1 SOUTHLANDS

Title
Relevant Chemical Monitoring Locations

Figure: **1.1**

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Legend:

WG41S Monitoring Well

585 Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW Approved: DRAFT Date: 17/01/2008

Job No: 43217741 File: 43217741.001wor

Client
ORICA AUSTRALIA PTY LTD

Project
REVIEW OF SHALLOW GROUNDWATER CHLORINATED HYDROCARBON DATA BLOCK 1 SOUTHLANDS

Title
1,2-Dichloroethane

Figure: 1.2



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Legend:

WG41S Monitoring Well

585 Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW Approved: DRAFT Date: 17/01/2008

Job No: **43217741** File: 43217741.001wor

Client
ORICA AUSTRALIA PTY LTD

Project
**REVIEW OF SHALLOW GROUNDWATER
CHLORINATED HYDROCARBON DATA
BLOCK 1 SOUTHLANDS**

Title
Vinyl Chloride

Figure: **1.3**



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Legend:

- WG41S** Monitoring Well
- 585** Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW | Approved: DRAFT | Date: 17/01/2008

Job No: **43217741** | File: 43217741.001wor

Client

ORICA AUSTRALIA PTY LTD

Project

**REVIEW OF SHALLOW GROUNDWATER
CHLORINATED HYDROCARBON DATA
BLOCK 1 SOUTHLANDS**

Title

Tetrachloroethene

Figure: **1.4**

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Legend:
WG41S Monitoring Well
585 Concentration (mg/L)
 (Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
 Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW Approved: DRAFT Date: 17/01/2008

Job No: 43217741 File: 43217741.001wor

Client
 ORICA AUSTRALIA PTY LTD

Project
 REVIEW OF SHALLOW GROUNDWATER
 CHLORINATED HYDROCARBON DATA
 BLOCK 1 SOUTHLANDS

Title
 Trichloroethene

Figure: 1.5





Legend:

- WG41S** Monitoring Well
- 585** Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW	Approved: DRAFT	Date: 17/01/2008
Job No: 43217741	File: 43217741.001wor	
Client ORICA AUSTRALIA PTY LTD		
Project REVIEW OF SHALLOW GROUNDWATER CHLORINATED HYDROCARBON DATA BLOCK 1 SOUTHLANDS		
Title Carbon Tetrachloride		
Figure: 1.6		

URS

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Legend:

- WG41S** Monitoring Well
- 585** Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW | Approved: DRAFT | Date: 17/01/2008

Job No: **43217741** | File: 43217741.001wor

Client: **ORICA AUSTRALIA PTY LTD**

Project: **REVIEW OF SHALLOW GROUNDWATER CHLORINATED HYDROCARBON DATA BLOCK 1 SOUTHLANDS**

Title: **Chloroform**

Figure: **1.7**

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Legend:

WG41S Monitoring Well

585 Concentration (mg/L)
(Maximum concentration in groundwater 1- 8 m bgl)

Source: Aerial Image from Ausimage SKM 2005
Datum: GDA94, Projection: UTM, Grid: MGA Zone 55

Drawn: AZW Approved: DRAFT Date: 17/01/2008

Job No: 43217741 File: 43217741.001wor

Client
ORICA AUSTRALIA PTY LTD

Project
REVIEW OF SHALLOW GROUNDWATER
CHLORINATED HYDROCARBON DATA
BLOCK 1 SOUTHLANDS

Title
cis-1,2-Dichloroethene

Figure: 1.8



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Appendix B

Toxicity Summaries

Chromium

General

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms. The most common forms are chromium(0), trivalent (or chromium(III)), and hexavalent (or chromium(VI)). Chromium(III) occurs naturally in the environment and is an essential nutrient required by the human body to promote the action of insulin in body tissues so that sugar, protein, and fat can be used by the body. Chromium(VI) and chromium(0) are generally produced by industrial processes. No known taste or odour is associated with chromium compounds.

Chromium compounds show a wide range of solubilities with chromium(III) salts essentially insoluble (with the exception of chromium(III) chloride and nitrate that are soluble) and chromium(VI) soluble (with the exception of zinc and lead chromates). Hexavalent compounds are reduced to the trivalent form in the presence of oxidisable organic matter. This anaerobic decomposition may increase the mobility of chromium by forming more soluble complexes. The mobility on soil depends on the sorption characteristics of the soil with most chromium in soil strongly adsorbed onto soil particles (UK EA, 2002).

The fraction of chromium present in the hexavalent form is greater in industrial environments compared with ambient environments (UK, EA, 2002). Hexavalent and trivalent states are in dynamic equilibrium, with the degree of oxidation depending on various factors; for example in soil - moisture content, pH and the presence of reducing and oxidising agents. However, in most circumstances, chromium(VI) tends to be converted to chromium(III) when in contact with the natural environment, and for all practical purposes the oxidation of chromium(III) to chromium(VI) never occurs in biological systems. Chromium in foodstuffs is generally considered to be in the trivalent form.

Exposure and Health Effects

The following has been summarised from UK EA (2002) and WHO (2006):

Exposure to chromium may occur through the ingestion of food products containing chromium, inhalation of chromium particulates in air, ingestion of contaminated water or soil. Dermal absorption of chromium, particularly from soil, is not a significant route of exposure. In general, food appears to be the major source of intake. The toxicity of chromium depends on its oxidation state with chromium(VI) more toxic than chromium(III). Chromium(VI) compounds penetrate biological membranes much more readily than do chromium(III) compounds. After crossing cellular membranes, chromium(VI) may be reduced to chromium(III) via a number of reactions with a number of intermediates (pentavalent and tetravalent species) that may interact with essential constituents of the cells (including genetic material), which they can damage through oxidation and complexation with the resulting chromium species. As well as the inherently greater toxicity of hexavalent compared with trivalent chromium, the former is the more readily absorbed by both the inhalation and oral routes.

Chromium(III) is an essential nutrient that helps the body use sugar, protein, and fat. Deficiency of chromium(III) in the diet can result in weight loss or decreased growth, improper function of the nervous system, and a diabetic-like condition.

Chromium

In general, Inhalation of high concentrations of chromium(VI) can cause irritation to the nose, such as runny nose, sneezing, itching, nosebleeds, ulcers, and holes in the nasal septum. Breathing in chromium(III) does not cause irritation to the nose or mouth in most people.

Long-term exposure to chromium, particularly chromium(VI) has been associated with lung cancer in workers exposed to levels in air that were 100 to 1,000 times higher than those found in the natural environment. Breathing in small amounts of chromium(VI) for short or long periods does not cause a problem in most people.

In the same way, ingestion of small amounts of chromium(VI) is not considered to be toxic; however, accidental or intentional swallowing of larger amounts has the potential to cause stomach upsets and ulcers, convulsions, kidney and liver damage.

Besides its capacity to induce primary skin irritation and corrosion, direct contact with small amounts of chromium may be the cause of an allergic contact dermatitis. It is clear from occupational exposures that hexavalent chromium compounds are responsible for the majority of the cases of allergy to chromium. This is most likely explained by the fact that chromium(VI) penetrates the skin tissue better and is therefore more able than trivalent chromium to induce an allergic reaction. The role of the oxidation state of chromium in allergy is, however, complex.

Toxicity Classification

Chromium(VI) is classified as a "known" human carcinogen (Category A) by the USEPA for the inhalation route of exposure. Carcinogenicity by the oral route of exposure cannot be determined and is classified as Group D. Chromium(III) is not classified as to carcinogenicity and is in Group D.

The International Agency for Research on Cancer (IARC) has determined that chromium(VI) in air is carcinogenic to humans, while chromium(0), chromium(III) compounds and oral exposures to chromium(VI) are not classifiable as to their carcinogenicity to humans.

Hexavalent compounds have been shown to have mutagenic potential with positive results in a number of in vitro and in vivo studies. There is no consistent evidence that trivalent compounds have genetic activity.

Exposure Limits and Toxicity Evaluations

The NHMRC has presented a guideline value of 0.05 mg/L for chromium in the Australian Drinking Water Guidelines (2004) on the basis of the guideline value derived by the WHO and the point noted that the guideline value of 0.05 mg/L has been adopted by many countries with no known cases of chromium toxicity.

Review of the WHO provisional guideline of 0.05 mg/L indicates that the value is not derived on the basis of a specific threshold or non-threshold toxicity approach. Review by WHO in 2004 indicated that the guideline, while considered provisional, remained protective of human health as available toxicity data did not support revision of the guideline.

The US EPA derived a reference dose (RfD) of 0.003 mg/kg-day based on a NOAEL of 2.5 mg/kg-day from a drinking water study in rats. An uncertainty factor of 300 was used to account for interspecies and interhuman variability and the less-than-lifetime exposure in the principal

Chromium

study; a modifying factor of 3 was used to account for uncertainties related to reports of gastrointestinal effects following drinking water exposures in a residential population in China. This RfD is limited to soluble salts of hexavalent chromium (VI). The same study (from 1958) was used by the RIVM (2001) to establish a TDI of 0.005 mg/kg/day.

EPA derived a reference dose (RfD) of 1.5 mg/kg-day for chromium III. The critical study was conducted using chromic oxide. The NOAEL from this study (1,800 g/kg) was converted to a NOAEL for chromium (III) of 1,468 mg/kg-day by adjusting by a factor of 0.6849 g Cr/g Cr₂O₃ and then adjusting for continuous exposure. An uncertainty factor of 100 was used to account for interspecies and interhuman variability; a modifying factor of 10 was used to reflect database deficiencies (total = 1000). This RfD is limited to metallic chromium (III) of insoluble salts. Examples of insoluble salts include chromic III oxide (Cr₂O₃) and chromium (III) sulfate (Cr₂[SO₄]₃). The USEPA has also established an inhalation unit risk of 0.012 (µg/m³)⁻¹ for chromium(VI).

RIVM (2001) has established an oral TDI for chromium(III) for both soluble compounds (0.005 mg/kg/day) and metallic and insoluble compounds (5 mg/kg/day). In addition a tolerable concentration in air of 0.06 mg/m³ has been established for metallic and insoluble chromium(III) in air. For chromium(VI) a TDI of 0.005 mg/kg/day has been established as a provisional oral value with an inhalation risk concentration of 0.0025 µg/m³ based on a 1 in 10,000 excess lifetime cancer risk. This value was established on the same basis as the WHO.

The WHO (2000) provides an inhalation unit risk of 0.04 (µg/m³)⁻¹ for the assessment of inhalation exposures to chromium, based on lung cancer effects (i.e. for an air concentration of 1 µg/m³, the lifetime risk is estimated to be 4.0x10⁻²). This unit risk is the geometric mean of estimated lifetime risks calculated from various epidemiological data sets, which ranged from 0.13 to 0.011. The incremental risks calculated from these data sets have estimated lifetime risks from a lifetime exposure to chromium (VI) at a concentration of 1µg/m³. This is equivalent to the following slope factor (based on a 70kg body weight):

$$\begin{aligned}
 SF \text{ (mg/kg/day)}^{-1} &= \text{Risk/Intake(mg/kg/day)} \\
 &= [\text{Risk} \times \text{Body Weight}]/[\text{Concentration (in air)} \times \text{Inhalation Rate}] \\
 &= [0.04 \times 70\text{kg}]/[0.001\text{mg/m}^3 \times 20 \text{ m}^3/\text{day}] \\
 &= 140 \text{ (mg/kg/day)}^{-1}
 \end{aligned}$$

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. For chromium a background intake of 13 µg/day has been estimated by the UK (2002) based on data for levels of chromium in food (most significant), water and air. It is also conservatively assumed that 10% of chromium in food and water is present in the hexavalent state. The RIVM (2001) has estimated background intakes of trivalent chromium to be 1 µg/kg/day and hexavalent chromium background intakes that vary from 0.01 to 30% of total chromium in air.

Chromium

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for chromium following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	TDI = 0.003 mg/kg/day (USEPA current) for chromium(VI) TDI = 1.5 mg/kg/day (USEPA current) for chromium(III)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity. However it is noted that the assessment does not adequately address dermal sensitivity (allergic contact dermatitis [ACD]). RIVM recommends a 10% threshold value be used to address ACD.
Inhalation	Inhalation Unit Risk = $0.04 (\mu\text{g}/\text{m}^3)^{-1}$ (WHO, 2000), with an equivalent inhalation slope factor of $140 (\text{mg}/\text{kg}/\text{day})^{-1}$. Occupational inhalation exposure (ASCC/NOHSC): TWA: $0.5 \text{ mg}/\text{m}^3$ for chromium III and $0.05 \text{ mg}/\text{m}^3$ for chromium VI STEL: NA
Background	Conservatively assumed to be 30% (based on background levels from the UK and a child body weight)

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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	6/10/2007
Previous Revisions:	2005A	16/6/2005

Lead

General

Lead has been used from very early times due to the fact that it can be easily extracted from its ores. It is soft and malleable and both metallic lead and lead compounds are used for a number of industrial, domestic and rural purposes. In uncontaminated environments, lead is generally present at concentrations in the soil of less than 50 mg/kg. When lead is released into the environment it has a long residence time compared with other pollutants and has a low mobility within soil and groundwater.

Lead is neither an essential nor beneficial element for plants, animals or humans. There are many different sources of environmental lead that can result in human exposure. These include drinking water, soil, dust, particulates emitted from industrial and incineration activities, food and paint. The concentration of lead in soils is only one element of potential human exposure. Appendix A presents a summary of the different sources of lead in the environment.

Human exposure to lead occurs primarily through diet, air, drinking water, and ingestion of dirt and paint chips. Lead may be present within soils and other environments in the form of elemental lead or various other lead compounds. These may include lead oxide, lead carbonate, lead sulfide and lead acetate (for example). Lead sulfate forms in lead storage batteries during the discharge cycle. On addition, lead sulfate may form naturally from lead and its compounds in the atmosphere and also in soil. Speciation studies show that lead sulfate is a major lead compound in soil and air (at least near smelters). The following information on the intake of lead, its potential health effects are relevant to lead and its compounds, i.e. exposures to all forms of lead in the workplace.

The efficiency of lead absorption depends on the route of exposure, age, and nutritional status. Adult humans absorb about 10-15% of ingested lead, whereas children may absorb up to 50%, depending on whether lead is in the diet, dirt, or paint chips. More than 90% of lead particles deposited in the respiratory tract are absorbed into systemic circulation. Inorganic lead is not efficiently absorbed through the skin; consequently, this route does not contribute significantly to the total body lead burden (US EPA, 1986).

Potential Effects of Lead Exposure

Biological Fate of Lead

Once in the blood, lead is distributed primarily among three areas:

- Blood;
- Soft tissue (kidney, bone marrow, liver, and brain); and
- Mineralising tissue (bones and teeth).

In single-exposure studies with adults, lead has a half-life, in blood, of approximately 25 days; in soft tissue about 40 days; and in the non-labile portion of bone, more than 25 years. Consequently, after a single exposure a person's blood lead level may begin to return to normal; the total body burden, however, may still be elevated. In bone, there is both a labile component, which readily exchanges lead with the blood, and an inert pool. The lead in the inert pool poses a special risk because it is a potential endogenous source of lead. When the body is under

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physiological stress such as pregnancy, lactation, or chronic disease, this normally inert lead can be mobilised, increasing the lead level in blood. Because of these mobile lead stores, significant drops in a person's blood lead level can take several months or sometimes years, even after complete removal from the source of lead exposure.

For lead poisoning to develop, major acute exposures to lead need not occur. The body accumulates this metal over a lifetime and releases it slowly, so even small doses, over time, can cause lead poisoning. It is the total body burden of lead that is related to the risk of adverse effects.

Physiological Effects

Whether lead enters the body through inhalation or ingestion, the biological effects are the same. There is interference with normal cell function and with a number of physiological processes. These include:

- **Neurological Effects** - particularly in children where studies have indicated a link between lead exposure and deficits in psychometric intelligence scores, speech and language processing, attention, and classroom performance.
- **Haematological Effects** - Lead inhibits the body's ability to make haemoglobin by interfering with several enzymatic steps in the haeme pathway, which may then induce anaemia.
- **Endocrine Effects** - resulting in impairment of cell growth and maturation and tooth and bone development.
- **Renal Effects** - A direct effect on the kidney of long-term lead exposure is nephropathy.
- **Reproductive and Developmental Effects**
- **Potential Carcinogenic Effects**

The US EPA classifies lead as a probable human carcinogen (classification B2) based on sufficient evidence available from animal studies, but inadequate data on humans. Similarly, the International Agency for Research on Cancer (IARC) classifies lead as possibly carcinogenic in humans (Group 2B).

Measurement of Lead Absorption In Humans

For the purpose of setting standards relating to exposure to lead, it is important to know the lowest exposures that have produced detectable effects on the biological functions of humans.

Blood lead levels have been found to be a good indicator of exposure to lead. A blood lead level reflects lead's dynamic equilibrium between adsorption, excretion and deposition in soft and hard tissues. For chronic exposures, blood lead levels can under-represent the total body burden; however they are the most widely accepted and commonly used measure of lead exposure. Blood lead levels respond relatively rapidly to changes in lead intake, such as the ingestion of lead paint chips by children, and in general bear a linear relationship to these intakes.

Continued research on the biological effects and low level lead exposure has resulted in a progressive reduction in the blood lead levels of concern.

Lead

Goals for Lead Exposure in Australia

Standards or goals for exposure to lead in Australia are presented in Table 1. This table presents goals set for blood lead levels, total lead intake and exposure to lead in air, soil and water particularly with respect to occupational exposure.

Table 1 GOALS FOR LEAD EXPOSURE IN AUSTRALIA

LEVEL	FOCUS	AGENCY
Acceptable Daily Intake of Lead from All Sources		
25 µg/kg(bw)/week	FAO/WHO provisional tolerable weekly intake. Adopted by NHMRC in the derivation of drinking water guidelines (2004).	
Soil Investigation Level¹		
300 mg/kg	Residential "A" NEPM (Schedule 7(A), 1999) health based soil investigation level derived on the basis of the above PTWI and allowance for background intake.	
1200 mg/kg	Residential "D" NEPM (Schedule 7(A), 1999) health based soil investigation level The guideline was determined by applying an adjustment factor to the residential level.	
600 mg/kg	Parks/recreational "E" NEPM (Schedule 7(A), 1999) health based soil investigation level. The guideline was determined by applying an adjustment factor to the residential level.	
1500 mg/kg	Industrial "F" NEPM (Schedule 7(A), 1999) health based soil investigation level. The industrial guideline was determined by applying an adjustment factor to the residential level.	
Drinking Water Guideline		
0.01 mg/L	Health based guideline for drinking water (NHMRC 2004)	
Air Guideline for Workplace Environments		
0.15 mg/m ³	NOHSC TWA for inhalation of lead dusts (available on-line, 2007).	
Blood Lead Guidelines		
10 µg/dL	Goal for all individuals established by NHMRC 1993.	
50 µg/dL	Response level for males and females (not of reproductive capacity) in the workplace (ASCC/NOHSC).	
20 µg/dL	Response level for females of reproductive capacity in the workplace (ASCC/NOHSC).	

Notes:

- 1 Soil investigation levels are designed to provide a basis for screening soil information to identify whether further investigation is warranted. They should not be used as remediation or response levels for assessing potential exposure to lead concentrations in soil.
- 2 National Standard for the Control of Inorganic Lead at Work [NOHSC:1012(1994)] and National Code of Practice for the Control and Safe Use of Inorganic Lead at Work [NOHSC:2015(1994)] available on-line from Worksafe Australia web-site.

Potential for Exposure to Lead to be Unacceptably High

There are three approaches that can be taken to provide an assessment as to whether potential exposure to lead may be unacceptably high, these are:

1. Comparison of the estimated intake with the ADI. As discussed in Section 1.4, this is the method that is recommended by the NEPC (1999), as well as ANZECC and NHMRC (1992). The method takes into account exposure from all sources including food, drinking water, air and contaminated soil (by direct and indirect means) and compares this intake with the ADI. Intakes less than the ADI are acceptable while intakes greater than the ADI may be unacceptable. It should be noted, however, that the ADI does not represent a trigger value above which adverse health effects would be expected; rather, it represents a

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value that is regarded as being safe. Intakes much greater than the ADI would normally be expected before adverse health effects occurred.

2. Calculation of the blood lead concentrations using a biokinetic uptake model. This method is the preferred approach by the US EPA who have developed the most commonly used model. The approach also takes into account all lead intakes, however, the assessment of whether intakes are acceptable is made on the basis of calculated blood lead concentrations and not an ADI. The use of blood lead models is not endorsed by NHMRC, ANZECC or the various EPAs in Australia and has not been used in this assessment.

Measurement of blood lead concentrations in the exposed populations and site-specific studies such as measurement of bioavailability. The monitoring of blood lead concentrations has been used both in Australia (for example Port Pirie, Broken Hill and Boolaroo) and in the US to manage sites that are widely affected by elevated concentrations of lead in soil and dusts. The blood data is used to identify various levels of intervention to manage the lead exposure. Lead is unique in comparison to most other environmental contaminants, in that the measurement of concentrations in blood is widely accepted as an appropriate basis for assessing whether the actual intakes are unacceptable. The monitoring of blood lead levels represents the most definitive way of assessing whether an actual harm has taken place. This is also consistent with the management of occupational exposures.

Suggested Toxicity Values for Risk Characterisation

As indicated above, the approach that is currently required to be taken in Australia, when assessing exposure to lead and most other environmental contaminants, is to compare the total intake to the ADI value using the methodology outlined in the NEPC (1999).

Toxicity data relevant for use in the characterisation of risk to human health have been selected for chromium following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

- **Oral intake** - Potential ingestion of lead has been assessed using the PTWI of 25 $\mu\text{g}/\text{kg}/\text{week}$ which was established by the WHO for potential exposure to lead by children on the basis of blood lead data (i.e. an absorbed dose). The PTWI is used in the derivation of WHO Drinking Water Guidelines (2004) and Australian Drinking Water Guidelines (NHMRC 2004). CSMS (1991) indicates that background sources of lead (food, water, air, etc.) contributes approximately 48% of the total daily intake of lead, hence 52% of the PTWI should be used in determining potential exposure to lead in other sources. Hence the adjusted PTWI is 13 $\mu\text{g}/\text{kg}/\text{week}$, equivalent to a TDI of 0.0019 $\text{mg}/\text{kg}/\text{day}$. It is noted that the PTWI for lead has been derived on the basis of an absorbed dose and hence application of oral bioavailability is considered relevant to enable calculation of an absorbed intake from an ingested intake. Based on data from Maynard (CSMS, 1991) 50% bioavailability can be assumed for children and 10% for adults.
- **Dermal** absorption of lead from soils is essentially negligible (CSMS 1991 and USEPA 1986) and does not significantly contribute to the total intake of lead from soils. Potential dermal intake of lead in water must be considered.

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- **Inhalation** The WHO (2000) has established a guideline value of $0.5 \mu\text{g}/\text{m}^3$ of lead in air on the basis of an annual average. This GV was derived on the bases of a critical level of lead in blood that is protective of at least 98% of the population including preschool children.

In summary the toxicity values selected are:

Oral	PTWI = $0.025 \text{ mg}/\text{kg}/\text{week}$ (NHMRC and WHO 2004) – refer to note below Equivalent adjusted TDI = $0.00357 \text{ mg}/\text{kg}/\text{day}$
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation Guideline Value = $0.5 \mu\text{g}/\text{m}^3$ (WHO, 2000), with an equivalent inhalation TDI of $0.000143 \text{ mg}/\text{kg}/\text{day}$. Occupational inhalation exposure for lead (ASCC): TWA: $0.15 \text{ mg}/\text{m}^3$ for lead, inorganic dusts and fumes STEL: NA
Background	48%

Note of PTWI Adopted (from UK EA, 2002):

- *“In 1972, Joint FAO/WHO Expert Committee on Food Additives (JECFA) set the provisional tolerable weekly intake (PTWI) for adults at 3 mg per person from all sources (WHO, 1972), which was equivalent to $50 \mu\text{g kg}^{-1} \text{ bw}$. In 1987, the corresponding PTWI for infants and children was $25 \mu\text{g kg}^{-1} \text{ bw}$, again for all sources (WHO, 1987a). Although the toxicological effects of lead were reviewed and considered, the PTWI for children was based on dietary balance studies (Ziegler et al, 1978; Ryu et al, 1983), which showed that infants fed $3\text{--}4 \mu\text{g Pb kg}^{-1} \text{ bw day}^{-1}$ did not increase their blood lead and were in negative balance for lead. The Committee were simply attempting to ensure that there would be no accumulation of lead in the body.*
- *At their 41st meeting, JECFA (WHO, 1993a) stated that “the most significant health effect from lead is the association of lead exposure with reduced cognitive development and intellectual performance in children”. While noting a wide statistical variation in the results of a number of studies on children with blood lead concentrations below $25 \mu\text{g dL}^{-1}$, they observed that the IQ is reduced on average by 1–3 points for each $10 \mu\text{g dL}^{-1}$ increment in blood lead. Although the limits of precision in analytical and psychometric measurement and the presence of confounding variables were seen to increase the uncertainty associated with any effects related to lead, they noted that there is “some evidence of an association between lead exposure and cognitive deficits even in the 7–8 $\mu\text{g}/\text{dl}$ range”.*
- *The Committee estimated that the previously recommended PTWI of $25 \mu\text{g kg}^{-1} \text{ bw}$ for infants and children would have been responsible for a blood lead concentration of $5.7 \mu\text{g dL}^{-1}$ for a 10 kg child. (They assumed that $1 \mu\text{g}$ of lead intake per day produced $0.16 \mu\text{g}$ of lead per decilitre of blood.) As this concentration was thought then to be below that associated with effects on intellectual performance, the PTWI for infants and children was reconfirmed.*
- *The separate PTWI for adults was withdrawn (WHO, 1993a). The Committee recognised that the foetus was as least as sensitive as the newborn infant to lead’s neurotoxicity. As lead was known to cross the placenta readily, they considered that the PTWI for women of*

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child-bearing age should ideally be as low as that of children. Thus the PTWI of 25 $\mu\text{g kg}^{-1}\text{ bw}$ was extended to all age groups.

- In 1999, when JECFA again evaluated lead, the main objective was to assess the risk of dietary exposure of infants and children on the most critical effect, which was considered to be impaired neurobehavioural development. The then current PTWI [of 25 $\mu\text{g kg}^{-1}\text{ bw}$] “was not reconsidered and was retained at its present value”. The Committee noted that “as a conservative estimate ... a dietary intake of 1 $\mu\text{g/kg bw/day}$... would result in an increase in the lead concentration of blood of 1 $\mu\text{g/dl}$, the upper estimate for infants, and that this relationship was valid during the long-term exposure period (in utero + 10 years)”.

The EC (2000) has provided a review of lead toxicity and intake, summarising potential lead intake levels expressed as tolerable daily intakes that are associated with “no risk” and intervention levels of lead in blood. The following was presented:

Population	No-risk Blood Lead Level (g/dL)	TDI (g/kg/day)
Children (1 yrs)	100	60
Children (7 yrs)	100	150
Pregnant women	100	250
Adult	300	750

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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	1/7/2007
Previous Revisions:	2006A	4/04/2006
	2005A	15/6/2005

Mercury

General

Mercury is a heavy metal which exists in three oxidation states: 0 (elemental), +1 (mercurous) and +2 (mercuric). As well as the common mercurous and mercuric inorganic salts, mercury can also bind covalently to at least one carbon atom. Thus the most commonly encountered exposures associated with mercury are with elemental mercury, inorganic mercuric compounds and methyl mercury.

Mercury occurs naturally as a mineral is widely distributed by natural and anthropogenic processes. The most significant natural source of atmospheric mercury is the degassing of the Earth's crust and oceans and emissions from volcanoes. Man-made sources such as mining, fossil fuel combustion and industrial emissions generally contribute less on a global scale, but more on a local scale. Wet and dry deposition to land and surface water result in mercury sorption to soil and sediments.

Uses of mercury include use in the electrical and chlor-alkali industry (lamps, batteries and as cathodes in the electrolysis of sodium chloride to produce caustic soda and chloride), industrial and domestic instruments, laboratory and medical instruments and dental amalgam (mixed in proportion of 1:1 with a silver-tin alloy).

Properties

Elemental mercury is a dense, silvery white metal which is liquid at room temperature, readily volatilises and is considered to be the predominant form of mercury in the atmosphere. Mercury compounds differ greatly in general properties and solubility. Due to the wide range in properties associated with the forms of mercury, key properties have not been listed here. These are available from many sources including the ATSDR review (1999).

Exposure

Exposure of the general population to mercury may occur via inhalation, oral or dermal contact. Exposure to elemental mercury may occur in the workplace or home if mercury is spilled. Inorganic mercury compounds are found in some batteries, pharmaceuticals, ointments and herbal medicines. Exposure to inorganic mercury can occur via inhalation or ingestion. Methylmercury is most commonly found in fish, especially larger fish at the top of the food chain with exposure typically associated with ingestion.

If released into the environment the following can be noted with respect to mercury (USEPA 1997, WHO 1989 and 1991):

- **Air:** Mercury is released into the atmosphere from anthropogenic emissions as either vapour (elemental or oxidized mercury) or as particles (oxidized compounds). Natural emissions are mainly in elemental mercury form. Mercury may reside in the atmosphere for about one year, allowing global circulation systems to transport elemental mercury emissions from source of emission to anywhere on earth before transformation and deposition take place. Mercury is transferred from the atmosphere to the earth's surface via wet or dry deposition.

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- **Soil:** The majority of mercury in surface soil is in the form of oxidized mercury complexes/compounds; however, a small fraction is methyl mercury and elemental mercury. Mercury complexes deposited in soils can be transformed back into gaseous mercury by light and humic substances and re-enter the atmosphere. Studies have consistently shown that plant uptake is negligible and consequently, animals foraging on plants accumulate little mercury. In addition to direct deposition, mercury can also reach water from soil run-off, although the amount partitioning to run-off is expected to be small since mercury binds to soil; run-off is probably in the form of suspended sediments.
- **Water:** Once in water, mercury can either enter the food chain, settle into sediment, or volatilise back into the atmosphere. Entrance into the food chain begins with bacteria in water which can take up mercury in its inorganic form and metabolise it to methyl mercury. The methyl mercury-containing bacteria may be consumed by the next level in the food chain, or they may excrete the methyl mercury into the water where it can adsorb to plankton, which are also consumed by the next level in the food chain. Even small environmental concentrations of mercury in water can readily accumulate to potentially harmful concentrations in fish and fish-eating people. Fish higher in the food chain have much higher mercury concentrations than fish lower on the food chain.
- Mercury is continuously mobilized, deposited and re-mobilized in the environment. The only sinks for removal from the biosphere are deep-seas sediments or well-controlled landfills. If the release of mercury into the environment is reduced, resultant decreases in mercury concentrations in the environment would occur slowly, most likely over many decades or centuries.

On the basis of the potential for long-range transport, persistence in water, soil and sediment, bioaccumulation, toxicity and ecotoxicity, mercury is considered persistent and is addressed in the 1998 UN-ECE Convention on Long-Range Transboundary Air Pollution on Heavy Metals (UN-ECE, 1998). The United Nations Environment Programme (UNEP) Governing Council concluded, at its 22nd session in February 2003, after considering the key findings of the Global Mercury Assessment report, that there is sufficient evidence of significant global adverse impacts from mercury to warrant further international action to reduce the risks to humans and wildlife from the release of mercury to the environment. The UN Governing Council decided that national, regional and global actions should be initiated as soon as possible and urged all countries to adopt goals and take actions, as appropriate, to identify populations at risk and to reduce human-generated releases. While mercury is not listed as one of the 12 chemicals listed in the Stockholm Convention on Persistent Organic Pollutants (POPs), it chemical meets criteria listed (annex D) in the convention for consideration as persistent and bioaccumulative.

Health Effects

The following information is available from UK (2002) and ATSDR (1999).

Elemental Mercury

General

Limited data is available concerning the absorption of elemental mercury. Inhaled mercury vapour by humans indicates approximately 80% of the vapour crosses the alveolar

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membranes into the blood. Liquid metallic mercury is poorly absorbed via the oral route with studies indicating less than 0.01% absorption, possibly because of its enterogastric conversion to divalent mercury and subsequent binding to sulfhydryl groups. Dermal absorption of mercury vapour is limited and may only contribute approximately 2.5% of absorbed mercury following inhalation exposures. No data are available concerning dermal absorption of liquid metallic mercury.

Absorbed mercury is lipophilic and rapidly distributed to all tissues and able to cross the blood-brain and foetal barriers easily. Mercury is oxidised in the red blood cells by catalase and hydrogen peroxide to divalent ionic mercury. Approximately 7-14% of inhaled mercury vapour is exhaled within a week after exposure. The rest of the elemental mercury is either excreted via sweat and saliva, or is excreted as mercuric mercury. Approximately 80% is excreted as mercuric mercury via faeces and urine. Half-life elimination is approximately 58 days.

Acute exposure to high concentrations of mercury vapour has been associated with chest pains, haemoptysis, breathlessness, cough and impaired lung function with the lung identified as the main target following acute exposure.

The central nervous system is generally the most sensitive indicator of toxicity of metallic mercury vapour. Data on neurotoxic effects are available from many occupation studies.

Chronic exposure to metallic mercury may result in kidney damage with occupational studies indicating an increased prevalence of proteinuria.

Genotoxicity and Carcinogenicity

Both USEPA and IARC indicate that elemental mercury is not classifiable as to its human carcinogenicity.

No adequate animal studies are available for elemental mercury and occupational studies have indicated conflicting results.

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Inorganic Mercury Compounds

General

Limited data is available concerning the absorption of inhaled mercury compounds, however it is expected to be determined by the size and solubility of the particles. Absorption of ingested inorganic mercury has been estimated to be approximately 5 to 10% with absorption be children greater than for adults. Absorption via the dermal route is considered low and not significant, however indirect evidence of dermal absorption is provided by case studies following dermal application of ointments that contained inorganic mercury salts.

Inorganic mercury compounds are rapidly distributed to all tissues following absorption. The fraction that crosses the blood-brain and foetal barriers is less than for elemental mercury due to poor lipid solubility. The major site of systemic deposition of inorganic mercury is the kidney. Most inorganic mercury is excreted in the urine or faeces.

Acute exposure to high concentrations of ingestion of inorganic mercury has been associated with gastrointestinal damage, cardiovascular damage, acute renal failure and shock.

The kidney is the critical organ associated with chronic exposure to inorganic mercury compounds. The mechanism for the end toxic effect on the kidney, namely autoimmune glomerulonephritis, is the same for inorganic mercury compounds and elemental mercury and results in a condition sometimes known as nephrotic syndrome.

There is some evidence that inorganic mercury may cause neurological effects, particularly associated with studies of mercuric chloride. Reproductive and developmental effects have been observed in rats given mercuric chloride.

Genotoxicity and Carcinogenicity

IARC have considered inorganic mercury compounds not classifiable as to human carcinogenicity. The USEPA has classified mercuric chloride as a possible human carcinogen (Class C) based on increased incidence of squamous cell papillomas of the forestomach and marginally increased incidence of thyroid follicular cell adenomas and carcinomas from a long term oral studies in rats. Mercuric chloride has produced some evidence of an action on the chromosomes, and mixed results associated with mutagenic activity has been reported. The USEPA evaluation of mercuric chloride indicate that a linear low-dose extrapolation is not appropriate as kidney tumour seen in mice occurred at doses that were also nephrotoxic.

Methylmercury

General

Limited data are available concerning the absorption of inhaled methyl mercury compounds, however studies on rats indicates rapid and almost complete absorption of inhaled methyl mercury vapour. Ingested methyl mercury is almost completely absorbed. No dermal absorption data are available.

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Methyl mercury is distributed via the blood to all tissues. It can cross into the brain and foetus. The major site of systemic deposition of methyl mercury is the kidney. Hair levels are typically used as an index of exposure to mercury and there is a proportional relationship between mercury intake, blood mercury and hair mercury. Methyl mercury is converted to mercuric mercury in animals and humans, though less readily than for elemental mercury.

The key target of methyl mercury in humans is the CNS, particularly the brain. Evidence from animal and human studies indicates that the embryo and foetus are more sensitive to methyl mercury than adults.

Other effects associated with methyl mercury include damage to other tissues and organs including the lung, cardiovascular system, liver and kidney. In animals, the most sensitive indicator of damage other than CNS effects, are renal effects.

Genotoxicity and Carcinogenicity

USEPA and IARC have classified methyl mercury as a possible human carcinogen (USEPA Class C and IARC Group 2B) on the basis of long term animal studies. Both agencies consider that the evidence for carcinogenicity of methyl mercury in humans is inadequate. The USEPA (2001) have concluded that methyl mercury is not a potent genotoxic agent. Methyl mercury induced tumours in mice were considered likely to have a non-genotoxic origin.

Exposure Limits and Toxicity Evaluations

Australia

The Australian Drinking Water Guidelines (2004) have derived a drinking water guideline of 0.001 mg/L for total mercury using the provisional tolerable weekly intake (***PTWI***) of ***0.0033 mg/kg*** (equivalent to ***TDI of 0.00047 mg/kg/day***) for methyl mercury recommended by JECFA and used by the WHO (as below). The guideline was considered sufficient to be protective of pregnant women and nursing mothers.

Worksafe Australia (ASCC/NOHSC, available in 2007) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For mercury the following have been established:

Elemental mercury: $TWA = 0.025 \text{ mg/m}^3$, STEL = NA

Inorganic monovalent mercury: $TWA = 0.1 \text{ mg/m}^3$, STEL = NA (skin notification)

Inorganic divalent mercury: $TWA = 0.025 \text{ mg/m}^3$, STEL = NA

Alkyl mercury compounds: $TWA = 0.01 \text{ mg/m}^3$, STEL = 0.03 mg/m^3 (skin notification)

Aryl mercury compounds: $TWA = 0.1 \text{ mg/m}^3$, STEL = NA (skin notification)

Mercury

WHO

The WHO (Drinking Water Guideline 1993 and 1996) provide a guideline value for total mercury of 0.001 mg/L based on the provisional tolerable weekly intake (**PTWI**) of **0.005 mg/kg** for total mercury for the general population of which no more than **0.0033 mg/kg** should be present as methyl mercury recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA, 2003). The value for methyl mercury was used in the derivation of the drinking water guideline “to be on the conservative side”. JECFA note that pregnant women and nursing mothers may be at greater risk. The TDI remained unchanged in the WHO 2004 documentation (WHO, 2004). The PTWI for methyl mercury was revised by the JECFA in June 2003 to **0.0016 mg/kg** to be “sufficient to protect the developing foetus, the most sensitive subgroup of the population”.

The rolling revision to the WHO Drinking Water Guidelines (2005) derived a drinking water guideline value of 0.006 mg/L for inorganic mercury on the basis of a **TDI of 0.002 mg/kg/day**. The TDI for inorganic mercury is based on kidney effects in a 26-week study in rats, uncertainty of 100 and adjustment for 5 day/week dosing. This value is consistent with the TDI presented in the WHO (2003) review.

WHO (2000) have derived a **guideline value of 0.001 mg/m³** for mercury in air as an annual average based on a LOAEL derived from occupational studies on mercury vapour. The WHO note that “since cationic inorganic mercury is retained only half as much as the vapour, the guideline also protects against mild renal effects caused by cationic inorganic mercury”. “Present knowledge suggests, however, that effects of the immune system at lower exposures cannot be excluded”. The WHO have not proposed an air quality guideline value for methyl mercury due to the potential for adverse health impacts associated with post-depositional methyl mercury formation and bioaccumulation. A tolerable concentration (TC) for elemental mercury in air has been derived by WHO (2003). The **TC of 0.0002 mg/m³** is derived for long-term inhalation exposure to elemental mercury vapour based on CNS effects in workers.

EU

No assessment of toxicity of mercury is available from the EU. However it is noted that EU reviews of dietary intakes of mercury in the EU adopt the WHO PTWI of 0.005 mg/kg for total mercury and the JEFCA (2003) PTWI of 0.0016 mg/kg for methyl mercury.

RIVM

Review by RIVM (2001) has identified the following with respect to mercury:

- Elemental mercury, Tolerable Concentration in Air (**TCA**) of **0.0002 mg/m³** based on CNS effects (utilising the same study used by ATSDR).
- Mercuric Chloride (Inorganic Mercury), a **TDI of 0.002 mg/kg/day** has been derived based on kidney effects in rats.
- Methyl mercury, a **TDI of 0.0001 mg/kg/day** has been derived for oral exposures based developmental effects (utilising the same study used by ATSDR but a larger uncertainty factor).

Mercury

Background intake of inorganic and elemental mercury in The Netherlands is estimated to be 0.0001 mg/kg/day. For organic mercury the exposure is estimated to be 0.00002 mg/kg/day.

US

The USEPA have provided separate evaluations for elemental mercury, mercuric chloride and methylmercury.

- Elemental mercury: The USEPA have derived an inhalation reference concentration (**RfC**) of **0.0003 mg/m³** based on CNS effects in occupationally exposed workers.
- Mercuric chloride: The USEPA have derived an oral reference dose (**RfD**) of **0.0003 mg/kg/day** based on autoimmune glomerulonephritis observed in rats (sub-chronic studies).
- Methyl mercury: The USEPA have derived an oral **RfD of 0.0001 mg/kg/day** based on neurological effects in children exposed in utero as a consequence of maternal intake of methylmercury in food.

The ATSDR has established Minimal Risk levels (MRLs) associated with non-carcinogenic effects associated with mercury. The levels established are:

- Chronic inhalation MRL for mercury (metallic, vapour) = **0.0002mg/m³** based on CNS effects in occupational workers;
- Acute oral MRL for inorganic mercury = **0.007 mg/kg/day** based on renal effects in rats exposed to mercuric chloride
- Intermediate oral MRL for inorganic mercury = **0.002 mg/kg/day** based on kidney effects in rats exposed to mercuric chloride; and
- Chronic oral MRL for methyl mercury = **0.0003 mg/kg/day** based on developmental effects in children.

The California Air Resources Board (CARB and OEHHA) has established the following:

- Acute inhalation reference exposure level (**acute REL, 1999**) for mercury and **compounds of 0.0018 mg/m³** based on CNS disturbances in offspring following inhalation of metallic mercury vapours. The same value is also presented for mercuric chloride;
- **Chronic inhalation REL (2000) of 0.00009 mg/m³** for mercury and compounds (mercuric chloride) and elemental mercury based on CNS effects in occupational studies;
- **Chronic oral REL (2000) of 0.0003 mg/kg/day** for mercury and compounds (mercuric chloride) adopted from the USEPA RfD;
- **Chronic inhalation REL (1991)** of 0.001 mg/m³ for methyl mercury

Mercury

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Based on data available on background intakes for elemental and inorganic mercury compounds from Imray P. and Neville G. (CSMS, 1996), it has been calculated that background may contribute 50% of the TDI for total mercury with 42% derived from dental amalgams. More recent reviews (NHMRC 1999) of intakes associated with amalgam fillings in Australian children and adults (based on average number of fillings of 0.5 and 8 respectively) provides a reasonable estimate of daily mercury absorption per person of about 0.3 µg for children and 3.5 µg for adults. Dietary intakes of mercury is about 10 fold and 2 fold higher than these amounts (NHMRC 1999). If these values are considered (with a child body weight of 20 kg and adult body weight of 70kg), background intakes are approximately 0.00017 mg/kg/day. This is approximately 24% of the oral TDI for inorganic mercury.

Review of methyl mercury intake by Food Standards Australia (2004) has indicated that background intake of methyl mercury from non-sea food sources in the diet accounts for 0.09% of the PTWI for adults and children over 2 years of age and 0.01% of the PTWI for children 2-6 yrs.

The assessment of methyl mercury has assumed 0.1 % background intake where the assessment is for seafood. For the assessment of recreationally caught fish, it is expected that commercially caught seafood will still form part of the diet and hence a higher background intake of 15% (CSMS 1996 for methyl mercury) has been assumed.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for mercury following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	TDI = 0.00071 mg/kg/day for total mercury (based on WHO PTWI of 0.005 mg/kg for total mercury, 2004) TDI = 0.00023 mg/kg/day for methyl mercury (based on revised PTWI of 0.0016 mg/kg provided by JECFA 2003 for the protection of the developing foetus)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity. Dermal absorption of inorganic and methyl mercury from soils is assumed to be 0.001 (unitless).
Inhalation	TC = 0.0002 mg/m ³ as an annual average (WHO 2003) for elemental mercury. GV = 0.001 mg/m ³ as an annual average (WHO 2000) for inorganic mercury. As no inhalation value has been established for methyl mercury the TDI adopted for oral exposures should be considered when inhalation

Mercury

	<p>exposures are assessed.</p> <p>Occupational inhalation exposure (ASCC/NOHSC):</p> <p><u>Elemental mercury</u>: TWA = 0.025 mg/m³, STEL = NA</p> <p><u>Monovalent mercury</u>: TWA = 0.1 mg/m³, STEL = NA</p> <p><u>Divalent mercury</u>: TWA = 0.025 mg/m³, STEL = NA</p> <p><u>Alkyl mercury compounds</u>: TWA = 0.01 mg/m³, STEL = 0.03 mg/m³</p> <p><u>Aryl mercury compounds</u>: TWA = 0.1 mg/m³, STEL = NA</p>
Background	<p>24% inorganic and elemental exposures</p> <p>15% methyl mercury exposures</p>
Notes	Mercury is persistent and bioaccumulative

Most information on the toxicity of inorganic mercury compounds comes from studies of mercuric chloride. As the water solubility and bioavailability of many other inorganic compounds, notably mercurous compounds, are much less than those of mercuric chloride, such compounds are likely to be clearly less toxic, and the tolerable intake adopted above is likely to err on the conservative side.

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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	10/09/2007
Previous Revisions:	2006A	31/7/2006

Total Petroleum Hydrocarbons

General

Total petroleum hydrocarbons (TPH) is a term used to describe a wide group of chemicals typically derived from petroleum products. TPH is the most common reporting acronym used by environmental laboratories in Australia to describe the measurable amount of petroleum based hydrocarbons in relevant media such as soil and water. TPH data is used extensively as the basis for the assessment of contamination from petroleum products. TPH is also referred to as mineral oil, hydrocarbon oil, extractable hydrocarbon and oil and grease. There are several hundred individual hydrocarbon chemicals defined as petroleum based. In addition each petroleum product has its own mix of constituents which will vary. Crude oil itself varies in composition which is reflected to some degree in the finished petroleum product. Petroleum products are the source of many components but do not define what TPH is. They help define the potential hydrocarbons that become environmental contaminants, but ultimately exposure is determined also by how the product changes with use, by the nature of the release and the hydrocarbon's environmental behaviour.

There are many analytical methods available that measure and report TPH in the environment with no single method providing the whole range of petroleum-derived hydrocarbons. Hence the available methods provide a measure of different sub-sets of the petroleum-derived hydrocarbons on each sample. Hence the definition, measure and interpretation of TPH is highly dependent on the analytical method. If used incorrectly TPH data can be misleading. Hence TPH data must be used (particularly within a risk assessment) and interpreted in conjunction with site-specific data, other analytical data and evaluation of the laboratory method used.

Despite the potential inadequacies of TPH as a contaminant indicator, it is possible to complete meaningful health risk assessments and to develop site-specific remediation criteria for TPH. For a meaningful assessment of potential risks associated with the presence of TPH to be made, the following is necessary:

- An understanding of the analytical method used and the presence of interfering non-petroleum product compounds such as chlorinated solvents and polar compounds. This is a key issue, which affects how TPH will be assessed with respect to exposure and toxicity.
- Some knowledge of the source of the contamination. In many cases, the source of the contamination will be known (e.g. diesel, unleaded fuel etc.). It may also be possible to characterise the source by comparison of the chromatogram against product standards. This can be undertaken on a qualitative basis or can be a detailed almost quantitative assessment using a fingerprinting analysis.
- Measurement of the concentration of the potentially carcinogenic components of the product, which is usually benzene and the carcinogenic PAHs often expressed as benzo(a)pyrene equivalents.
- A conceptual site model describing potential exposure pathways and receptors associated with the contamination identified in various media (such as soils, groundwater and surface water).
- Sufficient data, of sufficient quality to allow quantification of exposure for the key pathways and receptors identified.

Total Petroleum Hydrocarbons

Because TPH is a complex mixture with variable composition depending on sources and time, a generic assessment of the toxicity of TPH is difficult. The assessment of risks associated with exposure to constituents of TPH involves a process of calculating intake of the various fractions distinguishing between volatile fractions and semi volatile fractions (as necessary to enable evaluation of relevant exposure pathways). As toxicity values are not generally available for TPH fractions, the risk assessment typically utilises surrogates or reference chemicals (ie. a single chemical) to provide an estimate of the potential toxicity of specific groups of TPH fractions. Custance *et al* (1992) proposed that the evaluation of the fate of chemical mixtures could be examined by three approaches:

1. Use of indicator chemicals within the specific TPH fractions;
2. Use of surrogates; or
3. Use of toxicity evaluations associated with whole products (such as petrol/gasoline or diesel). Very few whole product toxicity studies have been undertaken. In addition, there are so many variations (between companies and countries) within the one product type that the relevance of the whole product toxicity studies can be questioned. Hence this approach is typically not adopted in Australia or in other countries.

The TPHCWG (1997-1999) details an approach that utilises both individual indicator compounds (for specific compounds such as benzene) and data for whole products or well-defined mixtures that are representative of TPH fractions. The TPHCWG methodology is an extension of the Massachusetts Department of Environment Protection: "Development of Health-Based Alternative to the Total Petroleum Hydrocarbon (TPH) parameter" (MADEP, 1994) approach. This approach is summarised by Turczynowicz in the Contaminated Sites Monograph Series (No.7, 1998) and the WHO (2005) and is typically adopted for use in risk assessments in Australia.

The choice of the surrogate is dependent on the type of product being assessed. The toxicity value for the surrogate is taken to be representative of the whole TPH fraction. In general, the selection of a surrogate will reflect the more toxic (or more closely studied) hydrocarbon within the fraction assessed. The surrogate may be an aliphatic¹ compound (e.g. hexane) or an aromatic² compound such as toluene or a non-carcinogenic PAH such as naphthalene or pyrene. It is important when using this technique to account for highly toxic or potentially carcinogenic individual chemicals. These should be quantified and assessed separately. The concentration of the carcinogenic constituents may be subtracted from the relevant TPH fraction group with the remainder assessed on the basis of the toxicity of an appropriate surrogate.

The commonly reported TPH fraction groups in Australia (C₆-C₉, C₁₀-C₁₄, C₁₅-C₂₈ and C₂₉-C₃₆) do not follow the approach used by TPHCWG and other sources. The TPHCWG reports TPH bands as the following for aliphatic constituents TPH C₅-C₆, TPH C₆-C₈, TPH C₈-C₁₀, TPH C₁₀-C₁₂, TPH C₁₂-C₁₆ and TPH C₁₆-C₂₁. For aromatic constituents it is TPH C₅-C₇, TPH C₇-C₈, TPH C₈-C₁₀, TPH C₁₀-C₁₂, TPH C₁₂-C₁₆, TPH C₁₆-C₂₁ and TPH C₂₁-C₃₅. The TPHCWG have made

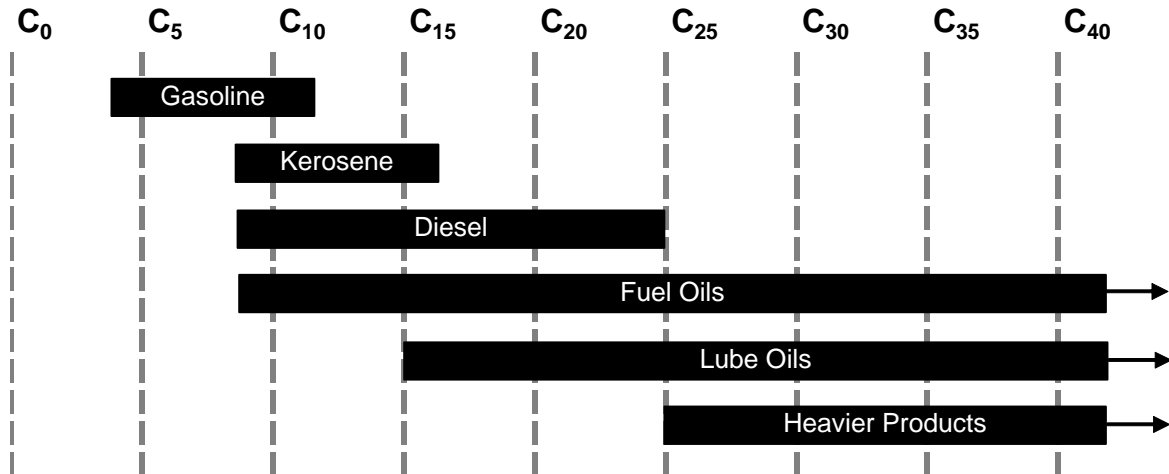
¹ Aliphatic hydrocarbons consist of linear chains and branched chains of carbon atoms.

² Aromatic hydrocarbons consist of closed rings of carbon atoms. The building block of aromatic hydrocarbons is the benzene ring. Many aromatics have distinct odours (but not all). Aromatics that contain several fused (jointed) rings are called polycyclic aromatics.

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preliminary recommendations for toxicity values for TPH for the following bands aliphatic C₅-C₈, C₈-C₁₆ and C₁₆-C₃₅ and aromatics C₇-C₈, C₈-C₁₆ and C₁₆-C₃₅. These groupings simplify the TPH characteristics, for example Equivalent Carbon (EC) > 8-16 range for which toxicity criteria have been selected include some 45 petroleum hydrocarbons identified in the compositional analysis of petroleum streams reviewed by TPHCWG. Use of the TPHCWG bands with data provided by Australian laboratories requires some degree of approximation. The following figure presents a general range of TPH fractions that constitute key petroleum products evaluated on most sites.

TPH Fractions



(Source: CONCAWE report no. 3/03: European Oil Industry Guideline for Risk-Based Assessment of Contaminated Sites (revised), July 2003)

Properties

The evaluation of TPH has focused on the fraction groups typically provided by analytical methods adopted in Australia (C₆-C₉, C₁₀-C₁₄, C₁₅-C₂₈ and C₂₉-C₃₆) with further spilt into aromatic and aliphatic fractions to enable further assessment of properties and toxicities that are associated with these groupings. It is noted that at present these groupings differ from those presented in key review documents such as TPHCWG (1997/1999) and MADEP (1994), hence the values presented in this summary are based in review of the available information and professional judgement as to the parameters that are relevant to the TPH groups assessed.

Key properties are presented below for the TPH fractions. It is noted that many properties are not available for the general TPH fractions identified and hence properties associated with the key surrogate identified for each group have been used (ref: CSMS 1998, TPHCWG 1997-1999, ATSDR 1999, USEPA 2004 and RAIS):

Total Petroleum Hydrocarbons

Aromatic TPH Fractions

	C ₆ -C ₉	C ₁₀ -C ₁₄	C ₁₅ -C ₂₈	C ₂₉ -C ₃₆
Key Surrogate	toluene	naphthalene	pyrene	pyrene
Volatile Fractions	yes	yes	no	no
Molecular Weight	120	130	190	240
Vapour Pressure (mmHg)	29	0.48	0.00084	0.00000033
Vapour Density	3.2*	4.4*	NA	NA
Density (g/ml)	0.867 *	1.15*	1.27	1.27
Solubility (mg/L at 20-25°C)	520	25	0.65	0.0066
Air Diffusion Coefficient (cm ² /s)	0.086*	0.048	0.027*	0.027*
Water Diffusion Coefficient (cm ² /s)	8.6x10 ⁻⁶	7.7x10 ⁻⁶	7.2x10 ⁻⁶ *	7.2x10 ⁻⁶ *
Henry's Law Coefficient (unitless)	0.27	0.14	0.412	0.412
Koc (cm ³ /g)	251	2510	62900	62900
Log Kow	2.73*	3.3*	4.88*	4.88*
Odour Threshold	30.6 mg/m ³ *	0.44 mg/m ³ *	NA	NA
Dermal Absorption (unitless)	0.01*	0.13*	0.13*	0.13*
Permeability Constant (cm/hr)	0.0453*	0.0694*	0.324*	0.324*

* Data for surrogate chemical rather than TPH fraction group

Aliphatic TPH Fractions

	C ₆ -C ₉	C ₁₀ -C ₁₄	C ₁₅ -C ₂₈	C ₂₉ -C ₃₆
Key Surrogate	n-hexane	nonane and JP-8	eicosane and mineral oil	eicosane and mineral oil
Volatile Fractions	yes	yes	no	no
Molecular Weight	100	160	270	280
Vapour Pressure (mmHg)	48	0.48	0.00084	0.00084
Vapour Density	2.97*	4.41*	NA	NA
Density	0.66 *	0.775 – 0.84*	0.875 – 0.905*	0.875 – 0.905*
Solubility (mg/L at 20-25°C)	5.4	0.034	0.000025	0.000015
Air Diffusion Coefficient (cm ² /s)	0.067	0.046	0.033	0.033
Water Diffusion Coefficient (cm ² /s)	6.9x10 ⁻⁶	5.2x10 ⁻⁶	3.3x10 ⁻⁶	3.3x10 ⁻⁶
Henry's Law Coefficient (unitless)	50	130	110	110
Koc (cm ³ /g)	3160	316000	400000000	400000000
Log Kow	3.29 *	3.3 – 7.06*	NA	NA
Odour Threshold	458 mg/m ³ *	3.5 mg/m ³ *	NA	NA
Dermal Absorption (unitless)	0.01*	0.01*	0.01*	0.01*
Permeability Constant (cm/hr)	0.334*	NA	NA	0.0453

* Data for surrogate chemical rather than TPH fraction group

Exposure

Petroleum products are an integral part of modern life with exposure to hydrocarbons from many common sources including refuelling cars, spilled oils, asphalt, solvents used at home or in workplace or pesticide applications that use petroleum products as carriers. Petroleum products are released into the environment through accidents, managed released or as unintended by-products of industrial, commercial or private activities. Exposure to petroleum hydrocarbons may therefore include inhalation of volatile components as well as ingestion and dermal contact with hydrocarbons present in water and soil as well as many products.

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The nature of the chemical composition of a petroleum release into the environment is affected and complicated by hydrodynamic, abiotic and biotic processes that act on the release to change the chemical character. The longer the release is exposed in the environment the greater the change in chemical character and the more difficult the assessment becomes. After significant weathering detailed knowledge of the original bulk product becomes less valuable and more current site-specific data associated with individual hydrocarbon components and TPH fractions become more relevant.

If released into the environment the following general points can be noted with respect to petroleum hydrocarbons (ATSDR, 1999):

- Bulk product migration: petroleum products released into soil migrates as a bulk product flow via gravity and capillary action and as individual compounds separating from the bulk petroleum mixture and dissolving in air or water. With bulk flow, little or no separation of individual compounds occurs. Compounds that are insoluble and immobile in water are soluble in the bulk product and will migrate with the bulk product flow. With this migration some mass will be retained by soil particles as “residual saturation” that may remain for years. When the release is large the product may migrate downwards to groundwater (water saturated zone) where the product will float due to the density of hydrocarbon products (referred to as LNAPL).
- Compound migration: As the bulk product moves through soil, individual compounds may separate from the mixture and migrate independently. This is dependent on volatility, solubility and sorption potential. The volatility of compounds is primarily a function of the vapour pressure. As petroleum products are a complex mix of hundreds of compounds, the compounds that have relatively high vapour pressure will volatilise and enter the vapour phase. The composition of the vapours depends on the composition of the product. In petrol volatile compounds such as butane, propane, benzene, toluene, ethylbenzene and xylene are preferentially volatilised. The propensity to volatilise from an aqueous solution can be roughly estimated using Henry’s law. The greater the solubility of a compound the greater the likelihood that the chemicals will dissolve into rainwater or groundwater and migrate further. In general solubility decreases with increasing molecular weight of hydrocarbons. The ability of a compound to partition between water and organic carbon in soil affects mobility. In general benzene is considered mobile while toluene, ethylbenzene and xylenes are considered to have intermediate mobility.
- Biodegradation: the rate of biodegradation of hydrocarbons depends on the chemical composition of the product as well as site-specific factors. In general the straight chain hydrocarbons and aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics and aromatics in the C₁₀-C₂₂ range are the most readily biodegradable; the n-alkanes, n-alkyl aromatics and aromatics in the C₅-C₉ range are biodegradable at low concentrations by some micro organisms but preferentially removed via volatilisation; n-alkanes in the C₁-C₁₁ range are biodegradable by specialised hydrocarbon degraders; and n-alkanes, n-alkyl aromatics and aromatics above C₂₂ are not available for degradation. Hydrocarbons with condensed ring structures such as PAHs have been shown to be resistant to degradation. PAHs with only 2 or 3 rings such as naphthalene and anthracene are more easily degraded. Many of the water soluble fractions may be degraded as the compounds go into solution. Environmental factors affect biodegradation with oxygen being essential for effective biodegradation of oils. Anaerobic degradation rates are very low.

Total Petroleum Hydrocarbons

- Bioaccumulation: In general lower molecular weight petroleum hydrocarbons do not bioaccumulate. Some TPH fractions, particularly PAHs, bioaccumulate to some extent however depuration does occur if the source of contamination is removed

Health Effects

As TPH consists of hundreds of individual chemicals specific health effects associated with the presence of TPH will vary depending on the nature of the product and composition of individual hydrocarbons within the TPH. In the application of TPH assessment approaches provided by TPHCWG and MADEP the presence of individual chemicals that are associated with non-threshold carcinogenic risk must be assessed on an individual basis. This typically includes benzene and carcinogenic PAHs (using benzo(a)pyrene equivalents). Hence the further evaluation of potential exposure to TPH is associated with the assessment of typical health effects associated with key or indicator chemicals within the hydrocarbon fractions and assessed on the basis of threshold effects.

With regards to typical health effects associated with key or indicator chemicals within the approximate TPH fractions quantified in Australia, the following is provided by ATSDR (1999) and TPHCWG (1997-1999):

Aromatic C ₆ -C ₉	<p>Primarily consists of benzene (evaluated separately), toluene, ethylbenzene and xylenes (BTEX). This fraction can be assessed by quantifying exposure associated with each individual chemical. Fractions may be absorbed via inhalation, oral or dermal exposure and distributed widely to tissues and organs. Metabolism represents toxification pathway for some BTEXs (benzene) and a detoxification pathway for others (such as toluene).</p> <p>Inhalation – BTEX causes neurological effects, primarily central nervous system depression. Developmental effects have also been associated with exposure to ethylbenzene and xylenes.</p> <p>Oral – BTEX also causes neurological effects (central nervous system depression) via the oral route or exposure. Renal and hepatic effects have also been reported for toluene and xylenes.</p> <p>Dermal – Limited information available. Skin and eye irritation are associated with BTEX but no effects associated with absorption.</p> <p>Surrogate or indicator chemical: toluene</p>
Aromatic C ₁₀ -C ₁₄	<p>Primarily consists of cumene (isopropylbenzene), n-propylbenzene, methyl-ethylbenzene, trimethylbenzenes, butyl and pentylbenzene, methylindanes and naphthalene, alkyl benzenes and some methylnaphthalenes. Fractions may be absorbed via inhalation, oral or dermal exposure and distributed widely.</p> <p>Inhalation – Critical effects associated with key chemicals include hepatic and renal effects, haemolytic anaemia and respiratory effects (naphthalene).</p> <p>Oral – Critical effects associated with key chemicals include renal, hepatic and other systemic effects. Naphthalene produces haemolytic anaemia following ingestion.</p> <p>Dermal – Known to be irritating to skin but little data available to suggest systemic toxicity from dermal exposure.</p> <p>Surrogate or indicator chemical: naphthalene</p>

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Aromatic C ₁₅₊	<p>Primarily consists of PAHs including anthracene, fluorene, phenanthrene, pyrene, benzo(a)anthracene, benzo(b)-, benzo(j)-, and benzo(k)fluorathene, benzo(ghi)perylene, benzo(a)- and benzo(e)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-c,d)pyrene. Carcinogenic PAHs must be evaluated individually and remaining considered within TPH fraction. These fractions are absorbed to varying extents via inhalation, oral or dermal exposure depending on lipophilicity and molecular size of the compound. Once absorbed they are widely distributed to tissues and organs.</p> <p>Inhalation – Limited data available for chemicals other than benzo(a)pyrene. Not volatile fractions.</p> <p>Oral – Critical effects associated with key chemicals include hepatic effects. Renal effects have been reported with some PAHs as well as aplatic anaemia and immunological/lymphoreticular effects.</p> <p>Dermal – Known to be irritating to skin but little data available to suggest systemic toxicity from dermal exposure except for benzo(a)pyrene.</p> <p>Surrogate or indicator chemical: pyrene</p>
Aliphatic C ₆ -C ₉	<p>Primarily consists of n-pentane, n-hexane, dimethylbutanes, methylpentanes, cyclopentane, n-heptane, n-octane, trimethylpentanes and cyclohexane, methylcyclopentane and methylcyclohexane. These fractions may be readily absorbed in the lungs, absorption via oral and dermal routes not well characterised. Once absorbed, the fractions are widely distributed to tissues, preferentially to fatty tissues and perfused tissues, metabolised in the liver.</p> <p>Inhalation – acute exposure has been associated with respiratory and neurological effects. Chronic and intermediate exposures to n-hexane (most significant) cause peripheral neuropathy in humans and animals. Other chronic effects include renal, respiratory and reproductive effects (animal studies).</p> <p>Oral – Limited data is available but n-hexane has resulted in peripheral neuropathy in animal studies. Higher doses have results in reproductive and developmental effects.</p> <p>Dermal – Limited information available. Some compounds in this fraction group are irritating to the skin and eyes, but there is little information suggesting systemic toxicity from dermal exposure.</p> <p>Surrogate or indicator chemical: n-hexane</p>
Aliphatic C ₁₀ -C ₁₄	<p>Primarily consists of n-nonane, n-decane, a few substituted cycloalkanes, n-undecane, n-dodecane, pentylcyclopentane, n-tri-, tetra-, penta- and hexadecane and the products JP-8, JP-7 and kerosene (fuel oil #1), stoddard solvent and dearomatised petroleum streams. These fractions may be readily absorbed by the lungs, widely distributed to tissues (preferentially to fatty tissues) and slowly eliminated from fatty tissues.</p> <p>Inhalation – Critical effects associated with key chemicals include hepatic effects. Other effects include neurological effects (central nervous depression) and neuropathy.</p> <p>Oral – Critical effects associated with key chemicals include renal, hepatic and neurological effects.</p> <p>Dermal – Known to be irritating to skin and eyes but little data available to suggest systemic toxicity from dermal exposure.</p> <p>Surrogate or indicator chemical: JP-8</p>
Aliphatic C ₁₅₊	<p>Primarily consists of n-hepta, n-octa and n-nonadecane and n-eicosadecane, n-heneicosane, n-docosane, n-tetracosane and n-hexacosane and the products</p>

Total Petroleum Hydrocarbons

mineral-based crankcase oil and hydraulic fluids as well as other mineral oils. These fractions are poorly absorbed, regardless of the route of exposure and preferentially distributed to the liver and fatty tissues and slowly metabolised.

Inhalation – No data available as TPH within this fraction are not considered volatile and no data available on inhalation exposures to compounds or products in these fraction grouping.

Oral – Purified mineral oils have been used medicinally and in foods. Critical effects associated with key chemicals include hepatic and lymph node effects. hepatic effects.

Dermal – No data available.

Surrogate or indicator chemical: mineral oils

There are no toxicity data relevant to the highest-molecular weight compounds in the fractions C₃₅₊, however these compounds are not volatile, compounds C₂₀₊ are not soluble in water and C₃₅₊ compounds are not likely to be absorbed via oral or dermal routes of exposure (WHO, 2005).

Toxicity Classification

No carcinogenic classification is available for TPH as a mixture, however individual compounds within TPH have individual classifications available from USEPA and IARC (refer to individual chemical profiles for details)

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to TPH, intakes from air, soil, water and food can be considered to be low. However actual background values are not available and based on the expected widespread prevalence of many TPH compounds (as products of combusting) a background intake of 10% has been assumed for all TPH fractions assessed. This evaluation does not include potential background intake that may be derived from specific workplace activities such as motor vehicle maintenance or areas located closer to significant combustion sources.

Toxicity Values

Toxicological data relevant for key indicator or surrogate chemicals identified in aromatic or aliphatic TPH groupings noted above are adopted in the quantification of potential exposure. The values selected are those presented by the WHO (2005) in the derivation of drinking water guidelines with data not presented by the WHO obtained from the TPHCWG (1997-1999), used as a principal review source by the WHO. It is noted that where individual surrogate compounds have been used as the basis for a toxicity value, and the available toxicity data for

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that surrogate is reviewed and updated (since publication of the TPHCWG in 1997 -1999), the revised toxicity value is adopted (as referenced and noted).

The values adopted for the effective carbon (EC) ranges presented (typically reported by laboratories in Australia) are based on the most dominant EC group (ie the group that overlaps the most with respect to EC ranges) presented from the WHO (2005) or TPHCWH (1997-1999) references.

Workplace exposure values are not available for specific TPH groups, however values relevant to the appropriate surrogate compound for each group have been adopted. These values have been derived from ASCC or other international bodies such as ACGIH or NIOSH (current to 2005).

The following presents the toxicity values identified for the evaluation of potential exposure to TPH fractions (aromatic or aliphatic). These values are threshold values relevant for the TPH groups identified. It is noted that carcinogenic compounds, such as benzene or benzo(a)pyrene (equivalents) are required to be quantified and assessed on an individual basis as the application of the TPH threshold values does not address these specific non-threshold carcinogenic effects.

It is noted that no dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity where appropriate in the evaluation of exposure to the following TPH fractions.

Aromatic TPH Fractions

	C ₆ -C ₉	C ₁₀ -C ₁₄	C ₁₅ +
Key Surrogate	toluene	naphthalene	pyrene
Volatile	yes	yes	no
Oral TDI (mg/kg/day)	0.2	0.03	0.03
Inhalation Guideline (mg/m ³)	0.4	0.2	NA. For the evaluation of inhalation (particulates) adopt oral value
Occupational Inhalation Values			
TWA	50ppm = 191 mg/m ³	10ppm = 52 mg/m ³	NA
STEL	150ppm = 574 mg/m ³	15ppm = 79 mg/m ³	
Critical Effect	Hepatotoxicity, nephrotoxicity	Decreased body weight	Nephrotoxicity
Background	10%	10%	10%

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Aliphatic TPH Fractions

	C ₆ -C ₉	C ₁₀ -C ₁₄	C ₁₅ +
Key Surrogate	n-hexane	nonane and JP-8	eicosane and mineral oil
Volatile Fractions	yes	yes	no
Oral Value	5	0.1	2
Inhalation Value	0.7 (based on 2005 review of n-hexane from USEPA [IRIS])	1.0	NA. For the evaluation of inhalation (particulates) adopt oral value
Occupational Inhalation Values			
TWA	20ppm = 72 mg/m ³	For JP-8 (NIOSH): PEL = 350 mg/m ³	From ACGIH for mineral oil: TLV = 5 mg/m ³ (mineral oil mist – no data for particulates)
STEL	NA	1800 mg/m ³	
Critical Effect	Nephrotoxicity	Hepatic and haematological	Hepatic (foreign body reaction) granuloma
Background	10%	10%	10%

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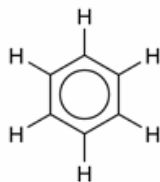
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Revision Dates

Document	Revision	Date of Review	Comments
Current Toxicity Summary	2008A	13/02/2008	Incorporated revised inhalation data from US review of n-hexane
Previous Revisions:	2006B	12/12/2006	
	2006A	16/8/2006	
	2005A	19/09/2005	

Benzene

General



Benzene found in the environment is from both human activities and natural processes. Benzene was first discovered and isolated from coal tar in the 1800s. Today, benzene is made mostly from petroleum sources. Because of its wide use, benzene is produced in large volumes in many countries.

Various industries use benzene to make other chemicals, such as styrene (for Styrofoam® and other plastics), cumene (for various resins), and cyclohexane (for nylon and synthetic fibres). Benzene is also used for the manufacturing of some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene, which include volcanoes and forest fires, also contribute to the presence of benzene in the environment. Benzene is also a natural part of crude oil, petrol and cigarette smoke (ATSDR, 1997).

Properties

Benzene, also known as benzol is a volatile, colourless liquid with a characteristic "aromatic" odour. Benzene evaporates into air very quickly and dissolves slightly in water. Benzene is highly flammable. Key properties are presented below (ref: ATSDR 1997, USEPA 2004, HSDB 2007 and RAIS):

CAS No	71-43-2
Chemical Formula	C ₆ H ₆
Molecular Weight	78.12
Vapour Pressure	95.2 mmHg at 25°C
Vapour Density	2.77
Density	0.8787 g/ml at 20°C
Solubility	1780 mg/L
Air Diffusion Coefficient	0.088 cm ² /s
Water Diffusion Coefficient	9.8 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.00555 atm.m ³ /mol = 0.228 at 25°C (unitless)
Koc	58.9 cm ³ /g
Log Kow	2.13
Odour Threshold	4.9 mg/m ³ (ATSDR, 1997)
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0207

Exposure

Exposure of the general population to benzene may occur in all urban areas, as motor vehicle emissions are a contributor to benzene levels. Inhalation is the primary route of exposure in industrial and everyday settings. Cigarette smoke contains benzene and is a significant exposure for active smokers. Other exposures include furnishings, solvents, adhesives, pumping petrol and residential areas near chemical manufacturing sites. Trace amounts are typically found in food and water.

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If released into the environment the following can be noted with respect to benzene (NICNAS, 2001):

- **Air:** Benzene is volatile and models indicate that 99% of benzene will partition to air, with 0.88% and 0.05% partitioning to water and soil respectively. In air benzene may be removed via rainwater, however it is expected to rapidly volatilise back into the atmosphere where it may be available for abiotic breakdown. Direct photolysis of benzene is unlikely to occur. The major degradation pathway for benzene in air is indirect photolysis through reaction with hydroxyl radicals with an estimated half life of 8 days.
- **Water:** Benzene is largely abiotically stable in water, with the major removal process expected to be volatilisation. Benzene has a high water solubility and low Kow indicating that it is not expected to absorb significantly to organic matter or sediments.
- **Soil:** Adsorption of benzene to soil and sediment is limited and based on Koc values, benzene is expected to be highly mobile in soils and may migrate to groundwater. The primary mechanism for loss of benzene from soils is via volatilisation to the atmosphere and runoff to surface water.
- **Biodegradation:** In surface and groundwater benzene is biodegradable by microorganisms under both aerobic and anaerobic conditions, although under anaerobic conditions biodegradation is expected to be very slow. Biodegradation in groundwater and river water appears to follow first-order rate kinetics with reported half-lives of 28 and 16 days respectively.
- **Bioconcentration** in aquatic or terrestrial organisms is estimated to be low.

Health Effects

There is no clinical disease which is unique to benzene toxicity. However the effects on the haematopoietic and immune systems are well recognised. Data from animal and human studies indicates that benzene is rapidly absorbed through the lungs. Definitive scientific data on the rate of absorption after ingestion of benzene in humans are not available. However case studies of accidental or intentional poisoning indicate that it is absorbed readily. Benzene can be absorbed through the skin, however the rate of absorption is much lower than that for inhalation.

Once absorbed, benzene partitions to lipid-rich tissues due to the lipophilic nature of the chemical with total uptake dependant on fat content and metabolism. Benzene accumulates in the adipose tissue, bone marrow and brain. The metabolism of benzene is rapid with water-soluble metabolites excreted within 2 hrs of exposure. A substantial proportion of absorbed benzene is eliminated unchanged in exhaled air, with the remainder eliminated in the urine, principally as metabolites. Benzene is metabolised primarily in the liver and to a lesser extent, in the bone marrow. There is no evidence that the route of administration has any substantial effect on subsequent metabolism of benzene in humans or animals.

Acute benzene exposure produces central nervous system excitation and depression. Acute exposure to high concentrations of benzene in air results in neurological toxicity and may sensitize the myocardium to endogenous catecholamines. Acute ingestion of benzene causes gastrointestinal and neurological toxicity (WHO, 1993).

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Chronic exposure to benzene results primarily in haematotoxicity, including aplastic anaemia, pancytopenia, or any combination of anaemia, leukopenia, and thrombocytopenia. Chronic benzene exposure is associated with an increased risk of leukaemia. In chronic exposures, benzene metabolites are considered the toxic agents, not the parent compound. The relative contribution of different benzene metabolic pathways may be dose related, with more toxic agents produced by high affinity low capacity pathways (WHO, 1993).

The following summary of more specific health effects has been derived from (Toronto Public Health, 2002).

Death	Acute inhalation and oral exposure of benzene has been known to result in death in humans. Cause of death is typically asphyxiation, respiratory arrest, CNS depression or cardiac collapse.
Gastrointestinal Effects	Ingestion of benzene has been associated with gastrointestinal effects which include congestive gastritis, intense toxic gastritis and pyloric stenosis.
Haematological Effects	Exposure by all routes causes toxic effects in the haematological system. All blood cells are susceptible. Less severe toxicity is characterised by specific reductions in the counts of individual blood elements. More severe toxicity is characterised by a reduction in all blood elements.
Hematopoietic Effects	Chronic exposure to benzene can lead to bone marrow damage which may manifest initially as anaemia, leukopenia or thrombocytopenia. Continued exposure may result in marrow aplasia and pancytopenia, an often fatal outcome.
Reproductive Effects	Reproductive effects have been observed in women occupationally exposed to benzene.
Developmental Effects	In animals, exposure to benzene has been shown to cause adverse effects (low birth weight, delayed bone formation and bone marrow damage) on the developing foetus.
Dermal/Ocular Effects	Exposure to high doses of benzene through contact with the air or skin and result in dermal effects which include erythema, oedema, frank burns and necrosis. Ocular effects such as moderate conjunctival irritation and transient corneal damage have been observed in occupational environments.
Immunological Effects	Effects have been identified following inhalation exposures. These include humoral (antibody) and cellular (leukocyte) response damage.
Genotoxic Effects	Studies show that benzene and/or its metabolites are genotoxic in humans. The targets of genotoxicity are peripheral lymphocytes and bone marrow cells.
Cancer	Epidemiologic studies and case studies provide clear evidence of a causal association between exposure to benzene and acute nonlymphocytic leukemia (ANLL) and also suggest evidence for chronic nonlymphocytic leukemia (CNLL) and chronic lymphocytic leukemia (CLL). Other neoplastic conditions that are associated with an increased risk in humans are hematologic neoplasms, blood disorders such as preleukemia and aplastic anemia, Hodgkin's lymphoma, and myelodysplastic syndrome (MDS). These human data are supported by animal studies. The experimental animal data add to the argument that exposure to benzene increases the risk of cancer in multiple species at multiple organ sites (hematopoietic, oral and nasal, liver, forestomach, preputial gland, lung, ovary, and mammary gland). It is likely that these responses are due to interactions of the metabolites of benzene with DNA. Recent evidence supports the viewpoint that there are likely multiple mechanistic pathways leading to cancer and, in particular, to leukemogenesis from exposure to benzene. Based on available data, benzene has the potential to cause cancer in humans following inhalation or oral exposure.

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Toxicity Classification

Benzene is classified as a “known” human carcinogen (Category A) by the USEPA under the Risk Assessment Guidelines of 1986 (and USEPA 1996) for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies.

IARC has classified benzene in Group 1 (known human carcinogen).

NICNAS (2001) has classified benzene as a “Carcinogen, Category 1” and “Toxic: Danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed”. In addition benzene is classified as “Irritating to eyes, respiratory system and skin” and as a mutagenic substance in Category 3 “Possible risks of irreversible effects”.

Exposure Limits and Toxicity Evaluations

Australia

The Australian Drinking Water Guidelines (2004) note that “benzene is a genotoxic human carcinogen and there is no safe or acceptable concentration in drinking water”. The guidelines provide a value of 0.001 mg/L based the limit of determination. The guideline values is also noted to be equal to the value that would be derived following the WHO Drinking Water Guidance for benzene (refer below) and adopting a maximum lifetime risk of one extra case of leukaemia per million people.

A benchmark dose for benzene has been derived (Dempsey in EPHC, 2003) on the basis of malignant lymphomas observed in male and female mice (regarded as relevant to haematopoietic tumours observed in humans) $BMD_{0.5} = 16.64$ to 26.31 mg/kg/day. A modifying factor of 10,000 (has been applied to derive a guideline BMD of 1.7 to 2.6 μ g/kg/day. The guideline dose suggested is 1.7 μ g/kg/day. A BMD based on human studies has also been derived. The BMD is derived from acute myloid leukaemia in benzene workers from China (inhalation exposures) and used with a modifying factor of 2000. On the basis of this approach a guideline value of 0.3 ppm has been derived. These guideline values have not been endorsed by NHMRC and have not been accepted for quantification of risk by Australian regulators.

Worksafe Australia (NOHSC, available in 2007) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment”. For benzene, the following have been established:

TWA: 1 ppm, equivalent to 3.2 mg/m³

STEL: not established

WHO

The WHO Drinking Water Guidelines (2004) have established a guideline value of 0.01 mg/L based on a linear extrapolation model applied to leukaemia and lymphomas in female mice and oral cavity squamous cell carcinomas in male rats from a 2 year study. The guideline value has been derived utilising a 10^{-5} upper bound excess lifetime cancer risk. The value derived is the same as derived in previous version of the drinking water guidelines derived from human

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leukaemia data from inhalation exposures (1984), carcinogenicity in mice and rats (1993) and also noted to be equal to the estimate derived from epidemiological data. The drinking water guidelines have been derived using an equivalent slope factor of:

$$\begin{aligned}
 SF \text{ (mg/kg/day)}^{-1} &= \text{Risk/Intake(mg/kg/day)} \\
 &= [\text{Risk} \times \text{Body Weight}]/[\text{Concentration (in water)} \times \text{Ingestion Rate}] \\
 &= [1 \times 10^{-6} \times 70 \text{kg}]/[0.001 \text{mg/L} \times 2 \text{L/day}] \\
 &= 0.035 \text{ (mg/kg/day)}^{-1}
 \end{aligned}$$

WHO (2000) established an inhalation unit risk based on a risk estimate using data on leukaemia from epidemiological studies involving inhalation exposures to benzene in air of $4.4 - 7.5 \times 10^{-6} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ with a geometric mean of $6 \times 10^{-6} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ (i.e. for an air concentration of $1 \text{ }\mu\text{g/m}^3$, the lifetime risk is estimated to be 6×10^{-6}). As no value within the range is recommended, the geometric mean has been used, which is equivalent to the following slope factor:

$$\begin{aligned}
 SF \text{ (mg/kg/day)}^{-1} &= \text{Risk/Intake(mg/kg/day)} \\
 &= [\text{Risk} \times \text{Body Weight}]/[\text{Concentration (in air)} \times \text{Inhalation Rate}] \\
 &= [6 \times 10^{-6} \times 70 \text{kg}]/[0.001 \text{mg/m}^3 \times 20 \text{ m}^3/\text{day}] \\
 &= 0.021 \text{ (mg/kg/day)}^{-1}
 \end{aligned}$$

EU

The EU has prepared a draft review of benzene (EU, 2003) that reviews occupational, consumer and environmental exposures. Benzene is identified as a genotoxic carcinogen where “no safe level of exposure is recommended” by the EU. Limited quantification of exposure to carcinogenic effects has been presented with a starting point for occupational exposure of 1ppm in air and 16 mg/person/day internal dose used to evaluate carcinogenic exposures. A Margin of Exposure (MOE) of at least 10 is recommended. No quantification of lifetime risk is presented for occupational, consumer or environmental exposures on the basis of carcinogenic effects.

Evaluation of exposure to non-neoplastic effects (using a NOAEL of 3.2 mg/m^3), fertility effects (NOAEL of 96 mg/m^3) and developmental toxicity (NOAEL of 31.9 mg/m^3) has been undertaken to determine relevant Margins of Safety (MOS).

US

The USEPA (available from IRIS 2007) provides the following with respect to non-cancer and carcinogenic evaluations of benzene:

- Oral reference dose (RfD) of 0.004 mg/kg/day has been established from route-to-route extrapolation from the results of benchmark dose modelling on the basis of decreased lymphocyte count from a human occupational inhalation study.
- Inhalation reference dose of 0.03 mg/m^3 established from the same study as used for the RfD above.

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- Oral slope factor of 0.015 to 0.055 $(\text{mg}/\text{kg}/\text{day})^{-1}$ has been derived on the basis of leukaemia from human inhalation occupational exposure data following route extrapolation from inhalation model below.
- Inhalation unit risk of 2.2×10^{-6} to 7.8×10^{-6} $(\mu\text{g}/\text{m}^3)^{-1}$ has been derived on the basis of leukaemia from human inhalation occupational exposure data and use of a linear dose-response model.

The ATSDR (1997) has established Minimal Risk Levels (MRLs) for the evaluation of non-cancer effects. The following MRLs have been derived for benzene:

- Acute inhalation MRL of 0.05 ppm based on immunological effects in mice;
- Intermediate inhalation MRL of 0.004 ppm based on neurological effects in mice

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. However, as benzene has been evaluated to be a genotoxic carcinogen by all routes of exposure it is considered appropriate to evaluate exposure using a slope factor for oral, inhalation and dermal exposures. Hence background intake is not relevant to this assessment.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for benzene following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	Slope Factor = 0.035 $(\text{mg}/\text{kg}/\text{day})^{-1}$ (WHO 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation Unit Risk = 6×10^{-6} $(\mu\text{g}/\text{m}^3)^{-1}$ (WHO, 2000), with an equivalent inhalation slope factor of 0.021 $(\text{mg}/\text{kg}/\text{day})^{-1}$. Occupational inhalation exposure (NOHSC, current): TWA: $1\text{ppm} = 3.2 \text{ mg}/\text{m}^3$ STEL: NA

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BTEX Mixtures

Where benzene is present in conjunction with toluene, ethylbenzene and/or xylenes potential effects associated with the mixture should be addressed. This has followed on from a review of interactions of mixtures undertaken by ATSDR (2004) that identified the potential issues associated with BTEX mixtures. Further detail associated with the approach adopted for the assessment of BTEX mixtures is presented in the BTEX Mixture toxicity summary.

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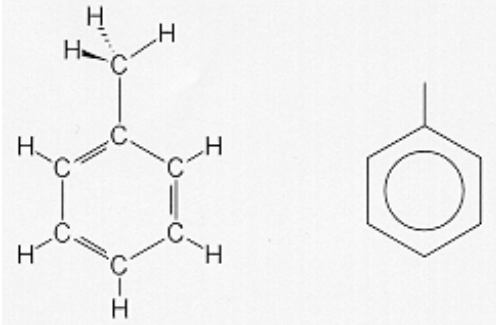
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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	28/8/2007
Previous Revisions:	2006A	31/7/2006
	2005A	29/3/2005

Toluene

General



Toluene is a clear, colourless liquid with a distinctive smell. Toluene occurs naturally in crude oil and in the tolu tree. It is produced in the process of making petrol and other fuels from crude oil, in making coke from coal, and as a by-product in the manufacture of styrene. Toluene is used in making paints, paint thinners, fingernail polish, lacquers, adhesives, and rubber and in some printing and leather tanning processes. It is disposed of at hazardous waste sites as used solvent (a substance that can dissolve other substances) or at landfills where it is present in discarded paints, paint thinners, and fingernail polish (ATSDR, 2000).

Properties

Toluene, also known as methylbenzene and phenylmethane, is a colourless liquid with a sweet pungent, benzene-like odour. Toluene is highly volatile and is subject to microbial degradation. Toluene is flammable. Key properties are presented below (ref: ATSDR 2000, USEPA 2004, HSDB and RAIS):

CAS No	108-88-3
Chemical Formula	C ₆ H ₅ CH ₃
Molecular Weight	92.12
Vapour Pressure	28.4 mmHg at 25°C
Vapour Density	3.2
Density	0.867 g/ml at 20°C
Solubility	526 mg/L
Air Diffusion Coefficient	0.087 cm ² /s
Water Diffusion Coefficient	9.6 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.00664 atm.m ³ /mol = 0.272 at 25°C (unitless)
Koc	182 cm ³ /g
Log Kow	2.73
Odour Threshold	30.6 mg/m ³ in air
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0453 cm/hr
Air Conversion factors	1 ppm = 3.75 mg/m ³ (at 20 °C)

Exposure

Inhalation is the primary route of toluene exposure for the general population and for occupationally exposed individuals. Evaporation of petrol and vehicle exhaust is the largest source of toluene in the environment, and industries that use toluene as a solvent are the second largest source. Toluene is also a common indoor contaminant due to releases from common household products and from cigarette smoke. Non-atmospheric releases of toluene are relatively small (e.g., to water and soil) and are estimated to comprise less than 1% of total toluene releases. In the atmosphere, toluene is degraded by reaction with hydroxyl radicals,

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with a typical half-life of about 13 hours. It is highly volatile, and volatilisation is likely to be the predominant removal process from soil and water. Trace amounts are typically found in food and water. Toluene is absorbed via ingestion, inhalation, and skin application.

If released into the environment the following can be noted with respect to toluene (WHO, 1985):

- **Air:** Toluene is volatile and most of the chemical released into the environment enters the atmosphere. Atmospheric oxidation of toluene removes 50% of the compound in less than 2 days (half-life was estimated to be 12.8 h). Because of this rapid removal, toluene will most probably not remain in the atmosphere long enough to be removed by air to surface transfer mechanisms, such as dry deposition or precipitation. Toluene is removed from the atmosphere primarily through free radical chain processes, of which reactions with hydroxy radicals are the most important processes.
- **Water:** Toluene is expected to volatilise from surface water as well as from groundwater. Factors affecting toluene levels in surface water and groundwater include volatilisation, solubility, and, where groundwater is concerned, degradation and/or adsorption
- **Soil:** Based on log K_{oc} coefficient (2.25), toluene is expected to adsorb to soils rich in organic matter, however the chemical will leach from soils low in organic content. Soil desorption is expected to be slow. Toluene probably exists in soils in the adsorbed state. It can be anticipated that a portion of toluene in soil will be transferred to air and water. The part that stays in soil may participate in chemical reactions (including photochemical reactions) and biological degradation and transformation.
- **Biodegradation:** Toluene appears to be aerobically and anaerobically biodegradable at high concentrations and when exposed to adapted micro-organisms, however under aerobic and oligotrophic conditions toluene seems not to be biodegradable if the concentration is low.
- **Bioconcentration** in aquatic or terrestrial organisms is estimated to be low.

Health Effects

General

There is no clinical disease which is unique to toluene toxicity. However the effects on the central nervous system (CNS) are well recognised and associated with acute, intermediate and chronic exposures.

With respect to oral exposures the following are the primary target organs:

1. Central nervous system:
2. Kidneys: Subchronic exposure to toluene produced nephrosis, damage to tubular epithelium, and increased kidney weights in rats.
3. Liver: Subchronic exposure to toluene produced increased liver weights in rats and mice and hepatocellular hypertrophy in rats.

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4. **Reproduction:** Embryonic deaths, reduced fetal weights, and cleft palates were observed in mice exposed to toluene during gestation.

Other target organs associated with oral exposure to toluene include the immune system, urinary and bladder systems.

With respect to inhalation exposures the following are the primary target organs:

1. **Central nervous system:** Chronic occupational exposure to toluene has resulted in headaches, dizziness, and impaired neurobehavioral performance. Brain dysfunction, abnormal encephalograms, brain atrophy, mental retardation, and visual and hearing impairment have been reported in long-term abusers of toluene. Subchronic exposure of rats produced a high frequency hearing loss.
2. **Kidneys:** Chronic exposure of workers to toluene has resulted in abnormalities of kidney function. Renal tubular effects have been associated with abuse of toluene-containing solvents. Chronic exposure to toluene has caused nephropathy in rats.
3. **Liver:** Hepatomegaly has been reported in workers chronically exposed to toluene. Increased liver weights were reported in rats following subchronic exposure to toluene.
4. **Respiratory system:** Sore throat was one of the symptoms reported in workers chronically exposed to toluene. Chronic exposure of rats has produced lesions in olfactory and respiratory epithelia.

Other target organs associated with inhalation exposure to toluene include the reproductive system.

Repeated or prolonged contact with toluene via skin contact may cause drying and dermatitis.

Toluene is readily absorbed from the lungs and gastrointestinal tracts and, to a lesser extent, through the skin.

Following absorption into the body, toluene is widely distributed to tissues with total uptake dependant on fat content and metabolism with accumulation in adipose tissue, other tissues with high fat content, and highly vascular tissues. There is no evidence that the route of administration has any substantial effect on subsequent metabolism of toluene in humans or animals. It is metabolised in the liver, primarily to hippuric acid and benzoyl glucuronide, compounds that are rapidly excreted in the urine.

Humans and animals excrete inhaled toluene via expiration. In addition, toluene metabolites are excreted primarily in urine.

The following summary of more specific health effects has been derived from the Health Effects Summary presented in ATSDR 2000.

Death	Limited data are available on toluene associated death due to solvent abuse or occupational exposure. Death associated with exposure to toluene may be attributed to cardiac arrhythmias, CNS depression, asphyxia, hepatic and renal failure.
Respiratory	Inhalation of toluene has been associated with irritation of the upper airways, throat and degeneration of the nasal epithelium.

Toluene

Effects	
Cardiovascular Effects	Proarrhythmic response has been associated with some solvent abuse (inhalation and solvent ingestion). Animal studies do not provide adequate evidence to support a direct effect of toluene exposure and effects on the cardiovascular system.
Gastrointestinal Effects	No studies are available regarding gastrointestinal effects in humans following inhalation. No gastrointestinal effects were reported in rats of mice following oral toluene exposure.
Haematological Effects	Haematological effects were not reported after oral or inhalation exposure to toluene in humans and animals. Some effects were noted in inhalation studies before the mid-1950's related to concurrent exposure to toluene and solvent mixtures. Toluene was noted to affect the blood but blood was not considered a critical target tissue following toluene exposure.
Hepatic Effects	Studies of chronic toluene abusers or occupationally exposed humans have provided little evidence of serious liver damage due to inhaled toluene. Acute and intermediate exposure to toluene in animals has produced biochemicals and ultrastructural changes in the liver. Increased liver weights has been reported from oral ingestion studies in mice.
Renal Effects	Studies of chronic toluene abusers or occupationally exposed humans have provided little evidence of serious kidney damage due to inhaled toluene. No changes were reported from oral animal studies.
Reproductive Effects	Current data do not provide sufficient evidence of reproductive effects associated with exposure to toluene.
Developmental Effects	Exposure to high levels of toluene may be toxic to the developing foetus, but the findings are not conclusive due to lack of data and confounding exposures.
Dermal/Ocular Effects	Exposure to toluene in air can result in irritation of the eyes. No dermal effects have been noted following oral or inhalation exposure to toluene.
Immunological Effects	Dermal contact with toluene may cause skin damage because it removes skin lipids. Toluene is slightly to moderately irritating to the eyes. Limited data is available. Exposure to toluene may have a slight effect on immunoglobins, leukocytes and lymphocytes, the significance in humans is uncertain. Toluene exposure has been observed to attenuate the immunotoxic effects of benzene.
Neurological Effects	Dysfunction of the CNS is a critical human health concern following acute, intermediate and chronic inhalation exposure to toluene. Chronic abuse of toluene has been associated with neurotoxic symptoms, narcosis and death. CNS depression has been noted in oral ingestion studies.
Genotoxic Effects	Human data are inconclusive with regard to the genotoxicity of toluene. Genotoxicity testing in animals has produced mostly negative results.
Cancer	Human data are insufficient with regard to the evaluation of carcinogenicity of toluene. Testing in animals has produced no evidence to support toluene as a potential carcinogen.

Toxicity Classification

Toluene is "not classifiable as to human carcinogenicity" (Category D) on the basis of no human data and inadequate animal data by the USEPA under the Risk Assessment Guidelines of 1986 (and USEPA 1996) for all routes of exposure.

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IARC has classified toluene in Group 3 (not classifiable as to human carcinogenicity) on the basis that there is inadequate evidence for the carcinogenicity of toluene in humans and there is evidence suggesting lack of carcinogenicity of toluene in experimental animals.

Exposure Limits and Toxicity Evaluations

Australia

The Australian Drinking Water Guidelines (2004) establish a health based guideline of 0.8 mg/L on the basis of an acceptable daily intake (**ADI**) of **0.22 mg/kg/day**. This is based on a no effect level of 312 mg/kg/day from a 13 week oral rat study associated with marginal hepatotoxicity, applying a safety factor of 1000 and a study duration conversion factor of 5/7.

The NEPC (2004) has established air investigation levels for toluene of 1 ppm (3.75 mg/m³) as a 24-hour average and 0.1 ppm (0.38 mg/m³) as an annual average. These values are investigation levels intended to be utilised as part of regional air quality monitoring. In addition the basis on which the values have been derived is not clear. However it is noted that the annual average value adopted is equal to the inhalation RfC derived by the USEPA and similar to (but slightly greater than) the guideline value established by the WHO and considered relevant for the assessment of chronic inhalation exposures (refer to notes at end of review).

Worksafe Australia (ASCC, available in 2007) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For toluene, the following have been established:

TWA: 50 ppm, equivalent to 191 mg/m³

STEL: 150 ppm, equivalent to 574 mg/m³

WHO

The WHO Drinking Water Guidelines (2004) have established a guideline value of 0.7 mg/L based on a tolerable daily intake (TDI) of 0.223 mg/kg on the same basis as used in the ADWG (2004).

WHO (2000) provide a review of inhalation exposures to toluene, utilising a LOAEL associated with central nervous effects from an occupational study, establishing a guideline value (GV) (based on 1 week average) of **0.26 mg/m³**. A guideline value is a concentration in air, linked to an averaging time, below which no adverse effects of (in the case of odorous compounds) no nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant time-specific studies on animals or humans and relevant factors to account for inter and intra species variations and uncertainty factors.

EU

The EU has prepared a review of toluene (EU, 2003) that reviews occupational, consumer and environmental exposures. Evaluation of exposure for a number of toxicological end points has been presented using margin of safety (MOS) determined from identified no effect levels. With

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respect to the evaluation of environmental exposure, the assessment is based on a subchronic NOAEL of 625 mg/kg/day using intake from air, drinking water and food.

US

The USEPA (available from IRIS, current) provides the following with respect to toluene:

- Oral reference dose (RfD) of 0.2 mg/kg/day has been established using a 13 week oral study with rats.
- Inhalation reference concentration of 0.4 mg/m³ established on the basis of an inhalation occupational exposure study based on adverse neurological effects (same study as used by WHO).

The ATSDR (2000) has established Minimal Risk Levels (MRLs) for the evaluation of non-cancer effects. The following MRLs have been derived for toluene:

- Acute inhalation MRL of 1 ppm (3.8 mg/m³) based on neurological effects in humans;
- Chronic inhalation MRL of 0.08 ppm (0.3 mg/m³) based on neurological effects in humans;
- Acute oral MRL of 0.8 mg/kg/day based on neurological effects in rats; and
- Intermediate oral MRL of 0.02 mg/kg/day based on neurological effects in mice.

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to toluene, intakes from soil, water and food can be considered to be insignificant. Intakes from air have been calculated from residential urban air concentrations in Sydney (NEPC 2002), with the overall mean concentrations reported between 0.008 mg/m³ and 0.019 mg/m³. Personal air monitoring of a large number of individuals undertaking normal daily activities in urban environment report (DEH, 2003) a mean toluene concentration of 8.19 ppb = 0.03 mg/m³. Review of these concentrations with the adopted inhalation guideline value (refer below) suggests that background exposures may comprise up to 10% of the allowable intake.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for toluene following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Toluene

Oral	ADI = 0.22 mg/kg/day (ADWG 2004 and WHO 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation GV = 0.26 mg/m ³ (WHO, 2000), assuming relevance to chronic exposures (refer to discussion below). Occupational inhalation exposure (NOHSC, current): TWA: 50ppm = 191 mg/m ³ STEL: 150ppm = 574 mg/m ³
Background	10%

Note with respect to inhalation value recommended:

WHO (2000) provide a review of inhalation exposures to toluene, utilising a LOAEL associated with central nervous effects from an occupational study, establishing a guideline value (based on 1 week average) of 0.26 mg/m³.

The USEPA also provides an inhalation reference concentration (associated with potential chronic exposures) based on the same study, associated with CNS effects, of 0.4 mg/m³. This is equivalent to the air investigation level provided by NEPC (2004) for an annual average (the basis of this value is unclear).

Both WHO and USEPA have used the same study in the derivation of a guideline value or RfC. The difference in the values derived is associated with the use of modifying and uncertainty factors. The WHO have used the LOAEL available from the occupational study, applied a factor to adjust for continuous exposure and then an uncertainty factor of 300. The USEPA has reviewed the available LOAEL with other studies available and observed that the LOAEL derived from the study shows a lack of duration response with respect to CNS effects and hence the LOAEL derived from the study has been taken to be relevant for all durations of exposure (sub-chronic and chronic). Hence the USEPA has not used a modifying factor for continuous exposure, only an uncertainty factor of 300.

Both values have been reviewed by a number of agencies (RIVM, EU, Canada, NZ and NEPM) with review in the NEPM indicating no guidance as to the validity of the assumptions used. Following guidance from ehHealth (and NEPC) on the preference of available data, the inhalation guideline value available from the WHO is recommended. While the WHO indicate that the guideline value is relevant for a 1 week averaging period, it is considered appropriate to adopt the value for the assessment of chronic exposures (USEPA not the lack of duration response in the most sensitive effect – CNS effect).

On this basis chronic exposure to toluene in air is evaluated using a guideline value of **0.26 mg/m³**.

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BTEX Mixtures

Where toluene is present in conjunction with benzene, ethylbenzene and/or xylenes potential effects associated with the mixture should be addressed. This has followed on from a review of interactions of mixtures undertaken by ATSDR (2004) that identified the potential issues associated with BTEX mixtures. Further detail associated with the approach adopted for the assessment of BTEX mixtures is presented in the BTEX Mixture toxicity summary.

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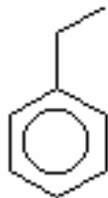
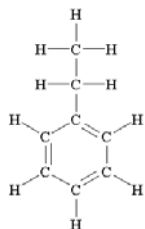
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Revision Dates

Document	Revision	Date of Review	Comments
Current Toxicity Summary	2008A	10/02/2008	Incorporated information from NEPC (2004)
Previous Revisions:	2007B	28/8/2007	
	2007A	24/1/2007	
	2006A	31/7/2006	
	2005A	21/6/2005	

Ethylbenzene

General



Ethylbenzene is a colourless liquid that smells like petrol. Ethylbenzene evaporates at room temperature and burns easily. Ethylbenzene occurs naturally in coal tar and petroleum. It is also found in many products, including paints, inks, and insecticides. Petrol contains about 8% (by weight) ethylbenzene. Ethylbenzene is used primarily in the production of styrene. It is also used as a solvent, a component of asphalt and naphtha, and in fuels. In the chemical industry, it is used in the manufacture of acetophenone, cellulose acetate, diethylbenzene, ethyl anthraquinone, ethylbenzene sulfonic acids, propylene oxide, and -methylbenzyl alcohol. Consumer products containing ethylbenzene include pesticides, carpet glues, varnishes and paints, and tobacco products (ATSDR, 1999).

Properties

Ethylbenzene, also known as ethylbenzol and phenylethane, is a colourless liquid with an aromatic odour. Ethylbenzene is a flammable and combustible liquid. Vapours are heavier than air and may travel to an ignition source. Combustion may produce irritants and toxic gases. Key properties are presented below (ref: ATSDR 1999, USEPA 2004, HSDB 2007 and RAIS):

CAS No	100-41-4
Chemical Formula	C ₈ H ₁₀
Molecular Weight	106.17
Vapour Pressure	9.53 mmHg at 25°C
Vapour Density	3.66
Density	0.867 g/ml at 20°C
Solubility	169 mg/L at 20°C
Air Diffusion Coefficient	0.075 cm ² /s
Water Diffusion Coefficient	7.8 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.00788 atm.m ³ /mol = 0.323 at 25°C (unitless)
Koc	363 cm ³ /g
Log Kow	3.15
Odour Threshold	2 – 2.6 mg/m ³ in air
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0453

Exposure

Inhalation is the primary route of ethylbenzene exposure for the general population and for occupationally exposed individuals. However, it is also present in trace amounts in some water supplies, hence ingestion (and dermal contact) may be an important exposure pathway. Exposure of the general population to ethylbenzene may occur through contact with petrol, evaporation of petrol and vehicle exhaust, solvents, pesticides, printing inks, varnishes, coatings and paints. Ethylbenzene is also a common contaminant due to releases from cigarette smoke.

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If released into the environment the following can be noted with respect to ethylbenzene (WHO, 1996):

- **Air:** In the atmosphere, ethylbenzene is degraded by reaction with hydroxyl radicals, with a typical half-life of about 1.4 to 2 days. It is highly volatile with a high Henry's law constant, indicating volatilisation and partitioning between water into air are likely to be the predominant removal process from soil and water. Ethylbenzene has a low water solubility and a relatively high vapour pressure. This means that only a very small proportion of ethylbenzene in the atmosphere is likely to be removed by precipitation.
- **Water:** The major removal process of ethylbenzene in water is expected to be volatilisation. Aerobic biodegradation has also been shown to be important for the removal of ethylbenzene from water systems. Ethylbenzene has a low Kow indicating that it is not expected to absorb significantly to organic matter or sediments.
- **Soil:** Ethylbenzene is considered to be moderately mobile in soils. Particularly in soils with a low organic content, ethylbenzene will tend to leach into groundwater.
- **Biodegradation:** In surface and groundwater ethylbenzene is biodegradable under aerobic conditions. Under anaerobic conditions (under denitrifying conditions) biodegradation is expected to be very slow.
- **Bioconcentration** in aquatic or terrestrial organisms is estimated to be low.

Health Effects

General

There is no clinical disease which is unique to ethylbenzene toxicity. However the effects on the central nervous system (CNS) are well recognised and particularly associated with acute exposures. The primary target organs following chronic oral exposures are likely to be the liver and kidney with CNS depression identified following acute oral exposure. The primary target organs following chronic oral exposures are likely to be the liver and kidney with CNS depression identified following inhalation exposure include:

- Adverse developmental effects (skeletal variants and reduced number of live offspring per litter) associated with exposure to ethylbenzene during gestation.
- Histopathological changes in the liver and kidney have been observed in experimental animals following prolonged inhalation exposures.
- Acute exposures to high concentrations are likely to cause irritation of the respiratory tract, and CNS effects such as dizziness and vertigo.

Ethylbenzene is readily absorbed from the respiratory and gastrointestinal tracts and, to a lesser extent, through the skin. It also crosses the placenta.

Following absorption into the body, ethylbenzene is widely distributed to tissues with total uptake dependant on fat content and metabolism. There is no evidence that the route of

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administration has any substantial effect on subsequent metabolism of ethylbenzene in humans or animals. Ethylbenzene is almost completely converted to soluble metabolites, which are excreted rapidly in urine. Acute oral toxicity is general considered to be low.

The following summary of more specific health effects has been derived from Health Effects Summary presented in ATSDR 1999 and WHO 1996:

Death	No data are available on ethylbenzene associated death following inhalation exposure only. Deaths have been reported which are associated with petrol abuse, solvent abuse or occupational exposure however it has not been possible to determine whether death was associated with exposure to ethylbenzene or other components in petrol and solvents.
Respiratory Effects	Inhalation of high concentrations of ethylbenzene has been associated with irritation of the upper airways, nose, throat and chest. Animal studies support these findings.
Systemic Effects	Some studies have identified systemic effects associated with ethylbenzene exposure by animals via inhalation. The target organs appear to be the lungs, liver, kidney with transient effects on the haematological system. Conclusions cannot be drawn due to the limitations of the studies.
Hepatatic Effects	Mild, transient liver and kidney toxicity have been reported in humans exposed to high vapour concentrations of ethylbenzene.
Reproductive Effects	Current data are not sufficient to provide conclusions as to the effect of ethylbenzene on the reproductive competence in humans. However animal studies suggest that male reproductive tissues may be a target for ethylbenzene toxicity.
Developmental Effects	Exposure to high levels of ethylbenzene may be toxic to the developing foetus, but the findings are from animal studies and are the potential effects on humans cannot be ascertained from these studies.
Dermal/Ocular Effects	Exposure to high concentrations of ethylbenzene in air can result in irritation of the eyes (irritation, burning and lacrimation). Repeated or prolonged contact with ethylbenzene may result in dermatitis.
Neurological Effects	Acute exposure to ethylbenzene has been associated with dizziness. The neurological effects of long-term exposure by humans to ethylbenzene are unknown, however based on animal data there is considerable likelihood that acute exposures have the potential for the development of neurological effects.
Genotoxic Effects	Long-term exposure to solvents in general, which contain ethylbenzene may cause permanent effects on the central nervous system (CNS). The signs and symptoms are ill-defined and include headaches, memory loss, fatigue and altered emotional reactivity. This syndrome is commonly known as Organic Solvent Syndrome. There are no specific studies that implicate ethylbenzene as a causal agent, although it is present in many of the paints and other solvent-containing products. The available studies tend to have a number of deficiencies including concurrent exposure to many different chemicals, and lack of exposure data. Human data are inconclusive with regard to the genotoxicity of ethylbenzene. Genotoxicity studies have provided negative results in a number of assays, two studies show positive results (mutagenic effects) suggesting ethylbenzene may cause an increased potential for genotoxicity in humans.
Cancer	No association has been found between the occurrence of cancer in humans and occupational exposure to ethylbenzene. There is some evidence of carcinogenicity in animal studies (based in the incidence of renal and testicular lesions).

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Toxicity Classification

Ethylbenzene is “not classifiable as to human carcinogenicity” (Category D) due to the lack of animal bioassays and human studies.

IARC (2000) has classified ethylbenzene in Group 2B (possibly carcinogenic to humans) on the basis that there is sufficient evidence in animals but inadequate evidence for the carcinogenicity in humans.

Exposure Limits and Toxicity Evaluations

Australia

The Australian Drinking Water Guidelines (2004) has established a guideline value of 0.3 mg/L on the basis of health. This value has been derived using an acceptable daily intake (**ADI**) of **0.097 mg/kg/day**. This is based on a No Effect Level of 136 mg/kg/day from a 6-month oral rat study associated with hepatotoxicity and nephrotoxicity, applying a safety factor of 1000 and a study duration conversion factor of 5/7.

Worksafe Australia (ASCC [NOHSC], available in 2007) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment”. For ethylbenzene, the following have been established:

TWA: 100 ppm, equivalent to 434 mg/m³

STEL: 125 ppm, equivalent to 543 mg/m³

WHO

The WHO Drinking Water Guidelines (2004) have established a guideline value of 0.3 mg/L based on an ADI of 0.0971 using the same approach as presented in the ADWG (above).

WHO (1996 and 2000) provide a review of inhalation exposures to ethylbenzene, utilising a NOEL associated with increased organ weight, establishing a guideline value (based on 1 year average) of **22 mg/m³**. It is noted that the guideline value is approximately 10 times higher than the odour threshold and hence chronic exposures near the guideline value maybe associated with odour recognition or annoyance.

EU

The OECD has prepared a review of ethylbenzene (OECD, 2002) that reviews occupational, consumer and environmental exposures. Ethylbenzene is considered to be an animal carcinogen however, the relevance of these findings to humans is currently unknown. Occupational exposures to ethylbenzene are expected to be low as ethylbenzene is primarily produced and further reacted to make styrene in a closed continuous process. Ethylbenzene is also present in crude oil and solvents where exposures are not well characterised, but expected to be low. No quantitative evaluation was presented.

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US

The USEPA (available from IRIS, current) provides the following with respect to non-cancer evaluations of ethylbenzene:

- Oral reference dose (RfD) of 0.1 mg/kg/day has been established based on liver and kidney toxicity from an oral rat study.
- Inhalation reference concentration of 1 mg/m³ established based on developmental toxicity from a rat and rabbit inhalation studies.

The ATSDR (1999) has established Minimal Risk Levels (MRLs) for the evaluation of non-cancer effects. The following MRLs have been derived for ethylbenzene:

- Intermediate inhalation MRL of 1.0 ppm based on developmental effects in rats.

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to ethylbenzene, intakes from soil, water and food can be considered to be insignificant. Intakes from air have been calculated from residential urban air concentrations (WHO 1996) and are low (up to 0.1 mg/m³ in urban air). Concentrations of ethylbenzene reported during personal air monitoring of a large number of individuals undertaking normal activities in urban areas (DEH, 2003) report a mean ethylbenzene concentration of 1.13ppb = 0.005 mg/m³, considered to be low is not significant with respect to the air guideline value adopted (see below). Hence background intakes of ethylbenzene can be considered to be low and do not affect the use of available ADI, TDI or RfD values.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for ethylbenzene following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	ADI of 0.097 mg/kg/day (ADWG 2004 and WHO 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation guideline value (based on 1 year average) of 22 mg/m ³ (WHO 2000). Occupational inhalation exposure (NOHSC, current): TWA: 100 ppm, equivalent to 434 mg/m ³

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	STEL: 125 ppm, equivalent to 543 mg/m ³
Background	Low

BTEX Mixtures

Where ethylbenzene is present in conjunction with benzene, toluene and/or xylenes potential effects associated with the mixture should be addressed. This has followed on from a review of interactions of mixtures undertaken by ATSDR (2004) that identified the potential issues associated with BTEX mixtures. Further detail associated with the approach adopted for the assessment of BTEX mixtures is presented in the BTEX Mixture toxicity summary.

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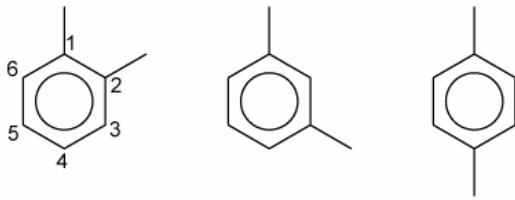
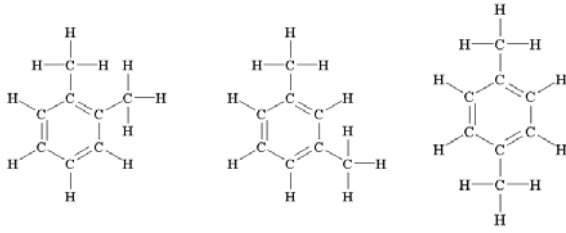
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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007B	28/8/2007
Previous Revisions:	2007A	24/1/2007
	2006A	31/7/2006
	2005A	29/3/2005

Xylenes

General



1,2-dimethylbenzene (ortho-xylene) 1,3-dimethylbenzene (meta-xylene) 1,4-dimethylbenzene (para-xylene)

There are three forms of xylene in which the methyl groups vary on the benzene ring: *meta*-xylene, *ortho*-xylene, and *para*-xylene (*m*-, *o*-, and *p*-xylene). These different forms are referred to as isomers. The term total xylenes refer to all three isomers of xylene (*m*-, *o*-, and *p*-xylene). Mixed xylene is a mixture of the three isomers and usually also contains 6–15% ethylbenzene. Xylene is primarily a synthetic chemical. Chemical industries produce xylene from petroleum. Xylene also occurs naturally in petroleum and coal tar and is formed during forest fires. It is used as a solvent (a liquid that can dissolve other substances) in the printing, rubber, and leather industries. Along with other

solvents, xylene is also used as a cleaning agent, a thinner for paint, and in varnishes. It is found in small amounts in airplane fuel and is a major component of petrol. Xylene is used as a material in the chemical, plastics, and synthetic fibre industries and as an ingredient in the coating of fabrics and papers. Isomers of xylene are used in the manufacture of certain polymers (chemical compounds), such as plastics. (ATSDR, 2007).

Properties

Xylene, also known as xylol or dimethylbenzene, is a colourless, flammable liquid with a sweet odour. Xylene evaporates and burns easily. Xylene does not mix well with water; however, it does mix with alcohol and many other chemicals. Combustion may produce irritants and toxic gases. Key properties are presented below (ref: ATSDR 2007, USEPA 2004, HSDB 2007 and RAIS):

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CAS No	1330-20-7 (for mixed xylene)
Chemical Formula	C ₈ H ₁₀ (single isomer)
Molecular Weight	106.16
Vapour Pressure	6 to 16 mmHg at 20°C
Vapour Density	3.7
Density	0.864 g/ml at 20°C
Solubility	130 mg/L at 25°C
Air Diffusion Coefficient	0.07 cm ² /s
Water Diffusion Coefficient	7.8 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.00734 atm.m ³ /mol = 0.301 at 25°C (unitless)
Koc	407 cm ³ /g
Log Kow	3.12 (mixture)
Odour Threshold	4.34 mg/m ³ in air
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0704 cm/hr (mixture)
Air Conversion factors	1ppm = 4.34 mg/m ³ , 1 mg/m ³ = 0.23 ppm

Exposure

Inhalation is the primary route of xylene exposure for the general population and for occupationally exposed individuals. However, xylenes are ubiquitously distributed in the environment and have been detected in air, rainwater, soils, surface waters, sediments, drinking water, aquatic organisms, human blood, urine and expired breath.

Exposure of the general population to xylene may occur through contact with petrol, evaporation of petrol, vehicle exhaust, workplace air, solvents and ingestion of contaminated drinking water. Xylene is also a common contaminant due to releases from cigarette smoke.

If released into the environment the following can be noted with respect to xylenes (WHO, 1997):

- **Air:** The majority of xylene released into the environment enters the atmosphere directly. In the atmosphere, xylene is degraded by reaction with hydroxyl radicals, with a typical half-life of about 0.4 to 1 day. It is volatile with a low water solubility and high Henry's law constant, indicating volatilisation and partitioning between water into air are likely to be the predominant removal process from soil and water. Xylene isomers are readily degraded in the atmosphere with photooxidation being the most important degradation process.
- **Water:** The major removal process of xylenes in water is expected to be volatilisation. In surface water, xylene may transform through photo-oxidation and biodegradation.
- **Soil:** The major removal process of xylenes in soils is expected to be volatilisation. Xylenes may adsorb to soil and sediments to a limited extent depending on the organic carbon content and water content.

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- Biodegradation: In soil and water, *o*- and *p*-xylene are readily biodegraded under a wide range of aerobic and anaerobic conditions, but *o*-xylene is much more persistent under similar conditions.
- Xylene has been found to bioaccumulate to low to modest levels, however food-chain biomagnification has not been observed

Health Effects

There is no clinical disease which is unique to xylene toxicity. Health effects of mixed xylenes, *o*-xylene, *m*-xylene and *p*-xylene appear to be similar, although the individual isomers are not necessarily equal in potency with respect to a particular effect. Studies indicate that the central nervous system (CNS) is a major and sensitive target of xylene toxicity via inhalation and oral routes. The primary target organs following chronic oral and inhalation exposures are likely to be the CNS and development. Some studies indicate enlargement of the liver and kidneys following oral exposure to mixed xylene. Other target organs identified following inhalation exposure include the respiratory system, altered haematological parameters, nose and throat irritation.

Xylene is readily absorbed from the respiratory and gastrointestinal tracts and, to a lesser extent, through the skin (following exposure to vapours and contact with liquid).

Following absorption into the body, xylene is mainly distributed to lipid-rich tissues, particularly adipose and brain. High uptake also occurs in well-perfused organs such as the liver and kidneys. There is no evidence that the route of administration has any substantial effect on subsequent metabolism of xylene in humans or animals. Xylene is almost completely (95%) converted to soluble metabolites, which are excreted rapidly in urine, with most of the remaining amount eliminated in exhaled air.

The following summary of more specific health effects has been derived from Health Effects Summary presented in ATSDR 2007 and WHO 1997:

Death	Death has been reported following paint fume abuse (where xylenes comprised 90% of the solvent). The data was confounded by the presence of other solvents and no other data is available. Death has also been reported following ingestion of a large quantity of xylene (death associated with depression of respiratory centre in the brain). Similar effects have been noted in acute animal studies. No deaths were reported in animal studies following intermediate and chronic studies.
Respiratory Effects	Inhalation of high concentrations of xylene has been associated with irritation of the nose and throat. Animal studies support these findings indicating potential for decreased respiration, laboured breathing, irritation to the respiratory tract, pulmonary oedema, pulmonary haemorrhage and inflammation. These observations are based on acute and intermediate studies. No chronic studies are available.
Cardiovascular Effects	Acute oral ingestion of xylenes can result in respiratory depression. Limited data is available from animal and human studies, none of which provide conclusive data associated with inhalation exposures. No effects were identified from limited data available for oral exposures. No specific (where inhalation is excluded) dermal studies are available.
Gastrointestinal	Workers exposed to xylene vapours have reported nausea, vomiting and gastric

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Effects	discomfort. No adverse effects have been reported in the limited animal studies available for the inhalation pathway. No effects were identified from limited data available for oral exposures. No specific (where inhalation is excluded) dermal studies are available.
Haematological Effects	Available studies (human and animal) indicate that exposure to xylenes does not cause haematological effects. Exposure to BTEX may result in effects due to the toxicity of benzene. No effects were identified from limited data available for oral exposures. No specific (where inhalation is excluded) dermal studies are available.
Hepatic and Renal Effects	Some studies have identified some hepatic effects and effects on the kidneys associated with xylene exposure via inhalation. Conclusions cannot be drawn due to the limitations of the studies. Mild, transient liver toxicity have occasionally been reported in humans exposed to high vapour concentrations of xylenes. Studies in animals following oral ingestion indicate mostly adaptive changes including increased activity of liver enzymes without histopathological changes in the liver tissue. Some studies have indicated hepatic enzyme induction, increased hepatic weight and increased kidney weight. No specific (where inhalation is excluded) dermal studies are available.
Dermal/Ocular Effects	Exposure to high concentrations of xylene in air can result in mild (transient) irritation of the eyes. Acute dermal exposure to xylene has been associated with transient skin irritation, vasodilation of the skin and dryness and scaling of the skin. Urticaria was reported following exposure to xylene vapours. Similar acute effects have been reported from animal studies. No intermediate or chronic studies are available.
Immunological Effects	Limited data is available. Possible toxic effect of mixed xylene on the immune system was a decrease in spleen and thymus weight observed in rats following oral ingestion. Urticaria was reported following exposure to xylene vapours, which was identified as an immunological reaction.
Neurological Effects	Xylenes appear to have a very limited capacity to adversely affect organs other than the central nervous system (CNS). Acute exposure to xylene has been associated with dizziness. The neurological effects of long-term exposure by humans to xylene are unknown, however based on occupational animal data there is considerable likelihood that acute exposures have the potential for neurological effects. Long-term exposure to solvents in general, which contain xylenes may cause permanent effects on the CNS. The signs and symptoms are ill-defined and include headaches, memory loss, fatigue and altered emotional reactivity. This syndrome is commonly known as Organic Solvent Syndrome. There are no specific studies that implicate xylenes as a causal agent, although it is present in many of the paints and other solvent-containing products. The available studies tend to have a number of deficiencies including concurrent exposure to many different chemicals, and lack of exposure data. Available data associated with xylene exposure (mixed or isomers) indicate the potential for increased prevalence of anxiety, forgetfulness, inability to concentrate and dizziness. Exposure levels associated with neurological effects in animals exposed to xylene are well defined. No chronic studies are available. No specific (where inhalation is excluded) dermal studies are available.
Reproductive Effects	Current data are not sufficient to provide conclusions as to the effect of xylene on the reproductive competence in humans. However animal studies suggest that there may be reduced fertility following inhalation exposure. No specific (where inhalation is excluded) dermal studies are available.
Developmental	Both mixed and individual isomers produce foetotoxic effects in laboratory animals, These include skeletal variations in foetuses, delayed ossification,

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Effects	foetal resorptions, haemorrhages and decreased foetal weight following inhalation exposure. Oral (maternal) exposure studies in animals have reported increased incidence of cleft palate and decreased foetal body weight.
Genotoxic Effects	Limited data are available with regard to the genotoxicity of xylene. Results of various assays indicate mixed xylene and individual xylenes indicate are nongenotoxic following in vitro and in vivo exposure.
Cancer	No conclusive association has been found between the occurrence of cancer in humans and occupational exposure to xylene via inhalation, oral or dermal exposure.

Toxicity Classification

Xylene is classified by the USEPA as “*not classifiable as to human carcinogenicity*” (Category D) due to the lack of animal bioassays and human studies.

IARC (1999) has classified xylene in Group 3 (*not classifiable as to their carcinogenicity to humans*) on the basis that there is inadequate evidence for the carcinogenicity in animals and humans.

Exposure Limits and Toxicity Evaluations

Australia

The Australian Drinking Water Guidelines (2004) have established a guideline of 0.6 mg/L on the basis of health effects using an acceptable daily intake (**ADI**) of **0.179 mg/kg/day**. This is based on a No Effect Level of 250 mg/kg/day from a 2 year oral rat study (based on eye, nose and throat irritation), applying a safety factor of 1000 and a study duration conversion factor of 5/7. It is noted that the guideline exceeds the taste threshold for xylenes in water of 0.02 mg/L.

The NEPC (2004) has established air investigation levels for total xylenes of 0.25 ppm (1.1 mg/m³) as a 24-hour average and 0.2 ppm (0.87 mg/m³) as an annual average. These values are investigation levels intended to be utilised as part of regional air quality monitoring. In addition the basis on which the values have been derived is not clear. However it is noted that the annual average value adopted is the same as the guideline value established by the WHO based on an annual average (see below).

Worksafe Australia (NOHSC, available in 2007) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment”. For xylenes, the following have been established:

TWA: 80 ppm, equivalent to 350 mg/m³

STEL: 150 ppm, equivalent to 655 mg/m³

WHO

The WHO Drinking Water Guidelines (2004) have established a guideline value of 0.5 mg/L based on an ADI of 0.179 using the same approach as presented in the ADWG (above) using a body weight of 60kg instead in the 70kg used in the ADWG.

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WHO (1997 and 2000) provide a review of inhalation exposures to xylenes, utilising a LOAEL associated with developmental neurotoxicity from animal studies, establishing a guideline value (based on 1 year average) of **0.87 mg/m³**.

EU

No review of xylene (mixed or individual isomers) are available from the EU or OECD.

US

The USEPA (available from IRIS 2007) provides the following with respect to non-cancer evaluations of xylenes:

- Oral reference dose (RfD) of 0.2 mg/kg/day has been established based on decreased body weight and increased mortality from an oral rat study.
- Inhalation reference concentration of 0.1 mg/m³ established based on impaired coordination in a subchronic inhalation rat study.

The ATSDR (2007) has established Minimal Risk Levels (MRLs) for the evaluation of non-cancer effects. The following MRLs have been derived for xylenes:

- Acute, intermediate and chronic inhalation MRLs for mixed xylenes are 2, 0.6 and 0.05 ppm respectively; and
- Acute, intermediate and chronic oral MRLs for mixed xylenes are 1, 0.4 and 0.2 mg/kg/day respectively.

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to xylenes, intakes from soil, water and food can be considered to be insignificant. Intakes from air have been calculated from residential urban air concentrations (WHO 1997 and DEP 2000) and are low (up to 0.01 mg/m³ in urban air). Personal air monitoring of a large number of individuals undertaking normal daily activities in urban environment report (DEH, 2003) a mean xylene concentration of 6.23 ppb = 0.03 mg/m³. Review of these concentrations with the adopted inhalation guideline value (refer below) suggests that background exposures may comprise up to 2% of the allowable intake.

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Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for xylenes following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	ADI of 0.179 mg/kg/day (ADWG 2004 and WHO 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation guideline value (based on 1 year average) of 0.87 mg/m ³ (WHO 1997 and 2000 and NEPC 2004). Occupational inhalation exposure (NOHSC\ASCC, current): TWA: 80 ppm, equivalent to 350 mg/m ³ STEL: 150 ppm, equivalent to 655 mg/m ³
Background	Low – 2%

BTEX Mixtures

Where xylenes are present in conjunction with benzene, toluene and/or ethylbenzene potential effects associated with the mixture should be addressed. This has followed on from a review of interactions of mixtures undertaken by ATSDR (2004) that identified the potential issues associated with BTEX mixtures. Further detail associated with the approach adopted for the assessment of BTEX mixtures is presented in the BTEX Mixture toxicity summary.

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Revision Dates

Document	Revision	Date of Review	Comments
Current Toxicity Summary	2008A	10/02/2008	Incorporated information from NEPC (2004) and revised profile from ATSDR (2007)
Previous Revisions:	2007B	28/8/2007	
	2007A	24/1/2007	
	2006A	31/7/2006	
	2005A	21/6/2005	

BTEX Mixtures

Introduction

The assessment of mixtures is an area where clear guidance has yet to be provided in Australia or internationally. EnHealth (2004) notes that where data are available on the interaction of chemicals, the data should be considered in the risk assessment. However, how this is to be addressed is not presented in the guidance. Some guidance is available from the NHMRC in the derivation of the Australian Drinking Water Guidelines (2004), where the assessment focuses on two categories based on health effects:

- Chemicals where the effects are observed above a certain threshold dose; and
- Chemicals that do not appear to have a threshold, namely genotoxic carcinogens.

The approach adopted provides for assessment of chemicals on the basis of either a threshold or non-threshold approach. The total risk is calculated summing the individual non-threshold carcinogenic risk or threshold hazard quotients to obtain a total non-threshold risk and total threshold hazard index. With respect to interactions between chemicals the use of large margins of safety within the values adopted for the quantification of toxicity is considered sufficient to account for potential interactions with other chemicals. This approach assumes additivity irrespective of whether the toxic endpoints are the same or different. This approach is taken in lieu of specific information or guidance on actual additive effects. The result is conservative when it is also considered that the toxicity values also incorporate a range of safety factors. This differs from an approach that calculates risks to human health based on end-points.

A number of reviews of the assessment of mixtures has identified that this area is highly complex and there is no consistent approach that can be applied universally (Feron et al 2002¹, Carpenter et al 1998², Hansen et al 1998³, Suk et al 2002⁴, Health Council of the Netherlands 2002⁵, Kortenkamp et al, 2007⁶). In essence many of the reviews identify that mixture effects should be assessed based on the nature of the chemicals identified in each media, with any available information on the potential for chemical interactions to be considered. For many of the chemicals considered in a risk assessment, there is limited data concerning chemical interactions. In addition there is difficulty in comparing derived toxicological values for chemicals as the threshold values derived may not be based on the same experimental method, conditions, assay or end points or that the combination of exposures below the relevant NOAELs is of any significance (Kortenkamp et al, 2007).

¹ Feron V.J., Cassee F. R., Groten J.P. van Vliet P.W and van Zorge J.A., 2002. International Issues on Human Health Effects of Exposure to Chemicals Mixtures. Environmental Health Perspectives, Volume 110, Supplement 6, December 2002.

² Carpenter D.O., Arcaro K.F., Bush B., Niemi W.D., Pang S and Vakharia D.D., 1998. Environmental Health Perspectives, Volume 106, Supplement 6, 1998.

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⁴ Suk W.A., Olden K. and Yang R.S.H, 1998. Chemical Mixtures Research: Significance and Future Perspectives. Environmental Health Perspectives Volume 110, Supplement 6, December 2002.

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BTEX Mixtures

Assessment of BTEX Mixtures

With respect to BTEX, ATSDR (2004⁷) has undertaken a review of the interaction of these chemicals. In summary, the review identified that exposures to BTEX are best assessed using an individual chemical approach that considers shared neurological effects (where an additive approach is appropriate) and unique haematologic/carcinogenic effects (where the carcinogenic assessment of benzene alone is adequate). Application of this approach, however requires that the threshold values adopted for the assessment of BTEX is relevant to the protection of neurological effects.

Relevant toxicity summaries are presented for benzene, toluene, ethylbenzene and xylenes separately. The approach adopted for the identification of relevant toxicity values for the quantification of oral, inhalation and dermal exposures follows guidance provided by enHealth (2004⁸) and NEPC (1999⁹). To provide a meaningful understanding of whether simply adding the calculated hazard indices calculated for BTEX provides an adequate assessment of the potential effects associated with exposure to BTEX mixture and neurological effects, the basis for the toxicity values adopted requires review. The ATSDR review of BTEX mixtures has focused on inhalation exposures, however the following noted the basis for both inhalation and oral data.

Toluene

The inhalation guideline value (GV) identified (0.26 mg/m³) available from the WHO (2000¹⁰) and adopted for the quantification of exposure is based on a LOAEL associated with central nervous effects from an occupational study. The ATSDR has also established a chronic inhalation Minimal Risk Level (MRLs) for toluene of 0.3 mg/m³ based on neurological effects in humans. As this value is similar to the GV adopted the approach adopted to the quantification of threshold effects for toluene is considered relevant to the protection of neurological effects.

The oral ADI adopted is based on a LOAEL associated with marginal hepatotoxicity in rats and mice.

Ethylbenzene

The inhalation GV identified (22 mg/m³) available from the WHO (1996¹¹) and adopted for the quantification of exposure is based on a NOEL associated with increased organ weight based on animal studies. The USEPA (available on IRIS) has established a reference concentration of

⁷ ATSDR, 2004. Interaction Profile for Benzene, Toluene, Ethylbenzene and Xylenes (BTEX). Agency for Toxic Substances and Disease Registry, May 2004.

⁸ enHealth, 2004. Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards, 2004.

⁹ National Environment Protection Council (NEPC), 1999. National Environment Protection Measure (Assessment of Site Contamination), Schedule B(4), Guideline on Health Risk Assessment Methodology, 1999.

¹⁰ WHO, 2000. *Air Quality Guidelines for Europe*. Second Edition, WHO Regional Office for Europe, Copenhagen. WHO European Publication Series No. 91.

¹¹ WHO, 1996. *Environmental Health Criteria 186, Ethylbenzene*. International Programme on Chemical Safety, World Health Organisation 1996.

BTEX Mixtures

1 mg/m³ based on development effects in rats and rabbits. It is noted that the WHO review included the same studies as considered in the USEPA review. ATSDR has not established a chronic inhalation Minimal Risk Level (MRLs) for ethylbenzene, however they have derived an intermediate inhalation MRL of 1ppm (4.35 mg/m³) based on one of the studies used for the USEPA value. The ATSDR (2004) reviews suggests this value be used as a guide for neurological effects and that a guideline for chronic exposures of 0.1 ppm (0.44 mg/m³) be applied. As this chronic values has not been reviewed it is not considered relevant for use in QRAs at this stage.

Hence the values adopted for the quantification of inhalation exposures to ethylbenzene are not specifically derived based on neurological end points.

The oral TDI adopted is based on a NOAEL associated with hepatotoxicity and nephrotoxicity in a limited study in rats.

Xylenes

The inhalation GV identified (0.87 mg/m³) available from the WHO (1997¹²) and adopted for the quantification of exposure is based on a LOAEL associated with developmental neurotoxicity.

The oral ADI adopted is based on a NOAEL associated with irritation effects (eye, nose and throat)in rats.

Benzene

With respect to the quantification of threshold effects associated with benzene, the following values are available from relevant sources:

- Oral RfD = 0.004 mg/kg/day available from IRIS (USEPA) based on decreased lymphocyte count;
- Inhalation RfC = 0.03 mg/m³ available from IRIS (USEPA) based on decreased lymphocyte count;
- Chronic inhalation GV = 0.0013 mg/m³ available from ATSDR (2004) based on neurotoxicity.

The inclusion of the assessment of threshold effects that may be associated with neurotoxicity effects of benzene assuming additivity with TEX threshold values is questionable, particularly for the assessment of oral and dermal exposures as the basis for the relevant threshold values differs and neither value is based on neurotoxicity. The assessment of inhalation exposures could be undertaken considering neurotoxicity if all the GVs from the ATSDR are adopted however a number of the values are not a peer reviewed and only a guidance value estimated from an intermediate MRL. Hence the ATSDR value is not considered relevant for use in this assessment and hence the assessment of additive effects for inhalation exposures does not

¹² WHO, 1997. *Environmental Health Criteria 190, Xylenes*. International Programme on Chemical Safety, World Health Organisation 1997.

BTEX Mixtures

address the same end point. On this basis the assessment of benzene has also included the following threshold values:

- Oral RfD = 0.004 mg/kg/day available from IRIS (USEPA) based on decreased lymphocyte count; and
- Inhalation RfC = 0.03 mg/m³ available from IRIS (USEPA) based on decreased lymphocyte count.

Background intakes of benzene relevant for urban and rural areas are based on inhalation exposure being the major contributor to background intakes. The average concentration in air reported in western Sydney is 0.00128 mg/m³, approximately 4% of the RfC adopted. If the assessment of benzene exposures is being undertaken in a major city, then background intakes are expected to be higher contributing between 5 and 10% of the RfC.

It is noted that there is no guidance as to the interaction of BTEX with other TPH compounds, hence additivity of BTEX and TPH is not certain and likely to be conservative.

1,2,4- and 1,3,5-Trimethylbenzene

General

The following is summarised from information presented in the database HSDB and review documents available from the USEPA (1994) and OEHHA.

1,2,4-Trimethylbenzene (pseudocumene) is an isometric compound of trimethylbenzene. Production of 1,2,4-trimethylbenzene occurs during petroleum refining as a major component of the C₉ aromatic hydrocarbon fraction (or simply the C₉ fraction). The primary use of the C₉ fraction, approximately 99% of its production volume, is as a gasoline additive. Uses of the remaining C₉ fraction include those as a solvent in coatings; cleaners; pesticides; and printing and inks. Isolated 1,2,4-trimethylbenzene is used in a number of industrial applications. It is used chiefly as the raw material for production of trimellitic anhydride. It is also used in the manufacture of pharmaceuticals and dyes.

1,3,5-Trimethylbenzene (mesitylene) is used as a dyestuff intermediate, solvent, paint thinner, and a UV oxidation stabilizer for plastics. It is released directly into the environment as a component of gasoline and by emission from gasoline-powered vehicles, municipal waste treatment plants, and coal-fired power stations.

1,2,4- and 1,3,5-Trimethylbenzene is present predominantly in the atmosphere. It is also present in surface water and drinking water. Trimethylbenzene volatilises rapidly from surface waters with a volatilisation half-life of approximately 3.4 hours. The chemical also volatilizes from soils, however, moderate adsorption to soils and sediments may occur. In air trimethylbenzene degrades by reaction with hydroxyl radicals, with a half life of 6-12 hours. In soil and water, volatilisation is the major route of removal of trimethylbenzene from soils; although, biodegradation may also occur. Bioaccumulation of the chemical is not considered to be significant.

Exposure and Health Effects

The main routes of exposure to 1,2,4- and 1,3,5-trimethylbenzene are via inhalation of contaminated air (including exposure during the use of self-service petrol bowsers) and ingestion/dermal contact with contaminated water. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal.

Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. The two principle metabolites excreted are 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid.

Direct contact with liquid 1,2,4-and 1,3,5-trimethylbenzene is irritating to the skin and breathing the vapour is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapour causes headache, fatigue, and drowsiness.

1,2,4-Trimethylbenzene depresses the central nervous system. Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Exposure to 1,3,5-trimethylbenzene may cause an asthma like allergy with future exposure potentially

1,2,4- and 1,3,5-Trimethylbenzene

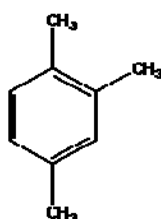
resulting in further asthma attacks, shortness of breath, wheezing, cough and/or chest tightness. 1,3,5-Trimethylbenzene may damage the liver and cause anaemia.

No information was found on the carcinogenicity or genotoxicity of 1,2,4- or 1,3,5-trimethylbenzene.

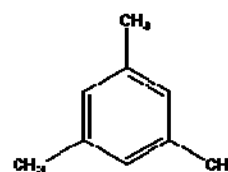
Properties

Trimethylbenzene is a colourless liquid with a distinct odour. Key properties are presented below (USEPA 1994, HSDB online database 2007, USEPA 2004 and RAIS):

	<u>1,2,4-trimethylbenzene</u>	<u>1,3,5-trimethylbenzene</u>
Cas No	95-63-6	108-67-8
Chemical Formula	C ₉ H ₁₂	C ₉ H ₁₂
Molecular Weight	120.2	120.2
Vapour Pressure	2.1 mmHg at 25°C	2.1 mmHg at 25°C
Vapour Density	4.15	4.1
Density	876 g/ml at 20°C	864 g/ml at 20°C
Solubility	57 mg/L at 20°C	48.2 mg/L at 20°C
Air Diffusion Coefficient	0.064 cm ² /s	0.0602 cm ² /s
Water Diffusion Coefficient	7.92 x 10 ⁻⁶ cm ² /s	8.67 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0057 atm.m ³ /mol = 0.252 at 25°C (unitless)	0.00771 atm.m ³ /mol = 0.316 at 25°C (unitless)
Koc	717.6 cm ³ /g	703 cm ³ /g
Log Kow	3.63	3.42
Odour Threshold	0.4 ppm = 1.97 mg/m ³	NA
Dermal Absorption	0.01 (unitless)	0.01 (unitless)
Permeability Constant	0.133	0.0944



1,2,4-trimethylbenzene



1,3,5-trimethylbenzene

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of trimethylbenzenes. Ambient levels of 1,2,4-trimethylbenzene in air have been reported by the NSW DEC with the average concentration reported in the Sydney CBD (data from January 1996 to August 2001) of 0.7 ppbv (with lower values reported in outer suburbs and rural areas). 1,3,5-

1,2,4- and 1,3,5-Trimethylbenzene

trimethylbenzene was less frequently detected in air samples we the average concentration reported in the Sydney CBD of 0.2 ppbv. As environmental exposures are expected to be dominated by the presence of this chemical in air, the potential for background concentrations in water is assumed to be negligible. Based on the average concentration in Sydney CBD of 0.7 ppbv = 0.003 mg/m³ (at 25°C), which is up to 50% of the available reference concentration, hence background intakes of 1,2,4-trimethylbenzene have been assumed to comprise up to 50% of the threshold intake levels. While lower concentrations of 1,3,5-trimethylbenzene were reported in ambient air, it has also been assumed that background intakes may comprise up to 50% of the threshold intake levels for this chemical.

Toxicity Values

Review of available data with respect to 1,2,4- or 1,3,5-trimethylbenzene indicates that these chemicals are not carcinogenic or genotoxic. On this basis it is considered relevant (in accordance with guidance from enHealth (2004)) to consider potential exposures to 1,2,4- and 1,3,5-trimethylbenzene on the basis of a threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for trimethylbenzenes following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

1,2,4- and 1,3,5-Trimethylbenzene

Classification USEPA: Not classified
IARC: Not classified

Toxicity Values:	
Oral	Reference Dose = 0.05 mg/kg/day (USEPA OEHHA ³ provisional value derived for 1,2,4- and 1,3,5-trimethylbenzene and is the only threshold value available from Level 1 sources as per enHealth (2004)).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Reference concentration = 0.006 mg/m ³ (USEPA OEHHA ³ provisional value derived from a study of human exposures to 1,2,4-trimethylbenzene that showed increased vertigo, headaches, drowsiness, chronic asthma-like bronchitis, anaemia, altered blood clotting in workers exposed up to 10 years (derived from a LOEL and 3000 fold uncertainty factor). This is the only threshold value available from Level 1 sources as per enHealth (2004)). This value has also been adopted for 1,3,5-trimethylbenzene. RfC equivalent to 0.0017 mg/kg/day assuming an air intake of 20 m ³ /day and 70 kg bodyweight. Occupational inhalation exposure for trimethylbenzenes (ASCC, current to 2007): TWA: 25 ppm = 123 mg/m ³ STEL: NA
Background	50%

References

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enHealth (2004). *Environmental Health Risk Assessment Guidelines for Assessing Human Health Risks from Environmental Hazards*.

HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2007, from Toxnet, Toxicology Data Network, National Library of Medicine: <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>

IARC (n.d.). *IARC - Summaries and Evaluations*. Retrieved 2007, from IPCS INCHEM: <http://www.inchem.org/pages/iarc.html>

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NEPC (1999). *National Environment Protection Measure, Schedule B(4), Guideline on Health Risk Assessment Methodology*. Australia: National Environment Protection Council.

1,2,4- and 1,3,5-Trimethylbenzene

NHMRC (2004). *Australian Drinking Water Guidelines - 6, National Water Quality Management Strategy*. Australia: National Health and Medical Research Council and the Agriculture and Resource Management Council of Australia and New Zealand.

NSW DEC (2004). Ambient Air Quality Research Project (1996-2001), Internal Working Paper No. 2. Ambient Concentrations of Toxic Organic Compounds in NSW. Department of Environment and Conservation. December 2004.

OEHHA (2001). Proposed Action Levels for 1,2,4-Trimethylbenzene and 1,3,5-Trimethylbenzene. Office of Environmental Health Hazard Assessment, California, May 24, 2001.

RAIS (n.d.). *The Risk Assessment Information System*. Retrieved 2007, from <http://rais.ornl.gov/index.shtml>

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USEPA, (2004). *Region IX Preliminary Remediation Goals*. Retrieved October 2007, from <http://www.epa.gov/region09/waste/sfund/prg/index.html>

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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007B	19/10/2007
Previous Revisions:	2007A	2/05/2007
	2006A	31/7/2006

Trichlorobenzenes

General

The following is summarised from information from a range of available sources (HSDB, RIVM (2001), Health Canada (1993), OEHHA).

1,2,3-Trichlorobenzene (1,2,3-TCB), **1,2,4-trichlorobenzene** (1,2,4-TCB) and **1,3,5-trichlorobenzene** (1,3,5-TCB) are isomers of trichlorobenzene (TCB) which are chlorinated cyclic aromatic compounds. TCBs are used as a chemical intermediate, solvent for oil-soluble dyes, degreasing solvent, dielectric fluid, lubricating oil adhesive and heat transfer medium. It has also been used as an accelerant for dyeing triacetates and was formerly used in termite preparations. The isomers are impurities of monochlorobenzene and therefore, may also enter the environment as a result of the production and use of monochlorobenzene. 1,2,4-TCB is economically the most important isomer.

Based on vapour pressures in the range of 0.21-0.46 mm Hg at 25 deg C, TCB isomers are expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase TCB is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals with an estimated atmospheric half-life of 24-57 days. TCB is expected to have low mobility in soils with volatilisation expected to occur from soil surfaces. TCB is expected to biodegrade slowly in soils. In water, TCB is expected to adsorb to sediment or particulate matter with volatilisation expected to occur from water surfaces. The potential for bioconcentration in aquatic organisms is considered high.

Exposure and Health Effects

The main routes of exposure to TCB are via inhalation of contaminated air and ingestion/dermal contact with contaminated water or food. Occupationally, inhalation and dermal exposures are the most important routes of absorption.

TCB are rapidly absorbed following oral, dermal and inhalation exposures. Following oral exposure TCB are found in fat, skin and the liver with high levels of metabolites found in the kidney and muscle. The major metabolites are trichlorophenols and corresponding glucuronides, sulphates and mercapturic acids.

Available data on the toxicity of the TCB are limited. Epidemiological studies of exposed populations are not available and information on chronic toxicity or carcinogenicity in adequate studies in experimental animals has not been identified. The available data do not provide evidence of mutagenicity or carcinogenicity for the TCBs.

In subchronic oral studies all three isomers of TCB caused a similar toxicity pattern: significant increases in liver/body weight ratios and mild to moderate histopathological changes in the liver, thyroid and kidneys.

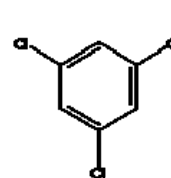
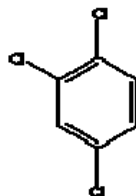
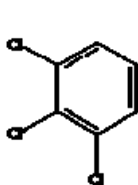
Inhalation studies are limited to 1,2,4- and 1,3,5-isomers. These studies indicate squamous metaplasia and hyperplasia of the respiratory epithelium of the nasal passages, hepatic and renal toxicity as the critical effects.

Trichlorobenzenes

Properties

1,2,4-TCB is colourless liquid at room temperature. 1,2,3-TCB and 1,3,5-TCB are white, crystalline solids. TCB are moderately volatile. Key properties are presented below (HSDB, Health Canada (1993), WHO (2004) and RAIS):

	1,2,3-TCB	1,2,4-TCB	1,3,5-TCB
Cas No	87-61-6	120-82-1	108-70-3
Chemical Formula	C ₆ H ₃ Cl ₃	C ₆ H ₃ Cl ₃	C ₆ H ₃ Cl ₃
Molecular Weight	181.5	181.5	181.5
Vapour Pressure	0.17 mmHg at 25°C	0.46 mmHg at 25°C	0.24 mmHg at 25°C
Vapour Density	6.26	6.26	6.26
Density	1.45 g/ml at 25°C	1.46 g/ml at 20°C	1.36 g/ml at 20°C
Solubility	12 mg/L at 25°C	49 mg/L at 20°C	4 mg/L at 25°C
Air Diffusion Coefficient	NA	0.03 cm ² /s	NA
Water Diffusion Coefficient	NA	8.23 x 10 ⁻⁶ cm ² /s	NA
Henry's Law Coefficient	0.00125 atm.m ³ /mol = 0.051 at 25°C (unitless)	0.00142 atm.m ³ /mol = 0.058 at 25°C (unitless)	0.0019 atm.m ³ /mol = 0.078 at 25°C (unitless)
Koc	3680 cm ³ /g	2670 cm ³ /g	NA
Log Kow	4.04	4.02	4.49
Odour Threshold	NA	1.4 ppm	NA
Dermal Absorption	NA	0.01 (unitless)	NA
Permeability Constant	NA	0.0173 cm/hr	NA
Conversion			



Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of TCB isomers. Ambient levels of 1,2,4-trichlorobenzene in air have been reported in Sydney and surrounding areas (DEC, 2004) with the average concentration reported in the Sydney CBD (data from January 1996 to August 2001) of 0.1 ppbv (consistent with low levels reported in outer suburbs and rural areas). More detailed information is available from Health Canada (1993) on background intakes of TCBs from air, drinking water, breast milk and food¹. This estimates intakes of 0.1 to 1.2 µg/kg/day of 1,2,4-TCB, approximately 0.3 µg/kg/day of 1,2,3-TCB and approximately 0.3 µg/kg/day 1,3,5-TCB. These intakes comprise less than 5% of the TDI. To be conservative a background intake of 5% has been assumed for the assessment of exposures to TCBs.

Trichlorobenzenes

Toxicity Values

Review of available data with respect to TCB indicates that these chemicals are not carcinogenic or genotoxic. On this basis it is considered relevant to consider potential exposures to all three isomers of TCB on the basis of a threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for TCB following review of the available information in general accordance with relevant Australian guidance (enHealth (2004) and NEPC (1999)).

Classification

USEPA: Not classified

IARC: Not classified

Toxicity Values:			
	<u>1,2,3-TCB</u>	<u>1,2,4-TCB</u>	<u>1,3,5-TCB</u>
Oral	TDI = 0.0077 mg/kg/day (ADWG based on subchronic dietary study on rats with the liver and thyroid as critical effects). This values is relevant to all isomers of TCB.		
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.		
Inhalation	No inhalation data available. Oral TDI can be adopted as the basis for the derivation of the oral TDI considers exposure (oral) to 1,2,3-TCB.	Chronic TC = 0.004 mg/m³ (Health Canada) based on a NOEL from an inhalation study on 1,2,4-TCB based on kidney effects.	Chronic TC = 0.0017 mg/m³ (Health Canada) based on a NOEL from an inhalation study on 1,3,5-TCB based on respiratory effects.
Occupational Inhalation	NA	Occupational inhalation exposure for trichlorobenzene (1,2,4-) (ASCC): TWA: 5 ppm = 37 mg/m ³ peak limitation STEL: NA	NA
Background	5%	5%	5%

References

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Trichlorobenzenes

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NEPC (1999). *National Environment Protection Measure, Schedule B(4), Guideline on Health Risk Assessment Methodology*. Australia: National Environment Protection Council.

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RIVM (2001). *Re-evaluation of human-toxicological maximum permissible levels, RIVM Report 711701 025*.

WHO (2004). *CICAD 60 Chlorobenzenes other than Hexachlorobutadiene: Environmental Aspects*. Retrieved 2007, from <http://www.inchem.org/documents/cicads/cicads/cicad60.htm#1.0>

Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007B	10/12/2007
Previous Revisions:	2007A	19/10/2007

GENERAL

Chlorobenzenes are cyclic aromatic compounds formed by the addition of 1-6 atoms of chlorine to the benzene ring. This yields 12 compounds: monochlorobenzene (chlorobenzene), three isometric forms of dichlorobenzene, trichlorobenzene and tetrachlorobenzene as well as penta- and hexachlorobenzene.

Dichlorobenzenes belong to the group of organic halogen compounds replacing two hydrogen atoms in benzene by chlorine atoms. **Dichlorobenzenes** include isomers:

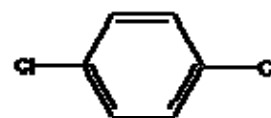
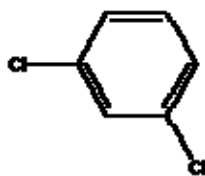
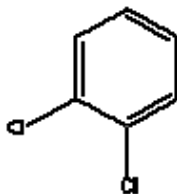
- 1,2-dichlorobenzene (1,2-DCB, o-dichlorobenzene), a colourless liquid boiling at 180 C, used as a solvent and chemical intermediate for dyes, pigment, agrochemicals, a wide range of organic synthesis, it is used as a solvent for organic materials and for oxides of non-ferrous metals; solvent carrier in the production of toluene diisocyanate; fumigant and insecticide; degreasing hides and wool; metal polishes; industrial odour controller; in cleaners for drains; preservative for wood. formulations for motor oil additives, lubricants and paints;
- 1,3-dichlorobenzene (1,3-DCB, m-dichlorobenzene), a colourless liquid boiling at 172 C, used as a fumigant and insecticide; solvent; chemical intermediate to manufacture dyes, agrochemicals, pharmaceuticals and other organic synthesis; and
- 1,4-dichlorobenzene (1,4-DCB, p-dichlorobenzene), a white solid with a with a characteristic penetrating odour. It is used mainly as an insecticidal fumigant against moths, as a space deodorizer, as a general insecticide and fungicide on crops, and as a chemical intermediate for plastics, dyes, pharmaceuticals and other organic compounds

All isomers are insoluble in water and denser than water. They are prepared by the chlorination reaction of benzene in the presence of iron(III) chloride. The chlorination reaction leads to similar ratio of ortho- and para-dichlorobenzene, but small amount of the meta isomer is performed. The ortho and para isomers are separated by fractional freezing. While the para isomer crystallizes, the ortho isomer remains liquid. The meta-dichlorobenzene is prepared by heating and pressure with aluminium chloride.

PROPERTIES

Key properties of dichlorobenzenes are presented below (ATSDR 2004, USEPA 2004 and ORNL Database 2006):

	1,2-DCB	1,3-DCB	1,4-DCB
CAS No	95-50-1	541-73-1	106-46-7
Chemical Formula	C ₆ H ₄ Cl ₂	C ₆ H ₄ Cl ₂	C ₆ H ₄ Cl ₂
Molecular Weight	147	147	147
Vapour Pressure	1.47 mmHg at 20°C	2.15 mmHg at 20°C	1.74 mmHg at 20°C
Vapour Density	5.1	5.07	5.08
Density	1.306 g/ml at 20°C	1.288 g/ml at 20°C	1.46 g/ml at 20°C
Solubility	80 mg/L at 20°C	125 mg/L at 20°C	81.3 mg/L at 20°C
Air Diffusion Coefficient	0.069 cm ² /s	0.069 cm ² /s	0.069 cm ² /s
Water Diffusion Coefficient	7.9 x 10 ⁻⁶ cm ² /s	7.9 x 10 ⁻⁶ cm ² /s	7.9 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0019 atm.m ³ /mol = 0.0779 at 25°C (unitless)	0.0028 atm.m ³ /mol = 0.108 at 25°C (unitless)	0.00243 atm.m ³ /mol = 0.0996 at 25°C (unitless)
Koc	443.1 cm ³ /g	434 cm ³ /g	434 cm ³ /g
Log Kow	3.43	3.53	3.44
Odour Threshold	301 mg/m ³	NA	1.1 mg/m ³
Dermal Absorption	0.01 (unitless)	0.01 (unitless)	0.01 (unitless)
Permeability Constant	0.0659 cm/hr	0.0775 cm/hr	0.0669 cm/hr



EXPOSURE

Exposure of the general population to DCB may be by inhalation, oral or dermal routes. Exposure is most likely to occur in occupational environments which handle or produce the chemical. The general public are more likely to be exposed to 1,4-DCB mainly by breathing vapours from 1,4-DCB products used in the home, such as mothballs and toilet-deodorizer blocks. 1,2- and 1,3-DCB are not found frequently in the air of homes and buildings because, unlike 1,4-DCB, these chemicals are not used in household products. Other exposures may include drinking water, food and soils. DCB have not been found in Australian drinking waters (NHMRC, 2004).

1,2-, 1,3-, and 1,4-DCB have similar physical and chemical properties, and consequently are expected to have similar environmental fates. If released into the environment the following can be noted with respect to DCB (ATSDR, 2004):

- Air: DCBs will exist predominantly in the vapour-phase in the atmosphere. They are degraded in the atmosphere by reaction with hydroxyl radicals, with atmospheric lifetimes (theoretically calculated) of about 1 month. The detection of these chemicals in rainwater suggests that atmospheric removal via washout is possible.

- Soil: Depending on soil type, DCBs are expected to be moderately mobile in soil and to volatilize from surface water and soil surfaces to the atmosphere. Volatilization, sorption, biodegradation (slow), and bioaccumulation are likely to be competing processes, with the dominant fate being determined by local environmental conditions. DCM that partitions to sediments can persist for long periods of time with little likelihood of anaerobic degradation.
- DCBs are removed from the environment principally by biological, and, to a lesser extent, by non-biological mechanisms; however, they are considered moderately persistent in water, air, and sediments.
- Water: DCBs released into the aquatic environment will be redistributed preferentially to the air and to sediment (particularly organically rich sediments).
- Furthermore, DCB was not found to bioconcentrate in significant quantities in aquatic biota.

HEALTH EFFECTS

General

The following information is available from ATSDR (2004) and RIVM (2001). The database for the DCB-isomers is limited, especially for 1,3-DCB for which insufficient data is available to derive toxicity values. There is no clinical disease which is unique to DCB toxicity.

The limited data does not identify particular differences in the kinetics (including biotransformations) of the DCBs. They are almost completely absorbed from the gastrointestinal tract, while some 20% is absorbed via inhalation. Once absorbed they are readily distributed, primarily to adipose tissue (because of their lipophilicity) and to kidney, liver and lungs.

Metabolism is mainly by hepatic oxidation to dichlorophenols and their glucuronide and sulphate conjugates. These are eliminated via the urine. It is note that in rats, almost 100% of 1,4-DCB was excreted in 5 days.

The liver is the primary target of animals orally exposed to DCBs with other effects identified in animal studies including decreased survival, reduced body weight, liver and kidney toxicity and induction of porphyria. More specifically, the key issues associated with 1,2-DCB are liver effects and respiratory effects. The most sensitive adverse health effects identified by studies of 1,3-DCB were effects on the endocrine system. Liver effects have also been identified. Effects on the liver have been reported to be a sensitive end point following exposure to 1,4-DCB. Other effects associated with exposure to 1,4-DCB include respiratory effects, renal effects and developmental effects.

All three isomers were reported to be non-teratogenic. Some relatively minor embryonic and fetotoxic effects have been observed for DCBs, however only at doses that were toxic for the mother.

Genotoxic Effects

DCBs were non-mutagenic in several bacterial tests both in the presence and absence of metabolic activation. Only 1,4-DCB has been tested in a number of *in vitro* and *in vivo* assays. Review of the available data by RIVM (2001) and WHO (1991) indicated that DCBs (all isomers) are not genotoxic.

Cancer

Limited data is available, however review of the data by RIVM (2001) and WHO (1991) indicates that there is no evidence of carcinogenicity for 1,2-DCB. The available studies provided limited evidence of carcinogenicity by 1,4-DCB based on the development of liver and kidney tumours in rats and mice

TOXICITY CLASSIFICATION

1,2-DCB:

USEPA: Group D: not classifiable as to human carcinogenicity; and

IARC: Group 3: not classifiable as to its carcinogenicity.

1,3-DCB

No evaluation available from USEPA or IARC.

1,4-DCB

USEPA: has not evaluated 1,4-DCB; and

IARC: Group 2B: possibly carcinogenic to humans.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have derived the following for DCBs:

1,2-DCB

NHMRC has derived a drinking water guideline of 1.5 mg/L using a ***TDI of 0.43 mg/kg/day*** based on a no effect level of 60 mg/kg/day from a 2-year oral study on mice, a safety factor of 100 and a 5/7 conversion factor to adjust from a 5day per week feeding study to 7 days.

1,3-DCB

Insufficient data is available to establish a health based guideline. The level of 0.02 mg/L was established based on aesthetic considerations of taste and odour.

1,4-DCB

NHMRC has derived a drinking water guideline of 0.04 mg/L using a *TDI of 0.0107 mg/kg/day* based on a lowest effect level for kidney tumours of 150 mg/kg/day from a 2-year oral study on rats, a safety factor of 10 000 and a 5/7 conversion factor to adjust from a 5day per week feeding study to 7 days.

Worksafe Australia (NOHSC) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment”. For DCBs, the following have been established:

	<u>1,2-DCB</u>	<u>1,3-DCB</u>	<u>1,4-DCB</u>
TWA:	25ppm = 150 mg/m ³	NA	25ppm = 150 mg/m ³
STEL:	50ppm = 301 mg/m ³	NA	50ppm = 300 mg/m ³

WHO

The WHO (Drinking Water Guideline 2004) provides the following for DCBs:

1,2-DCB

WHO has derived a drinking water guideline of 1 mg/L using a *TDI of 0.43 mg/kg/day* based on the same study as used by the NHMRC (as above).

1,3-DCB

Insufficient data is available to establish a health based guideline.

1,4-DCB

WHO has derived a drinking water guideline of 0.3 mg/L using a *TDI of 0.107 mg/kg/day* based on the same study used by the NHMRC (as above) however a safety factor of 1000 has been adopted, rather than 10000 as adopted by the NHMRC.

The WHO (2000) has established a tolerable concentration (TC) for 1,4-DCB in air. The *TC of 1 mg/m³*, based on an annual average, is based on increased organ weight and urinary proteins in rats.

EU

The EU has provided an assessment of 1,4-DCB (EU, 2004). Based on review of toxicity data, threshold levels have been identified for the consideration of acute effects and repeated dose effects. Fore repeated dose effects threshold levels (essentially NOAELs) have been identified for inhalation exposures (NOAEL = 80 mg/m³ for continuous exposure which is considered protective of all effects including carcinogenicity) and ingestion (NOAEL = 10 mg/kg/day for general systemic effects and 30 mg/kg/day fro developmental effects). The margin of exposure for workers, consumers and the general public (via

environmental exposures) was calculated and the conclusion of the assessment identified no issues of concern.

RIVM

The Dutch (RIVM, 2001) have provided a review of DCBs and identified the following:

1,2-DCB

- Oral TDI = 0.43 mg/kg/day based on the same study as considered by NHMRC and WHO in derivation of the DWG as above);
- Inhalation TC (provisional) = 0.6 mg/m³ based on a NOAEL of 60 mg/m³ and an uncertainty factor of 100;
- Background exposure up to 0.001 µg/kg/day.

1,3-DCB

No data available to establish guideline levels. Background intake likely to be the same as for 1,2-DCB.

1,4-DCB

- Oral TDI = 0.1 mg/kg/day based on data from a few studies;
- Inhalation TC (provisional) = 0.67 mg/m³ based on a NOAEL of 67 mg/m³ and an uncertainty factor of 100;
- Background exposure up to 0.006 µg/kg/day.

US

The USEPA (IRIS current in 2006) has derived the following for DCBs:

1,2-DCB: Oral reference dose (RfD) of 0.09 mg/kg/day based on NOAEL from 2-year rat study;

1,4-DCB: Inhalation reference concentration (RfC) of 0.8 mg/m³ based on increased liver weights in rat study.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with DCB. The levels established (valid in 2006) are:

- 1,2-DCB: Acute, intermediate and chronic oral MRLs of 0.8 mg/kg/day, 0.4 mg/kg/day and 0.4 mg/kg/day respectively;
- 1,3-DCB: Acute and intermediate oral MRLs of 0.4 mg/kg/day and 0.03 mg/kg/day respectively;
- 1,4-DCB: Acute, intermediate and chronic inhalation MRLs of 2 ppm, 0.1 ppm and 0.02 ppm respectively and an intermediate oral MRL of 0.1 mg/kg/day.

The California Air Resources Board (CARB and OEHHA) has established a reference exposure level of 0.8 mg/m³ for 1,4-DCB in air based on general effects (CNS effects, liver and kidney effects).

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of DCBs in Australia, noting that DCBs have not been found in drinking water in Australia (NHMRC). DCB has been reported in urban air in Australia (WA DEP 2000) at concentrations consistent with the range reported in Canada (CEPA, 1992). CEPA (1992) and RIVM (2001) provide a more comprehensive review of potential background intakes of DCBs where the contribution from background is estimated to be less than 1% of the TDI. On this basis background intakes are considered low and the threshold values adopted for the assessment of DCBs do not need to be adjusted for such low levels of background exposure.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for DCBs following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

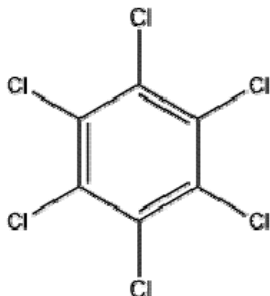
	1,2-DCB	1,3-DCB	1,4-DCB
Oral	TDI = 0.43 mg/kg/day (NHMRC 2004 and WHO 2004)	No data available, adopt data for 1,2- DCB	TDI = 0.0107 mg/kg/day (NHMRC 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.		
Inhalation	The only inhalation specific value derived is the provisional TC = 0.6 mg/m ³ (RIVM, 2001), equivalent to 0.17 mg/kg/day tolerable intake*.	No data available, adopt data for 1,2- DCB	TC = 1 mg/m ³ (WHO, 2000), which is equivalent to a tolerable intake of 0.29 mg/kg/day*
Occupational Inhalation Data	<u>Data from NOHSC:</u> TWA = 25ppm = 150 mg/m ³ STEL = 50ppm = 301 mg/m ³	No data available, adopt data for 1,2- DCB	<u>Data from NOHSC:</u> TWA = 25ppm = 150 mg/m ³ STEL = 50ppm = 300 mg/m ³
Background	<1%	<1%	<1%

* Tolerable intake in air calculated based on 70 kg body weight and 20 m³ inhaled per day.

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GENERAL



Hexachlorobenzene (also known as perchlorobenzene, pentachlorophenyl chloride and commonly abbreviated to **HCB**) is a synthetic organic compound that does not naturally occur. HCB can be produced by reacting benzene with excess chlorine in the presence of ferric chloride at 150-200°C. HCB was primarily used as a grain fumigant on wheat, barley, oats and rye for the control of bunts. In most countries its use as a fungicide has been discontinued. HCB was also used in the production of pyrotechnic and military ordinance and in the manufacture of nitroso rubber for car tyres.

HCB has also been incidentally produced as a by product in the manufacture of chlorinated solvents such as tetrachloroethylene and carbon tetrachloride, chlorinated pesticide and other chlorinated compounds. Small amounts of HCB can also be produced during combustion processes such as burning of city wastes.

PROPERTIES

HCB is a clear white crystalline solid at room temperature that is practically insoluble in water. When heated to decomposition, it emits toxic fumes of chlorides. HCB is slightly soluble in ethanol, soluble in ethyl ether and very soluble in benzene. Key properties are presented below (ATSDR 2002, USEPA 2004 and ORNL Database 2005):

CAS No	118-74-1
Chemical Formula	C ₆ Cl ₆
Molecular Weight	284.79
Vapour Pressure	0.000011 mmHg at 20°C
Vapour Density	9.8
Density	1.57 g/ml at 23°C
Solubility	0.006 mg/L at 20°C
Air Diffusion Coefficient	0.0542 cm ² /s
Water Diffusion Coefficient	5.91 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0017 atm.m ³ /mol = 0.0695 at 25°C (unitless)
Koc	4 to 5 cm ³ /g
Log Kow	5.73
Odour Threshold	Not available
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.408

EXPOSURE

Exposures to HCB may occur and in the workplace and within the general environment. Work place exposure may occur through inhalation and dermal contact with this compound at workplaces where HCB is produced or used.

The general population may be exposed to HCB via inhalation of ambient air, ingestion of food and drinking water. The general population is not likely to be exposed to large amounts of HCB, however

trace amounts have been reported in food and air with HCB reported in most people tested for HCB or its metabolite.

HCB is readily absorbed by the oral route and poorly via the skin. Intake from dietary sources is estimated to be the most significant intake mechanism in the general population, however intake from ambient air or drinking-water may increase in areas closer to emission sources.

The results of most studies of the levels of HCB in foods and human tissues over time indicate that exposure of the general population to HCB declined from the 1970s to the mid-1990s in many locations. Infants may be exposed to HCB from their mother *in utero* or via r human milk.

If released into the environment the following can be noted with respect to HCB (WHO, 2003):

- **Air:** HCB is expected to exist in both the vapour and particulate-phase in the ambient atmosphere. Vapour-phase HCB is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals with an estimated atmospheric half-life of about 2.6 years. Particulate-phase HCB may be physically removed from the air by wet and dry deposition. Due to its persistence in the troposphere HCB meets the criteria for long-range transport in the atmosphere.
- **Soil and Water:** HCB is considered to be immobile in soils and sediment. Volatilisation of HCB from dry soil surfaces is not expected on the basis of the vapour pressure of the chemical. Volatilisation from moist soil surfaces may occur, however this process may be attenuated due to adsorption on to soil particles. HCB is not expected to biodegrade based on a measured half-life in soil of 3 to 6 years. In water, HCB is expected to adsorb to sediment or particulate matter. This compound may volatilise from the surface of water bodies, however adsorption may attenuate this process. The volatilisation half-life is estimated to be approximately 5 years if adsorption is considered.
- Biodegradation is not expected in water on the basis of biodegradation half-lives which have been estimated to be in the order of several years (2 to 10 years) in fresh waters.
- Bioconcentration in aquatic organisms is estimated to be very high on the basis of bioconcentration factors (BCF) in the range of 1,600 to 20,000 measured in fish.

On the basis of the potential for long-range transport, persistence in air, water, soil and sediment, bioaccumulation, toxicity and ecotoxicity, HCB meets the UN-ECE Persistent Organic Pollutant (POP) criteria (UNECE, 1998).

HEALTH EFFECTS

General

The following information is available from WHO (1997 and 2003) and ATSDR (2004). There is no clinical disease which is unique to HCB toxicity.

There is a lack of toxicokinetic information for humans. HCB is readily absorbed by the oral route in experimental animals and poorly via the skin (there are no data concerning inhalation). In animals and humans, HCB accumulates in lipid-rich tissues, such as adipose tissue, adrenal cortex, bone marrow, skin and some endocrine tissues, and can be transferred to offspring both across the placenta and via mothers' milk. HCB undergoes limited metabolism, yielding pentachlorophenol, tetrachlorohydroquinone and pentachlorothiophenol as the major metabolites in urine. Elimination half-lives for HCB range from approximately one month in rats and rabbits to 2 or 3 years in monkeys.

Acute toxicity of HCB is considered to be low via the oral and inhalation exposure pathways. In humans, toxicity has been observed following short-term repeated ingested exposure: with the liver, immune system, skin, thyroid and nervous systems the target organs of toxicity. In animals, similar effects have been noted. The most pronounced effect in both humans and animals is liver toxicity. HCB accumulates in the body over time.

Most data on the effects of HCB on humans originate from accidental poisonings that took place in Turkey in 1955-1959, in which more than 600 cases of porphyria cutanea tarda were identified from oral ingestion of HCB in bread. In this incident, disturbances in porphyrin metabolism, dermatological lesions, hyperpigmentation, hypertrichosis, enlarged liver, enlargement of the thyroid gland and lymph nodes, and (in roughly half the cases) osteoporosis or arthritis were observed, primarily in children. Breast-fed infants of mothers exposed to HCB in this incident developed a disorder called pembe yara (pink sore) and most died within a year. Animal studies have shown that HCB causes reproductive toxicity and increases the risk of cancer formation.

The primary systems for HCB are hepatic toxicity (porphyria), reproductive toxicity, developmental toxicity and carcinogenicity.

Genotoxic Effects

HCB has little capability to induce directly gene mutation, chromosomal damage and DNA repair. It exhibited weak mutagenic activity in a small number of the available studies on bacteria and yeast, although it should be noted that each of these studies has limitations. There is also some evidence of low-level binding to DNA *in vitro* and *in vivo*, but at levels well below those expected for genotoxic carcinogens.

On the basis of the available evidence (WHO 1997 and Woodward-Clyde 1996) HCB can be considered to be non-genotoxic.

Cancer

No association has been found between HCB levels in humans and the incidence of breast or other cancers. Several animal studies have demonstrated an increase in the incidence of tumour formation following oral exposure to HCB. Evidence of carcinogenicity is strongest in the liver (hyperplasia, benign tumours and malignant tumours). In addition HCB has been shown to induce renal metaplasia, adenomas and renal cell carcinomas; lymphosarcomas; adrenal hyperplasia; parathyroid adenomas and hemangioendothelioma and thyroid tumours.

On the basis of available metabolic and toxicological information the WHO considered that a TDI approach was appropriate for the assessment of non-neoplastic effects and neoplastic effects.

TOXICITY CLASSIFICATION

HCB has been classified as a "probable" human carcinogen (Category B2) by the USEPA on the basis of the induction of tumours in the liver, thyroid and kidney in three rodent species following oral exposure.

IARC (2001) has classified HCB in Group 2B (possibly carcinogenic to humans) based on inadequate evidence in humans and limited evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) not evaluated HCB. NICNAS has classified not classified HCB.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have not derived a drinking water guideline for HCB.

Worksafe Australia (NOHSC) not evaluated HCB.

WHO

The WHO (1997) concluded that the available data were sufficient to develop guidance values for non-neoplastic effects and neoplastic effects for HCB.

For non-neoplastic effects a **TDI of 0.17 mg/kg/day** was derived based on primary hepatic effects in pigs and rats exposed via the oral route (NOEL of 0.05 mg/kg/day) and a 300 fold uncertainty.

The approach for neoplastic effects is based on the Tumorigenic Dose₅, or TD₅ i.e., the intake or exposure associated with a 5% excess incidence of tumours in experimental studies in animals. This is a benchmark approach in which the TD₅ is calculated directly from the experimental data rather than using the upper or lower confidence limits. Uncertainty factors are then applied to the TD₅ to obtain a guidance value. The TD₅ value was calculated from the results of a two-generation study in rats using a multistage model where the TD₅ values ranged from 0.81 mg/kg body weight per day for neoplastic liver nodules in females to 2.01 mg/kg body weight per day for parathyroid adenomas in males. The WHO Task Group decided that the most sensitive end-point (neoplastic nodules of the liver) would be used in the assessment. In calculating the suggested guidance value, it was agreed to use an uncertainty factor of 5000, based on consideration of the insufficient mechanistic data. The TD₅ was divided by this uncertainty factor to arrive at the **suggested guidance value of 0.16 µg/kg body weight per day**. However, it is fully realized that national authorities may choose other end-points or uncertainty factors depending upon data evaluation and future scientific findings.

Although infants may have a high intake of HCB via breast milk for a short time, the TD₅ and TDI were considered to be protective of the health of this population because one of the long-term studies used in deriving these values included lactational exposure. However, it should be noted that the TD₅ and TDI values derived above should not be compared directly with intakes from breast milk by nursing infants, since the guidance values are based on a lifetime intake, whereas the duration of breast-feeding is relatively short.

The WHO Drinking Water Guideline (2003 and 2004) provides the guideline value of 0.001 mg/L for HCB as noted in 1996 calculated using the same approach. However an alternate, benchmark dose approach for deriving exposure associated with neoplastic effects based on the *suggested guidance value of 0.16 µg/kg body weight per day* using the TD₅ value approach.

The WHO (Drinking Water Guideline 1996) provide a guideline value for HCB of 0.001 mg/L based on the application of the linearised multistage model to a 2 year dietary study in rats (liver tumours) with an excess lifetime cancer risk of 10⁻⁵. This approach was adopted because HCB has been shown to induce tumours in three animal species and at a variant of site, hence the linearised low-dose extrapolation model was considered appropriate in deriving a guideline value. The associated *oral slope factor for HCB is 0.3 (mg/kg/day)⁻¹*.

The WHO (2000, 2000b) has not published any review of inhalation exposures to HCB.

EU

No assessment of HCB is available from the EU.

US

The USEPA (IRIS current in 2006) has derived an *oral slope factor of 1.6 (mg/kg/day)⁻¹* for HCB based on a linear multistage model based on hepatocellular carcinoma in rats; and an *inhalation unit risk of 4.6x10⁻⁴ (µg/m³)⁻¹* using a linear multistage model based on oral data used to derive the oral slope factor. For the assessment of non-carcinogenic effects an oral reference dose (*RfD*) of *0.0008 mg/kg/day* has been established for HCB based on liver effects in rats. No reference concentration for the assessment of inhalation exposures (non carcinogenic effects) has been established.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with HCB. The levels established (valid in 2004) are:

- Acute oral MRL = 0.008 mg/kg/day based on hyperactivity in offspring rats (oral study)
- Intermediate oral MRL = 0.0001 mg/kg/day based on minimal ovarian effects in monkeys (oral study)
- Chronic oral MRL = 0.00005 mg/kg/day based on hepatic effects from a multigenerational study (oral study)

The California Air Resources Board (CARB and OEHHA) has derived an *inhalation unit risk of 5.1x10⁻⁴ (µg/m³)⁻¹* for HCB.

The ACGIH (American Conference of Governmental Industrial Hygienists, 1998) has established a Threshold Limit Value (TLV) of 0.002 mg/m³ for HCB based on skin effects.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Some data is available regarding levels of HCB reported in breast milk as well as food. No data are available concerning ambient air levels in Australia, however HCB is not regarded as a common urban air contaminant and hence background intake from air is considered to be negligible. WHO (1997) calculated that the total background intake by an adult of HCB is between 0.0004 and 0.003 µg/kg body weight per day mostly derived from dietary exposures. This intake is essentially negligible compared to the TDI and guidance values derived from the assessment of HCB. On this basis, the assessment of risk associated with potential intake of HCB does not need to be adjusted account for background.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for HCB following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

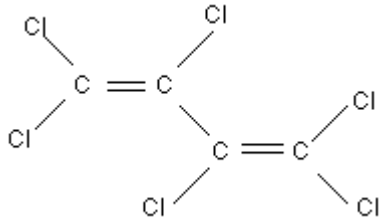
Oral	Guidance Value = 0.00016 mg/kg/day (WHO 1997, 2003 and 2004 based on neoplastic effects which is the most sensitive)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Guidance Value = 0.00016 mg/kg/day equivalent to the oral value as no inhalation specific data is available (US inhalation value is derived from oral study). In addition, there are no data to suggest that inhalation exposures to HCB should be evaluated using a non-threshold approach. Occupational inhalation exposure (ACGIH): TLV: 0.002 mg/m ³ STEL: NA
Background	Negligible (site specific)

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Hexachlorobutadiene

General



Hexachlorobutadiene (also known as perchlorobutadiene; 1,1,2,3,4,4-hexachloro-1,3-butadiene; 1,3-hexachlorobutadiene; dolen-pur; GP-40-66:120 and commonly abbreviated to **HCBD**) is a synthetic organic compound that does not naturally occur. HCBD is used as an intermediate in the production of rubber compounds. It is also used as a solvent, a fluid for gyroscopes, a heat transfer fluid, hydraulic fluid and has been used as a fumigant. HCBD has also been used as a means of recovering chlorine containing gas (snift) in chloride production plants. It is a by-product in the manufacture of chlorinated solvents such as tetrachloroethylene and carbon tetrachloride. (ATSDR, 1994).

Properties

HCBD is a colourless, oily liquid at room temperature with a turpentine like, pungent odour. HCBD is non-flammable, non-combustible, poorly soluble in water but miscible with ethanol and ether. Key properties are presented below (ref: ATSDR 1994, USEPA 2004, HSDB 2008 and RAIS):

CAS No	87-68-3
Chemical Formula	C ₄ Cl ₆
Molecular Weight	260.76
Vapour Pressure	0.22 mmHg at 25°C
Vapour Density	9
Density	1.55 g/ml at 20°C
Solubility	3.2 mg/L at 20°C
Air Diffusion Coefficient	0.0561 cm ² /s
Water Diffusion Coefficient	6.16 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0103 atm.m ³ /mol = 0.421 at 25°C (unitless)
Koc	5000 cm ³ /g (or l/kg, refer to note below)
Log Kow	4.78
Odour Threshold	12 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.121 cm/hr

Note: Koc for HCBD varies significantly. HSDB³ notes a range from essentially 5000 l/kg to 27500 l/kg. The lower end of this range is consistent with the value referenced by ATSDR (1994), Euro Chlor (2004) and USEPA (2003) that all reference a log Koc value of 3.67, equivalent to a Koc of 4680 l/kg. Health Canada (2000) references an even higher number of 80000 l/kg. RAIS Database provides a much lower value of 993.5 l/kg, however the source of this values is not available. Hence it is considered relevant and adequately conservative to adopt a value of 5000 l/kg for HCBD.

Hexachlorobutadiene

Exposure

Exposure of the general population to HCDB may be by inhalation, oral or dermal routes. Exposure is most likely to occur in occupational environments which handle or produce the chemical. Other environmental exposures may be associated with inhalation, ingestion of HCBD in drinking water or ingestion of fish or other foods. HCBD has not been found in Australian drinking waters (NHMRC, 2004).

If released into the environment the following can be noted with respect to HCBD (UNECE, 2002):

- **Air:** Inter-compartmental transport of HCBD will occur by volatilisation (limited), adsorption to particulate matter, and subsequent deposition or sedimentation. In addition to deposition, reaction with hydroxyl radicals is assumed to be an important sink of HCBD in the troposphere with an estimated atmospheric half-life of up to 2.3 years. Due to its persistence in the troposphere HCBD meets the criteria for long-range transport in the atmosphere.
- **Soil and Water:** HCBD is expected to bind with soil and sediments. In water, HCBD is considered persistent unless there is high turbulence. Information available on the persistence of HCBD in water, sediment and soil shows conflicting results, however expert judgement has identified HCBD as persistent. Half lives in natural waters and soils have been reported to be 4-52 and 4-26 weeks respectively. There is conflicting data available about biodegradation. Based on the Structure of HCBD it can be expected that dechlorination is required before aerobic biodegradation can occur. Model calculations indicate that HCBD does not biodegrade fast.
- HCBD has a high bioaccumulation potential as has been confirmed by both laboratory and field observations. Average steady-state bioconcentration factors of 5800 and 17 000, based on wet weight, have been determined experimentally in rainbow trout. Biomagnification has not been observed either in the laboratory or in the field (WHO, 1994).

HCBD is not listed as a key persistent organic pollutant under the Stockholm Convention. However, on the basis of the potential for long-range transport, persistence in water, soil and sediment, bioaccumulation, toxicity and ecotoxicity, HCBD meets the UN-ECE Persistent Organic Pollutant (POP) criteria (UNECE, 2002). Further review of the potential persistence and bioaccumulation potential of HCBD has been undertaken (WCC, 2005) that indicates that the persistence and bioaccumulation potential of HCBD does not necessarily meet the requirements as set out by the UN-ECE. However, while further data is obtained and debate continued, HCBD can be considered persistent in the environment and has the potential for bioaccumulation in the food chain.

Health Effects

General

The following information is available from WHO (1994) and ATSDR (1994). There is no clinical disease which is unique to HCBD toxicity. As there have been very few human studies, the evaluation of toxicity is mainly based on studies in experimental animals. However, limited

Hexachlorobutadiene

human *in vitro* data suggest that the metabolism of HCBd in humans is similar to that observed in animals.

There is limited data available on the absorption of HCBd in animals. Oral experiments indicate that HCBd absorption is rapid and complete, however little data are available concerning absorption following dermal and inhalation exposures.

When orally administered, HCBd or its metabolites have been observed to be distributed primarily in the kidney (outer medulla) and adipose tissue. HCBd has also been located in the liver following intraperitoneal administration. HCBd and its metabolites are excreted in exhaled air, urine and faeces.

HCBd vapour is considered to be irritating to the mucous membranes of humans, and the liquid is corrosive. The compound should also be regarded a sensitising agent.

The main target organs for toxicity are the kidney and, to a much lesser extent, the liver. Reduced birth weight and neonatal weight gain has only been observed at maternally toxic doses, as was developmental toxicity.

Biotransformation to a reactive sulphur containing metabolite probably accounts for the observed nephrotoxicity, genotoxicity and carcinogenicity.

Genotoxic Effects

HCBd has been found to induce gene mutations, chromosomal aberrations, increased sister chromatid exchanges and unscheduled DNA synthesis, although some studies have reported negative results. There is limited evidence for the genotoxicity of HCBd in animals, and insufficient evidence in humans.

Cancer

There is limited evidence for carcinogenicity in animals and insufficient evidence in humans. Review of carcinogenicity by OEHA (2000) indicated that there is sufficient evidence for the carcinogenicity of HCBd, based on the development of renal tubular neoplasms in rats. Review of HCBd by the WHO (guidelines 2004) also note the development of kidney tumours in a long-term oral study in rats. HCBd has not been shown to be carcinogenic by other routes of exposure. On the basis of available metabolic and toxicological information the WHO considered that a TDI approach was appropriate for the derivation of an oral drinking water guideline.

Toxicity Classification

HCBd has been classified as a "possible" human carcinogen (Category C) by the USEPA.

IARC (1999) has classified HCBd in Group 3 (not classifiable as to its carcinogenicity to humans) based on inadequate evidence in humans and limited evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) as Category 3 carcinogen (possibly carcinogenic to humans). NICNAS has classified not classified HCBd.

Hexachlorobutadiene

Exposure Limits and Toxicity Evaluations

Australia

The Australian Drinking Water Guidelines (2004) have derived a drinking water guideline of 0.0007 mg/L for HCBd using a **TDI of 0.0002 mg/kg/day** based on a NOAEL of 0.2 mg/kg/day based on renal effects in rats and a 1000 fold safety factor.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For HCBd, the following have been established:

TWA: 02 ppm, equivalent to 0.21 mg/m³

STEL: none established

Potential exposure via skin absorption is noted.

WHO

The WHO (Drinking Water Guidelines, 2004) provide a guideline value for HCBd of 0.0006 mg/L based on a **TDI of 0.0002 mg/kg/day** following the same approach outlined by NHMRC (as above with a body weight of 60kg rather than 70kg).

The WHO has not published any review of inhalation exposures to HCBd.

EU

No assessment of HCBd is available from the EU.

US

The USEPA (available from IRIS 2008) has derived an **oral slope factor of 0.078 (mg/kg/day)⁻¹** for HCBd based on a linear multistage model based on renal tubular adenomas and adenocarcinomas in rats; and an **inhalation unit risk of 2.2x10⁻⁵ (µg/m³)⁻¹** using a linear multistage model based on oral data used to derive the oral slope factor. The USEPA does not present any data relevant to the assessment of non-carcinogenic effects for HCBd. An oral reference dose of 0.0002 mg/kg/day was derived by the USEPA, however it was withdrawn in 1993.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with HCBd. The levels established (valid to 2008) are:

- Intermediate oral MRL = 0.0002 mg/kg/day based on kidney damage in mice

The California Air Resources Board (CARB and OEHHA) has not established any reference exposure levels (REL) or inhalation unit risk values for HCBd. OEHHA (2000) undertook a

Hexachlorobutadiene

review of carcinogenicity of HCBd. The review suggested that there is evidence of carcinogenicity of HCBd (renal tumours from oral studies). In addition the review suggested that HCBd and its metabolites were genotoxic in bacteria and mammalian cell cultures. The studies on pharmacokinetics and metabolism suggest that the carcinogenicity and genotoxicity of HCBd is primarily due to metabolism through a pathway that includes GSH, enzymes of the mercapturate pathway, deacylase activity and β -lyase activity. Both trichloroethene (TCE) and tetrachloroethene (PCE) have been shown to induce renal tubular neoplasms in long-term studies in rats. Similarities in the site and type of tumour induced in rodents, the genotoxic activity in short-term test systems, and the metabolism of HCBd, TCE and PCE suggest that the three chlorinated alkenes may share a common mechanism of action in the induction of kidney tumours. However, oxidative metabolism via CYP-dependent monooxygenases appears to play a greater role in the bioactivation of TCE and PCE than HCBd.

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. No data is available regarding environmental levels of HCBd in Australia, other than noting that HCBd has not been found in drinking water in Australia (NHMRC). HCBd is not a common urban air contaminant and as such background intakes of HCBd are considered to be negligible. On this basis, the assessment of risk associated with potential intake of HCBd does not need to be adjusted account for background unless other sources of HCBd are present in the study area.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for HCBd following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	TDI = 0.0002 mg/kg/day (NHMRC 2004 and WHO 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	TDI – 0.0002 mg/kg/day equivalent to the oral TDI as no inhalation specific threshold data is available. In addition, there are no data to suggest that inhalation exposures to HCBd should be evaluated using a non-threshold approach. Occupational inhalation exposure (NOHSC, current): TWA: 0.02 ppm = 0.21 mg/m ³ STEL: NA
Background	Negligible (study/site specific)

Hexachlorobutadiene

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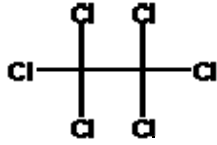
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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2008A	12/2/2008
Previous Revisions:	2007A	6/2/2007
	2006A	31/7/2006
	2005A	19/6/2005

GENERAL



Hexachloroethane (also known as perchloroethane; carbonhexachloride, 1,1,1,2,2,2-hexachloroethane; hexachloroethylene, phenohep and commonly abbreviated to **HCE**) is a synthetic chlorinated alkane compound that does not naturally occur. HCE is not produced in large quantities; however it is used in a number of industrial processes and products. Common uses include the manufacture of smoke candles and grenades, as a plasticiser for cellulose esters, as a minor component of rubber and insecticide formulations, as a moth repellent, retardant in industrial fermentation processes, camphor substitute in nitrocellulose solvent, flame retardant and in the manufacture of aluminium alloys. Historically HCE was also used as an antehelminthic agent (flukicide) in veterinary medicine. HCE can be released into the environment as a by-product of industrial processes, chlorination of drinking water (very small quantities) or incineration of chlorinated waste materials such as polyvinyl chloride (PVC).

PROPERTIES

HCE is a colourless, non-flammable solid (rhombic crystals) at room temperature with a camphor-like odour. HCE is insoluble in water, but soluble in ethanol, diethyl ether, chloroform, benzene and oils. Key properties are presented below (ATSDR 1997, USEPA 2004 and ORNL Database 2006):

CAS No	67-72-1
Chemical Formula	C ₂ Cl ₆
Molecular Weight	236.74
Vapour Pressure	0.4 mmHg at 20°C
Vapour Density	8.2
Density	2.09 g/ml at 20°C
Solubility	50 mg/L at 22°C
Air Diffusion Coefficient	0.0025 cm ² /s
Water Diffusion Coefficient	6.8 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0028 atm.m ³ /mol = 0.114 at 25°C (unitless)
Koc	224.7 cm ³ /g
Log Kow	4.14
Odour Threshold	0.15 ppm = 1.5 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0596 cm/hr

EXPOSURE

The primary routes of potential human exposure to HCE are through inhalation or drinking contaminated water. Occupational exposure of workers in industrial facilities manufacturing or using HCE may occur through inhalation or dermal absorption. HCE exposure to the general public is expected to be relatively low.

If released into the environment the following can be noted with respect to HCE (ATSDR 1997):

- Air: HCE is quite stable in air and hence atmospheric transport of HCE may occur. HCE is expected to diffuse slowly into the with a half life of about 30 years (noted within the stratosphere) and 73 years (within the troposphere). Deposition of HCE from air to water, plants and soil has been reported. It is not expected to react with hydroxyl radicals or ozone or to degrade in the troposphere. Degradation by photolysis may occur.
- Soil and Water: HCE released to water or soil may volatilise or adsorb to soil or sediments. Volatilisation appears to be the major removal mechanism from surface waters. HCE is expected to have a medium to low mobility in soil, hence leaching to groundwater may occur. Sorption of HCE to aquifer materials has been found to retard migration in groundwater. In aquatic systems moderate to slight adsorption to suspended solids and partitioning to sediments is likely. HCE is relatively resistant to degradation in aquatic environments. HCE may be reduced to tetrachloroethylene in the presence of sulphide and ferrous ions. HCE may biodegrade in soil but abiotic degradation processes are not expected to be significant. HCE is biotransformed in soil under aerobic and anaerobic conditions, but it is more rapid in anaerobic soils.
- HCE is expected to bioconcentrate to a moderate degree. However HCE appears to be rapidly metabolised and given low ambient levels of HCE in the environment, bioaccumulation and biomagnification in the food chain is not expected to be significant.

HCE is not listed by the UNEP as a persistent organic pollutant. However there is the potential for HCE to bioaccumulate and due to its persistence in the atmosphere there is the potential for long-range transport of HCE to occur. On this basis, for sites where significant releases of HCE may occur, the potential for HCE to be persistent and bioaccumulative within the environment should be considered.

HEALTH EFFECTS

General

The following information is available from ATSDR (1997) and Woodward-Clyde (1996). There is no clinical disease which is unique to HCE toxicity.

There is limited data available on the absorption of HCE. Once HCE is ingested it is rapidly absorbed. The degree of absorption following inhalation or dermal exposure is unknown.

Following absorption, HCE is rapidly distributed throughout the body via the systemic circulation. HCE is lipophilic and hence most partitions to fat. Degradation/detoxification of HCE is thought to occur primarily in the liver.

Acute Effects: HCE acts primarily as a central nervous system depressant (possibly resulting in mild paralysis) in humans acutely exposed to it and in high concentrations it causes narcosis. HCE is moderately irritating to the skin, mucous membranes, and liver in humans. Liver and kidney effects have been observed in animals acutely exposed to HCE by ingestion. Based on animal tests, HCE to have moderate acute toxicity from ingestion and low acute toxicity from dermal exposure.

Chronic Effects: No information is available on the chronic effects of HCE in humans. Animal studies have suggested that chronic inhalation exposure of animals to high concentrations resulted in neurobehavioral effects and chronically exposed to HCE by ingestion or gavage kidney effects have been observed.

Genotoxic Effects

Available studies indicate HCE is not mutagenic or genotoxic.

Cancer

No data are available on the carcinogenic effects of HCE in humans. Hepatocellular carcinomas were observed in mice following chronic oral exposure to HCE. An increased incidence of renal neoplasms was observed in orally-exposed male rats, but not in females.

Review of HCE by Woodward-Clyde (1996) indicates that based on the available evidence, it does not appear reasonable to conclude that HCE is a human carcinogen. The only evidence for carcinogenicity was observed in animals exposed to high doses of HCE and in nearly all cases these animals displayed evidence of nephrotoxicity. It is likely that the resulting tumours were due to cell proliferation that occurred in response to tissue injury rather than any mutagenic effect of HCE. No carcinogenic effects were seen at lower doses where tissue injury was not induced. Renal tumours observed in male rats were associated with hyaline droplet nephropathy which is thought to be specific to male rats, and is not observed in other species including humans. Hence the renal tumours observed have little relevance to humans. On this basis the most reasonable approach for evaluating HCE was considered to be to use the threshold approach.

TOXICITY CLASSIFICATION

HCE has been classified as a "possible" human carcinogen (Category C) by the USEPA.

IARC (1999) has classified HCE in Group 2B (possibly carcinogenic to humans) based on inadequate evidence in humans and sufficient evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) has classified HCE as Category 3 carcinogen (possibly carcinogenic to humans). NICNAS has not classified HCE.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have not established a guideline for HCE.

Worksafe Australia (NOHSC) has established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment” (NOHSC, current to 2006). For HCE, the following have been established:

TWA: 1 ppm, equivalent to 9.7 mg/m³

STEL: none established

WHO

The WHO (Drinking Water Guideline 2004) have not reviewed or derived a drinking water guideline for HCE.

The WHO (2000, 2000b) has not published any review of inhalation exposures to HCE.

EU

No assessment of HCE is available from the EU.

US

The USEPA (IRIS current in 2006) has derived the following for HCE:

- Non-cancer effects: An oral Reference Dose (*RfD*) of **0.001 mg/kg/day** based on atrophy and degeneration of the renal tubules in rats. No inhalation value has been derived.
- Cancer effects: an *oral slope factor of 0.014 (mg/kg/day)⁻¹* for HCE based on a linear multistage model based on hepatocellular carcinomas in mice and an *inhalation unit risk of 4.0x10⁻⁶ (µg/m³)⁻¹* using a linear multistage model based on oral data used to derive the oral slope factor.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with HCE. The levels established (valid in 2006) are:

- Acute inhalation MRL = 6 ppm (58.1 mg/m³) based on tremors in pregnant rats
- Intermediate inhalation MRL = 6 ppm (58.1 mg/m³) based on tremors in rats
- Acute oral MRL = 1 mg/kg/day based on hepatic necrosis and degeneration in rabbits
- Intermediate oral MRL = 0.01 mg/kg/day based on enlargement of the hepatocytes in rats
- Chronic oral MRL = 0.001 mg/kg/day based on atrophy and degeneration of renal tubules in rats

The California Air Resources Board (CARB and OEHHA) has established an *inhalation unit risk of 1.1x10⁻⁵ (µg/m³)⁻¹* for HCE.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. No data is available regarding environmental levels of HCE in Australia, other than noting that HCE is not a common urban air or water contaminant and as such background intakes of HCE are considered to be negligible. On this basis, the assessment of risk associated with potential intake of HCE does not need to be adjusted account for background.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for HCE following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	RfD = 0.001 mg/kg/day (USEPA oral RfD and equal to the ATSDR chronic MRL. No data is available from NHMRC or WHO)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	RfD = 0.001 mg/kg/day equivalent to the oral TDI as no inhalation specific data is available. In addition, there are no data to suggest that inhalation exposures to HCE should be evaluated using a non-threshold approach. Occupational inhalation exposure (NOHSC, current to 2006): TWA: 1 ppm = 9.7 mg/m ³ STEL: NA
Background	Negligible

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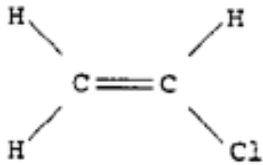
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GENERAL



Vinyl chloride (also known as, chloroethene, 1-chloroethylene, ethylene monochloride and vinyl chloride monomer and commonly referred to as VC) is man-made or results from the breakdown of other manmade substances, such as trichloroethene, trichloroethane, and tetrachloroethene. It is used mainly for the manufacture of polyvinyl chloride (PVC) plastics and resins, and VC copolymers. It is used as a monomer with vinyl acetate or vinylidene chloride in the production of resins. It is also used for the production of chlorinated solvents, such as methyl chloroform and 1,1,1-trichloroethane, and other chemicals; and in the production of adhesives. Other uses include furniture, automotive upholstery, wall coverings, house wares and automotive parts. Up until the mid 1970's it was used as a coolant, propellant in spray cans and in some cosmetics.

PROPERTIES

VC is a volatile, colourless gas with a pleasant, sweet, ethereal odour. It is a colourless liquid below -14°C. VC is slightly soluble in water and highly soluble in diethyl ether, soluble in ethanol, benzene and most organic liquids. Key properties are presented below (ATSDR 1997, USEPA 2004 and ORNL Database 2006):

CAS No	75-01-4
Chemical Formula	C ₂ H ₃ Cl
Molecular Weight	62.5
Vapour Pressure	2600 mmHg at 25°C
Vapour Density	2.16
Density	0.91 g/ml at 20°C
Solubility	2760 mg/L at 20°C
Air Diffusion Coefficient	0.106 cm ² /s
Water Diffusion Coefficient	1.2 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.027 atm.m ³ /mol = 1.107 at 25°C (unitless)
Koc	18.6 cm ³ /g
Log Kow	1.62
Odour Threshold	7650 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0113

EXPOSURE

The main route of exposure for the general public to VC is via inhalation (since VC commonly exists as a gas). Atmospheric concentrations of VC are generally low resulting in very little exposure to the general public. Similarly, the main route of occupational exposure is via inhalation. Dermal exposure is generally considered to be low.

In some countries exposure may also occur via ingestion of contaminated drinking water. In Australia, there are stringent requirements on the maximum permissible residual VC concentrations in PVC pipes and fittings used to carry potable water. Hence VC has not been reported in Australian drinking waters (ADWG 2004).

In the past, VC had been detected in food that was stored in materials that contained PVC. Many countries now regulate the amount of VC in food packaging materials.

If released into the environment the following can be noted with respect to VC:

- **Air:** Reaction with photochemically produced OH* radicals is the dominant atmospheric transformation process, which results in half-lives of 1 to 4 days in the troposphere. Several critical compounds, such as chloroacetaldehyde, formaldehyde and formyl chloride, are generated during experimental reactions.
- **Soil:** Volatilization half-lives are approx. 0.2-0.5 days. VC has a low soil sorption potential and therefore a high mobility in soil. VC may leach through soil into groundwater where it may persist for years.
- **Water:** With few exceptions, VC is not easily degraded. However under anaerobic conditions PCE and TCE can be intrinsically biodegraded to form DCE and VC (below).

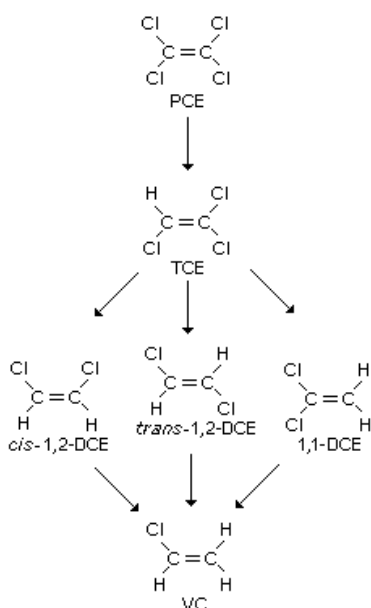


Figure 1. Pathway for anaerobic microbial degradation of chlorinated ethenes to form vinyl chloride (from: WHO, 1999)

NB: PCE=tetrachloroethene, TCE=trichloroethene, DCE=dichloroethene

HEALTH EFFECTS

General

Numerous human population studies and reports have led to the identification of significant long-term health effects which are sometimes collectively referred to as “*vinyl chloride disease*” and characterised by Raynaud’s Phenomenon, acroosteolysis, joint and muscle pain, enhanced collagen deposits, stiffness of the hands and scleroderma-like skin changes. Most of these effects are associated with inhalation exposures in the workplace (particularly during the 1970’s). Primary effects are associated with the liver/spleen, vascular, skeletal, immune system, skin, respiratory and higher central nervous system (CNS) effects. It is well recognised that VC is a genotoxic carcinogen.

VC is rapidly and well absorbed after inhalation or oral exposure. The primary route of exposure to VC is inhalation. Dermal absorption of VC in the gaseous state is not significant. Following exposure VC is distributed rapidly throughout the body. Placental transfer of VC occurs.

The main route of metabolism of VC after inhalation or oral uptake involves oxidation by cytochrome P-450 (CYP2E1) to form chloroethene oxide (CEO), a highly reactive, short-lived epoxide which rapidly rearranges to form chloroacetaldehyde (CAA).

After inhalation or oral exposure to low doses, VC is metabolically eliminated and non-volatile metabolites are excreted mainly in the urine. CEO is thought to be the most important metabolite *in vivo*, concerning the mutagenic and carcinogenic effects of VC.

The following summary has been derived from WHO (1999) and ATSDR (1997).

Death	Acute inhalation exposure of VC has been known to result in death in humans. Cause of death was associated with congestion of lungs and kidneys and failure of blood to clot. Increased mortality in rats following exposure to high concentrations of VC over different exposure duration periods has been observed. No studies indicate lethal effects associated with acute or intermediate ingestion of VC. Chronic ingestion of VC has been associated with decreased longevity in rats.
Respiratory Effects	Respiratory effects associated with occupational VC exposure are contradictory and inconclusive. Animal studies indicate high concentrations cause respiratory inflammation in a variety of species.
Cardiovascular Effects	Occupational exposure to VC has been associated with development of Raynaud’s Phenomenon. Increased incidence of hypertension, cardiovascular and cerebrovascular disease has been reported in workers. Animal studies indicate the potential for cardiac arrhythmias, myodegeneration in the heart and thickening of the walls of the arteries.
Haematological Effects	Exposure to high levels of VC has indicated that blood clotting is impaired. Occupational studies have indicated decreased platelets during early pregnancy, thrombocytopenia and increased levels of two plasma proteins. Animal studies have indicated a failure of blood to clot during exposure to high concentrations.
Musculoskeletal Effects	Acroosteolysis (shortening of the terminal digits) has been observed in occupationally exposed workers.

Hepatic Effects	The liver is a primary toxicity endpoint for VC. Liver damage and liver disease (including angiosarcoma of the liver) has been reported in animal and human studies with the effects correlated with increased concentration and duration of exposure.
Renal Effects	Animal studies (mice and rats) indicate acute exposure to VC can result in kidney congestion.
Endocrine Effects	Some endocrine effects have been reported in occupational workers and rats.
Dermal/Ocular Effects	Exposure to liquid VC has been associated with frostbite. Other skin effects associated with workers include scleroderma, thickening of the skin, while scaly appearance and Raynaud's Phenomenon. Burns to the conjunctiva and cornea have occurred following exposure to VC gas in the occupational environment.
Body Weight Effects	Workers exposed to high concentrations of VC have reported anorexia. Decreased body weights were also identified in some animal studies.
Immunological Effects	Some studies indicate immune system effects in VC workers. These effects include increased circulating immune complexes and presence of abnormal protein in the blood (cryoglobulinemia). Studies of workers who developed "vinyl chloride disease" indicate that the disease may have an immunologic basis.
Neurological Effects	VC was once used as an in halation anaesthetic. Exposure to very high concentrations of VC in air causes depression of the central nervous system (CNS). The most commonly observed CNS effects are weakness, dizziness, nausea, fatigue, headache, incoordination and loss of consciousness. Chronic occupational exposure to VC has been associated with peripheral neuropathy and Raynaud's Phenomenon.
Reproductive Effects	Reproductive effects (decreased male fertility, decrease in testicular weight) have been observed in animal studies following exposure to VC.
Developmental Effects	Studies associated with environmental exposures to VC and birth defects found no significant correlation. Results of animal studies indicate developmental effects at concentrations that are also toxic to maternal animals.
Genotoxic Effects	Genotoxic studies of VC include a large number of assays for occupationally exposed workers. Studies indicate VC is mutagenic and clastogenic in humans with the frequencies of chromosomal aberrations (CA), micronuclei (MN) and SCE in the peripheral blood lymphocytes of workers exposed to high levels of VC shown to be raised. The mutagenic and genotoxic effects of VC have been detected in a number of <i>in vitro</i> test systems in animals, predominantly after metabolic activation. VC showed clastogenic effects in rodents, increased SCE in hamsters and induced DNA breaks in mice. Review of VC by Woodward-Clyde (1996) indicates that there is sufficient evidence to indicate that VC is genotoxic.
Cancer	Exposure to VC via inhalation has been associated with increase in liver cancer including a rare form of angiosarcoma and biliary tract cancer. Other studies have indicated increase incidence of CNS and brain cancer. While most data is associated with inhalation exposures, ingestion studies suggest evidence of carcinogenicity via oral exposure.

TOXICITY CLASSIFICATION

VC is classified as a known human carcinogen (Category A) by the USEPA based upon sufficient evidence from animal studies. VC is a known human carcinogen via the inhalation and oral routes of exposure and a highly likely carcinogen via the dermal route of exposure.

IARC has classified VC in Group 1 (carcinogenic to humans) based in sufficient evidence from animal studies.

NICNAS has classified VC as a Carcinogen Category 1, which is a substance regarded as carcinogenic to humans.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (2004) set the guideline value based on practical limit of determination which is similar to the WHO guideline (noted below) based on an excess cancer risk of 1 in a million.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For VC, the following have been established:

TWA: 5 ppm, equivalent to 13 mg/m³

STEL: NA

WHO

The WHO Drinking Water Guidelines (2004) (also noted in the Australian Drinking Water Guidelines, 2004) have assessed VC as a genotoxic carcinogen and have established a guideline value of 0.0003 mg/L on the basis of linear extrapolation from an oral exposure study in rats associated with the upper-bound risk of 10⁻⁵ and assuming a doubling of the risk of exposure from birth. The WHO slope factor used in the derivation of the drinking water guideline can be calculated as follows:

$$\begin{aligned}
 SF \text{ (mg/kg/day)}^{-1} &= \text{Risk/Intake(mg/kg/day)} \\
 &= [\text{Risk} \times \text{Body Weight}]/[\text{Concentration (water)} \times \text{Ingestion Rate}] \\
 &= [2 \times 10^{-5} \times 70\text{kg}]/[0.0003\text{mg/L} \times 2 \text{ L/day}] \\
 &= 2.3 \text{ (mg/kg/day)}^{-1}
 \end{aligned}$$

The WHO (2000 and 2000b) provides a review of VC. Using human data, mainly occupational studies, associated with inhalation exposures, the WHO has derived *an inhalation unit risk of 1x10⁻⁶ (µg/m³)⁻¹* (i.e. for an air concentration of 1 µg/m³, the lifetime risk is estimated to be 1x10⁻⁶). This value is unchanged from that derived from the same studies in 1987.

The WHO notes that studies indicate that risks may be higher in childhood (i.e. ages less than 10 years) and the above (inhalation) value must be used with caution.

EU

An OECD SIDS assessment is available for vinyl chloride. The review provides a summary of available data and studies relevant to human health and the environment. No specific peer reviewed toxicity values (relevant for non-threshold or threshold effects) have been derived and presented in the report.

US

The USEPA undertook a comprehensive review of VC in 2000 where the following dose response values were established (based on animal studies):

Oral cancer slope factor = $0.72 \text{ (mg/kg/day)}^{-1}$ exposures in adulthood
 = $1.44 \text{ (mg/kg/day)}^{-1}$ exposures over a lifetime

Oral non-cancer reference dose (RfD) = 0.003 mg/kg/day based on liver cell polymorphism in animals, application of the PBPK model to derive a NOAEL and an uncertainty factor of 30.

Inhalation unit risk = $4.4 \times 10^{-6} \text{ (}\mu\text{g/m}^3\text{)}^{-1} = 0.016 \text{ (mg/kg/day)}^{-1}$ exposures during adulthood
 = $8.8 \times 10^{-6} \text{ (}\mu\text{g/m}^3\text{)}^{-1} = 0.03 \text{ (mg/kg/day)}^{-1}$ exposures over a lifetime

Inhalation non-cancer reference concentration (RfC) = 0.1 mg/m^3 based on the oral study to derive RfD.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with TCE. The levels established (current) are:

- Acute inhalation MRL = 0.5ppm based on developmental effects in mice;
- Intermediate inhalation MRL = 0.03ppm based on increased liver weights in rats; and
- Chronic oral MRL = 0.00002 mg/kg/day based on cellular alteration in livers of rats.

The California Air Resources Board (CARB 1990 and OEHHA 1999) has adopted an *acute reference exposure level (REL) of 180 mg/m^3* for VC based on effects identified in occupational studies. An *inhalation cancer slope factor of $0.27 \text{ (mg/kg/day)}^{-1}$* has also been established for VC based on lung carcinoma in mice.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. However, as VC has been evaluated to be a genotoxic carcinogen it is considered appropriate to evaluate exposure using a slope factor for oral, inhalation and dermal exposures where an incremental probability of cancer is calculated. Hence background intake is not relevant in the evaluation of non-threshold dose response chemicals.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for VC following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

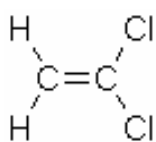
Oral	Oral Slope Factor = $2.3 \text{ (mg/kg/day)}^{-1}$ (WHO, 2004) which includes exposures from birth. For exposures during adult years only, the doubling of risk of exposure assumed from birth is not considered relevant and hence an oral slope factor of $1.15 \text{ (mg/kg/day)}^{-1}$ can be adopted.
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	<p>Inhalation slope factor = $0.016 \text{ (mg/kg/day)}^{-1}$ exposures during adulthood (USEPA 2000)*</p> <p>= $0.03 \text{ (mg/kg/day)}^{-1}$ exposures over a lifetime (USEPA 2000)*</p> <p>Occupational inhalation exposure (NOHSC):</p> <p><i>TWA: 5 ppm, equivalent to 13 mg/m^3</i></p> <p><i>STEL: NA</i></p>
Background	NA

* Following the approach outlined in NEPM (1999), the WHO inhalation assessment should be used in preference, however due to the limitations associated with the assessment of childhood exposure (noted by WHO), the more recent assessment presented by the USEPA is recommended for the evaluation of inhalation exposures to VC.

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GENERAL



1,1-Dichloroethene (vinylidene chloride, 1,1-dichloroethylene or 1,1-DCE) does not occur naturally. It is produced commercially by the dehydrochlorination of 1,1,2-trichloroethane in the presence of excess base or by thermal decomposition of methyl chloroform. 1,1-DCE is used as an intermediate in the production of

hydrochlorofluorocarbons (HCFCs), in the production of chloroacetyl chloride and in the production of latex and resins (also known as PVDC polymers). These polymers are produced as emulsion polymers, as solvent-soluble powders for coating applications, and as resins for extrusion and co-extrusion. PVDC co-polymers containing 79–90% 1,1-DCE are used to form moisture and vapour barrier coatings and films, with applications as food packaging products. PVDC co-polymers containing 10–70% 1,1-DCE are used to improve flame and ignition resistance properties. Residual 1,1-DCE in PVDC used for food packaging products typically ranges from 5 to <1 mg/kg, the limit of detection of the method. Other consumer products containing PVDC include PVDC-latex for carpet backing (<2 mg/kg residual 1,1-DCE), PVDC-latex for photographic film coating (<100 mg/kg residual 1,1-DCE), PVDC for flame retardant fibres for clothing and outdoor awnings (<100 mg/kg residual 1,1-DCE), and PVDC-fluorinated copolymers for application on textiles (<100 mg/kg residual 1,1-DCE). Further processing decreases the residual 1,1-DCE in the final consumer product.

If released to air, 1,1-DCE will exist solely as a vapour phase in the ambient atmosphere and will be degraded by reactions with photochemically produced hydroxyl radicals with a half life of approximately 16 hours. The major reaction products are formaldehyde, phosgene and hydroxyacetyl chloride. If released to soil, 1,1-DCE is expected to be highly mobile with volatilisation expected to be an important fate process. If released to water the chemical is not expected to adsorb to suspended solids or sediments with volatilisation expected to be an important process. 1,1-DCE is generally considered to be resistant to biodegradation. The potential for 1,1-DCE to bioaccumulate in aquatic organisms is low.

EXPOSURE AND HEALTH EFFECTS

1,1-DCE can be found in the environment from release during manufacture and use all from breakdown of polyvinylidene (PVDC) products, all from the biotic or abiotic breakdown of 1,1,1-trichloroethane, tetrachloroethene, 1,1,2-trichloroethene and 1,1-dichloroethane.

The main route of exposure to 1,1-DCE is via inhalation of contaminated ambient air. Exposure may also occur from ingestion of food, contaminated drinking water and use of consumer products containing 1,1-DCE such as plastic wrap which contains residual polymers. Occupational exposure to 1,1-DCE may occur through inhalation and dermal contact.

1,1-DCE is rapidly absorbed following inhalation and oral exposure. Because of its low relative molecular mass and hydrophobic nature, dermal absorption is also likely, but there are no relevant published data. Although 1,1-DCE is rapidly distributed to all tissues, most of the free 1,1-DCE, its metabolites, and covalently bound derivatives are found in the liver and kidney. 1,1-DCE is rapidly oxidized to 1,1-dichloroethene oxide (DCE-epoxide), 2-chloroacetyl chloride, and 2,2-dichloroacetaldehyde. The major metabolites, DCE-epoxide and 2-chloroacetyl chloride, can react with glutathione (GSH), water, or tissue macromolecules. It is not known if the metabolism of 1,1-DCE is the

same in humans, although *in vitro* microsomal preparations from human liver and lung form the same initial products.

The primary effect of acute exposure to high concentrations (approximately 4000 ppm) of 1,1-DCE vapour in humans is central nervous system (CNS) depression which may progress to unconsciousness. Occupational exposure has been reported to cause liver dysfunction in workers. 1,1-DCE is irritating when applied to the skin and prolonged contact can cause first degree burns. Direct contact with the eyes may cause conjunctivitis and transient corneal injury.

The following is provided by WHO (CICAD 51, 2003) for 1,1-DCE:

- The only existing epidemiological study is inadequate to assess the cancer or non-cancer health effects of 1,1-DCE.
- Following high-dose exposure by the oral or inhalation route, the target organs in experimental animals are the liver, the kidney, and the lung. Following low-dose, long-term exposure, the liver is the major target organ in rats following oral or inhalation exposure, but the kidney is the major target organ in mice following inhalation exposure.
- Bioassays for cancer by the oral route of exposure have been conducted in rats, mice, and trout. Although these bioassays have protocol limitations, none provides any significant evidence that 1,1-DCE is a carcinogen by the oral route of exposure.
- Bioassays for cancer by the inhalation route of exposure have been conducted in rats, mice, and hamsters. Most of these bioassays also have protocol limitations. One bioassay in male mice showed an increase in the incidence of kidney adenocarcinomas at one exposure level. There is evidence that the induction of kidney adenocarcinomas is a sex- and species-specific response. The results of the one bioassay showing an increase in tumours in one sex and at one exposure level in a single rodent species are not sufficient to justify an exposure–response assessment.
- 1,1-DCE causes gene mutations in micro-organisms in the presence of an exogenous activation system. Most tests with mammalian cells *in vitro* or *in vivo* show no evidence of genotoxicity.
- There is no evidence that reproductive toxicity or teratogenicity is a critical effect for 1,1-DCE. No reproductive or developmental toxicity was observed at an oral exposure that caused minimal toxicity in the liver of the dams. There is some evidence of developmental variations in the heart after oral exposure, but it is not clear if these effects are directly caused by exposure to 1,1-DCE. There is evidence of fetal toxicity (delayed ossification) following inhalation exposure in the absence of maternal toxicity.
- The toxicity of 1,1-DCE is associated with cytochrome P450-catalysed metabolism of 1,1-DCE to reactive intermediates that bind covalently to cellular macromolecules. The extent of binding is inversely related to loss of GSH, so that severities of tissue damage parallel the decline in GSH. Thus, the responses to 1,1-DCE at low doses with little depletion of GSH are expected to be very different from the responses at high doses causing substantial GSH depletion.

PROPERTIES

At room temperature, 1,1-DCE is a colourless liquid that is very volatile with an mild sweet chloroform like odour. Key properties are presented below (ATSDR 1994, HSDB 2006 and ORNL Database 2006):

CAS No	75-35-4
Chemical Formula	C ₂ H ₂ Cl ₂
Molecular Weight	96.95
Vapour Pressure	634 mmHg at 25°C
Vapour Density	3.25
Density	1.21 g/ml at 20°C
Solubility	2420 mg/L at 20°C
Air Diffusion Coefficient	0.09 cm ² /s
Water Diffusion Coefficient	1.04 x 10 ⁻⁵ cm ² /s
Henry's Law Coefficient	0.0261 atm.m ³ /mol
	1.07 at 25°C (unitless)
Koc	35.04 cm ³ /g
Log Kow	2.13
Odour Threshold	500 ppm = 1985 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0159 cm/hr

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of 1,1-DCE in Australia. It is noted in the Australian Drinking Water Guidelines (NHMRC, 2004) that DCEs have not been found in Australian drinking waters. Data available from the WA (WADEP, 2000) indicates 1,1-dichloroethane was rarely detected in air during sampling. Where detected in Duncraig (WA), the maximum concentration was reported to be 0.2 ppbV (0.00079 mg/m³). Data available from the NSW DEC (2004)WA (WADEP, 2000) indicates 1,1-DCE was never detected in air during sampling undertaken from 1996 to 2001 in a number of urban and rural areas. This is well below the adopted threshold tolerable concentration in air adopted below. Data suggest (from WHO CICAD 51 2003) that the mean exposure from drinking-water will not exceed 6–9×10⁻⁵ mg/kg body weight per day for a 70-kg individual consuming 2 litres per day; oral exposure from food and soil is most likely negligible (WHO, 2005); and data suggest that the upper end of the range for the mean concentration of 1,1-DCE in air will not exceed 0.004 mg/m³ (consistent with data available from Australia). Thus, human exposure is expected to be far below the threshold levels presented below and are considered negligible.

Toxicity Values

Review of available data with respect to 1,1-DCE indicates that the chemical is not considered to be genotoxic to humans (based on limited data) with limited data available with respect to carcinogenicity.

On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to 1,1-DCE on the basis of a threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,1-DCE following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Classification USEPA : Group C possible human carcinogen based on no human data and limited animal data, in particular tumours observed in one mouse strain after inhalation exposure.
IARC: Group 3 not classifiable as to human carcinogenicity based on limited evidence in animals.

Toxicity Values:	
Oral	TDI = 0.046 mg/kg/day (WHO 2005). It is noted that a lower TDI (0.009 mg/kg/day) is presented in the Australian Drinking Water Guideline (NHMRC 2004). The ADWG is noted to be adopted from the WHO without review. The guideline adopted was an old WHO guideline that has been subsequently revised as per WHO (2003 and 2005) and hence the revised TDI has been adopted.
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	TC = 0.2 mg/m ³ (WHO 2003). This is equivalent to an intake of 0.057 mg/kg/day (assuming inhalation of 20 m ³ per day and a body weight of 70 kg). Occupational inhalation exposure relevant for 1,1-dichloroethene (NOHSC, current to 2006): TWA: 5 ppm = 20 mg/m ³ STEL: 20 ppm = 79 mg/m ³
Background	Negligible

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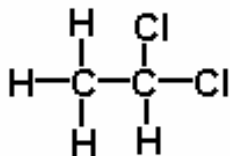
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GENERAL



1,1-Dichloroethane (ethylidene chloride or ethylidene dichloride) is a man made organic solvent. It does not occur naturally, but is manufactured for industrial uses. It is primarily used as an intermediate in the manufacture of other chemicals such as vinyl chloride (and associated byproducts such as EDC tars), 1,1,1-trichloroethane and to manufacture high vacuum rubber. 1,1-Dichloroethane is also used in paint removers and it is a coupling agent in antiknock fuel. In the past the chemical was used as an anaesthetic, however that use has been discontinued when effects on the heart, such as irregular heartbeats, were reported.

If released to air, 1,1-dichloroethane will exist solely as a vapour phase in the ambient atmosphere and will be degraded by reactions with photo chemically produced hydroxyl radicals with a half life of approximately 49 days. If released to soil, 1,1-dichloroethane is expected to be highly mobile with volatilisation expected to be an important fate process. If released to water the chemical is not expected to adsorb to suspended solids or sediments with volatilisation expected to be an important process. Halogenated aliphatic hydrocarbons, such as 1,1-dichloroethane, are generally considered to be resistant to biodegradation. The potential for 1,1-dichloroethane to bioaccumulate in aquatic organisms is low.

EXPOSURE AND HEALTH EFFECTS

The main route of exposure to 1,1-dichloroethane is via inhalation of contaminated ambient air. Exposure may also occur from ingestion of contaminated drinking water and use of consumer products such as paint removers that may contain this compound. Occupational exposure to 1,1-dichloroethane may occur through inhalation and dermal contact.

1,1-Dichloroethane can be absorbed via inhalation, ingestion and skin absorption. The chemical is believed to be rapidly absorbed. Information on the distribution of 1,1-dichloroethane to tissues and organs following exposure is limited. The anaesthetic effects of 1,1-dichloroethane indicate that the chemical reaches the central nervous system and is probably distributed throughout the rest of the body. Most of 1,1-dichloroethane is usually removed unchanged from the body within the breath within two days. A small part of the chemical is metabolised (with major metabolite being acetic acid, and minor metabolites include 2,2-dichloroethanol, chloroacetic acid and dichloroacetic acid) with breakdown products quickly are removed in the breath or urine.

Acute (short-term) inhalation exposure to high levels of 1,1-dichloroethane in humans results in central nervous system (CNS) depression and a cardio stimulating effect resulting in cardiac arrhythmias. Studies in animals have reported effects on the kidney. No information is available on the chronic (long-term), reproductive, developmental, or carcinogenic effects of 1,1-dichloroethane in humans. Some studies in animals have shown on that 1,1-dichloroethane can cause kidney disease after long term, high level exposure in air. An oral animal study reported a significantly positive dose-related trend in hemangiosarcomas, mammary tumours, liver tumours, and endometrial stromal polyps.

There are no human cancer data available. Limited animal studies have shown 1,1-dichloroethane to be a potential carcinogen. No studies are available regarding in vivo genotoxic effects in humans. Data

available from limited in vitro and in vivo (very few) assays in animals are conflicting with very few studies reporting conclusive results. In general 1,1-dichloroethane is considered to be non-genotoxic.

PROPERTIES

At room temperature, 1,1-dichloroethane is a colourless, orderly liquid that is very volatile with an aromatic chloroform/ether like odour. Key properties are presented below (ATSDR 1990, HSDB 2006 and ORNL Database 2006):

CAS No	75-34-3
Chemical Formula	C ₂ H ₄ Cl ₂
Molecular Weight	98.96
Vapour Pressure	227 mmHg at 25°C
Vapour Density	3.92
Density	1.17 g/ml at 20°C
Solubility	5040 mg/L at 20°C
Air Diffusion Coefficient	0.0742 cm ² /s
Water Diffusion Coefficient	1.05 x 10 ⁻⁵ cm ² /s
Henry's Law Coefficient	0.042 atm.m ³ /mol = 0.23 at 25°C (unitless)
Koc	35.04 cm ³ /g
Log Kow	1.79
Odour Threshold	120 - 200 ppm = 486 - 810 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00886

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of 1,1-dichloroethane in Australia. It is noted in the Australian Drinking Water Guidelines (NHMRC, 2004) that dichloroethanes have not been found in Australian drinking waters. Data available from the NSW DEC (2004) indicates 1,1-dichloroethane was rarely (<1% of samples) detected in air during sampling undertaken from 1996 to 2001 in a number of urban and rural areas. Where detected in Sydney's CBD, the maximum 24-hour average concentration was reported to be 0.9 ppbV (0.0036 mg/m³). Data available from the WA (WADEP, 2000) indicates 1,1-dichloroethane was never detected in air. Consideration of the maximum reported concentration of 1,1-dichloroethane in air suggests an intake of 1 µg/kg/day (assuming inhalation of 20 m³ per day and a 70 kg body weight) which is considered negligible compared with the adopted threshold levels presented below. No data is available regarding potential concentrations in food, with potential concentrations in water assumed to be negligible.

Toxicity Values

Review of available data with respect to 1,1-dichloroethane indicates that the chemical is not considered to be genotoxic to humans (based on limited data) with limited data available with respect to carcinogenicity. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to 1,1-dichloroethane on the basis of a threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,1-dichloroethane following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Classification USEPA : Group C possible human carcinogen based on no human data and limited evidence in two animal species (rats and mice) from inhalation studies.
IARC: no evaluation available.

Toxicity Values:	
Oral	No guidelines of toxicity values have been established by NHMRC, WHO, ATSDR or USEPA. RfD = 0.1 mg/kg/day (available from HEAST as referenced on the ORNL Database 2006. HEAST is a peer reviewed US source).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	RfC = 0.5 mg/m ³ (available from HEAST as referenced on the ORNL Database 2006. HEAST is a peer reviewed US source). This is equivalent to an intake of 0.14 mg/kg/day (assuming inhalation of 20 m ³ per day and a body weight of 70 kg). Occupational inhalation exposure relevant for 1,1-dichloroethane (NOHSC, current to 2006): TWA: 100 ppm = 412 mg/m ³ STEL: NA
Background	Negligible

REFERENCES

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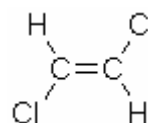
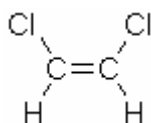
GENERAL

1,2-Dichloroethene (also known as **1,2-DCE**, 1,2-dichloroethylene, acetylene dichloride and dioform) exists in two isomeric forms, *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene, that are colourless, volatile liquids with a slightly acrid odour. 1,2-Dichloroethene is prepared commercially by either the direct chlorination of acetylene or by the reduction of 1,1,2,2-tetrachloroethane with fractional distillation used to separate the two isomers. 1,2-Dichloroethene can also be formed as a by-product during the manufacture of other chlorinated compounds. Commercial use is not extensive, but *trans*-1,2-dichloroethene and mixtures of *cis*- and *trans*-1,2-dichloroethene have been used as intermediates in the production of other chlorinated solvents and compounds, as well as low temperature extraction solvents for dyes, perfumes, and lacquers. Although not used extensively in industry, 1,2-dichloroethene is used in the production of other chlorinated solvents and as a solvent for dyes, perfumes, and lacquers.

PROPERTIES

Both *cis*- and *trans*-1,2-DCE are colorless, volatile liquids with ethereal and slightly acrid odors. 1,2-DCE is slightly soluble in water, but is very soluble in alcohol, ether, acetone and most other organic solvents. Both forms are moderately flammable and react with alkalis to form chloroacetylene gas, which spontaneously ignites in air. Additionally, *cis*- and *trans*-1,2-DCE react violently with potassium hydroxide, sodium, and sodium hydroxide and form shock-sensitive explosives when combined with dinitrogen tetroxide. 1,2-Dichloroethene emits chlorine gas when heated to decomposition (US DOE 1994). Key properties are presented below (ATSDR 1996, WHO 2003, USEPA 2004 and ORNL Database 2006):

Property	<i>cis</i> -isomer	<i>trans</i> -isomer
CAS No:	156-59-2	156-60-5
Chemical Formula	C ₂ H ₂ Cl ₂	C ₂ H ₂ Cl ₂
Molecular Weight	96.95	96.95
Vapour Pressure	180 mmHg at 20°C	265 mmHg at 20°C
Vapour Density	1.6	1.8
Density	1.28 g/ml at 20°C	1.26 g/ml at 20°C
Solubility	3500 mg/L at 20°C	6300 mg/L at 20°C
Air Diffusion Coefficient	0.0736 cm ² /s	0.0707 cm ² /s
Water Diffusion Coefficient	1.1 x 10 ⁻⁵ cm ² /s	1.2 x 10 ⁻⁵ cm ² /s
Henry's Law Coefficient	0.00408 atm.m ³ /mol = 0.167 at 25°C (unitless)	0.00938 atm.m ³ /mol = 0.384 at 25°C (unitless)
Koc	35.5 cm ³ /g	52.5 cm ³ /g
Log Kow	2.09	2.09
Odour Threshold	NA	0.332 to 68 mg/m ³
Dermal Absorption	0.01 (unitless)	0.01 (unitless)
Permeability Constant	0.0149	0.0149



EXPOSURE

Because of its volatility, the primary route of 1,2-DCE exposure to humans is by inhalation, however exposure via oral or dermal routes may occur but are expected to be insignificant. Exposure may occur in the workplace.

Cis-1,2-DCE may be released to the environment in emissions and wastewater during its production and use. Under anaerobic conditions that may exist in groundwater, landfills or sediment, 1,2-DCE can be formed as breakdown products from the reductive dehalogenation of trichloroethene, tetrachloroethene and 1,1,2,2-tetrachloroethane. The *cis*-1,2-DCE isomer is the more common isomer found although it is commonly mistakenly listed as the *trans*-isomer. The *trans*-isomer, is more commonly analysed for and the analytical procedures generally used do not distinguish the isomers.

If released into the environment the following can be noted with respect to 1,2-DCE (ATSDR, 1996):

- Air: In the atmosphere 1,2-DCE will be lost by reaction with photochemically produced hydroxyl radicals (half life 8 days for *cis*-isomer and 3.6 days for *trans*-isomer) and scavenged by rain. Most of the 1,2-DCE removed by rain will probably re-enter via volatilisation. Because it is relatively long lived in the atmosphere, considerable dispersal from source areas should occur.
- Soil: If 1,2-DCE (*cis* or *trans*) is released on soil, it should evaporate and/or leach into the groundwater where very slow biodegradation should occur. Adsorption of 1,2-DCE to soil, sediment or suspended solids in water is not a significant fate process.
- Water: If released into water, 1,2-DCE (*cis* or *trans*) will be lost mainly through volatilization (half life 3 hr in a model river).
- Biodegradation: Biodegradation, adsorption to sediment, and bioconcentration in aquatic organisms should not be significant. In groundwater 1,2-DCE may undergo anaerobic biodegradation with a biodegradation half life of approximately 13-48 weeks. Aerobic biodegradation processes have also been observed. 1,2-DCE is commonly found in mixtures with other chlorinated solvents and hence half-lives can only be approximated.
- 1,2-DCE has a low tendency to bioconcentrate in aquatic or marine organisms.

HEALTH EFFECTS

General

The following information is available from WHO (2003), USDOE (1994) and ATSDR (1996). There is no clinical disease which is unique to 1,2-DCE toxicity.

1,2-DCE is rapidly absorbed by the lungs. Once absorbed, the chemical is metabolised by the liver to form dichloroethanol and dichloroacetic acid via the epoxide intermediate. Animal studies indicate that the metabolism of the *cis*-isomer occurs faster than that of the *trans*-isomer. As the *cis* and *trans*-isomers are lipid soluble of low molecular weights, they would be expected to be readily absorbed by the oral or dermal routes.

Toxicokinetic data are very limited for both human and animals exposures to 1,2-DCE. Although the compound is relatively lipophilic, there is no good evidence of accumulation in the liver, brain, kidney and adipose tissue. 1,2-DCE is likely to be metabolised to more hydrophilic by-products and therefore eliminated quickly by the kidney as metabolites.

Workers exposed to 1,2-DCE have been reported to suffer from drowsiness, dizziness, nausea, fatigue, and eye irritation. Acute and sub-chronic oral and inhalation animal studies of *trans*-1,2-DCE and acute inhalation animal studies of *cis*-1,2-DCE suggest that the liver is the primary target organ. The toxicity is expressed in increased activities of liver associated enzymes, fatty degeneration, and necrosis. Secondary target organs include the central nervous system and lung. No information is available concerning the chronic, developmental, or reproductive toxicity of *cis*-1,2-DCE or *trans*-1,2-DCE.

Cancer and Genotoxic Effects

No studies are available regarding carcinogenicity.

In vitro investigations of the genotoxic potential of 1,2-DCE indicated negative results for both isomers. 1,2-DCE was not found to be mutagenic and neither isomer induced chromosomal aberrations or sister chromatid exchanges in a study in Chinese hamster lung fibroblasts (WHO, 2003).

In vivo studies indicate that *cis*-, and possibly the *trans*-, isomer may be genotoxic (WHO, 2003). The *cis*-isomer was found to be mutagenic in two mice studies where chromosomal aberrations in mouse bone marrow cells were observed. The *trans*-isomer yielded negative results in these studies.

Review of 1,2-DCE by RIVM (2001) with respect to genotoxicity and carcinogenicity conclude that *cis*-1,2-DCE should be considered as a genotoxic agent *in vivo*, producing gene mutations and chromosome aberrations. However, carcinogenicity data are not available and hence the estimation of risk has to be based on non-carcinogenic (threshold) toxicity data. The *trans*-isomer was negative in *in vivo* test systems, but has induced aneuploidy in an *in vitro* test. For the induction of this kind of genotoxic effect the threshold approach is applicable.

TOXICITY CLASSIFICATION

USEPA has placed both *cis*-1,2-DCE and *trans*-1,2-DCE in weight-of-evidence group D, not classifiable as to human carcinogenicity, based on the lack of human or animal carcinogenicity data and on essentially negative mutagenicity data. Oral and inhalation slope factors have not been calculated for these isomers.

1,2-DCE has not been evaluated by the IARC or the National Occupational Health and Safety Commission (NOHSC) or NICNAS with respect to carcinogenicity.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC 2004) have derived a drinking water guideline of 0.06 mg/L for 1,2-DCE (both isomers) following guidance from the WHO (refer below).

Worksafe Australia (NOHSC) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment” (available within the Hazardous Substances Information System, NIOSH 2006). For 1,2-DCE, the following have been established:

TWA: 200 ppm, equivalent to 793 mg/m³

STEL: NA

WHO

The WHO (Drinking Water Guidelines 2004) has derived a guideline of 0.05 mg/L based on a TDI of 0.017 mg/kg/day based on a NOAEL of 17 mg/kg from a 90 day study in mice administered *trans*-1,2-DCE in drinking water and an uncertainty factor of 1000. It is implied that the application of this guideline is relevant to the sum of both *cis*- and *trans*- isomers, however this is due to the WHO adopting a conservative approach where there is no data being available for the derivation of a *cis*- isomer value.

The WHO (2000, 2000b) have not evaluated or provided an inhalation guideline values for 1,2-DCE.

EU

No assessment of 1,2-DCE is available from the EU.

RIVM

Review of 1,2-DCE by RIVM (2001) has provided tolerable daily intakes and tolerable concentrations in are relevant for *cis*- and *trans*-isomers.

A TDI of 0.006 mg/kg/day has been established for *cis*-1,2-DCE based on a NOAEL of 32 mg/kg/day from a 90 day oral rat study and an uncertainty factor of 5000. For *trans*-1,2-DCE a TDI of 0.017 mg/kg/day was established using the same study and approach presented by the WHO.

Inhalation tolerable concentrations (TC) were derived for *cis*-1,2-DCE using route extrapolation from the oral study, resulting in a TC of 0.030 mg/m³. A TC of 0.060 mg/m³ was established for *trans*-1,2-DCE based on a LOAEL of 185 mg/m³ (continuous exposure) derived from liver and lung effects from an inhalation study on rats and applying an uncertainty factor of 3000. Both these inhalation values are considered to be provisional due to route extrapolation (*cis*-isomer) or poor database (*trans*-isomer).

US

The USEPA (IRIS current in 2006) has derived the following for 1,2-DCE:

- Non-cancer effects: oral reference dose (RfD) of 0.02 mg/kg/day for *trans*-1,2-DCE on the basis of a 90 day mouse study with drinking water (same study as used by WHO).
- No carcinogenic or review of inhalation effects for *trans*-1,2-DCE are provided.
- No data is available for oral or inhalation exposure (carcinogenic or non-cancer) to *cis*-1,2-DCE.

HEAST (1994) has provided a provisional peer reviewed chronic oral reference dose of 0.01 mg/kg/day for *cis*-1,2-DCE based on decreased haemoglobin and hematocrits in rats from a 90 day oral rat study. This value currently under review. The oral RfD available from IRIS for *trans*-1,2-DCE and the provisional RfD from HEAST for *cis*-1,2-DCE have been used by a number of state agencies (including Region IX) in the US in establishing remediation goals or screening levels. The oral values have been extrapolated for the evaluation of inhalation exposures.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with 1,2-DCE. The levels established (valid in 2006) are:

- Acute and intermediate inhalation MRL for *trans*-isomer = 0.2 ppm based on liver effects from inhalation study in rats (same study as used by RIVM);
- Acute oral MRL for *cis*-isomer = 1 mg/kg/day based on haematological effects;
- Intermediate oral MRL for *cis*-isomer = 0.3 mg/kg/day based on haematological effects;
- Intermediated oral MRL for *trans*-isomer = 0.2 mg/kg/day based on hepatic effects.

The California Air Resources Board (CARB and OEHHA) has not established an acute or chronic reference exposure level or unit risk values for 1,2-DCE.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to 1,2-DCE, intakes from soil, water and food can be considered to be

insignificant. 1,2-DCE is not considered to be a typical urban air contaminant and little data is available from data collected in Australian cities. *Cis*-1,2-DCE has been detected in VOC sampling from Perth (WA DEP 2000) with average concentrations of 0.2 ppb ($0.8 \mu\text{g}/\text{m}^3$) and a maximum reported concentration of 2.1 ppb ($8.3 \mu\text{g}/\text{m}^3$). These values were comparable to average concentrations reported in air in the US and used by RIVM (2001) following WHO methodology to estimate background intake of 1,2-DCE (both isomers) of approximately $0.13 \mu\text{g}/\text{kg}/\text{day}$. This intake is essentially negligible in comparison with the available TDI and RfDs available from WHO, RIVM and US authorities.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,2-DCE following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

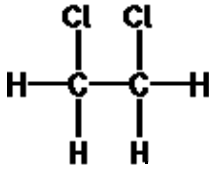
	<i>Cis</i>-1,2-DCE	<i>Trans</i>-1,2-DCE
	The <i>cis</i> -isomer is considered potentially genotoxic, however there is a lack of carcinogenic data – hence no non-threshold evaluation is available. Evaluation of exposure via all routes must be undertaken on the basis of a threshold approach.	The <i>trans</i> -isomer is not considered genotoxic and any effects identified in studies are considered appropriate to be evaluated on the basis of a threshold approach.
Oral	RfD = 0.01 mg/kg/day (USEPA, provisional peer reviewed value in HEAST based on oral exposure study using <i>cis</i> -isomer, which is similar to the TDI derived by RIVM of 0.006 mg/kg/day)	TDI = 0.017 mg/kg/day (WHO 2004 based on oral exposure study using <i>trans</i> -isomer. This value is used for both isomers, but is only derived from studies on the <i>trans</i> -isomer)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.	
Inhalation	<p>No inhalation studies are available for the evaluation of exposure to <i>cis</i>-1,2-DCE, hence the oral value has been used for the evaluation of inhalation exposures.</p> <p>RfD = 0.01 mg/kg/day (it is noted that the RIVM present a provisional TC for the <i>cis</i>-isomer based on the oral study. The value presented by RIVM is equal to the oral RfD adopted and hence the oral RfD has been used for the assessment of inhalation exposures).</p> <p>Occupational inhalation exposure (NOHSC) for 1,2-DCE (both isomers):</p> <p>TWA: 200 ppm, equivalent to $793 \text{ mg}/\text{m}^3$</p> <p>STEL: NA</p>	<p>Limited inhalation studies are available for the evaluation of exposure to <i>trans</i>-1,2-DCE, hence the oral TDI has been used for the evaluation of inhalation exposures.</p> <p>TDI = 0.017 mg/kg/day (it is noted that the RIVM present a provisional TC for the <i>trans</i>-isomer based on a limited inhalation study. The value presented by RIVM is equal to the oral TDI and hence the oral TDI has been used for the assessment of inhalation exposures).</p> <p>Occupational inhalation exposure (NOHSC) for 1,2-DCE (both isomers):</p> <p>TWA: 200 ppm, equivalent to $793 \text{ mg}/\text{m}^3$</p> <p>STEL: NA</p>
Background	Negligible	Negligible

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1,2-Dichloroethane

General



1,2-Dichloroethane (also known as ethylene dichloride, ethylene chloride, glycol dichloride, freon 150, dutch liquid, 1,2-ethylene dichloride, alpha, beta-dichloride and commonly abbreviated to **EDC**) is a synthetic product which is primarily used in the production of the vinyl chloride monomer. It is also an intermediate in the manufacture of fluorocarbons and chlorinated solvents such as trichloroethane, trichloroethylene, perchloroethylene and vinylidene.

These solvents are used to remove dirt, grease, resins and glue as well as in the manufacture of polystyrene and SBR latex. EDC is also added to leaded petrol as an anti-knock compound and has been used as a fumigant.

EDC is one of the most widely produced chemicals in the world. The majority of EDC released to the environment is in emissions to air. It is moderately persistent in the air, however it is not considered to be an ozone depleting substance.

Properties

EDC is a volatile, colourless liquid at room temperature with a pleasant smell and sweet taste. EDC evaporates into air very quickly and is soluble in water and several organic solvents such as alcohols, chloroform and ether. Key properties are presented below (ref: ATSDR, 2001 and RAIS):

CAS No.	107-06-2
Chemical Formula	C ₂ H ₄ Cl ₂
Molecular Weight	98.96
Vapour Pressure	78.9 mmHg at 25°C
Vapour Density	3.4
Density	1.23 g/ml at 20°C
Solubility	5100 mg/L at 25°C
Air Diffusion Coefficient	0.104 cm ² /s
Water Diffusion Coefficient	9.9 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0011 atm.m ³ /mol = 0.0482 at 25°C (unitless)
Koc	43.79 cm ³ /g
Log Kow	1.48
Dermal Absorption Factor	0.01 (unitless)
Permeability Constant	0.00534 (cm/hr)
Odour Threshold	48.6 to 405 mg/m ³ or 356 mg/m ³ (WHO, 1996)
Atmospheric Half-Life	63 days

Exposure

Exposure of the general population to EDC may be by inhalation, oral or dermal routes. In most cases inhalation is the primary route of exposure. Exposure may also occur through oral ingestion and dermal contact with drinking/household water and/or soils. Children may be exposed via the same pathways as for adults. EDC has been detected in human milk and hence infants could be exposed via breast-feeding. Intake from food sources is expected to be negligible. Occupational exposures (particularly inhalation and dermal contact) may occur in industries which handle the product (HSDB, ATSDR, 2001).

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If released into the environment the following can be noted with respect to EDC (HSDB):

- **Air:** EDC is expected to remain in vapour phase where it is moderately persistent with an estimated half-life of between 43 and 111 days. Once EDC reaches the troposphere, it undergoes photo-oxidation to produce formyl chloride, chloroacetyl chloride, hydrochloric acid, carbon monoxide and carbon dioxide. EDC is transported to the stratosphere where photolysis may produce chloride radicals which may in turn reach with ozone. EDC is not expected to contribute to ozone depletion. Due to its persistence in the troposphere there is the potential for long-range transport of EDC.
- **Soil and Water:** EDC is not expected to adsorb strongly in soils and may leach to groundwater where it has the potential to persist for years. EDC is expected to volatilise from surface soils and water.
- **Biodegradation:** Biodegradation is expected to occur slowly with hydrolysis and photolysis not expected to be important fate processes. The potential for bioaccumulation in aquatic or terrestrial organisms appears to be low.

Health Effects

The following has been summarised from information presented by ATSDR (2001) and WHO (1995):

General	<p>There is no clinical disease which is unique to EDC toxicity. Primary effects are associated with the liver, kidneys and neurological, cardiovascular and immune systems.</p> <p>EDC is readily absorbed into the body via inhalation, ingestion and dermal exposure. Following absorption into the body, EDC is widely distributed throughout the body. In animals the highest concentrations were generally within adipose tissue; however it is also distributed to the blood, liver, kidney, brain and spleen. EDC is metabolised extensively. Unmetabolised EDC is eliminated in expired air, while its metabolites (principally sulphur containing metabolites) are largely excreted in the urine. Although EDC is eliminated more slowly from adipose tissue than from blood or other tissues (lung and liver) following exposure, it is unlikely to bioaccumulate significantly.</p> <p>The following summary has been derived from ATSDR (2001).</p>
Death	<p>Acute inhalation and oral exposure of EDC has been known to result in death in humans. Cause of death is typically attributed to cardiac arrhythmia.</p>
Hepatic Effects	<p>Liver effects have been identified following acute inhalation or ingestion of EDC by humans and animals. Hepatic effects in animals were not limited to any specific route or duration of exposure and included increased levels of serum markers of liver dysfunction, increased liver weight and fatty degeneration.</p>
Renal Effects	<p>EDC is acutely nephrotoxic in humans following both inhalation and ingestion. Renal effects in humans include diffuse necrosis, tubular necrosis and kidney failure. Renal effects in animals include increased kidney weight, cloudy swelling of the tubular epithelium, tubular degeneration and regeneration, karyomegaly, dilation, protein casts and mineralisation.</p>
Immunological	<p>Immunological effects have not been reported in humans. In mice, immunological effects have been reported following both acute inhalation and oral exposure. Due</p>

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Effects	to interspecies differences in immunotoxicity, it is unclear whether the immune system may be a target of EDC in humans based on the mice studies.
Neurological Effects	Neurological effects reported by people acutely exposed to high concentrations of EDC via inhalation or ingestion include headache, irritability, drowsiness, tremors, partial paralysis and coma. Animal studies indicate the CNS is a target of high concentrations of EDC. Available data do not enable characterisation of the potential for EDC to cause more subtle neurotoxic effects following low-level prolonged exposures by inhalation, oral or dermal exposure.
Cardiovascular Effects	Cardiac effects (arrhythmias, insufficiency and haemorrhage) have been observed in humans acutely exposed to high concentrations of EDC. The available animal data suggests that the heart could be a target of EDC following acute high level exposure and possibly longer-term inhalation exposure.
Developmental Effects	Some developmental effects have been reported in humans and animals. However, the available information does not indicate that EDC is a developmental toxicant in animals at doses below those that cause other toxic effects.
Genotoxic Effects	The genotoxicity of EDC has been extensively investigated in non-mammalian and mammalian test systems. Following review of the available data by WHO (1998), EDC has been identified as genotoxic in <i>in vitro</i> and <i>in vivo</i> assays, and binds to DNA in rodents <i>in vivo</i> . Review of genotoxicity by Woodward-Clyde (1996) indicated that the available evidence in animals suggests that EDC is genotoxic.
Cancer	<p>Available data on the carcinogenicity of EDC in humans are limited. There are no epidemiological studies which show an associated between EDC exposure and cancer. There is convincing evidence of increases in the incidence of both common and rare tumours in experimental animals at several sites (including squamous cell carcinomas of the stomach, haemangiosarcomas, fibromas of the subcutaneous tissue and adenocarcinomas and fibroadenomas of the mammary gland in rats; and alveolar/bronchiolar adenomas, mammary gland adenocarcinomas, endometrial stromal polyp or endometrial stromal sarcoma combined and hepatocellular carcinomas in mice) following oral exposure studies (WHO, 1998).</p> <p>The incidence of benign lung papillomas was significantly increased in mice following long-term dermal application of EDC, while a non-significant increase in the number of pulmonary adenomas per animal was reported in a screening bioassay on mice and in the incidence of benign mammary gland tumours in rats exposed by inhalation for 2 years (WHO 1998). In addition more recent studies (Nagano et al (1998 and 2006) show carcinogenic outcomes associated with inhalation exposures.</p>

Toxicity Classification

EDC was classified as a "probable" human carcinogen (Category B2) by the USEPA (IRIS) for all routes of exposure based upon evidence from animal studies.

IARC has classified EDC in Group 2B (possibly carcinogenic to humans) based on inadequate evidence in humans for carcinogenicity and sufficient evidence in experimental animals.

NICNAS has not classified EDC.

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Exposure Limits and Toxicity Evaluations

Exposure limits and toxicity evaluations which are available in Australia, World Health Organisation, European Union and the US:

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have derived a drinking water guideline of 0.003 mg/L for EDC using an **oral slope factor of 0.01 (mg/kg/day)¹** and lifetime excess cancer risk of 1 in 1,000,000.

Worksafe Australia (ASCC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For EDC, the following have been established:

TWA: 10 ppm, equivalent to 40 mg/m³

STEL: NA

It should be noted that this chemical is flagged as a Category 2 carcinogen that requires further review based on carcinogenic potential.

WHO

The WHO (Drinking Water Guideline WHO, 2003) have established a guideline of 0.03 mg/L using a linearised multistage model and an excess lifetime cancer risk of 1 in 100,000. This corresponds to an **oral slope factor of 0.01 (mg/kg/day)¹** (as used by NHMRC). It is noted that a lower guideline was proposed as part of the review process, however this has not been included in the final, and current, WHO drinking water guideline.

The WHO (1998) also note that the available data indicate that EDC is less potent when inhaled.

WHO (2000a) has undertaken a review of 1,2-dichloroethane for inhalation exposures in the late 1990's. The review indicates that there is sufficient evidence of carcinogenicity in animals based on oral ingestion data. However, animal inhalation data reviewed at the time did not provide sufficient evidence of carcinogenicity and hence the WHO did not provide a carcinogenic assessment of inhalation exposures to EDC. Because of deficiencies in extrapolating oral data to inhalation, neither the oral slope factor or any inhalation value have been recommended by the WHO in this assessment. A guideline value of 0.7 mg/m³ for a 24-hour average has been derived for non-carcinogenic endpoints by the WHO (2000a) based on a lowest-observed-adverse-effect level from animal studies. It is noted that this guideline value recommended for the assessment of accidental release episodes or specific indoor pollution problems. The evaluation by WHO in this document was undertaken prior to the publication of a study by Nagano et al (1998 and 2006). The studies provided by Nagano et al show that inhalation exposures to EDC result in DNA damage and carcinogenic effects in animals. Hence it is considered reasonable to assume that EDC is a genotoxic carcinogen via inhalation routes of exposure as well as oral exposures.

Air guidance values established by the WHO (1998) based on the protection of carcinogenic endpoints, and considered representative of negligible risk, ranged from 3.6-20 µg/m³ (5000 fold

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safety margin) or 0.36-2.0 $\mu\text{g}/\text{m}^3$ (50000 fold safety margin and approximately equivalent to the use of the range of unit risk values identified below).

A web-based source WHO (2000b) provides a summary of available inhalation exposure assessments available from the WHO (EHC and CICAD documents). For EDC the document references the CICAD published by the WHO (1998) where a range of inhalation unit risk values for exposure to 1,2-dichloroethane in air have been presented based on tumour formation (based on oral studies). The range of inhalation unit risk values is $(0.6 \text{ to } 3) \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$ (i.e. for an air concentration of 1 $\mu\text{g}/\text{m}^3$, the lifetime risk is estimated to be $(0.6 \text{ to } 3) \times 10^{-6}$). This is equivalent to the following inhalation slope factor:

$$\begin{aligned} SF (\text{mg}/\text{kg}/\text{day})^{-1} &= \text{Risk}/\text{Intake}(\text{mg}/\text{kg}/\text{day}) \\ &= [\text{Risk} \times \text{Body Weight}]/[\text{Concentration (in air)} \times \text{Inhalation Rate}] \\ &= [(0.6 \text{ to } 3) \times 10^{-6} \times 70\text{kg}]/[0.001\text{mg}/\text{m}^3 \times 20 \text{ m}^3/\text{day}] \\ &= \mathbf{0.0018 \text{ to } 0.0098 (\text{mg}/\text{kg}/\text{day})^{-1}} \end{aligned}$$

The higher value in the range presented above for inhalation exposures is considered the more conservative (higher risk). This value is approximately equal to the NHMRC slope factor used to derive the drinking water guideline, namely $0.01 (\text{mg}/\text{kg}/\text{day})^{-1}$ and is recommended for a conservative evaluation of inhalation exposures.

EU

The European Commission published a directive in 1990 in which limit values for emission of EDC were specified for various types of industrial plants. These limits ranged from 0.1 mg/litre (monthly) for plants using EDC for degreasing metals away from an industrial site to 12 mg/litre (daily) for plants producing EDC and processing or using the substance at the site (WHO, 1998). No other assessment of EDC is available from the EU.

The UNECE has published a SIDS Initial Assessment Report (2002) for EDC. Based on review of data, EDC should be regarded as harmful following repeated inhalation exposure (with effects in lungs, liver and kidneys) and regarded as a suspected carcinogen. EDC is considered to be genotoxic. The cytochrome-P450 and glutathione dependent pathways are assumed to be responsible for the generation of intermediates capable of binding to and damaging DNA. Systemically toxic inhalation concentrations result in significantly lower blood and organ levels than toxic gavage doses and, therefore, are expected to be (hypothetically) less likely to form oncogenic intermediates.

RIVM

Review by RIVM, 2001 has identified the following with respect to EDC:

- EDC is considered a genotoxic compound and as such oral and inhalation exposure is assessed on the basis of a non-threshold approach adopting an acceptable cancer risk level of 1 in 10,000 lifetime risk.
- On the basis of the above, an oral cancer risk intake level of 0.014 mg/kg/day and a provisional inhalation cancer risk air concentration of 0.048 mg/m³ (based on oral data) have been established. If considered for a lifetime cancer risk level of 1 in 1,000,000, the air guideline would be equivalent to the lower guideline value established by the WHO (1998).

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US

The USEPA (IRIS) has derived an **oral slope factor of 0.091 (mg/kg/day)⁻¹** for EDC based on a linear multistage model based on hemangiosarcomas in rats; and an **inhalation unit risk of 2.6x10⁻⁵ (µg/m³)⁻¹** using a linear multistage model based on oral data used to derive the oral slope factor. The USEPA does not present any data relevant to the assessment of non-carcinogenic effects for EDC.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with EDC in the 2001 review, however the current listing provided by the ATSDR has no MRLs listed for EDC. The levels established in 2001 are:

- Chronic (and intermediate) inhalation MRL = 0.6ppm based on liver histopathology in rats; and
- Intermediate oral MRL = 0.2 mg/kg/day based on increased kidney weights in rats.

The California Air resources Board (OEHHA) has established **inhalation unit risk value of 2.1x10⁻⁵ (µg/m³)⁻¹** and a **chronic reference exposure level for EDC of 0.4 mg/m³** based on hepatotoxicity (elevated liver enzyme levels in serum of rats). No acute reference exposure levels have been established.

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. However, as EDC has been evaluated to be a genotoxic carcinogen it is considered appropriate to evaluate exposure using a slope factor for oral, inhalation and dermal exposures. Hence background intake is not relevant to this assessment.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for EDC following review of the available information in general accordance with guidelines from enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

Oral	Oral Slope Factor = 0.01 (mg/kg/day)⁻¹ (NHMRC, 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation unit risk of 0.6x10⁻⁶ (per µg/m³) , equivalent to 0.009 (mg/kg/day)⁻¹ (WHO 2000b). Occupational inhalation exposure (NOHSC): TWA: 10 ppm, equivalent to 40 mg/m³ STEL: NA
Background	NA

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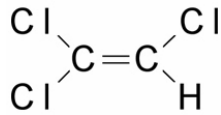
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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2008A	12/2/2008, provided additional discussion and references on inhalation carcinogenic effects
Previous Revisions:	2007A	14/12/2007
	2006A	31/7/2006
	2005A	16/6/2005

GENERAL

Trichloroethene (also known as 1,1,2-trichloroethylene, ethylene trichloride, and commonly abbreviated to **TCE**) is a synthetic product that was first prepared in 1864 by the reduction of hexachloroethane with hydrogen. It is mainly used as a liquid or vapour degreasing solvent, particularly in the metal fabricating industry. International concern about the environmental and health and safety concerns of chlorinated solvents has reduced the use of TCE.

TCE was manufactured in Australia from the 1950's to the early 1980's, with current demand met by imports of the chemical. TCE is also recycled in Australia. TCE is used widely in both large and small industries in Australia for vapour degreasing, cold cleaning as well as use in adhesives, waterproofing agents, paint strippers, carpet shampoos and some other cleaning products. It is also an effective cleaning agent for organic materials as it has a low latent heat of vaporisation and is non-flammable.

PROPERTIES

TCE is a volatile, colourless or blue mobile liquid with a sweet chloroform-like odour. TCE evaporates into air very quickly and dissolves slightly in water. Key properties are presented below (ATSDR 1995, USEPA 2004 and ORNL Database 2006):

CAS No	79-01-6
Chemical Formula	C ₂ HCl ₃
Molecular Weight	131.4
Vapour Pressure	74 mmHg at 25°C
Vapour Density	4.53
Density	1.465 g/ml at 20°C
Solubility	1100 mg/L at 20°C
Air Diffusion Coefficient	0.079 cm ² /s
Water Diffusion Coefficient	9.1 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0103 atm.m ³ /mol = 0.422 at 25°C (unitless)
Koc	166 cm ³ /g
Log Kow	2.42
Odour Threshold	115 mg/m ³ recognition in air (WHO 2000)
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0157

EXPOSURE

Exposure of the general population to TCE may be by inhalation, oral or dermal routes. In most cases inhalation is the primary route of exposure. Exposure may occur through oral ingestion of drinking water or soils, however exposure to TCE in food is generally low. Apart from occupational exposures, the primary concern is inhalation indoors. TCE in the outdoor air may originate from indoor or outdoor sources. Outdoor sources include outdoor air, contaminated soils or groundwater. Indoor air sources include new building construction materials or home cleaning products. The potential for bioaccumulation of TCE is considered to be low.

If released into the environment the following can be noted with respect to TCE:

- Air: TCE is expected to remain in vapour phase. Removal is primarily through reaction with hydroxyl radicals to produce low levels of phosgene, dichloroacetyl chloride, formyl chloride and other degradation products. Half-life of TCE varies from 1 day to months.
- Soil and Water: TCE is expected to volatilise from surface soils and water. TCE may leach through soil into groundwater where it may persist for years.
- Water: Depending on conditions reductive dehalogenation to vinyl chloride may occur. Under anaerobic conditions TCE can be intrinsically biodegraded to form DCE and vinyl chloride (below).

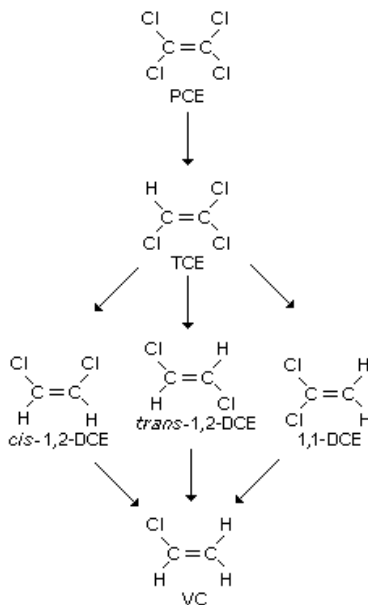


Figure 1. Pathway for anaerobic microbial degradation of chlorinated ethenes to form vinyl chloride (from: WHO, 1999)

NB: PCE=tetrachloroethene, TCE=trichloroethene, DCE=dichloroethene

HEALTH EFFECTS

General

There is no clinical disease which is unique to TCE toxicity. In the past, TCE was used as a human anaesthetic. TCE has also been inhaled by people intentionally for its narcotic effect. Hence most toxicological data is associated with inhalation exposures. Primary effects are associated with the central nervous system (CNS).

TCE can be absorbed into the body via inhalation, ingestion and dermal exposure. Following absorption into the body, TCE is distributed to the blood, then transported to various tissues where it is metabolised. The toxicities associated with TCE are thought to be mediated by metabolites rather than the parent compound. Major sites of TCE distribution appear to be the body fat and liver.

Humans and animals excrete un-metabolised TCE via expiration, while the metabolites are excreted primarily in urine. Urinary metabolites include trichloroacetaldehyde, trichloroethanol, and trichloroacetic acid; the reactive epoxide TCE oxide is an essential feature of the metabolic pathway.

The following summary has been derived from NICNAS (2000) and ATSDR (1995).

Death	Acute inhalation and oral exposure of TCE has been known to result in death in humans. Cause of death is typically attributed to hepatorenal failure (ingestion), ventricular fibrillation or CNS depression.
Gastrointestinal Effects	Acute inhalation exposure to TCE has results in nausea and vomiting. Chronic exposure to TCE in the occupation environment has been associated with anorexia and vomiting.
Hepatic Effects	There is some evidence for TCE induced hepatotoxic effects in humans. Reports (occupational) support the liver as the end point of TCE toxicity. Studies in animals (inhalation and oral) over acute and intermediate periods indicate liver enlargement.
Dermal/Ocular Effects	Exposure to high doses of TCE through contact with the air or skin has resulted in skin irritation and rashes. Stevens-Johnson syndrome (severe erythema), dermatitis and scleroderma have been reported in occupational environments. Adverse effects have not been reported from exposure to dilute aqueous solutions of TCE. Ocular effects such as mild eye irritation have been observed in occupational environments.
Body Weight Effects	Body weight loss has been reported in humans occupationally exposed to TCE in air for intermediate of chronic durations at concentrations resulting in neurological effects. No significant effects were observed from oral studies.
Immunological Effects	No significant effects have been reported following inhalation an oral exposures and animal studies.
Neurological Effects	Primary effects identified following inhalation exposures to TCE are associated with the CNS. Effects include headache, vertigo, fatigue, nausea, memory loss, decreased word associations, depression of the CNS, and anaesthesia. Animal studies have reported neurotoxicity and neuropathology effects following oral exposure studies. These effects in part are thought to be due to the sedative

Reproductive Effects	properties of the metabolite trichloroethanol (TCOH). Reproductive effects (increases in miscarriages) have been observed in following exposure to TCE in humans and animals.
Developmental Effects	Other than reproductive effects, no significant developmental effects have been identified following inhalation exposures to TCE. Evidence of birth defects following TCE exposure in drinking water is not clear, however animal studies indicate TCE can act as a developmental toxicant following oral exposure.
Genotoxic Effects	Studies are not conclusive but may be suggestive of clastogenic effects. No human oral studies are available, and animal oral studies indicate conflicting findings. Review of TCE by Woodward-Clyde (1996) indicates that the weight of evidence suggests that TCE has a limited ability to cause genotoxicity. TCE is only weakly mutagenic in bacteria and yeast and the ability of TCE to interact with DNA in whole animals is observed only at high doses. Review by NICNAS (2000) indicates that TCE can be classified a category 3 mutagen – <i>“as a substance which cause concern for humans owing to possible mutagenic effects, but in respect of which available information does not satisfactorily demonstrate heritable genetic damage.”</i>
Cancer	No clear unequivocal evidence is available that TCE inhalation exposure is linked to increased cancer risk. The link between oral exposure to TCE and cancer in humans is controversial. Studies in rats and mice have indicated TCE and its metabolites are carcinogenic in animals. TCE has been shown to induce lung and liver tumours in various strains of mice at toxic doses. However, there are no conclusive data that the chemical causes cancer in other species. Review of TCE by Woodward-Clyde (1996) indicates similar findings.

TOXICITY CLASSIFICATION

TCE was classified as a "probable" human carcinogen (Category B2) by the USEPA for all routes of exposure based upon evidence from animal studies. This classification has been withdrawn pending further review (not finalised as of December 2005).

IARC has classified TCE in Group 2A (probably carcinogenic to humans) based in limited evidence from several human epidemiological studies and on sufficient evidence from animal studies.

NICNAS has classified TCE as a Carcinogen Category 2, which is a substance regarded as if it is carcinogenic to humans, on the basis of the occurrence of tumours in experimental animals and limited evidence in workers.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have indicated that there are no long-term studies available to establish a no effect level associated with TCE, hence the available data was considered inadequate to establish an Australian guideline.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For TCE, the following have been established (including update adopted in August 2005):

TWA: 10 ppm, equivalent to 54 mg/m³

STEL: 40 ppm, equivalent to 216 mg/m³

WHO

The WHO (Drinking Water Guideline 1996 and 2004) established a **TDI of 23.8 µg/kg of body weight** (including allowance for 5 days per week dosing). This was calculated by applying an uncertainty factor of 3000 to a LOAEL of 100 mg/kg of body weight per day for minor effects on relative liver weight in a 6-week study in mice. The uncertainty factor components are 100 for inter- and intra-species variation, 10 for limited evidence of carcinogenicity, and an additional factor of 3 in view of the short duration of the particular study and the use of a LOAEL rather than a NOAEL.

Review of TCE by the WHO (issued as draft for review in 2004 and final in May 2005) as part of the rolling revision to the guidelines has provided a provisional guideline value that has been derived to be protective of both cancer effects and non-cancer effects. With respect to the evaluation of cancer effects, the linearised multistage (LMS) model was used to calculate a unit risk (slope factor) for kidney tumours observed in rates. Use of the LMS model is considered relevant based on possible genotoxicity associated with some TCE metabolites (particularly DCVC and DCVG). The slope factor derived was **7.8x10⁻⁴ (mg/kg/day)⁻¹** following review of data from oral and inhalation studies. Use of a non-threshold approach resulted in a higher drinking water guideline than calculated using a threshold approach that is based on the protection of reproductive-developmental effects.

Review of non-cancer effects resulted in the derivation of a TDI. This was undertaken using a LOAEL from a developmental toxicity study, applying a benchmark dose approach to estimate a NOAEL, and application of an uncertainty factor of 100. The TDI derived was **0.00146 mg/kg/day**. This TDI is lower than utilised in the WHO DWG from 1996 and 2004 and has been utilised in the derivation of the revised drinking water guideline as the guideline developed is lower than that developed on the basis of non-threshold effects.

The WHO (2000) provided toxicity data for a range of chemicals which were considered to have carcinogenic endpoints. TCE was one of those chemicals identified and an inhalation unit risk of 4.3x10⁻⁷ (per µg/m³) for the assessment of exposures to TCE in air has been established. (i.e. for an air concentration of 1 µg/m³, the lifetime risk is estimated to be 4.3x10⁻⁷). The unit risk has been established

by the WHO based on increase tumours in lungs and testes in animal bioassays. In utilising this data, the WHO note that “*it cannot be conclusively established whether a threshold with regard to carcinogenicity in the action of TCE may be assumed.*” Hence a conservative approach (deriving a unit risk) has been adopted by the WHO.

The unit risk value is equivalent to the following slope factor:

$$\begin{aligned}
 SF \text{ (mg/kg/day)}^{-1} &= \text{Risk/Intake(mg/kg/day)} \\
 &= [\text{Risk} \times \text{Body Weight}]/[\text{Concentration (in air)} \times \text{Inhalation Rate}] \\
 &= [4.3 \times 10^{-7} \times 70 \text{kg}]/[0.001 \text{mg/m}^3 \times 20 \text{m}^3/\text{day}] \\
 &= \mathbf{0.0015 \text{ (mg/kg/day)}^{-1}}
 \end{aligned}$$

EU

Review of TCE by the European Union (EU) in 2004 indicates that TCE gives rise to concern for humans owing to possible mutagenic and carcinogenic effects and because it is not possible to identify a threshold exposure level below which these effects would not be expressed. TCE is an *in vitro* mutagen in the presence of an exogenous metabolic activation system. Conflicting data exists, however the weight of evidence indicates that TCE can also exhibit genotoxic activity in somatic tissues *in vivo*. TCE is considered to have the potential to cause cancer in humans. The evaluation of exposure by the EU has focused on workers, consumers and environmental exposures. The evaluation has reviewed relevant toxicity end points, evaluated body burden associated with exposure and calculated a Margin of Exposure (MOE). The most sensitive threshold effect evaluated was associated with CNS disturbance following repeated dose where a NOAEL of 38 mg/kg/day was used.

The EU has presented a calculation of lifetime cancer risk based on the T25 method in relation to non-Hodgkin lymphoma. From an inhalation study in female mice a HT25 dose descriptor for humans was derived as 130 mg/kg/day. Following the approach presented the EU calculated increased cancer risk for TCE for all groups using an **equivalent slope factor of 0.0019 (mg/kg/day)⁻¹**. This value was used in the quantification of risk associated with exposure from oral, dermal and inhalation pathways.

US

The USEPA has withdrawn the slope factor and reference dose for TCE in 1994, pending review. Prior to being withdrawn, the USEPA had determined an oral slope factor of 0.013 (mg/kg/day)⁻¹ and an inhalation slope factor of 0.006 (mg/kg/day)⁻¹.

The USEPA issued an evaluation of TCE as a draft for review in 2001. The evaluation indicated that mechanistic research indicates that TCE-induced carcinogenesis is complex, involving multiple carcinogenic metabolites acting through multiple modes of action. Under EPA's proposed (1996, 1999) cancer guidelines, TCE can be characterized as “highly likely to produce cancer in humans.” For effects other than cancer, an oral reference dose (**RfD**) of **3x10⁻⁴ mg/kg/d** was based on critical effects in the liver, kidney, and developing fetus. An inhalation reference concentration (**RfC**) of **4x10⁻² mg/m³** was based on critical effects in the central nervous system, liver, and endocrine system. Several cancer slope

factors were developed, with most between 0.02 and 0.4 per mg/kg/d. Several sources of uncertainty have been identified and quantified. The review process has not been completed to date.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with TCE. The levels established (current) are:

- Acute inhalation MRL = 2ppm based on neurological effects in humans
- Intermediate inhalation MRL = 0.1ppm based on neurological effects in rats
- Acute oral MRL = 0.2 mg/kg/day based on developmental effects in mice

The California Air resources Board (CARB, 1990) has established an inhalation unit risk for the evaluation of chronic exposure to TCE. The inhalation unit risk is 2×10^{-6} to 3×10^{-6} ($\mu\text{g}/\text{m}^3$)⁻¹.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to TCE, intakes from soil, water and food can be considered to be insignificant. Intakes from air have been calculated from industrial air concentrations in reported in Brisbane (Hawas O. et. Al., 2001), with the maximum concentration reported of $0.000546 \text{ mg}/\text{m}^3$ (representing an intake of approximately $0.00018 \text{ mg}/\text{kg}/\text{day}$). Hence background intakes of TCE can be considered to be low and does not affect the use of available ADI, TDI or RfD values.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for TCE following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	TDI = 0.00146 mg/kg/day (WHO Drinking Water Guidelines, 2005)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation unit risk of 4.3×10^{-7} (per $\mu\text{g}/\text{m}^3$), equivalent to $0.0015 \text{ (mg/kg/day)}^{-1}$ (WHO 2000, also similar to that derived by EU 2004). Occupational inhalation exposure evaluated using the following (NOHSC current, update adopted August 2005): TWA: 10 ppm, equivalent to $54 \text{ mg}/\text{m}^3$ STEL: 40 ppm, equivalent to $216 \text{ mg}/\text{m}^3$
Background	Low

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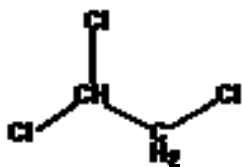
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GENERAL



1,1,2-Trichloroethane (also known as ethane trichloride; 1,1,2-TCE; beta-trichloroethane; 1,2,2-trichloroethane; vinyl trichloride; trichloroethane (non-specific name) and commonly abbreviated to **1,1,2-TCA**) is a predominantly man-made chemical. 1,1,2-TCA is a chemical intermediate in the production of 1,1-dichloroethene. 1,1,2-TCA has limited use as a solvent for fats, oils, waxes and resins. It is also released to the environment as a result of anthropogenic activity and it has also been identified as an intermediate in the biodegradation of 1,1,2,2-tetrachloroethane (another man-made chemical). It is formed commercially by the chlorination of ethylene with chlorine or by the oxychlorination of ethylene with HCl and oxygen.

PROPERTIES

1,1,2-TCA is a non-flammable, colourless, volatile liquid with a pleasant, sweet odour. It is insoluble in water and miscible with alcohol, ether and many organic liquids. Key properties are presented below (ATSDR 1989, USEPA 2004 and ORNL Database 2006):

CAS No	79-00-5
Chemical Formula	C ₂ H ₃ Cl ₃
Molecular Weight	133.41
Vapour Pressure	22.49 mmHg at 25°C
Vapour Density	4.63
Density	1.4 g/ml at 20°C
Solubility	1100 mg/L at 20°C
Air Diffusion Coefficient	0.078 cm ² /s
Water Diffusion Coefficient	8.8 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.000913 atm.m ³ /mol = 0.0374 at 25°C (unitless)
Koc	67.7 cm ³ /g
Log Kow	1.89
Odour Threshold	2.8 - 926.8 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00643 cm/hr

EXPOSURE

Exposure of the general population to 1,1,2-TCA may occur primarily through inhalation, however exposure via oral or dermal routes may occur but are expected to be insignificant. Exposure may occur in the workplace where it is used as a solvent.

If released into the environment the following can be noted with respect to 1,1,2-TCA (ATSDR, 1989):

- **Air:** Most of the 1,1,2-TCA released to the environment enters the atmosphere where it is fairly stable. In the atmosphere, 1,1,2-TCA is degraded by photochemically-produced hydroxyl radicals with a half-life of approximately 49 days.
- **Soil and Water:** Following releases to soil, 1,1,2-TCA is expected to partially volatilise, with the remainder leaching into the subsurface soil profile and groundwater. If released to surface water most would volatilise with the remainder dissolving in water. The chemical would not be expected to show appreciable adsorption to sediment or suspended organic material.
- **Biodegradation:** 1,1,2-TCA may undergo slow biodegradation under anaerobic. Anaerobic degradation occurs predominantly through reductive dehalogenation which forms vinyl chloride. Aerobic degradation occurs via substitutive and oxidative mechanisms with the production of trichloroethyl alcohol. Aerobic degradation and hydrolysis are not likely to be an important fate processes for 1,1,2-TCA.
- 1,1,2-TCA has a low tendency to bioconcentrate in aquatic or marine organisms.

HEALTH EFFECTS

General

The following information is available from USDOE (1995) and ATSDR (1989). There is no clinical disease which is unique to 1,1,1-TCA toxicity.

1,1,2-TCA is rapidly and extensively absorbed into the body following inhalation exposures (principal route of exposure) and dermal exposure.

One absorbed, 1,1,2-TCA is distributed widely in body tissues (including the liver, fatty tissue, kidneys, blood and brains, heart, spleen and lungs). The primary metabolites identified are chloroacetic acid, S-carboxymethylcysteine, and thiodiacetic acid. Elimination occurs via exhalation and urine (including elimination of metabolites).

No information is available on the acute effects of 1,1,2-TCA in humans from inhalation or oral exposures. Tests involving acute exposure of mice and rats have shown 1,1,2-TCA to have moderate and high acute toxicity from inhalation and oral exposures, respectively. Studies on dermal exposure to 1,1,2-TCA in humans have reported stinging and burning sensations and transient whitening of the skin.

Animal studies have reported effects on the liver, kidney, and central nervous system (CNS) from acute inhalation and oral exposure.

No information is available on the chronic effects of 1,1,2-TCA in humans from inhalation or oral exposure. Animal studies have not observed adverse effects from chronic inhalation exposure to 1,1,2-TCA, however effects on the liver and immune system have been noted in chronic oral studies.

No information is available regarding developmental or reproductive effects of 1,1,2-TCA in humans from inhalation or oral exposure. Animal studies have not reported developmental or reproductive effects from oral exposure to 1,1,2-trichloroethane.

Genotoxicity

Potential for genotoxicity of 1,1,2-TCA was reviewed by Woodward-Clyde (1996) which indicated that the available data were inadequate to enable a proper evaluation of genotoxic potential. In particular, there were a lack of gene mutation assays using mammalian cells and *in vivo* chromosome damage assays. Review of genotoxicity by OECD (2000) recommended further work such as an *in vivo* genotoxicity study. This was undertaken and reported in 2003 (OECD, 2003) which showed negative results. Hence the weight of evidence suggests that 1,1,2-TCA is not genotoxic *in vivo*.

Carcinogenicity

No studies are available regarding cancer in humans from inhalation or oral exposure. A study reported liver tumours and adrenal tumours in mice, but no tumours in rats from exposure to 1,1,2-TCA by gavage. Initiation/promotion screening studies on male rat liver demonstrated that the chemicals has neither initiation nor promotion activity. A carcinogenic study in skin of rats indicted no chemical related changes.

TOXICITY CLASSIFICATION

1,1,2-TCA has been classified as a “possible” human carcinogen (Category C) by the USEPA on the basis of hepatocellular carcinomas and pheochromcytomas in one strain of mice.

IARC (1999) has classified 1,1,2-TCA in Group 3 (not classifiable as to its carcinogenicity to humans) based on no epidemiological data and limited evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) and NICNAS have not classified the potential carcinogenicity of 1,1,2-TCA.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 1996 and 2004) have not derived a drinking water guideline for 1,1,2-TCA.

Worksafe Australia (NOHSC) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment”. For 1,1,2-TCA, the following have been established (current):

TWA: 10 ppm, equivalent to 55 mg/m³

STEL: none established

WHO

The WHO (Drinking Water Guideline 1996 and 2004) have not derived a drinking water guideline value for 1,1,2-TCA.

The WHO (2000, 2000b) have not evaluated or provided an inhalation guideline values for 1,1,2-TCA.

EU

No assessment of 1,1,2-TCA is available from the EU.

US

The USEPA (IRIS current) has derived the following for 1,1,2-TCA:

- Non-cancer effects: oral reference dose (*RfD*) of **0.004 mg/kg/day** on the basis of clinical serum chemistry in mice. No review of inhalation effects are provided.
- Cancer effects: *oral slope factor of 0.057 (mg/kg/day)⁻¹* has been derived using the linearised multistage procedure associated with hepatocellular carcinoma in mice. An *inhalation unit risk of 1.6x10⁻⁵ (µg/m³)⁻¹* has been derived using the oral exposure assessment.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with 1,1,2-TCA. The levels established (valid in 2006) are:

- Acute oral MRL = 0.3 mg/kg/day based on neurological effects
- Intermediate oral MRL = 0.04 mg/kg/day based on hepatic effects

The California Air Resources Board (CARB and OEHHA) has adopted the USEPA (IRIS) inhalation unit risk for the assessment of inhalation exposure to 1,1,2-TCA.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to 1,1,2-TCA, intakes from soil, water and food can be considered to be insignificant. Based on data available from urban air in Brisbane and Perth (Hawas, 2001, WA DEP 2000) 1,1,2-TCA is generally not detected in urban air and hence background intake can be considered to be negligible.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,1,2-TCA following review of the available information in general accordance with enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	RfD = 0.004 mg/kg/day (USEPA based on oral exposures study). 1,1,2-TCA is not a genotoxic carcinogen and hence use of a slope factor is not considered appropriate.
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	No threshold guidelines have been established based on inhalation exposure studies and hence the oral threshold guideline above has been adopted for the assessment of inhalation exposures. Occupational inhalation exposure (NOHSC, current to 2006): TWA: 10 ppm = 55 mg/m ³ STEL: NA
Background	Negligible

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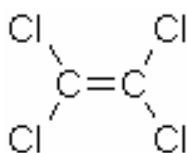
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GENERAL

Tetrachloroethene (also known as tetrachloroethylene, perchloroethylene, ethylene tetrachloride, per, perc, perchlor, 1,1,2,2-tetrachloroethylene and commonly abbreviated to **PCE**) is a synthetic chemical that is widely used for dry cleaning of fabrics and for metal-degreasing operations. It is also used as a building block for making other chemicals and is used in some consumer products. PCE manufacture in Australia ceased in 1991. Use in Australia has declined from 1995, consistent with declining use worldwide. PCE is primarily imported in its “pure” form with approximately 80 % used in the dry cleaning industry in Australia.

PCE is widespread in the environment and is found in trace amounts in water, aquatic organisms, air, foodstuffs, and human tissue. The highest environmental levels of PCE are found in the commercial dry-cleaning and metal-degreasing industries. The Australian Drinking Water Guidelines (2004) indicate that PCE has not been detected in Australian drinking water supplies.

PCE may degrade in the environment to more toxic compounds, including vinyl chloride. .

PROPERTIES

PCE is a volatile, colourless liquid. It is a non-flammable liquid at room temperature which evaporates easily into the air and has a sharp, sweet odour. PCE is practically insoluble in water but miscible with ethanol, ether and oils. Key properties are presented below (ATSDR 1997, USEPA 2004 and ORNL Database 2005):

CAS No	127-18-4
Chemical Formula	C ₂ Cl ₄
Molecular Weight	165.83
Vapour Pressure	18.5 mmHg at 25°C
Vapour Density	5.8
Density	1.62 g/ml at 20°C
Solubility	200 mg/L at 20°C
Air Diffusion Coefficient	0.072 cm ² /s
Water Diffusion Coefficient	8.2 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0184 atm.m ³ /mol = 0.754 at 25°C (unitless)
Koc	155 cm ³ /g
Log Kow	3.4
Odour Threshold	6.8 mg/m ³ (ATSDR) and 33.9 mg/m ³ (NOHSC) 180 mg/m ³ recognition (EU, 2005)
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0481

EXPOSURE

Exposure to PCE may be derived from environmental and occupational sources as well as from consumer products. Common background levels of PCE in the environment are generally several thousand times lower than levels found in some workplaces. Background levels are found in the air, water, and food. The most significant exposure pathway is via the air, particularly in the workplace. PCE gets into air by evaporation from industrial or dry cleaning operations and released from stores of chemical wastes. It is frequently found in surface water.

Common consumer products that may contain PCE include water repellents, silicone lubricants, fabric finishers, spot removers, adhesives, and wood cleaners. Although uncommon, small amounts of PCE have been found in food, especially food prepared near a dry cleaning facility. PCE has also been detected in the breast milk of mothers who have been exposed to the chemical. PCE is considered (NICNAS, 2001) to have a low potential for bioaccumulation.

If released into the environment the following can be noted with respect to PCE:

- Air: PCE is expected to remain in vapour phase. Removal is primarily through reaction with hydroxyl radicals, or chlorine atoms produced through photo-oxidation of PCE, which results in half-lives of 1 hour to 2 months.
- Soil and Water: PCE is expected to volatilise from surface soils and water. PCE has a low to medium mobility in soil and may leach slowly through soil into groundwater where it may persist for years. Depending on conditions reductive dehalogenation to vinyl chloride may occur. Under anaerobic conditions PCE and TCE can be intrinsically biodegraded to form DCE and vinyl chloride (below).

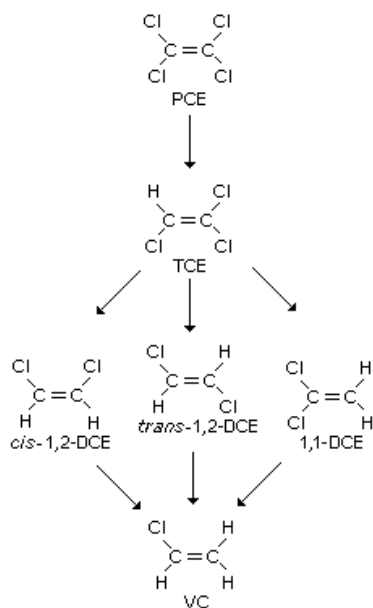


Figure 1. Pathway for anaerobic microbial degradation of chlorinated ethenes to form vinyl chloride (from: WHO, 1999)

NB: PCE=tetrachloroethene, TCE=trichloroethene, DCE=dichloroethene

HEALTH EFFECTS

General

There is no clinical disease which is unique to PCE toxicity. PCE is absorbed mainly through inhalation, causing both irritation and neurobehavioral effects. Skin burns, blistering and erythema can occur from severe direct contact with PCE. Some skin absorption can occur but does not appear to be of major significance. The amount of the chemical in the body increases with increasing exposure level and with an increase in physical exercise during exposure. It accumulates to a limited extent in the fatty tissues of man and of animals. Because of its affinity for fat, PCE is found in milk. PCE has also been shown to cross the placenta and distribute to the foetus.

PCE is eliminated slowly through the lungs. A small amount is metabolised to trichloroethanol and trichloroacetic acid. The concentrations of the compound in blood and breath can be used for estimating exposure levels in man.

At high concentrations, PCE causes central nervous system depression. Lower concentrations of PCE have been reported to damage the liver and the kidneys.

The following summary has been derived from ATSDR (1997).

Death	At high concentrations PCE is a potent anaesthetic agent and a cardiac sensitiser. Hence death resulting from excessive depression of the respiratory centre or the onset of fatal cardiac arrhythmia may occur. Deaths associated with PCE exposure (inhalation and ingestion) have been reported.
Respiratory Effects	Exposure to high concentrations of PCE has been associated with respiratory irritation.
Gastrointestinal Effects	Acute inhalation exposure to PCE has resulted in nausea and vomiting.
Hepatic Effects	The liver is a target organ in humans exposed to high concentrations of PCE in air. In animals, liver effects are characterised by hypertrophy, fatty degeneration and peroxisome proliferation. Hepatic lesions are also induced in experimental animals during inhalation exposure to PCE. The liver has not been shown to be a target organ in humans exposed via the oral route, however it is a target organ in animals exposed orally.
Renal Effects	Symptoms of renal dysfunction (including proteinuria and hematuria) have been associated with exposure to anaesthetic concentrations of PCE vapour. Weak (or no) effects have reported in people with chronic occupational exposures. Adverse renal effects have been observed in rodents exposed to PCE via inhalation and oral ingestion.
Dermal/Ocular Effects	Exposure to high doses of PCE through contact with the air or skin has resulted in burning or stinging in the eyes, transient eye irritation, acute burning and maculapapular rashes. Skin burns, blistering and erythema can occur from severe direct contact with PCE.
Body Weight Effects	Body weight loss has been reported in rats exposed to PCE in air and via oral ingestion.

Immunological Effects	No significant effects have been reported following inhalation exposures. Limited data supports immunotoxic effects on B cells/humoral immunity associated with oral exposures.
Neurological Effects	The nervous system is a major target organ in humans exposed to PCE via inhalation and ingestion. Anaesthetic and preanesthetic central nervous system effects (including mood changes, ataxia, faintness, dizziness, loss of motor coordination collapse, coma and seizures) have been reported from exposures over different periods of time. While acute symptoms seem to improve after cessation of exposure, chronic exposure has been associated with chronic encephalopathy (memory and concentration impairment) is persistent after cessation of exposure. Neurological effects and biochemical changes in the brain have been reported in animals exposed to PCE.
Reproductive Effects	Some adverse reproductive effects in occupationally exposed women have been reported which include increased risk of spontaneous abortion. Animal studies indicate reproductive effects associated with PCE exposure.
Developmental Effects	Limited animal studies indicate the potential for a slight increase in maternal and foetal toxicity following inhalation exposure to PCE. Animal studies associated with oral exposure to PCE indicate maternal toxicity, increased numbers of postnatal deaths and increased micro/anophthalmia. Acute studies indicate developmental neurotoxicity (with the LOAEL utilised by the ATSDR in the establishment of an acute oral MRL).
Genotoxic Effects	Assays of clastogenic effects in humans have shown inconsistent results in occupational human studies. No animal studies show genotoxic effects. From weight of evidence, PCE is considered to be non-genotoxic (WHO 2000, 2006). Lack of strong genotoxic effects is considered (by ATSDR) to be consistent with the metabolism of the compound. Review of PCE undertaken by Woodward-Clyde (1996) indicated that the weight of evidence indicates that PCE is non-genotoxic. However, genotoxicity is observed when PCE is stabilised with known genotoxicants such as compounds containing epoxide groups.
Cancer	<p>Some epidemiological studies indicate a possible association between chronic exposure to PCE and an increased cancer risk, however the evidence provided is considered to be inconclusive. This is mainly due to concurrent exposure to other petroleum solvents as well as PCE, confounding factors (smoking, alcohol, socio-economic status) and small numbers of cancers in the studies.</p> <p>An association between exposure to PCE (inhalation and ingestion) and an increased risk of cancer (mononuclear cell leukaemia and hepatic tumours) in animals has been suggested. Review of PCE by Woodward-Clyde (1996) indicates that PCE is a non-genotoxic animal carcinogen. Review of the possible mechanisms of tumour formation by PCE in animals suggests that the tumours observed may have little relevance for humans. Therefore a threshold type of exposure parameter would be relevant as a basis for human health risk assessment.</p>

TOXICITY CLASSIFICATION

PCE was classified as a "probable" human carcinogen (Category B2) by the USEPA for all routes of exposure based upon evidence from animal studies. This classification has been withdrawn pending further review (not finalised as of June 2004).

IARC has classified PCE in Group 2A (probably carcinogenic to humans) based in limited evidence in humans (epidemiological studies showed elevated risks for oesophageal cancer, non-Hodgkin's lymphoma and cervical cancer) and sufficient evidence in experimental animals (induce peroxisome proliferation in mouse liver and induced leukaemia in rats).

NICNAS has classified PCE as a Carcinogen Category 3, which is a substance regarded as a possible risk of irreversible effects.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC 2004) has followed the WHO Drinking Water Guidelines (1996) which established health based guidelines derived from a **TDI of 0.014 mg/kg/day**. The TDI was derived from both a 6 week mice study and 90 day rat oral drinking water study, both of which indicated a NOAEL of 14 mg/kg/day. An uncertainty factor of 1000 was applied to the NOAEL (100 for inter- and intraspecies variation and 10 for carcinogenic potential). On this basis, the TDI established by WHO can be used for the evaluation of oral exposures to PCE.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For PCE, the following have been established:

TWA: 50 ppm, equivalent to 340 mg/m³

STEL: 150 ppm, equivalent to 1020 mg/m³

WHO

Oral TDI used to derive drinking water guidelines (1996) as outlined above in the derivation of Australian Drinking Water Guidelines. The guideline has remained unchanged in the latest WHO guideline (WHO 2004).

Review of inhalation evaluations for PCE as presented by the WHO (2000, 2000b) indicates the following:

Reference	Inhalation Guideline Value	Averaging Time	Basis
WHO 2000	GV = 0.25 mg/m ³	24 hours	Non-carcinogenic LOAEL associated with kidney effects from long-term occupational study.
WHO 2000	GV = 8 mg/m ³	30 minutes	Non-carcinogenic odour annoyance level.
WHO 2000b	GV = 0.25 mg/m ³	Annual	Non-carcinogenic kidney effects in workers (as per WHO 2000) above.

There appears to be some inconsistency in air quality guideline values published by the WHO, particularly with respect to the relevant averaging time for the GV of 0.25 mg/m³. It should also be noted that the WHO (2000b) indicates that the guideline value is established based on non-carcinogenic end-points and that review of possible carcinogenic end points should be undertaken in the future.

Further review by WHO (2006) provided a guideline value of 0.2 mg/m³ based on the protection of neurotoxicity effects and is considered to be adequately protective of liver toxicity and human reproductive toxicity.

EU

The EU has released a Risk Assessment Report in 2005 that provides general information in PCE, exposures and the environment. The report does not provide an evaluation of risks or exposures to human health.

US

The USEPA have established an oral reference dose (RfD) of 0.01 mg/kg/day (available from IRIS, current) based on hepatotoxicity in mice and increased liver and kidney weights in rats over 13 weeks. An uncertainty factor of 1000 was used to derive the RfD. The USEPA provides no data relevant to non-carcinogenic inhalation or carcinogenicity. The slope factor previously provided by the USEPA (0.051 mg/kg/day)⁻¹) based on mouse liver tumour data has been withdrawn (1990).

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with PCE. The levels established (current) are:

- Acute inhalation MRL = 0.2ppm based on neurological effects in humans;
- Chronic inhalation MRL = 0.04ppm based on neurological effects in rats; and
- Acute oral MRL = 0.05 mg/kg/day based on developmental effects in mice.

The California Air Resources Board (CARB, current) has listed PCE as a toxic air contaminant and evaluated cancer and non cancer effects. Cancer effects for PCE have been evaluated on the basis of an inhalation unit risk of 5.9x10⁻⁶ (µg/m³)⁻¹ (equivalent to 0.021 (mg/kg/day)⁻¹, provided in 1991). Values

established to evaluate non cancer effects include and acute inhalation value of 20000 $\mu\text{g}/\text{m}^3$ (reviewed 1999) based on CNS effects and a chronic inhalation value of 35 $\mu\text{g}/\text{m}^3$ (reviewed in 2000) based on effects to the kidney, liver and gastrointestinal system.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI, GV or RfD in assessing potential exposures to site related chemicals. With respect to PCE, intakes from soil, water and food can be considered to be insignificant. Intakes from air have been calculated from industrial air concentrations in reported in Brisbane (Hawas O. et. al., 2001), with the average and maximum concentrations reported of 0.015 mg/m^3 and 0.085 mg/m^3 respectively (consistent with data from other cities, NICNAS 2001). This represents up to 34% intake from background air sources. On this basis, the oral TDI and inhalation GV identified should be reduced to account for approximately 34% background intake.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for PCE following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	Oral TDI = 0.014 $\text{mg}/\text{kg}/\text{day}$ (Australian Drinking Water Guidelines 2004 and WHO 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation GV = 0.2 mg/m^3 (WHO, 2006). Occupational inhalation exposure levels (NOHSC): TWA: 50 ppm, equivalent to 340 mg/m^3 STEL: 150 ppm, equivalent to 1020 mg/m^3
Background	34%

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GENERAL

Tetrachloroethane (TCA) is a chemical compound occurring in two isomers: 1,1,1,2- and 1,1,2,2-.

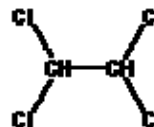
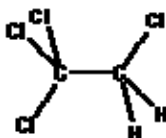
1,1,1,2-Tetrachloroethane (also known as asym-tetrachloroethane and abbreviated to **1,1,1,2-TCA**) is a synthetic chemical that does not occur naturally in the environment. 1,1,1,2-TCA is not produced on an industrial scale, however it is a common by-product of many chlorination reactions of C2 hydrocarbons including the production of 1,1,2,2-TCA. The primary sources of 1,1,1,2-TCA emissions are the industries that manufacture it or use it in production that include the chemical industry and wood stain and varnish manufacturers (NPI Database).

1,1,2,2-Tetrachloroethane (also known as acetylene tetrachloride; sym-tetrachloroethane; s-tetrachloroethane; tetrachloroethane, 1,1-dichloro-2,2-dichloroethane and commonly abbreviated to **1,1,2,2-TCA**) is a synthetic chemical not known to occur naturally. 1,1,2,2-TCA is widely used as an industrial solvent. It is also used as an intermediate in the production of trichloroethylene and tetrachloroethylene. Other minor uses of 1,1,2,2-TCA include the use as an insecticide (moth-proofing and as a fumigant). 1,1,2,2-TCA is synthesised by direct chlorination or oxychlorination of ethylene. 1,1,2,2-TCA can be a by-product generated during the production of vinyl chloride or ethylene dichloride.

PROPERTIES

At room temperature both isomers of TCA are non-flammable, colourless, low to moderate volatile liquids with a sweetish, suffocating chloroform-like odour. It is miscible with ethanol, methanol, ether, acetone, benzene, petroleum, carbon tetrachloride, chloroform, carbon disulfide, dimethyl formamide and oils. Key properties are presented below (ATSDR 1996, USEPA 2004 and ORNL Database 2006):

	<u>1,1,1,2-Tetrachloroethane</u>	<u>1,1,2,2-Tetrachloroethane</u>
CAS No.	603-20-6	79-34-5
Chemical Formula	C ₂ H ₂ Cl ₄	C ₂ H ₂ Cl ₄
Molecular Weight	167.85	167.85
Vapour Pressure	12 mmHg at 21°C	13.3 mmHg at 21°C
Vapour Density	5.32	5.32
Density	1.54 g/ml at 20°C	1.59 g/ml at 20°C
Solubility	1070 mg/L at 20°C	2870 mg/L at 20°C
Air Diffusion Coefficient	0.0423 cm ² /s	0.071 cm ² /s
Water Diffusion Coefficient	9.14 x 10 ⁻⁶ cm ² /s	7.9 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.0989 at 25°C (unitless)	0.000345 atm.m ³ /mol = 0.0141 at 25°C (unitless)
Koc	96.63 cm ³ /g	106.8 cm ³ /g
Log Kow	2.93	2.39
Odour Threshold	NA	21 to 35 mg/m ³
Dermal Absorption	0.01 (unitless)	0.01 (unitless)
Permeability Constant	0.0218	0.00897



EXPOSURE

Exposure of the general population to both isomers of TCA may occur primarily through inhalation, however exposure via oral or dermal routes may occur but are expected to be insignificant.

If released into the environment the following can be noted with respect to TCA (ATSDR, 1996):

- **Air:** Most of the TCA released to the environment enters the atmosphere where it is fairly stable in the lower atmosphere. The dominant process for removal of TCA is reaction with photochemically generated hydroxyl radicals with an estimated half-life of 53 days for 1,1,2,2-TCA and 550 days for 1,1,1,2-TCA. Removal may also occur through washout by precipitation, however most TCA is expected to re-enter the atmosphere via volatilisation. Atmospheric degradation of TCA is slow enough for it to be transported long distances with slow diffusion into the stratosphere expected to occur. TCA is not expected to contribute to the depletion of stratospheric ozone or to global warming.
- **Soil and Water:** Following releases to soil, some TCA is expected to volatilise, with the remainder leaching into the subsurface soil profile and possibly to groundwater. If released to surface water, part would volatilise with the remainder dissolving in water where it would undergo degradation through hydrolysis. In groundwater the major degradation processes involve anaerobic degradation and chemical hydrolysis.
- **Biodegradation:** TCA may undergo degradation through hydrolysis and/or anaerobic degradation. Chemical hydrolysis is very sensitive to pH and is much more rapid under basic or neutral conditions. Trichloroethylene is the major (if not only) product of chemical hydrolysis with half lives reported in neutral pHs from 29 to 102 days for 1,1,2,2-TCA. Biodegradation occurs through dehydrodehalogenation with the products of biodegradation including trichloroethylene, 1,2-dichloroethene and vinyl chloride.
- Since TCA is volatile and can be expected to be transformed to other compounds such as TCE, TCA would not be expected to accumulate in sediments. TCA has a low tendency to bioconcentrate in aquatic or marine organisms.

HEALTH EFFECTS

General

The following information is available from WHO (1998) and ATSDR (1996). There is no clinical disease which is unique to TCA toxicity. Owing to the significant decline in the use of this substance, the toxicological profile of TCA has not been well characterised, with the available data being confined primarily to early limited studies.

TCA is well absorbed from the gastrointestinal and respiratory tract in animals and humans. Dermal absorption of TCA occurs.

No studies are available on the distribution of TCA via inhalation, oral or dermal routes of exposure.

The metabolism of 1,1,1,2-TCA proceeds through both oxidative and reductive pathways resulting in CO₂ in exhaled air and trichloroethanol and trichloroacetic acid in urine as main metabolites. The data shows that in rats there is a difference in the metabolism of 1,1,1,2- and 1,1,2,2-TCA. The amount of trichloro compounds in the urine of rats exposed to 1,1,1,2-TCA was 20 times higher than the amount of trichloro compounds in the urine of rats similarly exposed to the 1,1,2,2-isomer (HCN, 2006).

Based on data on the metabolism of 1,1,2,2-TCA in mice (WHO, 1998) it is suggested that the principal pathway of degradation involves stagewise hydrolytic cleavage of the carbon-chlorine bonds and oxidation to dichloroacetaldehyde hydrate, dichloroacetic acid (the major metabolite), and eventually glyoxylic acid. The glyoxylic acid is then metabolized to oxalic acid, glycine, formic acid, and carbon dioxide. A small proportion of the parent compound is probably non-enzymatically dehydrochlorinated to trichloroethylene, which is further converted to trichloroacetic acid and trichloroethanol. In addition to the liver, metabolism may also occur in the epithelia of the respiratory tract and upper alimentary tract.

The metabolites of TCA are eliminated in the urine, faeces, skin and expired air.

Based on the results of studies in experimental animals, the acute toxicity of TCA is slight to moderate. The chemical may induce skin, eye, and mucosal irritation.

Limited long-term human data are not available, hence the evaluation of critical effects is based on animal studies.

The results of available studies on the non-neoplastic effects of TCA in experimental animals exposed by ingestion or inhalation indicate that the central nervous system and liver are the principal target organs.

Genotoxic Effects

The weight of evidence of available (WHO 1998 and Woodward-Clyde 1996) *in vitro* and *in vivo* assays suggests that 1,1,2,2-TCA is not genotoxic or that it is, at most, weakly genotoxic.

Similarly review of the available *in vitro* and *in vivo* study data (HCN, 2006) suggests that 1,1,1,2-TCA is not genotoxic.

Cancer

Human data on the carcinogenic potential of TCA are limited, hence data available from animal studies have been used by various agencies in reviewing carcinogenicity.

Long-term exposure to 1,1,2,2-TCA resulted in a significantly increased incidence of hepatocellular carcinomas in both male and female mice. However, no significant increases in tumours were observed in similarly exposed rats. The relevance of increased incidence of liver tumours in mice to humans in the absence of a carcinogenic response in other species or other mouse tissues and without clear evidence of genotoxicity is highly questionable.

The WHO (1998) has proposed “that the liver tumours in mice may be induced by mechanisms that may not be relevant to humans, for which humans are less susceptible, or for which there may be a threshold of exposure. In addition, it has been hypothesized that the carcinogenicity of 1,1,2,2-TCA may be associated with the formation of free radicals, lipid peroxidation, or hepatic damage (such as focal necrosis associated with intense cellular proliferation). Therefore, on the basis of data currently available, it is not possible to draw any firm conclusions with respect to the potential carcinogenicity of 1,1,2,2-TCA in humans”.

There is limited evidence from studies in rats and mice that 1,1,1,2-TCA is carcinogenic. Increased incidence of fibroadenomas in female rats and hepatocellular adenomas in mice were observed in some studies.

TOXICITY CLASSIFICATION

TCA (both isomers) has been classified as a "possible" human carcinogen (Category C) by the USEPA based on increase incidence of hepatocellular carcinomas in mice.

IARC (1999) has classified TCA (both isomers) in Group 3 (not classifiable as to its carcinogenicity to humans) based on inadequate evidence in humans and limited evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) or NICNAS have not classified the potential carcinogenicity of TCA.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 1996 and 2004) have not derived a drinking water guideline for 1,1,1,2- or 1,1,2,2-TCA.

Worksafe Australia (NOHSC) have established “Exposure Standards for Atmospheric Contaminants in the Occupational Environment”. For 1,1,2,2-TCA, the following have been established:

TWA: 1 ppm, equivalent to 6.9 mg/m³

STEL: NA

Potential exposure via skin absorption is noted.

No occupational exposure limit is available for 1,1,1,2-TCA.

WHO

Review of 1,1,2,2-TCA by the WHO was undertaken in 1998. Based on the limitations associated with the available studies a TDI cannot be derived with confidence for non-neoplastic effects or neoplastic effects. The toxicological end-point for which the dose-response relationship is best characterized is the increase in hepatocellular carcinomas observed in the long-term bioassay in mice, however as noted

above, the relevant to humans is questionable. A range of guidance values have been derived using multistage modelling associated with the doses associated with a 5% increase in tumour incidence ($TD_{0.05}$) and applying a safety margin. Guidance values derived are:

- Inhalation: 3.4 to 16 $\mu\text{g}/\text{m}^3$ (applying 5000 margin) or 0.34 to 1.6 $\mu\text{g}/\text{m}^3$ (applying 50000 margin) – derived from oral study. These values correspond to those considered by some agencies to represent "essentially negligible" risk (i.e. 10^{-5} to 10^{-6}).
- Ingestion: 1.2 to 5.6 $\mu\text{g}/\text{kg}/\text{day}$ (applying 5000 margin) or 0.12 to 0.56 $\mu\text{g}/\text{kg}/\text{day}$ (applying 50000 margin) These values correspond to those considered by some agencies to represent "essentially negligible" risk (i.e. 10^{-5} to 10^{-6}).

The WHO (Drinking Water Guideline 1996 and 2004) does not derive a guideline value for 1,1,2,2-TCA.

The WHO (2000b) have derivation of an *inhalation unit risk of $(0.6 \text{ to } 3) \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$* for 1,1,2,2-TCA on the basis of the guidance values derived in the WHO (1998) document (noted above). It is noted that this evaluation is considered conservative as there is suggestive, but incomplete evidence that 1,1,2,2-TCA may induce tumours through a threshold mechanism. In addition, this value has been derived on the basis of an oral exposure study.

The WHO (Drinking Water Guideline 1996 and 2004) does not derive a guideline value for 1,1,1,2-TCA. There are no other reviews of 1,1,1,2-TCA available from the WHO.

EU

No assessment of TCA is available from the EU.

US

The USEPA (IRIS current in 2006) has derived the following for 1,1,2,2-TCA:

- Non-cancer effects: no data available.
- Cancer effects: an *oral slope factor of $0.2 (\text{mg}/\text{kg}/\text{day})^{-1}$* based on a linear multistage model based on hepatocellular carcinomas; and an *inhalation unit risk of $5.8 \times 10^{-5} (\mu\text{g}/\text{m}^3)^{-1}$* using a linear multistage model based on oral data used to derive the oral slope factor.

The US EPA Provisional Peer Reviewed Toxicity Values for Superfund (PPRTV, 2004) used an RfD of 0.06 $\text{mg}/\text{kg}/\text{day}$ for 1,1,2,2-TCA.

The USEPA (IRIS current in 2006) has derived the following for 1,1,1,2-TCA:

- Non-cancer *oral reference dose (RfD) of $0.03 \text{ mg}/\text{kg}/\text{day}$* based on a LOAEL of 89.3 $\text{mg}/\text{kg}/\text{day}$ from a chronic oral study in rats effects and an uncertainty factor of 3000. No inhalation non-cancer evaluation provided.

- Cancer effects: an *oral slope factor of 0.026 (mg/kg/day)⁻¹* based on a linear multistage model based on hepatocellular adenomas or carcinomas in mice; and an *inhalation unit risk of 7.4x10⁻⁶ (µg/m³)⁻¹* using a linear multistage model based on oral data used to derive the oral slope factor

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with 1,1,2,2-TCA. The levels established (valid in 2006) are:

- Intermediate inhalation MRL = 0.4 ppm (2.79 mg/m³) based on hepatic effects in rats
- Intermediate oral MRL = 0.6 mg/kg/day based on reduced body weight gain in rats
- Chronic oral MRL = 0.04 mg/kg/day based on respiratory effects in rats.

No MRLs are available for 1,1,1,2-TCA.

The California Air Resources Board (CARB and OEHHA) has adopted the USEPA (IRIS) inhalation unit risk value for the assessment of inhalation exposures to 1,1,2,2-TCA. No evaluation is provided for 1,1,1,2-TCA.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Review of available data with respect to TCA (as above) indicates that there is limited evidence for carcinogenicity for both isomers, with both considered non-genotoxic. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to TCA on the basis of a threshold approach.

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to TCA, intakes from soil, water and food can be considered to be insignificant. Based on data available from urban air in Brisbane and Perth (Hawas, 2001, WA DEP 2000) TCA is generally not detected or rarely detected. Hence background intake of TCA can be considered to be negligible.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for TCA following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

	1,1,1,2-Tetrachloroethane	1,1,2,2-Tetrachloroethane
Oral	RfD = 0.03 mg/kg/day (USEPA, current)	MRL = 0.04 mg/kg/day (ATSDR, 1996)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.	
Inhalation	No inhalation levels available, adopt oral value for the assessment of inhalation exposures. No occupational exposure limits available, adopt levels established for 1,1,2,2-TCA.	No threshold inhalation value available for 1,1,2,2-TCA, hence the available inhalation unit risk of $3 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$ has been adopted (conservative value from range presented by WHO 2000b and 1998, note value derived from oral study). Occupational inhalation exposure (NOHSC): TLV: 1ppm = 6.9 mg/m ³ STEL: NA
Background	Negligible	Negligible

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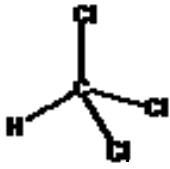
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GENERAL



Chloroform (also known as trichloromethane, methenyl chloride, methane trichloride, methyl trichloride and formyl trichloride) is both a synthetic and naturally occurring compound, with anthropogenic sources responsible for most of the chloroform in the environment. Chloroform is mainly used in the production of other materials, principally fluorocarbons, used in the synthesis of tetrafluoroethylene and polytetrafluoroethylene, and as a refrigerant and propellant. Chloroform is also widely employed as an organic solvent in industry and in analytical laboratories. It has also been used as an ingredient of pharmaceuticals, drugs, cosmetics, grain fumigants, dyes and pesticides.

In the past, chloroform has been extensively used as a surgical anaesthetic, but this use was discontinued because exposure to narcotic concentrations resulted in adverse side effects. The US Food and Drug Administration has banned the use of chloroform as an ingredient in human drug and cosmetic products in 1976.

PROPERTIES

It is a colourless liquid with a pleasant, non-irritating odour and a slightly sweet taste. It is only slightly soluble in water, but is miscible with alcohol, benzene, ether, petroleum ether, carbon tetrachloride, carbon disulfide, and oils. Decomposition may produce phosgene, carbon dioxide and hydrogen chloride. Key properties are presented below (ATSDR 1997, USEPA 2004 and ORNL Database 2006):

CAS No	67-66-3
Chemical Formula	CHCl ₃
Molecular Weight	119.38
Vapour Pressure	160 mmHg at 20°C
Vapour Density	4.1
Density	1.48 g/ml at 25°C
Solubility	7920 mg/L at 25°C
Air Diffusion Coefficient	0.104 cm ² /s
Water Diffusion Coefficient	1 x 10 ⁻⁵ cm ² /s
Henry's Law Coefficient	0.00367 atm.m ³ /mol = 0.15 at 25°C (unitless)
Koc	39.8 cm ³ /g
Log Kow	1.97
Odour Threshold	85 ppm (421 mg/m ³)
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00892

EXPOSURE

Human exposure to chloroform can occur orally, dermally, or by inhalation. Chloroform is the principal trihalomethane generated as by-products during the chlorination of drinking water. The primary sources of chloroform in the environment are chlorinated drinking water and wastewater, pulp and paper mills, and chemical and pharmaceutical manufacturing plants. The general population is exposed to chloroform mainly in food, drinking-water and indoor air. Most of the chloroform released to the environment

eventually enters the atmosphere, while much smaller amounts enter groundwater as a result of filtration through the soil. NHMRC indicate that concentrations of total trihalomethanes (including chloroform) in major Australian reticulates supplies are range up to 0.6 mg/L (NHMRC, 2004).

If released into the environment the following can be noted with respect to chloroform (WHO 1994):

- **Air:** Nearly all chloroform tetrachloride released to the environment will ultimately be present in the atmosphere due to its volatility. Chloroform has a residence time in the atmosphere of several months. In the atmosphere, chloroform may be transported long distances before degrading via indirection photochemicals reactions with free radicals such as hydroxyl (which form low levels of phosgene and hydrogen chloride).
- **Soil and Water:** Following releases to soil, most chloroform is expected to evaporate rapidly due to its high volatility and low soil adsorption. Most of the remaining chloroform will travel through the soil because of its low adsorption onto soils with leaching of chloroform to groundwater considered to be a significant pathway. Because of its volatility, evaporation is considered to be the main process for the removal of chloroform from aquatic systems. Chloroform is not expected to adsorb significantly to sediment or suspended organic matter in surface water.
- **Biodegradation:** Hydrolysis or direct photolysis are not considered to be significant degradation processes in water for chloroform. Chloroform is generally considered persistent in water and soils with a low potential for degradation. Under correct condition, chloroform may undergo anaerobic biodegradation. Concentrations of chloroform in soil or water above a certain threshold levels results in toxic conditions which inhibits bacteria, methane-fermenting bacteria under anaerobic conditions.
- Chloroform does not bioconcentrate in higher aquatic organisms

HEALTH EFFECTS

General

The following information is available from WHO (1999) and ATSDR (2003). There is no clinical disease which is unique to chloroform toxicity.

Chloroform is rapidly absorbed through the lungs and the gastrointestinal tract, and to some extent through the skin. In humans, the respiratory absorption of chloroform ranges from 49 to 77% and absorption from the gastrointestinal tract approximates 100%, with peak blood levels being reached within 1 hour.

Following its absorption, chloroform is distributed to all organs. The distribution of chloroform in the body does not differ qualitatively between the various routes of exposure. A number of studies have shown that chloroform to distribute to fat tissue. It is lipid soluble, readily passes through cell membranes, reaching relatively high concentrations in nervous tissue. Chloroform concentrations in tissues are dose-related and occur in the following order: adipose > brain > liver > kidney > blood. Chloroform passes

through the placenta and has been detected in fresh cow's milk and foetal blood at levels equal to or greater than those in maternal blood.

Chloroform is metabolised by oxidative dehydrochlorination of its carbon-hydrogen bond to form phosgene (CCl₂O). The reaction is P450-mediated and occurs in both the liver and the kidney. The major end product of chloroform metabolism is carbon dioxide (CO₂), most of which is eliminated via the lungs, but some is incorporated into endogenous metabolites and may be excreted as bicarbonate, urea, methionine and other amino acids, inorganic chloride ion, and carbon monoxide. Elimination of chloroform is not affected by the route of exposure. About 60 - 70% is eliminated unchanged in expired air; 30 - 40% is metabolised and excreted in urine and faeces. The extent of metabolism is dose-dependent.

Target organs for chloroform toxicity are the liver, kidneys, and central nervous system. The most universally observed toxic effect of chloroform is damage to the liver. Liver effects (hepatomegaly, fatty liver, and hepatitis) were observed in individuals occupationally exposed to chloroform. Several subchronic and chronic studies by the oral or inhalation routes of exposure documented hepatotoxic effects in rats, mice, and dogs. Renal effects have been reported in rats and mice following oral and inhalation exposures, but evidence for chloroform-induced renal toxicity in humans is sparse.

Chloroform is a central nervous system depressant, inducing narcosis and anaesthesia at high concentrations. Lower concentrations may cause irritability, lassitude, depression, gastrointestinal symptoms, and frequent and burning urination.

Genotoxicity

The weight of the available evidence (WHO 1994 and Woodward-Clyde 1996) indicates that chloroform has little, if any, capability to induce gene mutation, chromosomal damage and DNA repair. However, there is some evidence of low-level binding to DNA. Chloroform does not appear capable of inducing unscheduled DNA synthesis *in vivo*. Review of chloroform by USEPA (2001) indicates that chloroform is not a mutagen and is not likely to cause cancer through a genotoxic mode of action

Carcinogenicity

Human data on the carcinogenic potential of chloroform are limited and there have been no conclusive associated between chloroform exposure and cancer in humans. In experiments with mice and rats, chloroform induced liver and kidney tumours. The carcinogenic effects of chloroform on the mouse liver appear to be closely related to cytotoxic and cell replicative effects. Liver tumours in rat and mice studies have only occurred where signs of hepatotoxicity have been seen. In the rat and mice studies, the development of renal tumours in males is a consequence of nephrototoxicity of chloroform.

The pattern of development of tumours following chloroform treatment in animals is consistent with a tumour promoting mechanism rather than a genotoxic one. On the basis of available evidence, a dose threshold for the development of liver tumours is considered appropriate. It was considered plausible by the WHO (1996) that kidney tumours in rats may be associated with a threshold mechanism, however there are some limitations of the database.

Review of chloroform by the USEPA (2001) indicates that it is considered likely to be carcinogenic to humans by all routes of exposure under high-dose conditions that lead to cytotoxicity and regenerative hyperplasia. Chloroform is not likely to be carcinogenic to humans by any routes of exposure at doses that do not cause cytotoxicity and cell regeneration. Hence the USEPA has concluded that the threshold effects value established (RfD) is also protective against increased risk of cancer.

TOXICITY CLASSIFICATION

Chloroform has been classified as a "probable" human carcinogen (Category B2) by the USEPA based on carcinogenicity in animals. Under the Proposed Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1996; U.S. EPA, 1999), chloroform is *likely to be carcinogenic to humans by all routes of exposure* under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissues (U.S. EPA, 1998a,b). Chloroform is *not likely to be carcinogenic to humans by any route of exposure* under exposure conditions that do not cause cytotoxicity and cell regeneration.

IARC (1999) has classified chloroform in Group 2B (possibly carcinogenic to humans) based on inadequate evidence in humans and sufficient evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) as Category 3 carcinogen (possible human carcinogen). NICNAS has classified not classified chloroform

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have derived a drinking water guideline for total trihalomethanes, which included chloroform (as well as bromodichloromethane, dibromochloromethane and bromoform) of 0.25 mg/L as a total or individually using a ***TDI of 0.07 mg/kg/day*** derived from a no effect level based on a 90-day gavage study on rats and the application of 100 safety factor.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For chloroform, the following have been established (current, as amended 2001):

TWA: 2 ppm, equivalent to 10 mg/m³

STEL: none established

WHO

Review of chloroform by the WHO in 1994 has derived a number of tolerable daily intake (TDI) values for oral exposure. The values derived are:

- TDI = 0.015 mg/kg/day based on non-neoplastic effects (hepatotoxicity) in a 7.5 year study on dogs (lowest identified effects level of 15 mg/kg), 1000 uncertainty factor.
- TDI = 0.01 mg/kg/day for neoplastic effects (liver tumours) based on a 3 week study in mice (NOAEL of 10 mg/kg), 1000 uncertainty factor.
- Based on induction of renal tumours in male rats a total daily intake associated with a 10^{-5} excess cancer risk (linearised multistage model) is 0.0082 mg/kg/day.

The WHO (Drinking Water Guideline 1996 and 2004) provide a guideline value for chloroform of 0.2 mg/L based on a **TDI of 0.013 mg/kg/day** derived from a 7.5 year study on dogs (same study and derivation as noted above for non-neoplastic effects with the addition of a 5/7 conversion). It is noted that the guideline derived is approximately equal to that which would be derived using a linearised multistage model for renal tumours and a lifetime excess cancer risk of 10^{-5} .

The WHO have published a **TDI of 0.015 mg/kg/day (WHO 2000b)** based on hepatotoxicity in dogs (derived in 1994 from oral studies) and a **TC = 0.14 mg/m³ (WHO, 2004)**. WHO (2000b) have also published an inhalation unit risk of **$4.2 \times 10^{-7} (\mu\text{g}/\text{m}^3)^{-1}$** based on kidney tumours in rats (derived in 1994 from oral studies). No more relevant reviews of inhalation toxicity are available from the WHO.

EU

No assessment of chloroform is available from the EU.

US

The USEPA (IRIS current) has derived an oral reference dose (**RfD**) of **0.01 mg/kg/day** for the assessment of non-carcinogenic effects of chloroform. The RfD is based on liver effects in dogs. Evaluation of carcinogenicity of chloroform indicated that the derived RfD was considered to be protective of potential cancer effects. Hence no slope factor was derived for chloroform.

The USEPA (IRIS current) has derived an inhalation unit risk value for chloroform. The value derived is **$2.3 \times 10^{-5} (\mu\text{g}/\text{m}^3)^{-1}$** . It is noted that this value was not reviewed as part of the 2001 review for oral data and is currently being reviewed by the USEPA.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with chloroform. The levels established (current) are:

- Acute inhalation MRL = 0.1 ppm (0.496 mg/m³) based on hepatic effects in mice (inhalation study)
- Intermediate inhalation MRL = 0.05 ppm (0.248 mg/m³) based on toxic hepatitis in workers (inhalation study)
- Chronic inhalation MRL = 0.02 ppm (0.099 mg/m³) based on hepatic effects in workers (inhalation study)
- Acute oral MRL = 0.3 mg/kg/day based on hepatic effects in mice (oral study)

- Intermediate oral MRL = 0.1 mg/kg/day based on liver effects in dogs (oral study)
- Chronic oral MRL = 0.01 mg/kg/day based on liver effects in dogs (oral study)

The California Air Resources Board (CARB and OEHHA) has established the following with respect to chloroform:

- Inhalation unit risk of $5.3 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$.
- Chronic Inhalation reference Exposure Level (**REL**) = $0.3 \text{ mg}/\text{m}^3$ based on liver toxicity, kidney toxicity and developmental toxicity.
- Acute inhalation **REL** = $0.15 \text{ mg}/\text{m}^3$ (7 hour average) based on histological changes in nasal epithelium.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to chloroform the average intake from food, water and air has been estimated (WHO 2004) to be between 0.6 to 10 $\mu\text{g}/\text{kg}/\text{day}$. Data available from Australia indicate a similar range of potential intakes from water and air. Given the available TDI levels, it is considered relevant to assume a 50% intake from background. On this basis, the suggested threshold values should be adjusted to account for background intakes.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for chloroform following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	TDI = 0.013 mg/kg/day (WHO 2004)*
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	TC = 0.14 mg/m ³ (annual average, WHO 2004) Inhalation Unit Risk = $4.2 \times 10^{-7} (\mu\text{g}/\text{m}^3)^{-1}$ (WHO, 2000), with an equivalent inhalation slope factor of $0.0015 (\text{mg}/\text{kg}/\text{day})^{-1}$. Occupational inhalation exposure (NOHSC, current): TWA: 2ppm = 10 mg/m ³ STEL: NA
Background	50%

* Oral TDI value adopted from the WHO Drinking Water Guidelines (2004, also reviewed in CICAD 58, 2004) which provide a more recent evaluation of chloroform than presented in the Australian Drinking Water Guidelines (1996). The adoption of the new WHO value provides a more conservative evaluation.

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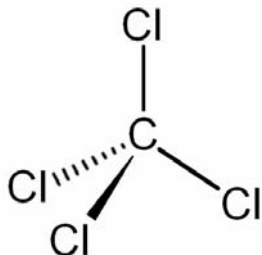
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GENERAL

Carbon Tetrachloride (also known as carbona, carbon chloride, tetrachloromethane, carbon tet, methane tetrachloride, perchloromethane, tetrachlorocarbon) is predominantly a man-made compound, however it has been detected in volcanic emission gasses. It has also been suggested that carbon tetrachloride can be formed in the troposphere by solar induced photochemical reactions of chlorinated alkenes (WHO, 1999). Production of carbon tetrachloride began in about 1907 in the US. Since 1990 the production of carbon tetrachloride has dropped due to the Montreal protocol which established a phase-out by 1996 of the production of carbon tetrachloride and chlorofluorocarbons (CFCs) by major manufacturing countries. Most of the carbon tetrachloride produced is used in the production of CFCs, which were primarily used as refrigerants, propellants, foam-blowing agents and solvents and in the production of other chlorinated hydrocarbons. Carbon tetrachloride has also been used as a grain fumigant, pesticide, solvent for oils and fats, metal degreaser, fire extinguisher and flame retardant, and in the production of paint, ink, plastics, semi-conductors and petrol additives. It was previously also widely used as a cleaning agent. All these uses have tended to be phased-out as production has dropped.

PROPERTIES

Carbon tetrachloride is a clear, colourless, volatile liquid with a characteristic, sweet odour. It is miscible with most aliphatic solvents and is itself a solvent. The solubility in water is low. Carbon tetrachloride is non-flammable and is stable in the presence of air and light. Decomposition may produce phosgene, carbon dioxide and hydrochloric acid. Key properties are presented below (ATSDR 1994, USEPA 2004 and ORNL Database 2006):

CAS No	56-23-5
Chemical Formula	CCl ₄
Molecular Weight	153.8
Vapour Pressure	115 mmHg at 20°C
Vapour Density	5.32
Density	1.59 g/ml at 25°C
Solubility	800 mg/L at 25°C
Air Diffusion Coefficient	0.078 cm ² /s
Water Diffusion Coefficient	8.8 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.03 atm.m ³ /mol = 1.2 at 25°C (unitless)
Koc	48.64 cm ³ /g
Log Kow	2.83
Odour Threshold	10 - 71,000 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0224 cm/hr

EXPOSURE

Exposure of the general population to carbon tetrachloride may be by inhalation, oral or dermal routes. Inhalation is expected to be the major route of exposure, particularly in occupational environment, but also in the general population. Dermal contact has not been shown to be a significant route of exposure to carbon tetrachloride (ATSDR, 2003). NHMRC indicate that concentrations of carbon tetrachloride in major Australian reticulates supplies are significantly less than 0.001 mg/L (NHMRC, 2004).

If released into the environment the following can be noted with respect to carbon tetrachloride (WHO 1999):

- **Air:** Nearly all carbon tetrachloride released to the environment will ultimately be present in the atmosphere, due to its volatility. Since the atmospheric residence time of carbon tetrachloride is long, it is widely distributed. Estimates of atmospheric lifetime are variable, but 45-50 years is accepted as the most reasonable value. Carbon tetrachloride contributes both to ozone depletion and to global warming.
- **Soil and Water:** Following releases to soil, most carbon tetrachloride is expected to evaporate rapidly due to its high vapour pressure. A small fraction of carbon tetrachloride may adsorb to organic matter. Carbon tetrachloride is expected to be moderately mobile in most soils, depending on organic carbon content, and leaching to groundwater may occur. Carbon tetrachloride introduced into water resources is transported by movement of surface water and groundwater. Because of its volatility, evaporation is considered to be the main process for the removal of carbon tetrachloride from aquatic systems. The amount of carbon tetrachloride dissolved in the oceans is reported to be less than 1-3% of that in the atmosphere.
- **Biodegradation:** Carbon tetrachloride is very stable in the troposphere primarily because carbon tetrachloride, in contrast to most other volatile halocarbons, has low reactivity towards hydroxyl radicals. The principal degradation process for carbon tetrachloride occurs in the stratosphere, where it is dissociated by short wave length (190- 220 nm) UV radiation to form the trichloromethyl radical and chlorine atoms. Simmonds et al. (1983) estimated a half-life of 18-80 years for this photo dissociation process. Carbon tetrachloride dissolved in water does not photodegrade or oxidize in any measurable amounts with the rate of hydrolysis calculated with a half-life of 7000 years (concentration of 1 ppm). Carbon tetrachloride has been shown to be resistant to aerobic biodegradation, however biodegradation may occur within 16 days under anaerobic conditions. Carbon tetrachloride may undergo reductive dechlorination to form chloroform and other products in the presence of free sulphide and ferrous ions.
- Carbon tetrachloride has a low tendency to bioconcentrate in aquatic or marine organisms. Most animals readily metabolise and excrete carbon tetrachloride following exposure and hence biomagnification is not expected.

HEALTH EFFECTS

General

The following information is available from WHO (1999) and ATSDR (2003). There is no clinical disease which is unique to carbon tetrachloride toxicity.

Carbon tetrachloride is well absorbed from the gastrointestinal and respiratory tract in animals and humans. Dermal absorption of liquid carbon tetrachloride is possible, but dermal absorption of the vapour is slow.

Carbon tetrachloride is distributed throughout the whole body, with highest concentrations in liver, brain, kidney, muscle, fat and blood. The parent compound is eliminated primarily in exhaled air, while minimal amounts are excreted in the faeces and urine.

Carbon tetrachloride has depressant effects on the central nervous system particularly following high levels of exposure. It can also produce irritation effects on the gastrointestinal tract and skin. Most other toxic effects associated with exposure to carbon tetrachloride are associated with its metabolism by mixed function cytochrome P-450 oxygenases.

The liver and kidney are target organs for carbon tetrachloride toxicity via oral and inhalation exposures. The severity of the effects on the liver depends on a number of factors such as species susceptibility, route and mode of exposure, diet or co-exposure to other compounds, in particular ethanol. Furthermore, it appears that pre-treatment with various compounds, such as phenobarbital and vitamin A, enhances hepatotoxicity, while other compounds, such as vitamin E, reduce the hepatotoxic action of carbon tetrachloride.

In humans, acute symptoms after carbon tetrachloride exposure are independent of the route of intake and are characterized by gastrointestinal and neurological symptoms, such as nausea, vomiting, headache, dizziness, dyspnoea and death. Liver damage appears after 24 h or more. Kidney damage is evident often only 2 to 3 weeks following the poisoning.

Epidemiological studies have not established an association between carbon tetrachloride exposure and increased risk of mortality, neoplasia or liver disease. Some studies have suggested an association with increased risk of non-Hodgkin's lymphoma and, in one study, with mortality and liver cirrhosis. However, not all of these studies pinpointed specific exposure to carbon tetrachloride, and the statistical associations were not strong.

Genotoxicity

It was concluded that carbon tetrachloride can induce embryotoxic and embryolethal effects, but only at doses that are maternally toxic, as observed in inhalation studies in rats and mice. Carbon tetrachloride is not teratogenic (WHO 1999).

Many genotoxicity assays have been conducted with carbon tetrachloride. On the basis of available data, carbon tetrachloride can be considered (WHO 1999) as a non-genotoxic compound. Review of genotoxicity associated with carbon tetrachloride by Woodward-Clyde (1996) supported this outcome.

Carcinogenicity

Human data on the carcinogenic potential of carbon tetrachloride are limited and there have been no conclusive associated between carbon tetrachloride exposure and cancer in humans. In experiments with mice and rats, carbon tetrachloride proved to be capable of inducing hepatomas and hepatocellular carcinomas. The doses inducing hepatic tumours were higher than those inducing cell toxicity. It is considered likely that the carcinogenicity of carbon tetrachloride is secondary to its hepatotoxic effects (WHO 1999) and may be related to its metabolism (ATSDR 2003).

The available data (WHO 1999 and review by Woodward-Clyde 1996) indicate that hepatic tumours are induced by a non-genotoxic mechanism, and it therefore seems acceptable to develop a tolerable daily intake (TDI) and a tolerable daily concentration in air (TC) for carbon tetrachloride.

TOXICITY CLASSIFICATION

Carbon tetrachloride has been classified as a "probable" human carcinogen (Category B2) by the USEPA based on carcinogenicity in rats, mice and hamsters.

IARC (1999) has classified carbon tetrachloride in Group 2B (possibly carcinogenic to humans) based on inadequate evidence in humans and sufficient evidence in experimental animals for carcinogenicity.

The National Occupational Health and Safety Commission (NOHSC) as Category 2 carcinogen (probable human carcinogen). NICNAS has classified not classified carbon tetrachloride.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have derived a drinking water guideline of 0.003 mg/L for carbon tetrachloride using a ***TDI of 0.00086 mg/kg/day*** derived from a no effect level based on a 90-day gavage study on mice and the application of 1000 safety factor and a 5/7 study duration adjustment factor.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For carbon tetrachloride, the following have been established (current, potential exposure via skin absorption is noted):

TWA: 0.1 ppm, equivalent to 0.63 mg/m³

STEL: none established

WHO

Review of carbon tetrachloride by the WHO in 1999 has derived a number of tolerable daily intake (TDI) values for oral exposure and tolerable concentrations (TC) for inhalation exposure. The values derived are:

- TDI = 0.00142 mg/kg/day based on a 12 week oral rat study (NOAEL of 1 mg/kg), 500 uncertainty factor and a 5/7 conversion.
- TDI = 0.00172 mg/kg/day based on a 90-day oral study on mice (NOAEL of 1.2 mg/kg), 500 uncertainty factor and 5/7 conversion.
- TC = 0.0061 mg/m³ based on 90-day inhalation study on rats (NOAEL 6.1 mg/m³) and 100 uncertainty factor.
- TC = 0.0067 mg/m³ based on 6-month inhalation study on rats (NOAEL 32 mg/m³), 1000 uncertainty factor and 5/7 conversion.
- TC = 0.0114 mg/m³ based on a 2-year inhalation study on rats (LOAEL 32 mg/m³), 500 safety factor and 5/7 conversion.

The WHO (Drinking Water Guideline 1996) provide a guideline value for carbon tetrachloride of 0.002 mg/L based on a **TDI of 0.00071 mg/kg/day** derived from a 12-week oral study on rats. The WHO revision to the Drinking Water Guideline (2004) derives a guideline of 0.004 mg/L based on a **TDI of 0.00142 mg/kg/day** derived from a 12-week oral study in rats (as per WHO 1999). It is noted that the guideline derived (1996 and 2004) is lower than values calculated using linear extrapolation and a lifetime excess cancer risk of 10⁻⁴ to 10⁻⁶.

The WHO (2000b) have published a **TC of 0.0061 mg/m³** based on 90-day inhalation study on rats, the lower TC value derived by WHO (1999, noted above) based on an annual average.

EU

No assessment of carbon tetrachloride is available from the EU.

US

The USEPA (IRIS current) has derived an **oral slope factor of 0.13 (mg/kg/day)⁻¹** for carbon tetrachloride based on a linear multistage model based on hepatocellular carcinomas and hepatomas; and an **inhalation unit risk of 1.5x10⁻⁵ (µg/m³)⁻¹** using a linear multistage model based on oral data used to derive the oral slope factor. The USEPA has also derived an oral reference dose (**RfD**) of **0.0007 mg/kg/day** for the assessment of non-carcinogenic effects. The RfD is based on liver lesions in a sub-chronic rat study.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with carbon tetrachloride. The levels established (valid in 2006) are:

- Intermediate inhalation MRL = 0.03 ppm (0.19 mg/m³) based on hepatic effects in animals (inhalation study).
- Chronic inhalation MRL = 0.03 ppm (0.19 mg/m³) based on hepatic effects in rats (inhalation study).
- Acute oral MRL = 0.05 mg/kg/day based on hepatic effects in rats (oral study).
- Intermediate oral MRL = 0.02 mg/kg/day based on hepatic effects in rats (oral study).

The California Air Resources Board (CARB and OEHHA) has established the following with respect to carbon tetrachloride:

- Inhalation unit risk of $4.2 \times 10^{-5} (\mu\text{g}/\text{m}^3)^{-1}$.
- Chronic Inhalation reference Exposure Level (**REL**) = **0.04 mg/m³** based on hepatic effects in guinea pigs.
- Acute inhalation REL = 1.9 mg/m³ (1 hour average).

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Intake of carbon tetrachloride from soil, water and food can be considered to be insignificant. Intakes from air can be calculated from urban air concentrations from a light industrial area in Brisbane (Hawas, 2001) which indicate a background concentration of 0.0025 mg/m³ (average) to 0.004 mg/m³ (max) which is approximately 40 to 65% of the tolerable concentration in air (equivalent to an ADI) as adopted from the WHO (2000b). On the basis of maximum concentrations of carbon tetrachloride in air from this study, background intake can be assumed to be up to 65% of the TC (WHO 2000b). On this basis, the suggested threshold values should be adjusted to account for background intakes.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for carbon tetrachloride following review of the available information in general accordance with guidance from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	TDI = 0.00142 mg/kg/day (WHO 1999 and 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	TC = 0.0061 mg/m ³ (annual average, WHO 1999 and 2000b) Occupational inhalation exposure (NOHSC, current to 2006): TWA: 0.1 ppm = 0.63 mg/m ³ STEL: NA
Background	65 %

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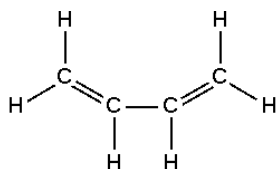
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1,3-Butadiene

General

The following is summarised from information from a range of available sources (HSDB, WHO (2001), OEHHA and Health Canada (2000)).



1,3-Butadiene (also known as butadiene, butadiene monomer, alpha-butadiene and vinyl ethylene and abbreviated to **BD**) is a product of incomplete combustion resulting from natural processes and human activity. It is also an industrial chemical used primarily in the production of polymers, including poly butadiene, styrene-butadiene rubbers (for use in latex) and lattices, and nitrile-butadiene rubbers. BD most commonly enters the environment from exhaust emissions from petrol- and diesel-powered vehicles, from non-transportation fuel combustion, from biomass combustion especially bushfires, and from industrial emissions. Minor releases occur in production processes, tobacco smoke, gasoline vapours, and vapours from the burning of plastics as well as rubber.

Exposure and Health Effects

The main routes of exposure to BD are via inhalation of contaminated indoor and ambient air (particularly in areas of heavy traffic or from tobacco products), particularly occupational exposures. Dermal exposures can also occur during consumer use of products containing BD. In comparison, other media, including food and drinking-water, contribute negligibly to exposure to BD.

While BD is not persistent, it is ubiquitous in the urban environment because of its widespread combustion sources. The highest atmospheric concentrations have been measured in air in cities and close to industrial sources

Since BD is released primarily to air, exposure and its fate in that medium is of primary importance. BD is not expected to persist in air, since it oxidizes rapidly with several oxidant species. The net atmospheric lifetime of BD is short, and there is generally limited potential for long-range transport of this compound. It is predicted from its physical/chemical properties that when BD is released into air, almost all of it will exist in the vapour phase in the atmosphere. Wet and dry deposition are not expected to be important as transfer processes. Evaporation from rain may be rapid, and the compound is returned to the atmosphere relatively quickly unless it is leached into the soil.

In water, BD is expected to volatilise readily. Volatilization of BD from soil and other surfaces is also expected to be significant. Butadiene's organic carbon/water partition coefficient indicates that it should not adsorb to soil particles to a great degree and would be considered moderately mobile. However, the rapid rate of volatilization and the potential for degradation in soil suggest that it is unlikely that butadiene will leach into groundwater.

The potential for BD to bioaccumulate in the terrestrial or aquatic environment is considered to be low.

Acute exposure to BD can cause eye, nose and throat irritation. Long-term exposures to BD may be associated with effects on the blood and bone marrow as well as the reproductive system. Few other adverse non-carcinogenic effects have been observed.

1,3-Butadiene

BD has been identified as a carcinogen to human via inhalation exposures. This has been based on the following findings:

- Increased lymphohematopoietic cancers in workers occupationally exposed via inhalation to BD based on epidemiologic studies (leukemias in polymer workers and non-Hodgkin's lymphoma in monomer workers);
- BD causes a variety of tumors in mice and rats by inhalation in various studies;
- Demonstration that BD is metabolized into genotoxic metabolites by experimental animals and humans.

Although the mode of action by which BD produces tumors is unknown, scientific evidence suggests that carcinogenic effects are mediated by genotoxic metabolites of BD.

Properties

BD is a colourless, flammable gas with a mild aromatic odour. It is soluble in ethanol, ether, acetone, benzene and organic solvents, and only slightly soluble in water. Key properties are presented below (HSDB, ATSDR (1992) and RAIS):

CAS No	106-99-0
Chemical Formula	C ₄ H ₆
Molecular Weight	54.09
Vapour Pressure	2110 mmHg at 25°C
Vapour Density	1.9
Density	0.6211 g/ml at 20°C
Solubility	735 mg/L at 20°C
Air Diffusion Coefficient	0.249 cm ² /s
Water Diffusion Coefficient	1.08 x 10 ⁻⁵ cm ² /s
Henry's Law Coefficient	0.0705 atm.m ³ /mol = 3.01 at 25°C (unitless)
Koc	43.79 cm ³ /g
Log Kow	1.99
Odour Threshold	1.0 – 1.6 ppm (recognition) 0.025 ppm (detection)
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0231 cm/hr
Conversion	1ppm = 2.21 mg/m ³ at 25°C

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of BD. Ambient levels of BD in air have been reported in a number of locations within Sydney, Newcastle and Wollongong (DEC, 2004) with the average concentration reported in the Sydney CBD (data from January 1996 to August 2001) of 0.4 ppbv (with lower values reported in outer suburbs and rural areas). The maximum 24-hour average concentration of BD reported was 1.4 ppbv. As environmental exposures are expected to be dominated by the presence of this

1,3-Butadiene

chemical in air, the potential for background concentrations in water is assumed to be negligible. Based on the average concentration in Sydney CBD of 0.4 ppbv = 0.9 $\mu\text{g}/\text{m}^3$ (at 25°C), which is approximately 5% of the available threshold chronic reference concentration, hence background intakes of 1 have been assumed to comprise up to 5% of the threshold intake levels.

Toxicity Values

Review of available data with respect to BD indicates that this chemical is considered to be carcinogenic and genotoxic via the inhalation exposure pathway. Hence the quantification of effects associated with exposure to BD needs to adequately address inhalation exposures on the basis of carcinogenicity.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for BD following review of the available information in general accordance with current Australian guidance (enHealth (2004) and NEPC (1999)), accounting for background intake where relevant.

The data presented with respect to the assessment of inhalation effects is relevant to both acute and chronic exposures (with relevant averaging periods identified).

Classification USEPA: Carcinogenic to humans via inhalation
IARC: Group 1 – Carcinogenic to humans (2007)

Toxicity Values:	
Oral	No data specifically available for oral exposures, hence assessment can be undertaken on the basis of data available for inhalation exposures.
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation unit risk = 0.00017 ($\mu\text{g}/\text{m}^3$) ⁻¹ (OEHHA) Chronic Reference Exposure Level (REL) = 20 $\mu\text{g}/\text{m}^3$ (OEHHA) (annual average) based on increased incidence of ovarian atrophy in mice (the same study identified by WHO (2001) for which the lower BMC can be derived). No acute air guideline currently available, however it is noted that a draft acute REL available from the TNRCC is 240 $\mu\text{g}/\text{m}^3$ (1-hour average) based on maternal toxicity. This is only a draft currently for review. Occupational inhalation exposure for BD (ASCC): TWA: 10 ppm = 22 mg/m^3 STEL: NA
Background	5%

1,3-Butadiene

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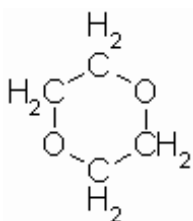
Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	10/12/2007
Previous Revisions:		

1,4-Dioxane

General

The following is summarised from information from a range of available sources (HSDB, ATSDR (2007) and OEHHA).



1,4-Dioxane (also known as **dioxane**, dioxyethylene ether, diethylene oxide, para-dioxane and tetrahydro-p-dioxane) is a cyclic ether used as a degreasing agent, a component of paint and varnish removers and as a wetting and dispersion agent in the textile industry. 1,4-Dioxane is also used as a solvent in chemical synthesis, stabiliser in chlorinated solvents, purification of drugs, preparation of tissue samples for histology and a working fluid for scintillation counter samples. 1,4-Dioxane may be released to the environment through various waste streams.

Exposure and Health Effects

The main routes of exposure to 1,4-dioxane are via inhalation of contaminated indoor and ambient air, particularly occupational exposures. Exposures may also occur through dermal contact with consumer products that contain 1,4-dioxane (including cosmetics, detergents and shampoos) and ingestion (and inhalation) of contaminated drinking water.

If released to the atmosphere, 1,4-dioxane will exist solely in the vapour phase where it will be degraded by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 35 hours. If released to soil, 1,4-dioxane is expected to have very high mobility with volatilisation expected to be an important fate process. 1,4-Dioxane is very slow to biodegrade and is considered recalcitrant in the environment. If released into water, 1,4-dioxane is not expected to adsorb to suspended solids and sediment based upon the Koc value. Volatilisation from water surfaces is expected to be an important fate process. The potential for bioconcentration in aquatic organisms is low.

1,4-dioxane is metabolized to β -hydroxyethoxyacetic acid (HEAA) by mixed-function oxidase enzymes; HEAA can be converted to 1,4-dioxane-2-one under acidic conditions. Both of these products are rapidly and extensively eliminated in the urine. Unchanged 1,4-dioxane can also be excreted in the urine and in exhaled air, but mainly after high-dose exposure. Studies have shown that the metabolism of 1,4-dioxane in rats is saturable at high doses. There is virtually no information regarding the toxicokinetics of 1,4-dioxane in humans following oral or dermal exposure. There is no indication that 1,4-dioxane or HEAA accumulates in the body.

Acute exposure to 1,4-dioxane can cause respiratory and eye irritation. Exposures to very high levels may cause severe kidney and liver effects. Little is known about the long term effects of exposure to low concentrations of 1,4-dioxane, however long-term exposures to high concentrations may be associated with effects on the liver, kidneys and circulatory system. These effects are the same regardless of the route of exposure.

1,4-Dioxane has been considered to be a possible human carcinogen based on data from studies on rats and mice (oral studies with high concentrations) that have shown liver and nasal tumours. However it is noted that a 2-year inhalation study on rats exposed to low concentrations of 1,4-dioxane provided no evidence of carcinogenicity or any other health effect.

1,4-Dioxane

The mechanism of carcinogenicity is not clear, however current data suggests that at dose levels that induce tumours, 1,4-dioxane may be acting through a non-genetic mode of action.

Properties

1,4-dioxane is a colourless liquid with a faint, pleasant odour. It is miscible with water, aromatic solvents and oils. Key properties are presented below (HSDB, ATSDR (2007) and RAIS):

CAS No	123-91-1
Chemical Formula	C ₄ H ₈ O ₂
Molecular Weight	88.11
Vapour Pressure	38.1 mmHg at 25°C
Vapour Density	3.0
Density	1.03 g/ml at 20°C
Solubility	1,000,000 mg/L at 20°C
Air Diffusion Coefficient	0.229 cm ² /s
Water Diffusion Coefficient	1.02 x 10 ⁻⁵ cm ² /s
Henry's Law Coefficient	4.8x10 ⁻⁶ atm.m ³ /mol = 0.000196 at 25°C (unitless)
Koc	1 cm ³ /g
Log Kow	-0.27
Odour Threshold	1.8 - 24 ppm
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.000356 cm/hr
Conversion	1ppm = 3.6 mg/m ³ at 25°C

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of 1,4-dioxane. No ambient air data is available from Australian studies that indicate the level of 1,4-dioxane in urban, industrial and rural areas. No data is available on drinking water quality. Background levels of 1,4-dioxane reported in urban areas in New Jersey ranged from 0.01 to 0.02 ppb, with an average outdoor air level reported across the US of 1.029 ppbv. Based on the average concentration in the US of 1.029 ppbv = 3.7 µg/m³ (at 25°C), which is <0.1% of the available threshold chronic reference concentration, background intakes have been assumed to be negligible.

Toxicity Values

Review of available data with respect to 1,4-dioxane indicates that this chemical is considered to be carcinogenic, however it is not currently considered to be genotoxic. Therefore it is considered appropriate that effects be evaluated on the basis of an appropriate threshold (where available) that addresses both carcinogenic and non-carcinogenic end-points.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,4-dioxane following review of the available information in general accordance with current Australian guidance (enHealth (2004) and NEPC (1999), accounting for background intake where relevant.

1,4-Dioxane

The data presented with respect to the assessment of inhalation effects is relevant to both acute and chronic exposures (with relevant averaging periods identified).

Classification USEPA: B2 – Possible human carcinogen
IARC: Group 2B – Possibly carcinogenic to humans

Toxicity Values:	
Oral	TDI = 0.016 mg/kg/day (WHO, 2004 and rolling revisions) based on the protection of cancer end-point (based on non-genotoxic carcinogenicity) as well as the protection of non-cancer endpoints (renal and hepatocellular effects).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation unit risk = $0.0000077 (\mu\text{g}/\text{m}^3)^{-1}$ (OEHHA) – adopted as the inhalation threshold value (below) has not considered carcinogenic effects. Chronic Reference Exposure Level (REL) = $3000 \mu\text{g}/\text{m}^3$ (OEHHA) (annual average) based on liver, kidney and haematologic changes in rats. Acute REL = $3000 \mu\text{g}/\text{m}^3$ (OEHH) (1-hour average) based on nasal and eye irritation effects in healthy human volunteers. Occupational inhalation exposure for 1,4-dioxane (ASCC): TWA: 10 ppm = $36 \text{ mg}/\text{m}^3$ STEL: NA Skin notification
Background	negligible

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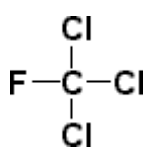
Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	10/12/2007
Previous Revisions:		

Trichlorofluoromethane (Freon 11)

General

The following is summarised from information from a range of available sources (HSDB, WHO (1990) and OEHHA).



Trichlorofluoromethane (also known as **freon 11**, **CFC-11**, fluorocarbon 11, fluorochloroform and fluorotrichloromethane) is a fully halogenated chlorofluorohydrocarbon (CFC). CFCs are commercially important because of their unique physical and chemical properties and relatively low physiological activity.

They are mainly used as refrigerants, solvents, blowing agents, sterilants, aerosol propellants, and as intermediates for plastics. Trichlorofluoromethane has been identified in emissions from volcanoes. The release of CFC-11 (and CFC-12) during use has caused the greatest concern environmentally because of their impact on ozone-depletion. During the mid-1970s, when aerosol propellant use was the major chlorofluorocarbon application, aerosols accounted for 75% of the immediate release of CFC-11 (and CFC-12), while refrigerants and blowing agents accounted for 14% and 12%, respectively. CFCs were scheduled for production phase-out under the Montreal Protocol and hence use has been reduced significantly with production now banned in many countries.

Exposure and Health Effects

Monitoring data indicate that the general population may be exposed to CFC-11 via inhalation of ambient air, ingestion of drinking water, and dermal contact with this compound and other consumer products containing CFC-11. Occupational exposure to CFC-11 may occur through inhalation and dermal contact with this compound at workplaces where CFC-11 is produced or used.

CFC-11's former production & use as a refrigerant, in fire extinguishers, chemical intermediate, and blowing agent resulted in its direct release to the environment through various waste streams. If released to air, CFC-11 will exist solely as a gas in the ambient atmosphere. CFC-11 is very stable in the troposphere having a half-life of 52-207 yr. As a result of its stability, CFC-11 is transported long distances and its concentration is fairly uniform around the globe away from known sources. The only major sink for CFC-11 is its slow diffusion into the stratosphere where photolysis occurs and subsequent reactions which destroy ozone.

If released to soil, CFC-11 is expected to have moderate mobility with volatilisation from soil surfaces is expected to be an important fate process. If released into water, CFC-11 is not expected to adsorb to suspended solids and sediment based upon the estimated K_{oc}. Volatilisation from water surfaces is expected to be important. Concentration profiles in oceans show that CFC-11 is primarily in surface layers, suggesting that the oceans are not a sink for this chemical. Biodegradation of CFC-11 in water and soils proceeds slowly under anaerobic conditions.

The potential for bioconcentration of CFC-11 in aquatic organisms is moderate.

The kinetics and metabolism of CFCs are characterised by rapid pulmonary absorption and distribution. There is no indication of any accumulation. Metabolic transformation of the CFCs is considered negligible, if it occurs at all. Therefore, toxic effects of metabolites are very unlikely.

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The acute toxicity of chlorofluorocarbons is very low, as demonstrated in studies on various animal species and by different routes of administration. It is characterized by effects on the heart, the respiratory system, and occasionally the liver. The effects are in accordance with those observed in acute intoxications in humans.

After repeated exposure, comparable clinical symptoms can be observed. Alterations in the liver and kidney occur occasionally. In humans, CNS, cardiovascular and respiratory symptoms occur in cases of severe abuse and in uncontrolled or accidental occupational exposure. Under conditions of use involving short-term exposures of up to 1000 ppm, no adverse health effects would be expected.

An evaluation of the animal studies indicates no carcinogenic risk to humans. This is underlined by the lack of genotoxicity in different mutagenic end-points and cell transformation. No effects on reproduction have been reported.

Increasing concern has been focused on the consequences of a reduction of ozone in the upper atmosphere, with the concomitant increase of UV-B radiation at the surface of the earth. Model calculations predict, for the next 50 years, ozone depletions of between 1 and 10%, depending on the scenario used for the release of chlorofluorocarbons and other trace gases.

Among the effects on human health, the induction of non-melanoma skin cancer has been investigated extensively, both in human epidemiology and in animal experimental work. It is a generally accepted conclusion that the incidence of non-melanoma skin cancer will increase as a result of ozone depletion. Ozone depletion by 5% would lead to an increase of the incidence by 16%. Indications are increasing that UV-B radiation also plays a role in the induction and growth of cutaneous melanomas, a more dangerous type of skin cancer. Uncertainty, however, especially with regard to the dose-effect relationship, makes quantitative predictions very difficult. The immune system of experimental animals is suppressed in specific ways by UV-B radiation. Although much still has to be learned through further research, the possibility that immune suppression effects and a consequent increase in the incidence of some infectious diseases might occur as a result of stratospheric ozone depletion should not be ignored. There are also indications that UV-B radiation increases cataract formation, an important cause of blindness especially in areas with limited medical facilities.

Trichlorofluoromethane (Freon 11)

Properties

CFC-11 is a colourless, stable, organic liquid (below 23.8°C) with a faint ethereal odour. Key properties are presented below (HSDB and RAIS):

CAS No	75-69-4
Chemical Formula	CCl ₃ F
Molecular Weight	137.37
Vapour Pressure	803 mmHg at 25°C
Vapour Density	4.7
Density	1.49 g/ml at 20°C
Solubility	1100 mg/L at 20°C
Air Diffusion Coefficient	0.087 cm ² /s
Water Diffusion Coefficient	9.7 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.11 atm.m ³ /mol = 3.97 at 25°C (unitless)
Koc	48.64 cm ³ /g
Log Kow	2.53
Odour Threshold	>20% in air detected
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0173 cm/hr
Conversion	1ppm = 5.71 mg/m ³ at 20°C

Suggested Toxicity Values for Risk Characterisation

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. Limited data is available regarding environmental levels of CFC-11. Ambient levels of CFC-11 in air have been reported in a number of locations within Sydney, Newcastle and Wollongong (DEC, 2004) with the average concentration reported in the Sydney CBD (data from January 1996 to August 2001) of 0.3 ppbv (with similar concentrations reported in outer suburbs and rural areas). The maximum 24-hour average concentration of CFC-11 reported was 2.7 (higher than in outer lying urban areas and rural areas). As environmental exposures are expected to be dominated by the presence of this chemical in air, the potential for background concentrations in water is assumed to be negligible. Based on the average concentration in Sydney CBD of 0.3 ppbv = 1.69 µg/m³ (at 25°C), which is <1% of the available threshold chronic reference concentration, background intakes have been assumed to be negligible.

Toxicity Values

Review of available data with respect to CFC-11 indicates that this chemical is not considered to be carcinogenic or genotoxic. Therefore it is considered appropriate that potential health effects be evaluated on the basis of an appropriate threshold (where available).

Toxicity data relevant for use in the characterisation of risk to human health have been selected for CFC-11 following review of the available information in general accordance with current

Trichlorofluoromethane (Freon 11)

Australian guidance (enHealth (2004) and NEPC (1999)), accounting for background intake where relevant.

The data presented with respect to the assessment of inhalation effects is relevant to both acute and chronic exposures (with relevant averaging periods identified).

Classification USEPA: Not classified
IARC: Not Classified

Toxicity Values:	
Oral	RfC = 0.3 mg/kg/day (IRIS) based on increased mortality in study on rats.
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Chronic Reference Concentration (RfC) = 0.7 mg/m ³ (formerly available from HEAST, a peer reviewed source that has not been updated ¹) (annual average)). No acute REL is available for CFC-11. Occupational inhalation exposure for CHC-11 (ASCC): TWA: 1000 ppm = 5620 mg/m ³ as a peak limitation STEL: NA
Background	negligible

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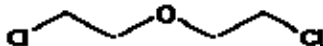
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Revision Dates

Document	Revision	Date of Review
Current Toxicity Summary	2007A	10/12/2007
Previous Revisions:		

GENERAL



Bis(2-chloroethyl) ether (also known as dichloroethyl ether, dichloroethyl oxide, and bis(β -chloroethyl) ether and abbreviated to **BCEE**) is a β -chloroalkyl ether. BCEE is reported to be used as an intermediate reactant in the synthesis of the methylthiocarbamic acid fungicide commonly known as metam-sodium. BCEE is believed to be present in trace amounts in the final product. Metamsodium. BCEE is also used as a solvent and in the production of polymers in a range of industrial processes.

The mobility and distribution of the selected chloroalkyl ethers is influenced by the water solubility and stability of BCEE. BCEE is soluble in water; rainfall removes it from the atmosphere and it tends to remain in water with very slow hydrolysis. BCEE evaporates from surface water within a week and is degraded in a little more than a day in the atmosphere by abiotic processes. BCEE may be persistent in air and water due to the relative stability of β -chloroalkyl ethers. BCEE is not expected to absorb to soil or sediment and is therefore considered to be mobile in these media. Bioaccumulation is not expected to occur.

EXPOSURE AND HEALTH EFFECTS

BCEE may enter the environment as a by-product from the chlorination of waste streams containing ethylene or propylene, and as a contaminant in the fungicide metam-sodium. The main routes of exposure to BCEE are via inhalation of contaminated air. The chemical can be ingested from drinking water or absorbed dermally. Exposures in occupational areas where the chemical is handled or produced are expected to be higher. BCEE is rapidly absorbed from all routes of exposure, metabolized, and excreted in the breath and urine. Most BCEE that enters to body is eliminated within 2 to 3 days and hence will not accumulate in the body.

People exposed to BCEE in air at high concentrations report that it is highly irritating to the eyes and the nose. Animal studies show that acute exposure to BCEE in air can cause severe injury to the lungs, and may lead to death. Mice given repeated oral doses of BCEE developed liver tumours but not in rats. Effects of BCEE on other organs and body functions have not been well studied, and it is not known if BCEE impairs reproduction or the development of foetuses. None of the long term studies in laboratory animals is of sufficient quality to provide quantitative information on either the potential of BCEE to cause cancer or the toxicological effects produced by long term exposure to this substance. Available data is generally considered to be inadequate for the assessment of BCEE.

PROPERTIES

BCEE is a colourless, volatile liquid with a "chlorinated solvent-like" odour. . Key properties are presented below (ATSDR 1989, USEPA 2004 and ORNL Database 2006):

CAS No	111-44-4
Chemical Formula	C ₄ H ₈ Cl ₂ O
Molecular Weight	143.04
Vapour Pressure	1.55 mmHg at 25°C
Vapour Density	4.93
Density	1.22 g/ml at 20°C
Solubility	17200 mg/L at 20°C
Air Diffusion Coefficient	0.0692 cm ² /s
Water Diffusion Coefficient	7.53 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	1.8x10 ⁻⁵ atm.m ³ /mol = 0.000738 at 25°C (unitless)
Koc	14.95 cm ³ /g
Log Kow	1.29
Odour Threshold	0.049 ppm = 0.287 mg/m ³
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00211

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. No data is available regarding environmental levels of BCEE in Australia. As BCEE has been evaluated on the basis of a non-threshold approach, background intakes are not relevant for the assessment of potential risks to human health.

Toxicity Values

Review of available data with respect to BCEE by the WHO (2001) indicates that very limited data is available for this chemical. There is insufficient data available to determine carcinogenicity or genotoxicity. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to BCEE on the basis of a threshold approach, however there are no threshold values that have been derived for this chemicals relevant to chronic exposures. The USEPA has established non-threshold values for BCEE for oral and inhalation exposure. In addition more data is available data for bis(2-chloromethyl)ether (BCME, also a chloroalkyl ether), which indicates it can be considered to be carcinogenic, with the WHO (1998) deriving an inhalation unit risk (non-threshold) value for the assessment of inhalation exposures to this chemical. In the absence of threshold data, and given the application of a non-threshold approach for the assessment of the alpha-chloroalkyl ether (BCME), the assessment of BCEE has adopted the non-threshold data available from the USEPA. This approach is considered to be conservative.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for BCEE following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Classification USEPA – B2 – possible human carcinogen
IARC: Group 3 not classifiable as to carcinogenicity to humans.

Toxicity Values:	
Oral	Oral slope factor= $1.1 \text{ (mg/kg/day)}^{-1}$ (USEPA, current).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation unit risk = $0.00033 \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ (USEPA, current) Occupational inhalation exposure (NOHSC, current to 2006): TWA: 5 ppm = 29 mg/m ³ STEL: 10 ppm = 58 mg/m ³

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GENERAL

PAHs are a large group of organic compounds with two or more fused aromatic rings made up of carbon and hydrogen atoms. PAHs are formed from incomplete combustion of organic materials such as processing of coal, crude oil, combustion of natural gas, refuse, vehicle emissions, heating, cooking and tobacco smoking as well as natural processes including carbonization. The natural background level is due to PAH production in plant species. Because of such widespread sources, PAHs are present almost everywhere. Food is considered to be the major source of human exposure to PAH due to the formation of PAH during cooking or from atmospheric deposition of PAHs on grains, fruits and vegetables.

There are several hundred PAHs, including derivatives of PAHs. The best known (and studied) is benzo(a)pyrene (BaP).

At ambient temperatures, PAHs are solids. The general characteristics common to the class are high melting- and boiling-points, low vapour pressure, and very low water solubility which tends to decrease with increasing molecular mass. PAH are soluble in many organic solvents and are highly lipophilic. Most PAHs have with a low vapour pressure are absorbed on particles in air. When absorbed onto particulates or in water, PAHs can undergo photodecomposition.

PAHs are considered to be persistent organic pollutants. The classification of PAHs as a persistent chemical has been undertaken in detail (Edlund, 2001) and while many of the individual PAHs are persistent in the environment, with half-lives in soils up to 9 years, and a number considered carcinogenic to humans, PAHs are not considered to be bioaccumulative. PAHs are metabolised by most organisms and hence do not bio-accumulate within species (animals, poultry, fish).

EXPOSURE

PAHs are present throughout the environment with exposure of the general population via inhalation of particulates (dust) or cigarette smoke, oral (including food) or dermal routes. In most cases the inhalation of PAHs from cigarette smoke, wood smoke, ambient air and ingestion of foods are the primary routes of exposure. Exposure may also occur through oral ingestion of drinking water or soils. Apart from occupational exposures, the primary concern is ingestion of PAHs in food.

If released into the environment the following can be noted with respect to PAHs:

- **Air:** PAHs released to the atmosphere are subject to short and long range transport and are removed by wet and dry deposition onto soil, water and vegetation. PAHs may be present in vapour phase (more relevant for naphthalene) and bound to particulates (most relevant for heavier PAHs such as BaP).
- **Water:** In water PAHs can volatilise (relevant for some PAHs only), oxidise, biodegrade, bind to suspended particles or sediments or accumulate in aquatic organisms.

- Soil and Sediments: In sediment, PAHs can biodegrade or accumulate in aquatic organisms. In soils PAHs can volatilise (relevant for some PAHs only), undergo abiotic degradation, biodegrade or accumulate in plants. PAHs in soils can also enter groundwater.
- PAHs can be accumulated in aquatic organisms from water, sediments and food. Bioconcentration is greater for higher molecular weight compounds, such as BaP. However the ability of fish to metabolise PAHs has resulted in BaP not frequently detected, or only detected at low concentrations, in fish from environments with heavily contaminated PAHs.
- Some plants can take up PAHs from soil via the roots or from the air (deposition). Biomagnification in plants is low.
- PAHs may accumulate in terrestrial animals via the food chain or ingestion of soils.

TOXICITY

General

Several PAHs may induce a number of adverse effects such as immunotoxicity, genotoxicity, carcinogenicity, reproductive toxicity, and possible development of atherosclerosis. The critical endpoint for these PAHs is generally considered to be carcinogenicity. The most potent carcinogens have been shown to be benzo[a]anthracene, benzo[a]pyrene and dibenz[ah]anthracene. Studies of various environmentally relevant matrices, such as coal combustion effluents, motor vehicle exhaust, used motor lubricating oil, and tobacco smoke, have shown that the PAH in these mixtures are mainly responsible for their carcinogenic potential.

PAH are absorbed through the pulmonary tract, the gastrointestinal tract, and the skin. The rate of absorption from the lungs depends on the type of PAH, the size of the particles on which they are absorbed, and the composition of the adsorbent. PAH are widely distributed throughout the organism after administration by any route and are found in almost all internal organs, but particularly those rich in lipids. The metabolism of PAH is complex.

There have been several pollutant classifications relating to PAHs. Although there are a vast number of PAHs, sixteen are routinely tested for and evaluated. These 16 PAHs have been designated by the United States Environmental Protection Agency (USEPA) as compounds of interest under a suggested procedure for reporting test measurement results (USEPA 1988). A subset of this includes PAHs identified by the International Agency for Research on Cancer (IARC) as probable (Group 2A) or possible (Group 2B) human carcinogens (IARC 1983 and 2002). Others have not been evaluated (NA) or are not classifiable (Group 3).

A further subset of PAHs are those to be used as indicators for the purposes of emissions inventories under the UN/ECE's Persistent Organic Pollutants (POP) Protocol (1998). The WHO (2003) lists all PAHs as POP to be evaluated with respect to risks to human health. WHO (1998) has evaluated a number of PAHs for their carcinogenic potential (C) and genotoxic potential (G) (+ is positive; - is negative; ? is

questionable; parentheses identifies that result is derived from small database). These classifications are summarised in the following table which lists the 16 priority PAHs in order of increasing molecular weight

Priority PAHs	IARC/USEPA Classification	WHO C/G	TEF	UN/ECE POP	Volatile?
Acenaphthene	NA/NA	(?)/(?)			
Acenaphthylene	NA/D	na/(?)			
Anthracene	3/D	-/-			
Benz(a)anthracene	2A/B2	+/+	0.1		
Benzo(a)pyrene	2A/B2	+/+	1	Yes	
Benzo(b)fluoranthene	2B/B2	+/+	0.1	Yes	
Benzo(g,h,i)perylene	3/NA	-/+			
Benzo(k)fluoranthene	2B/B2	+/+	0.1	Yes	
Chrysene	3/B2	+/+	0.1		
Dibenz(a,h,)anthracene	2A/B2	+/+	4		
Fluoranthene	3/D	(+?)/+			
Fluorene	3/NA	-/-			
Indeno(1,2,3-c,d)pyrene	2B/B2	+/+	0.1	Yes	
Naphthalene	3/D	(?)/-			Yes
Phenanthrene	3/C	(?)/(?)			
Pyrene	3/C	(?)/(?)			

IARC Classification: 2A: Probable Human Carcinogen; 2B: Possible Human Carcinogen; 3: Not Classifiable
 USEPA Classification: B2: Probable Human Carcinogen; D Not Classifiable

Evaluation of PAHs in Risk Assessment

There are three main ways in which PAHs can be evaluated in a risk assessment (WHO EHC, 1998).

1. Toxicity Equivalence Factors (TEF)
2. Comparative toxicity; and
3. Use of BaP as a surrogate.

Toxicity Equivalence Factors (TEF) Approach (Adopted by URS)

Relatively few mixtures containing PAH have been evaluated under conditions that are acceptable for use in the quantification of risk. For some processes, e.g. coal coking, the data on human exposure are sufficiently complete to allow quantitative estimates of risk (refer to second approach); however, changes in the parameters of the combustion process, such as temperature and amount of oxygen feedstock, may result in variations in the types, amounts, and physical status of PAH in the mixture. These variations may be sufficient to alter the risk posed by the mixture. One way of dealing with the uncertainties inherent in

differences in the composition of mixtures is to base quantitative estimates on considerations of individual components.

This approach is based on an assumption of additive risk, which leads, in principle, to an estimate of the risk associated with identified PAHs. In practice, the risks attributable to individual PAH are summed, or the risk posed by individual PAH is expressed relative to that for BaP, and then the levels of these equivalents are summed (referred to as the toxicity equivalence factor or TEF, approach). The underlying assumption is that the individual estimates of risk are additive. Although there may be interactions between PAH, the risks appear to be approximately additive, especially at low doses relevant for assessing environmental exposures.

This can be applied in two ways:

- 1. Identification of available toxicological values for individual PAHs, typically those not considered carcinogenic or considered non-genotoxic carcinogens. Then the remaining PAHs, typically considered genotoxic carcinogens (or potentially) are evaluated based on their relative toxicity to BaP (refer to table above for common TEFs, Fitzgerald 1998). This is the approach adopted by URS in quantifying potential risks associated with exposures to PAHs. Refer to toxicity summary relevant to BaP and above table for adopted TEFs. PAHs evaluated on the basis of threshold effects are quantified separately on the basis of relevant threshold values for oral, dermal and inhalation exposures. Refer to relevant toxicity summaries for each of these compounds.***
2. Evaluation of all PAHs based on the relative carcinogenic potential relative to BaP (i.e. applying TEFs for all PAHs).

The main advantages to using the TEF approach are: clearly defined chemical species are assessed, a good body of scientific literature is available to evaluate it, not affected by variability in the composition of mixtures, relatively easy to apply in ambient environments affected by many sources, and regulatory experience exists.

The main disadvantages are: may underestimate risk due to all PAH by considering only a few compounds, depends on extrapolation from animal models to humans (as data is limited for many PAHs), and it can be resource-intensive, as monitoring and analysis are required.

Comparative Potency of Mixtures

The comparative potency approach is used to estimate the potency of the PAH in mixtures without having to identify or quantify the individual compounds. The comparative potency approach was initially proposed as part of an approach to assessing the carcinogenic risk of PAH in diesel emissions. Risk assessments of relatively high quality are currently available only for the following mixtures: cigarette smoke, coke-oven emissions, and coal-tar.

Although the concept of comparative potency has been extensively validated, some outstanding issues remain, which are discussed below.

The main advantages to the methodology are: the risk of whole mixtures rather than only a few components is estimated, a good body of scientific literature is available for some mixtures, takes advantage of existing data on human carcinogenicity (available for mixtures only), and it is simple and requires inexpensive monitoring.

The main disadvantages are: does not define the contribution of PAHs to estimated overall risk, it is difficult to use for assessing speciated components of a mixture, risk estimates require estimates of the contributions of individual sources to the levels of organic compounds in the ambient environment, the assumption that mixtures from the same source are associated with similar risks may not be supported by the available data, the levels of compounds extractable in organic solvents are not usually reported, and the analytical methods are not standardized. On this basis this approach is not typically adopted for the quantification of potential exposures to PAHs in risk assessment.

BaP as Surrogate

The third approach assumes that the risk due to the PAH component of complex mixtures and the levels of individual PAHs in the mixtures are proportional to those of BaP in the mixture and vary proportionately. Using this approach, the risk due to the PAH component of mixtures can be estimated as the product of the environmental levels of BaP and the estimate of the risk attributable to mixtures per unit amount of BaP.

In general, the approach does not predict the potency of an ambient complex mixture as a whole but merely it's PAH component. The approach assumes that the levels of individual PAHs relative to BaP are relatively stable from mixture to mixture. It also assumes that the risk attributable to PAHs in any given mixture is proportional to the risk due to BaP. In other words, the level of BaP is sufficient to estimate the risk of the PAH fraction in a mixture.

The main advantages are: it can be used to estimate risk of entire PAH component of a mixture, it is simple and based on a few testable assumptions, it is well supported by the available data, relatively easy and inexpensive to apply, and regulatory experience exists (particularly in air assessments).

The main disadvantages are: may result in overestimate of the risk of PAHs within a mixture, some PAHs are not well represented by BaP and must be considered separately.

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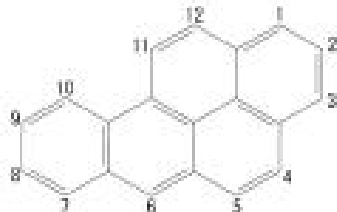
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GENERAL



Benzo(a)pyrene (also known as benzo(d,e,f)chrysene, 3,4-benzopyrene, 3,4-benzopyrene, benz(a)pyrene, 6,7-benzopyrene, BP and abbreviated to **BaP**) is a yellowish solid with very low vapour pressure and low water solubility. BaP is the most well known and most widely studied polycyclic aromatic hydrocarbon (PAH). PAHs are a large group of hydrocarbons containing two or more benzene rings fused to each other or to other hydrocarbon rings.

PAHs are formed mainly as a result of pyrolytic processes, especially the incomplete combustion of organic materials during industrial and other human activities, such as processing of coal and crude oil, combustion of natural gas, including for heating, combustion of refuse, vehicle traffic, cooking and cigarette smoking, as well as in natural processes such as carbonization. PAHs are also present in many foodstuffs. Natural sources include volcanoes and forest fires. Crude oil, shale oil and coal tar contain small amounts of PAHs.

There are several hundred individual PAH compounds.

PROPERTIES

BaP is a pale yellow solid (plates or needles) with a faint aromatic odour. BaP is not very soluble in water, sparingly soluble in ethanol and methanol, soluble in benzene, toluene, xylene and ether. Key properties are presented below (ATSDR 1995, USEPA 2004 and ORNL Database 2006):

CAS No	50-32-8
Chemical Formula	C ₂₀ H ₁₂
Molecular Weight	252.32
Vapour Pressure	5.49x10 ⁻⁹ mmHg at 25°C
Vapour Density	8.7
Density	1.351 g/ml at 20°C
Solubility	0.00162 mg/L at 20°C
Air Diffusion Coefficient	0.043 cm ² /s
Water Diffusion Coefficient	9 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	4.9x10 ⁻⁷ atm.m ³ /mol = 0.0000187 at 25°C (unitless)
Koc	787 000 cm ³ /g
Log Kow	6.13
Odour Threshold	NA
Dermal Absorption	0.13 (unitless)
Permeability Constant	1.24 cm/hr

EXPOSURE

PAHs are present throughout the environment with exposure of the general population via inhalation of particulates (dust) or cigarette smoke, oral (including food) or dermal routes. In most cases the inhalation of PAHs from cigarette smoke, wood smoke, ambient air and ingestion of foods are the primary routes of exposure. Exposure may also occur through oral ingestion of drinking water or soils. Apart from occupational exposures, the primary concern is ingestion of PAHs in food.

If released into the environment the following can be noted with respect to PAHs:

- Air: PAHs released to the atmosphere are subject to short and long range transport and are removed by wet and dry deposition onto soil, water and vegetation. As BaP is not considered volatile, it is generally present in the atmosphere sorbed to particulates with dry deposition responsible for most of the removal.
- Water: In water PAHs can volatilise (not relevant for BaP), oxidise, biodegrade, bind to suspended particles or sediments or accumulate in aquatic organisms.
- Soil and Sediments: In sediment, PAHs can biodegrade or accumulate in aquatic organisms. In soils PAHs can volatilise (Not relevant for BaP), undergo abiotic degradation, biodegrade or accumulate in plants. PAHs in soils can also enter groundwater.
- The low solubility, low vapour pressure and high K_{ow} of BaP results in partitioning mainly between soil (82%) and sediment (17%) with very minimal partitioning to water, air and biota.
- The estimated half-lives for BaP are less than 1-6 days in the atmosphere, less than 1-8 hours in water, 5-10 years in sediment, and greater than 14-16 months in soil (for complete degradation).
- PAHs can be accumulated in aquatic organisms from water, sediments and food. Bioconcentration is greater for higher molecular weight compounds, such as BaP. However the ability of fish to metabolise PAHs has resulted in BaP not frequently detected, or only detected at low concentrations, in fish from environments with heavily contaminated PAHs.
- Some plants can take up PAHs from soil via the roots or from the air (deposition). Biomagnification in plants is low.
- PAHs may accumulate in terrestrial animals via the food chain or ingestion of soils.

HEALTH EFFECTS

General

The following has been summarised from information presented by EA (2002), ATSDR (1995) and the WHO (2000).

PAHs are extremely lipophilic and are generally rapidly absorbed upon inhalation, ingestion or dermal exposure with rapid and wide distribution to the organs. The metabolism of BaP has been extensively studied in the literature and one of the most important pathway is the metabolism initially by the microsomal cytochrome P-450 monooxygenase system and epoxide hydrolases to several arene oxides including an intensely reactive intermediate (7,8-diol-9,10-epoxide). The intermediate is considered electrophilic and is considered an “ultimate” carcinogen as it can bind directly to informational macromolecules, forming adducts, Such adducts in DNA results in mutations when the nucleic acid is replicated and is considered a probable basis for PAH carcinogenic action.

Data from animal studies indicate that several PAHs may induce a number of adverse effects, such as immunotoxicity, genotoxicity, carcinogenicity, reproductive toxicity (affecting both male and female offspring), and may possibly also influence development of atherosclerosis. However, the critical endpoint for the health risk evaluation is the well-documented carcinogenicity of several PAHs.

BaP is considered to have fairly low acute toxicity, however exposures to high levels (mg range) may result in body weight loss, peripheral blood pattern changes, changes in the liver and kidney function and peribronchial pathologies. Following repeated exposures, adverse effects include chronic dermatitis, breathing problems, chest pains, chest and throat irritation, non-tumour pathology in the lung, cancer of the skin and lung and a depressed immune system.

There is an extensive literature on the epidemiology of workforces exposed to complex mixtures of PAHs in, for example, aluminium production and coke production, and in those handling coal tar, coal tar pitches and soot (UA, 2002). These studies conclusively demonstrate an elevated incidence of lung tumours on inhalation and of skin tumours from skin contact. It is not possible, however, to assess with confidence the contribution of individual PAHs to the observed cancer burden.

Genotoxicity

BaP has been thoroughly studies for genotoxicity. BaP induces genetic damage *in vitro* and produces a wide range of genotoxic effects. The results of *in vivo* studies indicate many of the same types of adverse effects observed *in vitro*. The available data also indicate that BaP is genotoxic in both somatic and germinal cells of animals. The formation of diol epoxides that covalently bond to DNA appears to be the primary mechanism for both genotoxicity and carcinogenicity.

The genotoxic activity of BaP is well established and is frequently used as a positive control to review the genotoxic action of unknown compounds and serves as the model compound for PAHs with the information available on the formation of metabolites and structure of BaP theoretically used to predict the potential genotoxicity and carcinogenicity of other PAHs that have not been as extensively studied.

Carcinogenicity

Evidence exists (primarily from occupational studies) that mixtures of PAHs are carcinogenic in humans. BaP is by far the most intensively studied PAH in experimental animals. It produces tumours of many different tissues, depending on the species tested and the route of application. BaP is the only PAH that has been tested for carcinogenicity following inhalation, and it produced lung tumours in hamsters, the only species tested. Induction of lung tumours in rats and hamsters has also been documented for BaP and

several other PAHs following direct application, e.g. intratracheal instillation into the pulmonary tissue. The lung carcinogenicity of BaP can be enhanced by coexposure to other substances such as cigarette smoke, asbestos and probably also airborne particles.

Because several PAHs have been shown to be carcinogenic, and many more have been shown to be genotoxic, due the level of knowledge associated with BaP, BaP is typically used as a suitable indicator for the carcinogenic fraction of the large number of PAHs.

TOXICITY CLASSIFICATION

BaP is classified as a "probable" human carcinogen (Category B2) by the USEPA for all routes of exposure based on evidence from animal studies. Human data specifically linking BaP to a carcinogenic effect are lacking.

IARC has classified BaP in Group 2A (probably carcinogenic to humans) based on sufficient evidence that BaP is carcinogenic to experimental animals.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have established a health based guideline of 0.00001 mg/L (10 ng/L) for BaP. The value was established based on the consideration of health effects (consideration of the WHO guideline established, refer below) and the determination for analysis using commonly available techniques.

The NEPC (2002) has established an ambient air investigation level for BaP as a marker for PAHs of 0.3 ng/m³ based on an annual average and an 8-year goal. As discussed in NEPM (2003), this value was derived in New Zealand from the WHO unit risk value and an incremental lifetime risk of 10⁻⁵. Due to the transparency provided in the derivation of the WHO unit risk value and the New Zealand air quality guideline the NEPC adopted the guideline value of 0.3 ng/m³. The available WHO unit risk is presented in the discussion below.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For BaP the NOHSC have identified the chemical as a Category 2, probable human carcinogen for which there is sufficient evidence to provide a strong presumption that human exposure may result in the development of cancer. As such it has been determined that it is not currently possible to establish an appropriate exposure standard and that for BaP, exposure should be controlled to the lowest practicable level.

WHO

The WHO (Drinking Water Guideline 2004) identified BaP as one of the most carcinogenic PAHs and established a drinking water guideline of 0.7 ug/L, corresponding to an excess lifetime cancer risk of 10^{-5} . This was based on an oral carcinogenicity study.

The oral slope factor associated with this guideline value:

$$\begin{aligned} SF (mg/kg/day)^{-1} &= Risk/Intake(mg/kg/day) \\ &= [Risk \times Body Weight]/[Concentration (in water) \times Ingestion Rate] \\ &= [1 \times 10^{-5} \times 70kg]/[0.0007 mg/L \times 2 L/day] \\ &= 0.5 (mg/kg/day)^{-1} \end{aligned}$$

The WHO (2000) provided a review of PAHs in air. With respect to establishing an air guideline, BaP was identified as an indicator chemical due to the more extensive database available. Based on epidemiological data from studies in coke-oven workers, a unit risk for BaP was estimated to be $8.7 \times 10^{-5} (ng/m^3)^{-1}$ (ie for an air concentration of $1 ng/m^3$, the lifetime risk is estimated to be 8.7×10^{-5}).

The unit risk value is equivalent to the following slope factor:

$$\begin{aligned} SF (mg/kg/day)^{-1} &= Risk/Intake(mg/kg/day) \\ &= [Risk \times Body Weight]/[Concentration (in air) \times Inhalation Rate] \\ &= [8.7 \times 10^{-5} \times 70kg]/[0.000001mg/m^3 \times 20 m^3/day] \\ &= 304.5 (mg/kg/day)^{-1} \end{aligned}$$

EU

The European Commission (2001) developed a limit value for PAH compounds in ambient air. Following review of available unit risk estimates derived from relevant epidemiological studies, the working group recommended the unit risk adopted by the WHO Air Quality Guideline for Europe from a US coke oven workers study (noted above) as a starting point. BaP was identified as a suitable marker for the carcinogenic risk of PAHs in air, despite not necessarily being the most potent carcinogen present. The EU set an air quality limit of 0.5 – 1.0 ng/m^3 (annual average) for PAHs in air on this basis.

US

The USEPA has established and **oral slope factor for BaP of $7.3 (mg/kg/day)^{-1}$** based on the geometric mean of four slope factors obtained by different modelling procedures undertaken on the combination of multiple data sets from two different reports.

No inhalation specific carcinogenic assessment is provided by the USEPA. Common practice in the US is to adopt the oral slope factor for the assessment of inhalation exposures.

No Minimum Risk levels (MRLs) have been derived by ATSDR for BaP.

The OEHHA (1994) has established a cancer potency factor of $11.5 \text{ (mg/kg/day)}^{-1}$ and an inhalation unit risk for the evaluation of chronic exposure to BaP that ranges from 1.1×10^{-3} to $3.3 \times 10^{-3} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$. The unit risk value of $1.1 \times 10^{-3} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ is considered the best value for the assessment of inhalation exposures as it is based on an inhalation study (hamsters).

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI or RfD in assessing potential exposures to site related chemicals. With respect to BaP, assessment is undertaken on the basis of a non-threshold approach for oral, dermal and inhalation exposures and hence potential background intakes are not relevant.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for BaP following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	Oral Slope Factor = $0.5 \text{ (mg/kg/day)}^{-1}$ (WHO Drinking Water Guidelines, 2004)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation unit risk of 8.7×10^{-5} (per ng/m^3), equivalent to $304.5 \text{ (mg/kg/day)}^{-1}$ (WHO 2000 and NEPC 2002). Occupational inhalation exposure evaluated using the following (NOHSC): <i>No guideline available, exposure to be controlled to the lowest practicable level.</i>

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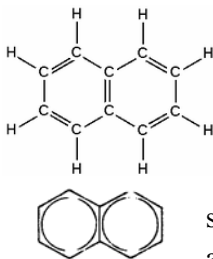
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GENERAL



Naphthalene (also known as tar camphor, albocarbon, naphthene, mothballs and white tar) is a white solid with a characteristic odour of mothballs. It is a polycyclic aromatic hydrocarbon (PAH) composed of two fused benzene rings. The principal end use of naphthalene is as a raw material for the production of phthalic anhydride. It is also used as an intermediate for synthetic resins, celluloid, lampblack, smokeless powder, solvents, and lubricants. Naphthalene is also used directly as a moth repellent, insecticide, anthelmintic, and intestinal antiseptic.

1-Methylnaphthalene is a naphthalene-related compound which is also called alpha methylnaphthalene. It is a clear liquid. Another naphthalene-related compound, 2-methylnaphthalene, is also called beta methylnaphthalene. It is a solid like naphthalene. 1-Methylnaphthalene and 2-methylnaphthalene are used to make other chemicals such as dyes, resins, and, for 2-methylnaphthalene and vitamin K. Along with naphthalene, they are present in cigarette smoke, wood smoke, tar, and asphalt, and at some hazardous waste sites.

PROPERTIES

Naphthalene is a white, crystalline solid, volatile and lipophilic. When mixed with air, naphthalene vapours burn easily. Naphthalene is slightly soluble in water but soluble in benzene, alcohol, ether and acetone. Key properties are presented below (ref: ATSDR 2005, USEPA 2004 and ORNL Database 2006):

CAS No	91-20-3
Chemical Formula	C ₁₀ H ₈
Molecular Weight	128.16
Vapour Pressure	0.087 mmHg at 25°C
Vapour Density	4.4
Density	1.15 g/ml at 20°C
Solubility	31.7 mg/L at 25°C
Air Diffusion Coefficient	0.059 cm ² /s
Water Diffusion Coefficient	7.5 x 10 ⁻⁶ cm ² /s
Henry's Law Coefficient	0.00048 atm.m ³ /mol = 0.0198 at 25°C (unitless)
Koc	1837 cm ³ /g
Log Kow	3.3
Odour Threshold	0.44 mg/m ³ in air
Dermal Absorption	0.13 (unitless)
Permeability Constant	0.0694 cm/hr

EXPOSURE

Exposure to naphthalene may be derived from environmental and occupational sources and from consumer products. The most likely pathway by which the general public is exposed to naphthalene is by inhalation due to the release of this substance from combustion fuels, moth repellents, and cigarette smoke. Available data also indicate that the general population may be exposed to naphthalene via ingestion of food and drinking water, although these pathways are considered minor as compared to inhalation.

Naphthalene is erratically absorbed when ingested. It is rapidly absorbed when inhaled. Dermal absorption in humans, especially in infants may be significant and further enhanced by prior application of oils. Effects of toxicity can be observed in infants following transplacental transfer of naphthalene or its oxidation products.

Naphthalene is metabolized in the liver to yield a variety of hydroxy and methylthio derivatives. In these two groups of metabolites alpha-naphthol and 1-methylthio naphthalene are the most prominent urinary constituents. The initial metabolite is apparently a 1,2-epoxide produced in the liver by mixed function oxidase enzymes. This reactive compound is subsequently converted to naphthalene dihydrodiol and to alphanaphthol. Both compounds are excreted as such and as glucuronide conjugates.

Naphthalene dihydrodiol may be further converted in the eye to yield 1,2-naphthoquinone, a known cataractogenic agent.

If released into the environment the following can be noted with respect to naphthalene (EU 2003 and ATSDR 2005):

- **Air:** Naphthalene is volatile and most airborne naphthalene is in the vapour phase. A small proportion of naphthalene in the atmosphere may be transported to surface water or soil via wet or dry deposition. Vapour-phase naphthalene will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals and nitrate radicals; the half-life for these reactions in air are estimated to be 18 and 60 hours, respectively.
- **Water:** If released into water, only a small fraction of naphthalene is expected to adsorb to suspended solids and sediment based upon the Koc data. Naphthalene has been shown to biodegrade in water with half-lives ranging from about 0.8 to 43 days. Volatilisation and photolysis in sunlit surface waters may be an important fate process. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyze under environmental conditions.
- **Soil:** If released to soil, naphthalene is expected to adsorb to soils and sediments to a moderate extent and have moderate to low mobility. Volatilisation from moist soil surfaces is expected to be an important fate process (particularly from aerated soils). Biodegradation is expected to be an important fate process based upon soil degradation half-lives of 2-18 days.
- **Biodegradation:** Standardised tests indicate that naphthalene is not inherently biodegradable. However, other non-standard tests indicate naphthalene is degraded under aerobic and denitrifying

conditions, particularly where micro-organisms are present (or used). Tests indicate that naphthalene is resistant to biodegradation under anaerobic conditions.

- Bioconcentration of naphthalene is moderate in aquatic organisms however naphthalene is reported to be rapidly eliminated and readily metabolised. Bioaccumulation in the food chain is not expected to occur.

HEALTH EFFECTS

General

Lipophilic PAHs can be absorbed through the lungs, the GI tract and the skin. This is observed with naphthalene. There are few data available regarding the distribution of naphthalene in either human or animal tissues, but rat studies indicate PAHs and their metabolites are found in most tissues, particularly those rich in lipids. Metabolism is complex with most metabolites excreted in the faeces or urine.

Reports that establish associations between naphthalene exposure and health effects in humans are restricted to numerous reports of haemolytic anaemia or cataracts following acute exposure or occupational exposure to naphthalene, either by ingestion or by inhalation of naphthalene vapours, but these reports have not identified exposure levels associated with these effects (ATSDR, 2005). A relationship appears to exist between an inherited deficiency in the enzyme, glucose 6-phosphate dehydrogenase (G6PD), and susceptibility to naphthalene-induced haemolysis. Newborn infants also appear to be susceptible to naphthalene-induced haemolysis presumably due to a decreased ability to conjugate and excrete naphthalene metabolites (ATSDR 2005). Results from animal studies exposed to naphthalene by oral administration, by inhalation exposure, or by parenteral administration identify several health effects of potential concern for humans, including maternal toxicity during pregnancy with acute oral exposure, decreased body weight (without lesions developing in any tissues or organs) with intermediate oral exposure, and increased incidence of non-neoplastic and neoplastic lesions in the nose (in rats and mice) and the lung (in mice only) with chronic inhalation exposure (ATSDR 2005).

Following oral exposure, the following have been identified as the primary target organs associated with toxicity of naphthalene (ORNL):

- Blood: Haemolytic anaemia associated with decreased haemoglobin and hematocrit values, increased reticulocyte counts, Heinz bodies, and bilirubin levels is the most commonly observed effect in humans following acute exposure to naphthalene. Haematological changes indicative of anaemia were observed in animals following subchronic exposure and dermal exposure.
- Gastrointestinal tract: Nausea, vomiting, abdominal pain, and diarrhoea has been reported in humans following acute exposure to naphthalene.
- Nervous system: Confusion, listlessness and lethargy, vertigo, muscle twitching, convulsions, decreased responses to painful stimuli, coma, and cerebral oedema have been reported in humans following acute exposure to naphthalene. Some effects may be secondary to hemolysis.

- Liver: Jaundice, enlarged liver, and increased serum enzyme activity have been reported in humans following acute exposure to naphthalene. Increased liver weight and increased liver enzyme activity have been reported in animals following subchronic exposure to naphthalene.
- Kidneys: Increased creatinine and blood urea nitrogen levels, proteinuria and hemoglobinuria, anuria, and tubular necrosis have been reported in humans following acute exposure to naphthalene. Some effects may be secondary to hemolysis. Cortical lymphocyte infiltration and tubular degeneration have been reported in animals following subchronic exposure.
- Eyes: Restricted visual fields, optic atrophy, and cataracts have been reported following acute exposure in humans. Cataracts have been reported in animals following subchronic exposure to naphthalene.
- Reproduction: Haemolytic anaemia has been reported in infants whose mothers were exposed to naphthalene during pregnancy. Decreased number of live pups, embryonic mortality, preimplantation losses, and subcutaneous hematomas in foetuses have been reported in animals following oral exposure to naphthalene during gestation.
- Spleen: Reduced spleen weight has been reported in animals following subchronic exposure to naphthalene.
- Thymus: Lymphoid depletion of the thymus has been reported in animals following subchronic exposure.

Following inhalation exposure, the following have been identified as the primary target organs associated with toxicity of naphthalene (ORNL):

- Blood: Acute haemolytic anaemia, high serum bilirubin levels, Heinz bodies, and fragmentation of erythrocytes have been reported in infants exposed to naphthalene vapours from blankets or clothes that were stored in or near the infants' room. Anaemia occurred in individuals exposed to large numbers of naphthalene-containing mothballs in their homes.
- Eyes: Occupational exposure to naphthalene has been associated with cataracts, retinal haemorrhage, and chorioretinitis. Also reported was corneal ulceration.
- Gastrointestinal tract: Vomiting and abdominal pain has been reported in individuals exposed to large numbers of naphthalene-containing mothballs in their homes.
- Nervous system: Nausea, headache, malaise, and confusion has been reported in individuals exposed to large numbers of naphthalene-containing mothballs in their homes.
- Liver: Jaundice was reported in individuals exposed to large numbers of naphthalene-containing mothballs in their homes.
- Kidneys: Renal disease (not specified) was reported in individuals exposed to large numbers of naphthalene-containing mothballs in their homes.

Acute (short-term) exposure of humans to naphthalene by inhalation, ingestion, and dermal contact is associated with haemolytic anaemia, damage to the kidneys, and, in infants, brain damage. Symptoms of acute exposure include headache, nausea, vomiting, diarrhoea, malaise, confusion, anaemia, jaundice, convulsions, and coma. Other acute effects include cataracts (reported in workers acutely exposed to naphthalene by inhalation and ingestion). Tests involving acute exposure of animals, have demonstrated naphthalene to have moderate to high acute toxicity from ingestion and low to moderate acute toxicity from dermal exposure.

Genotoxic Effects

Naphthalene has been tested for genotoxicity in a variety of *in vitro* and *in vivo* genotoxicity assays. Review of available data from genotoxicity studies by OEHHA (2004) indicate that naphthalene has not been shown to cause gene mutations, but has been demonstrated to cause chromosomal damage and may cause DNA damage. Review of genotoxicity by the UK (2003) and the EU (2003) indicate that the balance of evidence indicates that naphthalene is not genotoxic.

Carcinogenicity

Human data are insufficient with regard to the evaluation of carcinogenicity of naphthalene. Testing in animals has indicated that naphthalene was carcinogenic to rats and mice following inhalation exposure. Review of these studies by the UK (2003) and the EU (2003) indicate that the tumours observed following inhalation exposure did not arise by a direct genotoxic mechanism. On this basis, use of a non-threshold approach was not considered appropriate in the quantification of risk associated with naphthalene.

TOXICITY CLASSIFICATION

Naphthalene is classified as a "possible" human carcinogen (Category C) by the USEPA for all routes of exposure based upon limited evidence from animal studies.

IARC (review in 2002) has classified naphthalene in Group 2B (possibly carcinogenic to humans) based on inadequate evidence in humans but sufficient evidence in animals.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (2004 and earlier publications) have not established guidelines for naphthalene. Hence no ADI or other relevant toxicity value is available from these sources.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For naphthalene, the following have been established:

TWA: 10 ppm, equivalent to 52 mg/m³

STEL: 15 ppm, equivalent to 79 mg/m³

WHO

The WHO Drinking Water Guidelines (2004 and earlier publications) have not established guidelines for naphthalene. Hence no ADI or other relevant toxicity value is available from these sources.

The WHO (2000) has provided guidance on inhalation exposures to PAHs. However the guidance is focused on potential exposure to benzo(a)pyrene in the basis of non-threshold effects and use of toxicity equivalent factors for all other PAHs, including naphthalene where a TEF of 0.001 has been suggested.

EU

The EU has prepared a review of naphthalene (EU, 2003) that reviews occupational, consumer and environmental exposures. For the key health effect of haemolytic anaemia, repeated inhalation toxicity and carcinogenicity have been identified, however no NOAEL could be identified from available data. For other effects associated with inhalation exposure such as tissue damage a NOAEL could not be identified from available studies. A LOAEL of 5 mg/m³ has been identified on the basis of nasal lesion in rate for use in the risk characterisation for repeated inhalation toxicity including carcinogenicity. There are no concerns for irritation, sensitisation, mutagenicity or effects on reproduction.

US

The USEPA (1998) has evaluated exposures to naphthalene and established the following:

- A chronic oral reference dose (RfD) of 0.02 mg/kg/day based on a NOAEL (of 71 mg/kg/day and uncertainty factor of 3000) established for terminal body weight decrease in rats (confidence in RfD: low).
- An inhalation reference concentration (RfC) of 0.003 mg/m³ based on a LOAEL (9.3 mg/m³ and uncertainty factor of 3000) associated with nasal effects in a chronic mouse study (confidence in RfC: medium).
- Using the 1996 Proposed Guidelines for Carcinogen Risk Assessment, the human carcinogenic potential of naphthalene via the oral or inhalation routes "cannot be determined" at this time based on human and animal data; however, there is suggestive evidence (observations of benign respiratory

tumours and one carcinoma in female mice only exposed to naphthalene by inhalation. Additional support includes increase in respiratory tumours associated with exposure to 1-methylnaphthalene. At the present time the mechanism whereby naphthalene produces benign respiratory tract tumours are not fully understood, but are hypothesized to involve oxygenated reactive metabolites produced via the cytochrome P-450 mono-oxygenase system. However, based on the many negative results obtained in genotoxicity tests, a genotoxic mechanism appears unlikely. No quantitative cancer risk values have been derived for oral or inhalation exposures.

The ATSDR establish Minimal Risk Levels (MRLs) for the evaluation of non-cancer effects. The TSDR (2005) have derived the following MRLs for naphthalene:

- Chronic inhalation MRL of 0.0007 ppm (0.003 mg/m³) based on a LOAEL for nasal lesions in rats (LOAEL_[human equivalent concentration]= 0.2 ppm), and a total uncertainty factor of 300 (10 for the use of a LOAEL, 3 for extrapolation from animals to humans using dosimetric adjustment, and 10 for human variability);
- An acute and intermediate oral MRL of 0.6 mg/kg/day for s based on a minimal LOAEL of 50 mg/kg/day for clinical signs of toxicity in pregnant rats and a total uncertainty factor of 90 (3 for the use of a minimal LOAEL, 10 for extrapolation from animals to humans, and 3 for human variability). The acute-duration oral MRL of 0.6 mg/kg/day is also adopted as the intermediate-duration oral MRL for naphthalene.

The California Office of Environmental Health Hazard Assessment (OEHHA) provided a review of naphthalene (August 2004) where an inhalation unit risk value of 3.4×10^{-5} (µg/m³)⁻¹ and a slope factor of 0.12 (mg/kg/day)⁻¹ was derived based on data for the incidence of nasal respiratory epithelial adenomas and nasal olfactory epithelial neuroblastoma in male rats (inhalation study). The derivation of cancer risk values was undertaken following the classification of naphthalene as a possibly carcinogenic to humans by IARC in 2002.

SUGGESTED TOXICITY VALUES FOR RISK CHARACTERISATION

Background Intake

For common contaminants, intakes from background sources such as food, water and/or air must also be considered in the evaluation and use of the ADI, TDI, GV or RfD in assessing potential exposures to site related chemicals. With respect to naphthalene, intakes from soil, water and food can be considered to be less significant. ATSDR estimated the average daily intake for urban/suburban areas to be 19 µg based on an average air concentration of 0.95 µg/m³. Data from Australian cities (DEH, 1999) indicate the highest annual average concentration of naphthalene of 0.058 µg/m³. Similarly data from Brisbane (Berko, 1999) indicates average urban air concentrations of naphthalene of 0.075 µg/m³. These data from Australia are approximately 10 times lower than used in the ATSDR estimate. Daily intake from all background sources has been estimated by the UK (2003) as 7 µg/day for a 70 kg adult (0.0001 mg/kg/day) from oral sources and 2.8 µg/day (0.00004 mg/kg/day) for an adult from inhalation sources. Intake associated with potential background concentrations is low (negligible for oral intakes), however there is the potential for

inhalation intakes from background sources to comprise 3% to 5% (excluding any consideration of intakes associated with cigarette smoking) of the adopted threshold value (refer below). For the purpose of providing a reasonable value for potential background intakes, a value of 5% has been adopted.

Toxicity Values

Toxicity data relevant for use in the characterisation of risk to human health have been selected for naphthalene following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	RfD = 0.02 mg/kg/day (USEPA)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation RfC = 0.003 mg/m ³ (USEPA and ATSDR), equivalent to an intake of 0.00086 mg/kg/day. Occupational inhalation exposure (NOHSC): TWA: 10ppm = 52 mg/m ³ STEL: 15ppm = 79 mg/m ³
Background	Low, 5%

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Appendix C

Methodology for Quantifying Exposure

Appendix C

Methodology for Quantifying Exposure

C.1 Introduction

The following presents the general methodology adopted for the calculation of chemical intake via inhalation, ingestion and dermal exposure pathways. Most equations are derived from USEPA (1989).

C.2 Intakes via Ingestion

The assessment of ingestion exposures requires evaluation of a number of pathways that can loosely be grouped into the ingestion of water and the ingestion of soils and sediments.

C.2.1 Ingestion of Water

The following equation is used to calculate intake of chemicals via the ingestion of water pathways:

$$\text{Daily Chemical Intake}_{IW} = C_w \cdot \frac{IR_w \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

where:

- C_w = Concentration of chemical in water (as relevant for each pathway assessed) (mg/L)
- IR_w = Ingestion rate of water (dependant on age and activity and may be derived using ingestion rate per hour and number of hours undertaking activity or simply an intake per day) (L/day)
- B = Bioavailability or absorption of chemical via ingestion assumed to be 1 or 100% unless noted otherwise (unitless)
- EF = Exposure frequency (days/year)
- ED = Exposure duration (years)
- BW = Body weight (dependant on age) (kg)
- AT = Averaging time for threshold and non-threshold exposures (days)

C.2.2 Ingestion of Soils and Sediments

The following equation is used to calculate intake of chemicals via the ingestion of soils and sediment pathways:

$$\text{Daily Chemical Intake}_{IS} = C_s \cdot \frac{IR_s \cdot FI \cdot B \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

where:

- C_s = Concentration of chemical in soil or sediment (as relevant for each pathway assessed) (mg/kg)
- IR_s = Ingestion rate of soil or sediment (dependant on age and activity) (mg/day)
- FI = Fraction of daily ingestion that is derived from contamination source assumed to be 1 or 100% unless noted otherwise (unitless)
- B = Bioavailability or absorption of chemical via ingestion assumed to be 1 or 100% unless noted otherwise (unitless)
- CF = Conversion factor of 1x10⁻⁶ to convert mg to kg
- EF = Exposure frequency (days/year)
- ED = Exposure duration (years)
- BW = Body weight (dependant on age) (kg)
- AT = Averaging time for threshold and non-threshold exposures (days)

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C.3 Intakes via Dermal Exposures

The assessment of dermal exposures involved quantification of intakes from soils or sediments and water (where relevant).

C.3.3 Mechanisms of Dermal Absorption

Passive diffusion, as governed by Fick's First Law, is considered to be the main process whereby chemicals enter and permeate through the skin. The human skin is the largest organ of the body and it consists of a thin (approximately 100 µm) epidermal layer superimposed on a thick dermal layer (approximately 300-400 µm). The epidermis consists of four layers, the outermost layer being the stratum corneum (SC) (approximately 10-40 µm), which overlays the strata lucidum, granulosum, and germinativum (Figure 1). The SC layer is composed of flat highly keratinised squamous cells that are nonviable and are thought to maintain the barrier properties of the skin. If the SC layer is removed by tape stripping, for example, the permeability of the skin to chemicals increases dramatically.

- Factors that have an effect on the dermal absorption of chemicals through the skin include:
- Skin hydration with several studies indicating that skin hydration may increase skin permeability;
- Thickness of the SC and anatomic regions with higher absorption expected in areas unprotected by thick SC layers;
- Lipophilicity of chemical. The SC layer tends to be a lipid-rich milieu and hence provides a barrier to hydrophilic compounds but permits the entry of lipophilic compounds;
- Degree of sediment binding;
- The presence of surface slicks typically comprised of fatty substances (e.g., lipids from decomposed organisms and oil from petroleum contamination) which typically cover the surface of all natural bodies of water.
- Environmental factors that include water temperature, pH, turbidity, flow rate (current), and degree of solar illumination.
- Physiologic factors include genetic-related sensitivity (e.g., tendency to sunburn) and individual differences (e.g., age, the presence of skin disease, skin abrasions).

Models that are used to estimate dermal absorption of chemicals present in water and soils/sediment are simplifications of the more complex dermal absorption processes.

C.3.4 Dermal Contact with Water

The following equation (as detailed in USEPA 1989 and 1992b) is used to calculate intake of chemicals via dermal exposure to chemicals in water:

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Methodology for Quantifying Exposure

$$\text{Daily Chemical Intake}_{DW} = C_w \cdot \frac{SA_w \cdot ET \cdot K_p \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

where:

C_w = Concentration in water (mg/L)

SA_w = Surface area of body in contact with water (cm²)

ET = Exposure time in contact with water (hr/day)

K_p = Dermal permeability/absorption via skin for each chemical in water (cm/hr)

CF = Conversion factor of 1×10^{-3} to convert cm³ to L

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (dependant on age) (kg)

AT = Averaging time for threshold and non-threshold exposures (days)

K_p is a chemical-specific factor that has been derived for a wide range of chemicals. Values for chemicals evaluated have been obtained from the Risk Assessment Information System (RAIS) database available on the website (<http://risk.lsd.ornl.gov/index.shtml>) which is maintained by the Oak Ridge Operations of the US Department of Energy, as listed in the Toxicity Summaries presented in **Appendix B**.

C.3.5 Dermal Contact with Soils and Sediment

Dermal absorption of chemicals from soil or sediment depends on the area of contact, duration of contact, bond between chemical and the soil and the ability of the chemical to penetrate the skin. The assessment of dermal absorption of chemicals in sediments has followed the approach adopted for the evaluation of dermal absorption of chemicals in soils.

USEPA Approach

The USEPA (1989 and 2004) define a simple approach to the evaluation of dermal absorption associated with soil contact. This is presented in the following equation:

$$\text{Daily Chemical Intake} = C_s \cdot \frac{SAs \cdot AF \cdot ABSd \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

where:

C_s = Concentration in soil (mg/kg)

SAs = Surface area of body exposed to soil (cm²)

AF = Adherence factor, amount of soil that adheres to the skin per unit area which depends on soil properties and area of body (mg/cm² per event)

$ABSd$ = Dermal absorption fraction (unitless – refer to discussion below)

CF = Conversion factor of 1×10^{-6} to convert mg to kg

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (dependant on age) (kg)

AT = Averaging time for threshold and non-threshold exposures (days)

Dermal Absorption Fraction (ABSd)

The approach undertaken by the USEPA utilises a dermal absorption fraction typically derived from experimental studies on different chemicals. On the basis of the studies undertaken and a number of simplifications, $ABSd$ values have been recommended for a range of 10 chemicals and default values for other semi-volatile chemicals. No default is defined (in USEPA 2004) for volatile organic chemicals or

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inorganic chemicals. It is considered that, with regards to soil exposures, volatile organic chemicals would volatilise from soil on skin and should be accounted for in the assessment of inhalation exposures. For inorganics, the speciation is very important to dermal absorption and hence no generic default values have been determined.

More commonly used values for the assessment of dermal exposure to organics and inorganics utilise defaults for ABSd for organics of 0.01 and inorganics of 0.001 as presented on RAIS (Risk Assessment Information System derived from USEPA 1992), along with the few chemical-specific values available. With respect to the assessment of metals in soils, dermal absorption is considered to be negligible. The dermal ABSd values adopted for each COPC identified are presented in the Toxicity Summaries in **Appendix B**.

The value of ABSd has no consideration of exposure time. Experimental studies used to define ABSd values are associated with dermal application over 24 hours (i.e. the event is considered to be a 24 hour day by default). Due to the lack of information about the rate and relationship of absorption of chemicals through the skin over shorter exposure periods, the USEPA methodology does not recommend adjusting the ABSd to account for exposures over times less than 24 hours, rather it recommended adjusting exposure frequency and exposure duration to reflect site conditions. This approach is utilised to evaluate dermal intake per exposure event (commonly adopted as a day) and is considered to be conservative in the assessment of exposure events that are less than 24 hours in duration or multiple short-term exposure events where absorption would be "double-counted".

Further Review of Dermal Absorption

In reviewing the approach to the assessment of dermal exposure presented in source documents "The Health Risk Assessment and Management of Contaminated Sites" (CSMS, 1991, 1993, 1996, 1998 and enHealth 2002) that are referenced in enHealth (2002) it is noted that the approach suggested by Hawley (1985) has been adopted. The approach is similar to the USEPA approach presented above, with some differences with respect to the evaluation of absorption.

Hawley (1985) provides a review of dermal uptake from soils for organic chemicals based on studies of a number of compounds by humans, rats, rabbits and pigs. The absorption rates obtained for application of a pure compound (or in acetone solvent) were determined to be 11% for an adult over 24 hours. For a 12 hour contact time, the dermal absorption rate was taken to half or 6% (as rounded up from half of 11%) of the applied dose. For children a higher rate was assumed to account for more permeable skin of a young child. For children the absorption rate was taken to be twice that of the adult, or 12% of the compound on the skin over 12 hours.

Absorption of contaminants in soils or dust (and sediments) was discussed by Hawley to be limited by the physical-chemical binding to the matrix. On this basis Hawley also applied use of a factor to account for the matrix effect of absorption from soils (or relevant matrix) by comparison to the absorption study data that used the pure compound or solvent solution. The value used by Hawley (for TCDD and other organic compounds where no data is available) was 15%.

This approach is also adopted in the assessment of dermal intake of organic chemicals presented in the Dutch sediment exposure model SEDISOIL (1996) and the soil exposure model CSOIL (2001). These models express the dermal absorption of organics presented by Hawley as an absorption rate per hour. Hence for children the absorption rate is 0.01 or 1% per hour and for adults the absorption rate is 0.005 or 0.5% per hour.

This approach was generally utilised by Fitzgerald D.J (CSMS 1991) in the setting of a response level for benzo(a)pyrene where intake via skin absorption for a 2 ½ and 6 year old child utilises the dermal absorption value over 24 hours. For an adult the intake via skin absorption considers exposure to soils over 12 hours and adjusts the intake using a linear relationship for absorption. While many other response levels (in various papers in CSMS) have been established following this approach, they are typically set for a 2 ½ year old child where dermal absorption associated with soils over 24 hours is considered to be relevant. Other references where dermal absorption over shorter exposure periods than 24 hours (using the linear approach) are presented in the following:

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- Di Marco P.N. and Buckett K.J in CSMS 1996 in "Derivation of Health Investigation Levels for Beryllium and Beryllium Compounds"
- Di Marco P.N. in CSMS 1993 in "The Assessment and Management of Organochlorine Termiticides" where exposures over 24 hours (children), 8 and 10 hours (adults).
- Di Marco P.N. and Buckett K.J in CSMS 1993 in "Derivation of A Health Investigation Level for PCBs"

Following this approach dermal intake from exposure to soil and sediment is calculated using the following equation:

$$\text{Daily Chemical Intake}_{Ds} = C_s \cdot \frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

where:

C_s = Concentration in sediment (mg/kg)

SAs = Surface area of body exposed to sediment (cm^2)

AF = Adherence factor, amount of sediment that adheres to the skin per unit area which depends on soil/sediment properties and area of body (mg/cm^2 per event)

Abs = Dermal absorption rate (per hour) – based on chemicals specific information or use of default values of 0.01 per hour for children and 0.005 per hour for adults (as above)

ET = Exposure time to sediments (hours/day)

ME = Matrix effect (unitless) – dependant on the nature of dermal data available, by default 0.15 or 15% assumed (discussed above)

CF = Conversion factor of 1×10^{-6} to convert mg to kg

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (dependant on age) (kg)

AT = Averaging time for threshold and non-threshold exposures (days)

For the purpose of comparison, the assessment of dermal exposure will be presented using both methodologies with the maximum risk calculated presented in the report.

C.4 Intakes via Inhalation

The assessment of inhalation exposures presented is assumed to be associated with vapour phase contaminants. On this basis, the following equation is used to calculate intake of volatile chemicals via all inhalation pathways:

$$\text{Daily Chemical Intake}_V = C_a \cdot \frac{InhR \cdot ET \cdot FI \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

where:

C_a = Concentration of chemical in air (as relevant for each pathway assessed) (mg/m^3)

$InhR$ = Inhalation rate (dependant on age and activity) (m^3/hr)

ET = Exposure time (dependant on activity) (hr/day)

FI = Fraction inhaled from contaminated source assumed to be 1 or 100% unless noted otherwise (unitless)

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (dependant on age) (kg)

AT = Averaging time for threshold and non-threshold exposures (days)

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Concentrations in Air

The concentration of volatile COPC in air has been measured in some areas, however there are many issues identified where concentrations need to be modelled from measured soil gas or flux data, soil concentrations, groundwater concentrations or surface water concentrations. The vapour model models adopted for this assessment are noted in the main report. Details on these models are attached to this Appendix for reference. The modelling undertaken is presented in the risk calculations in **Appendix D**.

C.5 References

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Vapour Phase Partitioning and Diffusion

Introduction

The assessment of vapour migration and vapour intrusion into buildings can be undertaken using a number of different models depending of the building type considered. While the model and approach adopted for the different building types differs, the initial processes associated with partitioning from a source concentration in soil or groundwater to vapour phase (directly above the source) is the same. In addition, all the models currently used consider diffusion as a key mechanism for vapour phase transport through the subsurface. The methodology for estimating vapour diffusion is the same in each model.

The vapour phase concentration at the source can be estimated using the following relationships:

- Where soil gas data is available and relevant to the quantification of vapour intrusion, the measured soil gas concentration is considered to be the concentration at the source, with diffusion modelled through overlying soils (from point of measurement to the surface of building); and
- Where no soil gas data is available, the concentration at the source is based on theoretical partitioning from the groundwater or soil source, see below, with subsequent diffusion modelled through the overlying soils.

The following presents the equations (Johnson et al 1990¹ and Johnson and Ettinger 1991²) used to estimate the vapour phase concentration directly above the source and diffusion through overlying soils.

Vapour Phase-Partitioning

Groundwater Source

For a groundwater source, it is assumed that the vapour phase concentration directly above the groundwater is in equilibrium with the groundwater and the concentration is related to the groundwater concentration by Henry's Law:

$$C_{source} = C_{water} \cdot HL \quad (g/cm^3) \quad \dots \text{Equation VS1}$$

Where:

C_{water} = concentration in water (at top of groundwater, g/cm^3)
 HL = Henry's Law constant (unitless)

¹ Johnson, P.C., Hertz M.B. and Beyers D.L., 1990. Estimates for Hydrocarbon vapour emissions resulting from service station remediation and buried gasoline-contaminated soils. In: Petroleum Contaminated Soils, Vol. 3. Lewis Publishers, Michigan.

² Johnson, P.C. and Ettinger R.A., 1991. Heuristic model for predicting the intrusion rate of contaminant vapours into buildings. Environ. Sci. Technology, Volume 25: 1445-1452.

Vapour Phase Partitioning and Diffusion

The concentration within the vapour phase will increase proportionally with the concentration in groundwater (at the top of the groundwater table), until it reaches saturation. At some point the saturated vapour phase concentration will be reached, which is an upper limit of the vapour phase concentration. The saturated vapour phase concentration is estimated using the following relationship:

$$SVPC = \frac{VP \cdot MW}{T \cdot 62361} \quad (\text{g/cm}^3) \quad \dots \text{Equation VS2}$$

Where:

- VP = vapour pressure of the contaminant (mmHg)
- MW = molecular weight (g/mol)
- T = soil temperature (K)
- 62361 = conversion (mmHg/K* cm³/mol)

Soil Source

For a soil source, it is assumed that the vapour phase concentration directly above the soil is in equilibrium with the source and the concentration is related to the soil concentration by the following:

$$C_{source} = \frac{C_{soil} \cdot H' \cdot \rho_s}{\theta_{ws} + K_d \cdot \rho_s + H' \cdot \theta_{as}} \quad (\text{g/cm}^3) \quad \dots \text{Equation VS3}$$

where:

- C_{soil} = Concentration in soil source zone (g/g)
- H' = Henry's Law constant (unitless)
- ρ_s = Soil bulk density (g soil/cm³ soil)
- θ_{ws} = Volumetric water content in soil source zone (cm³ water/cm³ soil)
- θ_{as} = Volumetric air content in soil source zone (cm³ air/cm³ soil)
- K_d = Soil-water partition coefficient (cm³ air/g soil) = $K_{oc} \times f_{oc}$
- K_{oc} = Soil organic carbon partition coefficient, chemical specific (cm³/g)
- f_{oc} = Soil organic carbon fraction (unitless)

The equilibrium vapour phase concentration is proportional to the soil concentration up to the soil saturation limit (C_{sat}), which is calculated using the following (with the saturated vapour phase calculated using Equation VS2):

$$C_{sat} = \frac{S}{\rho_s} \cdot [H' \cdot \theta_{as} + \theta_{ws} + K_d \cdot \rho_s] \quad (\text{mg/kg}) \quad \dots \text{Equation VS4}$$

where:

- S = Pure component solubility in water (mg/L)

Vapour Phase Partitioning and Diffusion

When residual phase is present the vapour concentration is independent of the soil concentration but proportional to the mole fraction of the individual component of the residual phase mixture as below.

Vapour Phase above Free Phase (NAPL)

Where free phase or NAPL is present at the top of the groundwater or within a soil profile, the concentration of vapour directly above the NAPL is estimated using Raoult's Law:

$$C_{NAPL\ source} = \frac{x_i \cdot P_i(T_s) \cdot MW}{1000 \cdot R \cdot T_s} \quad \dots \text{Equation VS5}$$

where:

- x_i = mole fraction of chemical in NAPL (mol/mol);
- $P_i(T_s)$ = vapour pressure of chemical at average soil temperature (atm);
- M_w = molecular weight (g/mol);
- R = Universal Gas Constant, 0.08206 L (atm)mol⁻¹K⁻¹;
- T_s = Average soil temperature (°K);
- 1000 = Units conversion factor (L/ml).

Effective Diffusion

The total overall effective diffusion coefficient can be calculated for n different soil layers between the source and the enclosed floor (including the capillary fringe where relevant). This is estimated using Equation D1.

$$D_T^{eff} = \frac{L_T}{\sum_{i=1}^n L_i / D_i^{eff}} \quad \dots \text{Equation D1}$$

- L_T = separation distance between the source and the building (cm)
- L_i = thickness of the soil layer i (cm)
- D_i^{eff} = effective diffusion coefficient across soil layer i (cm²/s) – refer to Equation D2

$$D_i^{eff} = D_a \cdot \left[\frac{\theta_{ai}^{3.33}}{n_i^2} \right] + \left[\frac{D_w}{H'} \right] \cdot \left[\frac{\theta_{wi}^{3.33}}{n_i^2} \right] \quad \dots \text{Equation D2}$$

- D_a = diffusivity in air, chemical specific (cm²/s)
- θ_{ai} = soil air-filled volume of layer i (cm³/cm³)
- n_i = soil total porosity of layer i (cm³/cm³)
- = 1- ρ_b/ρ_s
- ρ_b = soil dry bulk density, (g/cm³)
- ρ_s = soil particle density, (g/cm³) - typically 2.65
- D_w = diffusivity in water, chemical specific (cm²/s)
- θ_{wi} = soil water-filled volume of layer i, (cm³/cm³)

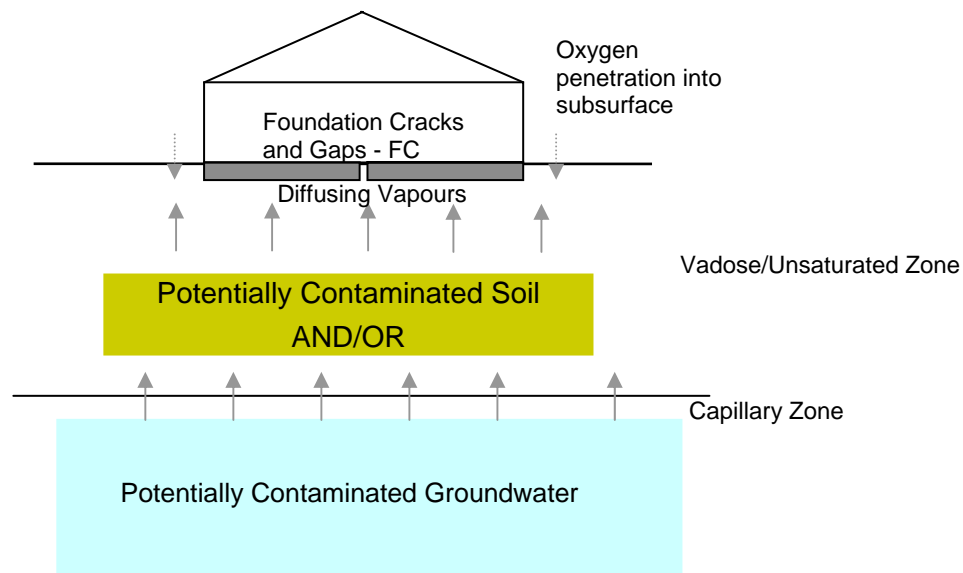
Farmer Vapour Intrusion Model

Introduction

The potential concentration of volatile chemicals inside a range of building types can be estimated using a box model as detailed by Farmer (1980¹) as modified by SD DEH (2000²). The model is simple and is relevant to a range of building types that are constructed on or above the ground. The model is not relevant for the estimation of concentrations in a building with a subsurface basement.

The model is used to assess vapour intrusion indoors only and assumed that the source is non-depleting.

Conceptual Model



Equations

The steady-state vapour-phase concentration of a contaminant inside a building (C_{building}) is calculated by using Equation F1.

¹ Farmer WJ, Yang MS, Letey J and Spencer AW, 1980. Land Disposal of Hexachlorobenzene Wastes – Controlling Vapour Movement of Soil, USEPA Report EPA-600/2-80-119.

² SD DEH, 2000. Users Guide to the Vapor Risk 2000, Version: December, 2000. San Diego County Department of Environmental Health, Land And Water Quality Division Site Assessment and Mitigation Program.

Farmer Vapour Intrusion Model

$$C_{indoors} = \frac{E_{surface} \cdot FS \cdot FC}{ER \cdot V} \quad \mu\text{g}/\text{m}^3 \quad \dots\text{Equation F1}$$

$E_{surface}$	= Emission rate measured from the surface of the ground beneath the building ($\mu\text{g}/\text{s}$), calculated using Equation F2 (or directly measured)
FS	= fraction of floor above source, unitless
FC	= fraction of emission entering through floor, unitless.
ER	= air exchange rate, (per s)
V	= volume of the building (m^3)

$$ER = \frac{C_{source} \cdot D_{eff} \cdot A_{area}}{D} \quad (\text{g}/\text{s}) \quad \dots\dots\text{Equation F2}$$

C_{source}	= estimated vapour phase concentration at source (g/cm^3) – refer to Equations VS1 to VS5 as relevant for the source assessed
D_{eff}	= effective diffusion coefficient through the vadose zone (cm^2/s), refer to Equations D1 and D2
A_{area}	= area of the emission (cm^2) - (width x length of area)
D	= depth to source (cm)

The fraction of emissions entering through the floor (FC) is also referred to as an attenuation factor that relates the concentration directly beneath the slab to that above the slab. A range of attenuation factors are relevant to buildings and include the following (SD DEH, 2000):

- Dirt floor (no slab) attenuation factor = 1.0
- Old slab attenuation factor = 0.1
- New/improved slab attenuation factor = 0.01
- Coated slab attenuation factor = 0.001 (note the use of the factor requires data to demonstrate this is achieved)
- Crawl-space attenuation factor = 0.3 (see details below)

An attenuation factor can also be used that is representative of a building constructed on piers with a crawl-space. Data from a study undertaken by Nazaroff and Doyle (1985³), which is summarised by Markey and Anderson (1996⁴) that examines the infiltration of radon into homes with crawl spaces has been reviewed. The study showed under a range

³ Nazaroff W.W. and Doyle S.M., 1985. Radon Entry Into Houses Having a Crawl Space. Health Physics Vol 48, No 3, p265-281, 1985.

⁴ Markey B. and Anderson R., 1996. Exposure Assessment and Volatilisation from Soil. In: The Health Risk Assessment and Management of Contaminated Sites, Contaminated Sites Monograph Series No. 5, 1996.

Farmer Vapour Intrusion Model

of conditions and ventilations of the crawl space that 30% to 65% of radon and a tracer gas released beneath the houses entered the indoor air environment. Utilising general data for a soundly constructed home or building with a ventilated crawl-space it can be assumed that 30% of the soil gas present within the emissions emitted from the surface of the ground (estimated from the measured soil gas data) may enter the house.

Model Assumptions

All vapour migration models are simplistic and are considered to be conservative. The general assumptions made in the above models in estimating concentrations of volatile chemicals which may migrate from a subsurface source to indoor air are:

- The contaminant is homogeneously distributed in the soil or groundwater source.
- Soil is homogeneous such that effective diffusion coefficient is constant.
- Contaminant loss from leaching downward does not occur.
- Source degradation and transformation is not considered.
- Contaminant concentration at the interface between the soil particle surface and the soil pore air space is zero.
- Soil gas migrates upwards only (one dimension).
- Convective vapour flow near the building foundation is uniform.
- Convective vapour flow rates decrease with increasing distance between the contaminant source and the building.
- Contaminant vapours enter the building through openings in the walls and foundation at or below the ground surface.
- Both the building ventilation rate and the difference in pressure between the building interior and the surrounding soil are constant.
- All contaminant vapours directly below the building will enter the building, unless the floor and walls are perfect vapour barriers.
- The building contains no other contaminant sources or sinks, and contaminant vapour dispersion is instantaneous and homogeneous.

Use of the Farmer Model as a first-tier screening tool to identify sites needing further assessment requires careful evaluation of the assumptions listed in the previous section to determine whether any conditions exist that would render the model inappropriate for the site. If the model is deemed applicable at the site, care must be taken to ensure reasonably conservative and consistent model parameters are used as input to the model. Considering the limited site data typically available in preliminary site assessments, the model can be expected to predict only whether or not a risk-based exposure level will be exceeded at the site. Precise prediction of actual concentration levels is not possible with this approach.

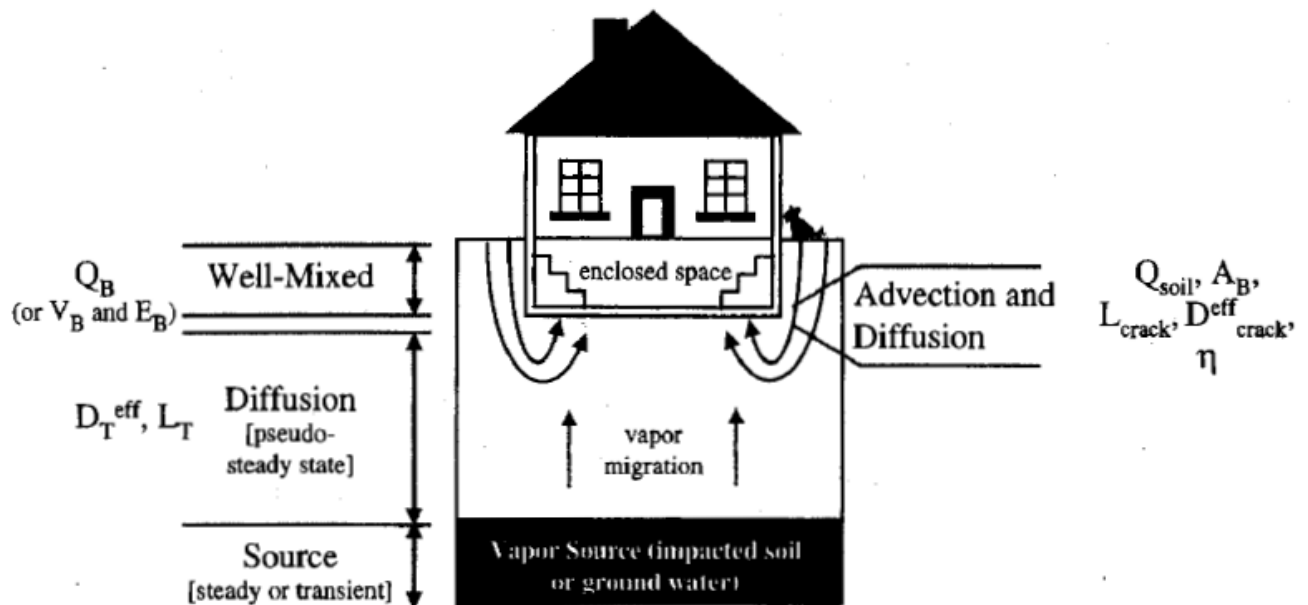
Johnson & Ettinger Vapour Intrusion Model

Introduction

The potential concentration of volatile chemicals inside a building constructed on a concrete slab with or without a sub-surface basement has been estimated using the Johnson & Ettinger Model (USEPA 2003¹). This model is consistent with the Johnson & Ettinger equations (1991²) recommended in the Soil Screening Guidelines (USEPA, 1996³) and the Risk Based Corrective Action at Petroleum Release Sites (ASTM, 2002⁴).

The model is used to assess vapour intrusion indoors only and assumed that the source is non-depleting.

Conceptual Model



(from Johnson and Ettinger 1991)

¹ USEPA, 2003. User's Guide for Evaluating Subsurface Vapor Intrusion into Buildings. June 2003.

² Johnson, P.C. and Ettinger, R.A. 1991. "Heuristic Model for Predicting the Intrusion Rate of Contaminant Vapours Into Buildings". Environmental Science and Technology, Vol 25 (8), 1991, pp.1445-1452.

³ USEPA 1996. Soil Screening Guidance. Publication 9355.4-23, July 1996

⁴ ASTM, 2002. Standard Guide for Risk-Based Corrective Action Applied at Petroleum Release Sites. ASTM E 1739-95 (2002)

Johnson & Ettinger Vapour Intrusion Model

Equations

The steady-state vapour-phase concentration of a contaminant inside a building (C_{building}) is calculated by applying the Johnson and Ettinger model assuming a steady-state mass transfer (i.e., infinite). This is calculated using Equation JE1.

$$C_{\text{indoor}} = C_{\text{source}} \cdot \alpha \quad \dots \text{Equation JE1}$$

Where

C_{indoor} = the steady-state vapor-phase concentration of a contaminant inside a building ($\mu\text{g}/\text{m}^3$)

α = attenuation coefficient [unitless], refer to Equation JE2

C_{source} = vapour concentration at the source ($\mu\text{g}/\text{m}^3$), refer to equations VS1 to VS5 (as relevant).

The attenuation factor is calculated using the following:

$$\alpha = \frac{\left[\frac{D_T^{\text{eff}} \cdot A_B}{Q_{\text{building}} \cdot L_T} \right] \cdot \exp \left[\frac{Q_{\text{soil}} \cdot L_{\text{crack}}}{D_{\text{crack}} \cdot A_{\text{crack}}} \right]}{\left[\exp \left[\frac{Q_{\text{soil}} \cdot L_{\text{crack}}}{D_{\text{crack}} \cdot A_{\text{crack}}} \right] + \left[\frac{D_T^{\text{eff}} \cdot A_B}{Q_{\text{building}} \cdot L_T} \right] + \left[\frac{D_T^{\text{eff}} \cdot A_B}{Q_{\text{soil}} \cdot L_T} \right] \cdot \exp \left[\frac{Q_{\text{soil}} \cdot L_{\text{crack}}}{D_{\text{crack}} \cdot A_{\text{crack}}} \right]^{-1} \right]} \quad \text{Equation JE2}$$

Where:

D_T^{eff} = total overall effective diffusion coefficient. Refer to Equations D1 and D2.

A_B = area of the enclosed space below the ground level which will vary depending on whether the building has a basement below the ground or not (cm^2).

Q_{building} = building ventilation rate which is calculated using building parameters and air exchange rate (cm^3/s). Refer to Equation JE3.

L_T = separation distance between the source or soil gas measurement and the building (cm).

Q_{soil} = volumetric flowrate of soil gas into the enclosed space. This represents the convective flow of vapours into a building through cracks in the floor and walls. It incorporates pressure driven flows and a default value of 5 L/min is recommended (2003), however it can be calculated using Equation JE5.

L_{crack} = enclosed space foundation or slab thickness (cm).

D_{crack} = effective diffusion coefficient through the cracks (cm^2/s).

A_{crack} = area of total cracks which varies depending on whether there is a basement or not (cm^2), refer to Equation JE4.

Johnson & Ettinger Vapour Intrusion Model

The building ventilation rate is calculated using Equation JE3 for the building dimensions representing the living space of the building. It assumes that the total air volume entering the structure is mixed and that the vapour entering the structure is instantaneously and homogeneously distributed.

$$Q_{building} = \frac{(L_B \cdot W_B \cdot H_B \cdot ER)}{3600} \quad \dots \text{Equation JE3}$$

Where:

- L_B = length of building, (cm)
- W_B = width of building, (cm)
- H_B = height of building, (cm)
- ER = air exchange rate, (per hour)
- 3600 = conversion from hours to seconds

$$A_{crack} = n \cdot AB \quad \dots \text{Equation JE4}$$

$$AB = L_B \cdot W_B + (2 \cdot L_B \cdot L_h + 2 \cdot W_B \cdot L_h)$$

Where:

- AB = area of enclosed space below ground, (cm²)
- n = ratio of crack area to total area (unitless)
- A_{crack} = total crack area, (cm²)
- L_h = depth below ground, (cm)

The volumetric flow rate of soil gas into the building is calculated using Equation JE5. This represents the advective/convective flow rate of contaminant vapours in soil surrounding the building via the cracks in the building floor and walls. It incorporates pressure driven flows into the building that may be associated with wind effects on the structure, stack effects due to heating or an unbalanced mechanical ventilation. This is of particular importance where a basement is present and where heating /ventilation effects are of significance.

Tracer testing of buildings where advection is the primary mechanism for intrusion into the building suggested a typical range for Q_{soil} from 1 to 10 L/min, with 5 L/min selected as a default by the USEPA (2003). The equation represents potential openings for soil vapour entry into a building. These openings include floor/wall joints associated with floating concrete slabs or a perimeter drain /sump system. The soil vapour permeability used is that for the type of material immediately under the slab.

Johnson & Ettinger Vapour Intrusion Model

$$Q_{soil} = \frac{2 \cdot \pi \cdot P \cdot k_v \cdot X_{crack}}{\mu \cdot \ln \left[2 \cdot \frac{Z_{crack}}{r_{crack}} \right]} \quad \dots \text{Equation JE5}$$

Where:

- P = pressure differential between the soil surface and the enclosed space, (g/cm.s²) which may range from negligible (0.001-20Pa, or 0.0001 to 2 g/cm.s²)
- k_v = soil vapour permeability, (cm²), calculated based on soil type beneath slab as per USEPA 2003
- X_{crack} = floor-wall seam perimeter, (cm)
- μ = viscosity of air, (g/cm.s)
- Z_{crack} = crack depth below ground level, (cm)
- r_{crack} = equivalent crack radius, (cm), refer to USEPA 2003 for approach.

However, for buildings constructed as slab-on-grade in climates where the potential for pressure differences to be driven by long term heating or unbalanced ventilation systems, the potential for pressure driven flows (advection) is considered negligible, consistent with the approach adopted in the ASTM guidance (2002). This results in Q_{soil} to be essentially negligible and hence the attenuation factor is simplified and can be calculated using the following (as per ASTM 2002):

$$\alpha = \frac{\left[\frac{D_T^{eff} / L_T}{ER \cdot L_B} \right]}{\left[1 + \left[\frac{D_T^{eff} / L_T}{ER \cdot L_B} \right] + \left[\frac{D_T^{eff} / L_T}{(D^{crack} / L^{crack}) \cdot \eta} \right] \right]} \quad \text{Equation JE6}$$

Where:

- D_T^{eff} = total overall effective diffusion coefficient. Refer to Equations D1 and D2.
- L_B = enclosed-space volume: infiltration area ratio (cm).
- ER = enclosed-space air exchange rate (1/sec).
- L_T = separation distance between the source or soil gas measurement and the building (cm).
- L_{crack} = enclosed space foundation or slab thickness (cm).
- D_{crack} = effective diffusion coefficient through the cracks (cm²/s).

Johnson & Ettinger Vapour Intrusion Model

Model Assumptions

The following represent the major assumptions/limitations of the J&E Model.

1. Contaminant vapours enter the structure primarily through cracks and openings in the walls and foundation.
2. Convective transport occurs primarily within the building zone of influence and vapour velocities decrease rapidly with increasing distance from the structure.
3. Diffusion dominates vapour transport between the source of contamination and the building zone of influence.
4. All vapours originating from below the building will enter the building unless the floors and walls are perfect vapour barriers.
5. All soil properties in any horizontal plane are homogeneous.
6. The contaminant is homogeneously distributed within the zone of contamination.
7. The aerial extent of contamination is greater than that of the building floor in contact with the soil.
8. Vapour transport occurs in the absence of convective water movement within the soil column (i.e., evaporation or infiltration), and in the absence of mechanical dispersion.
9. The model does not account for transformation processes (e.g., biodegradation, hydrolysis, etc.).
10. The soil layer in contact with the structure floor and walls is isotropic with respect to permeability.
11. Both the building ventilation rate and the difference in dynamic pressure between the interior of the structure and the soil surface are constant values.

Use of the J&E Model as a first-tier screening tool to identify sites needing further assessment requires careful evaluation of the assumptions listed in the previous section to determine whether any conditions exist that would render the J&E Model inappropriate for the site. If the model is deemed applicable at the site, care must be taken to ensure reasonably conservative and self-consistent model parameters are used as input to the model. Considering the limited site data typically available in preliminary site assessments, the J&E Model can be expected to predict only whether or not a risk-based exposure level will be exceeded at the site. Precise prediction of concentration levels is not possible with this approach.

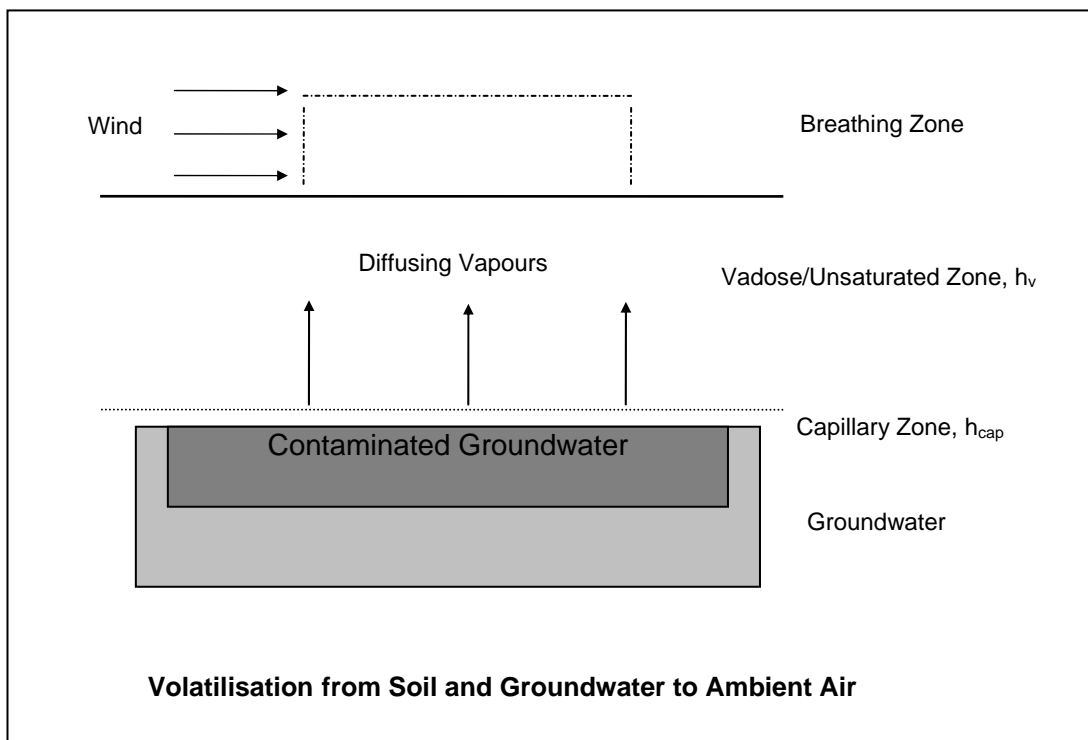
Outdoor Air and Excavations

Introduction

There are a number of models available for estimating potential concentrations of chemicals within the outdoor air environment associated with the migration from a subsurface source. Limited guidance is available for the estimation of concentrations in an excavation, hence the outdoor model adopted has also been utilised for calculations of concentrations within an excavation. The estimation of concentrations in outdoor air can be undertaken using two different methodologies outlined in the Soil Screening Guidelines (USEPA, 1996¹) and the Risk Based Corrective Action at Petroleum Release Sites (ASTM, 2002²).

The model is used to assess vapour intrusion indoors only and assumed that the source is non-depleting.

Conceptual Model



¹ USEPA 1996. Soil Screening Guidance. Publication 9355.4-23, July 1996

² ASTM, 2002. Standard Guide for Risk-Based Corrective Action Applied at Petroleum Release Sites. ASTM E 1739-95 (2002)

Outdoor Air and Excavations

Equations

The relevant equations associated with the estimation of outdoor air concentrations based on the approach outlined in the USEPA document "Soil Screening Guidance" (1996 and Supplement 2001 Exhibit D-3). This model uses air dispersion models to provide an estimate of potential dispersion of emissions above the ground as presented below.

$$C_o = \frac{J_s}{Q/C \cdot 10^{-9}} \quad \dots \text{Equation O1}$$

Where:

- C_o = Outdoor air concentration ($\mu\text{g}/\text{m}^3$)
 J_s = Contaminant flux from the surface of the ground (measured) ($\text{g}/\text{s}/\text{m}^2$)
 Q/C = Dispersion term calculated for area ($\text{g}/\text{s}/\text{m}^2$ per kg/m^3)
 10^{-9} = Units conversion from kg/m^3 to $\mu\text{g}/\text{m}^3$

$$Q/C = 11.91 \cdot \exp\left(\frac{(\ln(\text{Acres}) - 18.4385)^2}{209.7845}\right) \quad \dots \text{Equation O2}$$

Where:

- Q/C = Dispersion term calculated for area ($\text{g}/\text{s}/\text{m}^2$ per kg/m^3) based on climates similar to Los Angeles which is considered relevant for much of Australia, however for other areas, relevant parameters are selected.
 Acres = Area of the source outside (acres)

A simpler approach more commonly used for small subsurface sources is the outdoor model presented in the ASTM (2002) guidance. Outdoor air concentrations have been estimated using a simple box model, which accounts for some atmospheric mixing. The concentration of volatile contaminants within the breathing zone of outdoor air has been estimated using Equation O3.

$$C_{\text{outdoor}} = C_s \cdot VF \quad (\text{mg}/\text{m}^3) \quad \dots \text{Equation O3}$$

Where:

- C_s = concentration at the source (mg/m^3)
 VF = volatilisation factor calculated for emissions from the source to air, refer to Equation O4.

As noted with the indoor air model, the vapour phase concentration at the source can be estimated using the following relationships:

- Where soil gas data is available and relevant to the quantification of vapour migration, the measured soil gas concentration is considered to be the concentration at the source, with migration modelled through overlying soils (from point of measurement to the surface); and

Outdoor Air and Excavations

- Where no soil gas data is available, the concentration at the source is based on theoretical partitioning from the groundwater or soil source, as presented in Equations VS1 to VS5 (as required).

The volatilisation factor is calculated using the following:

$$VF = \frac{D_s^{eff} \cdot W}{U_{air} \cdot \delta \cdot L_{GW}} \quad \dots \text{Equation O4}$$

where:

- U_{air} = Wind speed above the ground surface in the ambient mixing zone (cm/s)
- δ_{air} = Ambient air mixing zone height (cm)
- L_{GW} = Depth to groundwater (= height of capillary zone, h_{cap} , + height of unsaturated zone, h_v) (cm)
- W = Width of source area parallel to wind or groundwater flow direction (cm) (i.e. width and breadth of breathing zone)
- D_{ws}^{eff} = Effective diffusion coefficient between the groundwater and soil surface (cm^2/s), refer to Equations D1 and D2.

Emissions into Excavation or Trench

Volatile COPC have the potential to accumulate within trenches or excavations in areas where excavations intersect or are located directly above contaminated soil or groundwater. Workers have the potential to be exposed to these COPC when working in or near the trench or excavation. It is unlikely that workers would spend an entire workday within any excavation or trench, and any exposure near the trench or excavation would result in exposure to significantly lower concentrations due to dilution.

Concentrations within an excavation have been estimated using the ASTM (2002) outdoor air model presented above, however the depth to the source is adjusted to reflect to depth from the base of the excavation to the source, the dimensions of the excavation are used and the wind speed is adjusted to reflect a more confined space scenario. A typical excavation is estimated as 1m x 10m x 1 to 1.5m depth (ANZECC 1992³ notes the depth of most services is between 1 to 2m below ground surface). A wind speed considered representative of a more confined space within an excavation is 0.5 m/s.

Where groundwater seeps into an excavation, concentrations of volatile chemicals in groundwater that could be inhaled during excavation work can be estimated using an upper-bound volatilization factor (VF). The VF is based on workers in trenches flooded with groundwater off-gassing volatile organic compounds (VOCs). A methodology developed by the

³ ANZECC 1992. Australian and New Zealand Guidelines for the Assessment and Management of Contaminated Sites. Australian and New Zealand Environment and Conservation Council, National Health and Medical Research Council, January 1992.

Outdoor Air and Excavations

USEPA has been used to estimate a VF from water (VF_w) (USEPA 1999⁴). The EPA method examines the mass of a chemical that could be transferred from water to air and assumes:

$$VF_w \left(\frac{l}{m^3} \right) = \frac{(k_{lg})}{(k) \left(\frac{\mu}{L} \right) (H)} \cdot \left(\frac{1000l}{m^3} \right) \quad (\text{mg}/\text{m}^3 \text{ air})/(\text{mg}/\text{L water})$$

where:

k_{lg} = a conservative estimate of the overall mass transfer coefficient from the liquid phase to the gas phase of 3.0×10^{-6} m (USEPA 1999)

L = an average trench length of up to 30 meters (USEPA 1999)

H = an average trench depth of 3 meters (USEPA 1999)

μ = average wind speed of 1 mph (0.45 m/sec) over a year's time (USEPA 1999)

L = air changes per day in the trench of 0.015/sec, assuming the wind flow is in the direction of the trench (USEPA 1999)

k = an air mixing rate between trench air and ambient air of 50 percent; uniform mixing of air occurs in the trench (USEPA 1999)

Using these assumptions, the USEPA's default, upper-bound volatilization factor (VF_w) is 0.133 litres per cubic meter (L/m^3) has been adopted (USEPA 1999). The VF_w is applied directly to the relevant groundwater concentrations assumed to seep into an excavation to estimate an air concentration in the flooded trench.

⁴ United States Environmental Protection Agency (USEPA) 1999. *Derivation of a volatilization factor to estimate upper bound exposure point concentration for workers in trenches flooded with groundwater off-gassing volatile organic chemicals*. Region 8. Ref: 8EPR-PS. July 29, 1999

Vapour Emissions and Concentrations from Surface Water

Introduction

The modelling of emissions from surface water as an open pond, lagoon (or pool) or river (drain or creek) has been undertaken utilising the models presented in USEPA Air Emissions Model for Waste and Wastewater (WATER9 2001¹). This model supersedes WATER8 (USEPA, 1994²). WATER8 is the emissions model recommended for estimating emissions to air from wastewater treatment collection and storage systems by Environment Australia (EA, 1999³). It is noted that the equations relevant to the assessment of emissions from a pond (or lagoon) have not changed in the updated model.

The modelling of emissions from such a water body assumes that the environment is non-aerated, non-biological and the surface is quiescent (i.e. non-turbulent) for most of the time.

The assessment of rivers (drains and creeks) has been undertaken using the equations presented for an open drain or trench. The open sections of the creek are assumed to be similar to an open drain assuming that the water that flows through the drain is exposed to the wind at the water surface. Emissions from open drain systems are dominated by volatilisation from the surface of the water. Volatilisation occurs when molecules of a dissolved substance escape into an adjacent gas phase in an attempt to reach equilibrium between liquid and vapour phases. The driving force in non-turbulent liquids is molecular diffusion. It has been assumed that water flowing in the creek is non-turbulent as most of the time the water flow in the drain is considered to be non-turbulent. Turbulent surfaces will result in a higher mass transfer to the air and generally a higher emission from the surface of the water.

Emission Rate from Surface

The rate of volatilisation of chemicals in water (or wastewater) can be estimated using mass transfer theory that incorporates two resistances to mass transfer in series, the liquid-phase resistance and the gas-phase resistance. The individual mass transfer coefficients depend on the individual COPC properties and the system parameters. The following equations have been used to calculate the emission rate from the water surface (USEPA 1994 and EA 1999).

$$E = -\frac{\partial C}{\partial t} = K \cdot A \cdot c$$

.....Equation P1

where:

E = emission rate of chemical from the surface (g/s)

C = concentration of chemical in liquid (g/m³)

K = overall mass transfer coefficient (m/s), refer to Equation P2

A = liquid surface area (m²)

t = time (s)

¹ USEPA WATER9, 2001. Users Guide for WATER9 Software, Office of Air Quality Planning and Standards, USEPA, February 2001.

² USEPA WATER8, 1994. Air Emissions Models for Waste and Wastewater, WATER8, EPA-453/R-94-080A, November 1994.

³ EA, 1999. National Pollutant Inventory, Emission Estimation Technique Manual for Sewage and Wastewater Treatment. Environment Australia 1999.

Vapour Emissions and Concentrations from Surface Water

$$K = \left[\frac{1}{k_L} + \frac{1}{k_g \cdot k_{eq}} \right]^{-1}$$

.....Equation P2

where:

k_L = liquid-phase mass transfer coefficient (m/s), refer to following sections for calculation

k_g = gas-phase mass transfer coefficient (m/s), refer to following sections for calculation

k_{eq} = equilibrium constant or partition coefficient

= $H/(RT)$

H = Henry's Law Constant ($\text{atm}\cdot\text{m}^3/\text{g}\cdot\text{mol}$)

R = universal gas constant = 8.21×10^{-5} ($\text{atm}\cdot\text{m}^3/\text{g}\cdot\text{mol}\cdot\text{K}$)

T = temperature of water (K)

Mass Transfer Rates

The gas-phase mass transfer coefficient for all surface water bodies considered is calculated using the following equation developed from experiments on evaporation of isopropyl benzene, gasoline and water to air:

$$k_g = 4.82 \times 10^{-3} \cdot U_{10}^{0.78} \cdot Sc_G^{-0.67} \cdot d_e^{-0.11} \quad (\text{m/s})$$

.....Equation P3

where:

k_g = gas-phase mass transfer coefficient (m/s)

U_{10} = windspeed at 10m height above the surface (m/s)

Sc_G = Schmidt number on gas side

= $\mu_G/(\rho_G \cdot Da)$

μ_G = viscosity of air = 1.81×10^{-4} g/cm/s

ρ_G = density of air = 1.2×10^{-3} g/cm³

Da = diffusivity of chemical in air (cm²/s)

d_e = effective diameter of water body (m)

= $2(A/\pi)^{0.5}$

A = area of water body (m²)

4.82×10^{-3} = empirical constant (m/s (m/s)^{-0.78}(m)^{0.11})

The liquid-phase mass transfer coefficient for **ponds and lagoons** is calculated using an equation which depends on the ratio between the fetch (linear distance across water body, F) to depth (D) ratio and the wind speed at 10m height. Based on these values one of the following equations is applicable:

$$k_L = (2.78 \times 10^{-6}) \cdot \left(\frac{Dw}{De} \right)^{2/3} \quad [0 < U_{10} < 3.25 \text{ m/s, all F/D ratios}] \quad \text{.....Equation P4}$$

Vapour Emissions and Concentrations from Surface Water

$$k_L = (2.605 \times 10^{-9} \cdot (F/D) + 1.277 \times 10^{-7}) \cdot U_{10}^2 \cdot \left(\frac{D_w}{D_e} \right)^{2/3}$$

$$[U_{10} > 3.35 \text{ m/s}, 14 < F/D < 51.2]$$

.....Equation P5

$$k_L = (2.611 \times 10^{-7}) \cdot U_{10}^2 \cdot \left(\frac{D_w}{D_e} \right)^{2/3}$$

$$[U_{10} > 3.25 \text{ m/s}, F/D > 51.2]$$

.....Equation P6

where:

k_L = liquid-phase mass transfer coefficient (m/s)

U_{10} = windspeed at 10m height above the surface (m/s)

D_w = diffusivity of chemical in water (cm^2/s)

D_e = diffusivity of ether in water = 8.5×10^{-6} (cm^2/s)

2.611×10^{-7} = empirical constant (per (m/s))

The liquid-phase mass transfer coefficient for **rivers (drains and creeks)** is calculated using an equation which was derived from a number of field studies on rivers and streams (as presented in WATER9, equations derived from Owens Model).

$$k_L = 7.62 \times 10^{-5} \cdot U_w^{0.67} \cdot d^{-0.85} \cdot \Psi \quad (\text{m/s})$$

.....Equation P7

where:

k_L = liquid-phase mass transfer coefficient (m/s)

U_w = water flow rate (ft/s)

d = depth of flow (ft)

Ψ = ration of mass transfer coefficient to oxygen ($D_w(\text{VOC})/D_{\text{oxygen}}$), where $D_{\text{oxygen}} = 0.000025$ cm^2/s

7.62×10^{-5} = conversion of units such that K_L is expressed in m/s

Concentration in Air

A simple box model has been used in estimating concentrations in air within the breathing zone above the water surface which may be available for inhalation during wading and swimming activities. The model assumes that emissions of COPC from the surface of the water are mixed with ambient air in a zone above the surface, known as a mixing zone. The concentration in air can be calculated using the following equation:

Vapour Emissions and Concentrations from Surface Water

$$C_b = \frac{ER}{H \cdot R}$$

.....Equation P8

where:

C_b = concentration in breathing zone (g/m³)

ER = emission rate of chemical from the surface (g/m²/s), this is the emission rate (E) calculated as above per unit area (E/A)

H = height of mixing zone (m),

R = rate of air exchange (1/s), this is the time taken for air to travel through the box assumed for the purpose of mixing. This is the wind-speed near the surface of the water divided by the distance across the mixing zone. In the case of a swimmer, the head will be close to the water surface and the mixing zone can be assumed to be small, say 1mx1m in area.

Key Modelling Parameters and Calculations

Calculations undertaken for the estimation of air emissions and concentrations associated with the presence of volatile COPC in surface water within a pond, pool or lagoon are included in **Appendix D**.

Appendix D

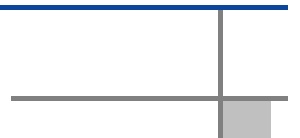
Risk Calculations

Appendix D

Risk Calculations

A.1 Calculations Relevant for Table 5-2 in Main Report

Note vapour models referenced in other calculations and tables are presented in this section only.



Inhalation of Volatile Chemicals - Emissions from Other Sources on BIP

Stage 1 and Stage 2 Development

General Data/ Equations		Units	Exposure Calculations				
			Indoors/Outdoors - Workers on Site (Tables 5-2 and 5-5, Scenario 1)				
Exposure Parameters							
Exposure Frequency (EF)		days/year	240	Work for 5 days per week for 48 weeks			
Exposure Duration (ED)		years	30	Working life at the one location			
Body Weight (BW)		kg	70	USEPA 1989 and CSMS 1996			
Averaging Time - NonThreshold (ATc)		days	25550	USEPA 1989 and CSMS 1996			
Averaging Time - Threshold (ATn)		days	10950	USEPA 1989 and CSMS 1996			
Inhalation Rate (InhR)		m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)			
Exposure Time (ET)		hours/day	10	Assume 9 hours indoors and 1 hr outdoors			
Bioavailability (B)		-	100%	Assume 100% bioavailability via inhalation			
Fraction Inhaled (FI)		-	100%	Assume all time spent on the site			
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$		m ³ /kg/day	5.1E-02	NonThreshold			
			1.2E-01	Threshold			
<i>Daily Intake = Concentration in Air x Intake Factor</i>							
<i>NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor</i>							
<i>Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)</i>							
Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		1.5E-9	4.7E-3
mercury (as total, inorganic)		2.2E-04	1.60E-06	8.2E-08	1.9E-07	--	8.8E-4
vinyl chloride (adult exposures)	1.5E-02		1.10E-06	5.6E-08	1.3E-07	8.7E-10	--
1,1-dichloroethene		5.7E-02	1.10E-07	5.6E-09	1.3E-08	--	2.3E-7
trans-1,2-dichloroethene		1.7E-02	7.40E-09	3.8E-10	8.8E-10	--	5.2E-8
1,1-dichloroethane		1.4E-01	5.50E-09	2.8E-10	6.6E-10	--	4.6E-9
cis-1,2-dichloroethene		1.0E-02	6.10E-08	3.1E-09	7.3E-09	--	7.3E-7
1,2-dichloroethane (EDC)	9.8E-03		8.40E-07	4.3E-08	1.0E-07	4.2E-10	--
trichloroethene (TCE)	1.5E-03		2.60E-06	1.3E-07	3.1E-07	2.0E-10	--
1,1,2-trichloroethane		4.0E-03	1.40E-08	7.2E-10	1.7E-09	--	4.2E-7
tetrachloroethene (PCE)		3.8E-02	4.10E-04	2.1E-05	4.9E-05	--	1.3E-3
chloroform	1.5E-03	2.0E-02	5.40E-07	2.8E-08	6.5E-08	4.1E-11	3.2E-6
dichloromethane		2.3E-01	4.60E-08	2.4E-09	5.5E-09	--	2.4E-8
hexachlorobutadiene (HCBD)		2.0E-04	1.00E-06	5.1E-08	1.2E-07	--	6.0E-4
hexachlorobenzene (HCB)		1.6E-04	6.10E-09	3.1E-10	7.3E-10	--	4.6E-6
hexachloroethane (HCE)		1.0E-03	1.60E-05	8.2E-07	1.9E-06	--	1.9E-3

↑
Modelled ground level concentrations - emissions from BIP including CPWE remediation
Concentrations considered presented in Table 3-11

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS

Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 1 Southlands - Average

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.5	Calculated from layers
Soil Temperature	[C]	T	17	Assumed
Vadose Zone Layer 1 Characteristics				
Depth of Layer 1	[m]	vd1	0.5	<i>Fill</i> Assume 0.5m fill (and soil gas data relevant for 0.5m depth)
Moisture Content	[g/g]	mocon	0.08	Conservative value for soil materials
Organic Carbon Fraction	-	foc	0.003	Assumed for fill
Soil Bulk Density	[g/ml]	rhob	1.7	Assumed for fill
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.36	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.136	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.222	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	emarea	625.0	Assumed size of building/room in area (25mx25m)
Height of Room	[m]	boxh	3	Assumed for commercial/industrial building
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum Australian Std for Commercial Bldgs
Air Exchange Rate	[exch/s]	er	0.00056	Calculated
Floor Attenuation Factor	-	faf	0.1	Conservative value for range of commercial bldgs
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	Assumed area contributing to outdoor air concentration
Width of Contaminated Area	[m]	width	20	Assumed area contributing to outdoor air concentration
Outdoor Area	[m ²]	oarea	400	calculated
Q/C Dispersion term	[g/m ² /s per kg/m ³]	qc	9.28E+01	Calculated for climate similar to East coast of Australia

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
carbon tetrachloride	800	153.8	170	0.078	8.8E-06	91.3	1.2
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Capillary Fringe (cm ² /s)	Total Effective Diffusion (GW to surface) (cm ² /s)
vinyl chloride (adult exposures)	5.50E-3			5.50E-3
1,2-dichloroethane (EDC)	5.40E-3			5.40E-3
trichloroethene (TCE)	4.10E-3			4.10E-3
tetrachloroethene (PCE)	3.74E-3			3.74E-3
chloroform	5.40E-3			5.40E-3
carbon tetrachloride	4.05E-3			4.05E-3
dichloromethane	5.25E-3			5.25E-3

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS

Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 1 Southlands - Average

Calculated Air Concentrations ASTM Guidance	Calculation Based on Measured Soil Gas			Measured Emission Rate from Surface of Ground - Average (g/s/m ²)	Concentration within Building (mg/m ³)	Outdoor Air Concentration Q/C (mg/m ³)
	Vapour Phase Concentration at Source (ug/m ³)	Vapour Phase Concentration at Source (g/cm ³)	Calculated Emission Rate from Surface of Ground (g/s/m ²)			
benzene				9.5E-10	5.7E-05	1.0E-05
toluene				4.4E-09	2.7E-04	4.8E-05
ethylbenzene				3.4E-10	2.1E-05	3.7E-06
xylene				1.7E-09	1.0E-04	1.8E-05
vinyl chloride (adult exposures)	200	2.0E-10	2.2E-10	2.0E-08	1.2E-03	2.2E-04
trans-1,2-dichloroethene				3.0E-09	1.8E-04	3.2E-05
cis-1,2-dichloroethene				1.1E-08	6.3E-04	1.1E-04
1,2-dichloroethane (EDC)				1.2E-08	7.3E-04	1.3E-04
trichloroethene (TCE)	91	9.1E-11	7.5E-11	1.2E-08	7.3E-04	1.3E-04
1,1,2-trichloroethane				1.4E-09	8.6E-05	1.5E-05
tetrachloroethene (PCE)	4.9	4.9E-12	3.7E-12	5.6E-08	3.3E-03	6.0E-04
1,1,2,2-tetrachloroethane				2.4E-09	1.5E-04	2.6E-05
chloroform	160	1.6E-10	1.7E-10	1.1E-08	6.7E-04	1.2E-04
carbon tetrachloride				7.3E-08	4.4E-03	7.8E-04
dichloromethane	5.1	5.1E-12	5.4E-12	3.9E-08	2.3E-03	4.2E-04
1,2,4-trimethylbenzene				1.1E-09	6.8E-05	1.2E-05
naphthalene				2.1E-08	1.3E-03	2.2E-04

Arithmetic average of detected concentrations (maximum where only detected once, refer to Tables 3-8 and 3-9)

Calculated based on the maximum of emission rate derived from soil gas or measured from flux emissions

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS

Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 1 Southlands - Maximum

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.47	Calculated from layers
Soil Temperature	[C]	T	17	Assumed
Vadose Zone Layer 1 Characteristics				
Depth of Layer 1	[m]	vd1	0.47	Fill
Moisture Content	[g/g]	mocon	0.08	Most shallow depth to soil gsa data
Organic Carbon Fraction	-	foc	0.003	Conservative value for soil materials
Soil Bulk Density	[g/ml]	rhob	1.7	Assumed for fill
Density of Solids	[g/ml]	sd	2.65	Assumed for fill
Total Soil Porosity	[ml/ml]	theta	0.36	default
Volumetric Water Content	[ml/ml]	wacon	0.136	1 - (rhob/sd)
Volumetric Air Content	[ml/ml]	acon	0.222	mocon*rhob
				theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	emarea	625.0	Assumed size of building/room in area (25mx25m)
Height of Room	[m]	boxh	3	Assumed for commercial/industrial building
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum Australian Std for Commercial Bldgs
Air Exchange Rate	[exch/s]	er	0.00056	Calculated
Floor Attenuation Factor	-	faf	0.1	Conservative value for range of commercial bldgs
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	Assumed area contributing to outdoor air concentration
Width of Contaminated Area	[m]	width	20	Assumed area contributing to outdoor air concentration
Outdoor Area	[m ²]	oarea	400	calculated
Q/C Dispersion term	[g/m ² /s per kg/m ³]	qc	9.28E+01	Calculated for climate similar to East coast of Australia

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
carbon tetrachloride	800	153.8	170	0.078	8.8E-06	91.3	1.2
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Capillary Fringe (cm ² /s)	Total Effective Diffusion (GW to surface) (cm ² /s)
vinyl chloride (adult exposures)	5.50E-3			5.50E-3
1,2-dichloroethane (EDC)	5.40E-3			5.40E-3
trichloroethene (TCE)	4.10E-3			4.10E-3
tetrachloroethene (PCE)	3.74E-3			3.74E-3
chloroform	5.40E-3			5.40E-3
carbon tetrachloride	4.05E-3			4.05E-3
dichloromethane	5.25E-3			5.25E-3

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS
Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)
Assessment of Vapour Migration - Block 1 Southlands - Maximum

Calculated Air Concentrations ASTM Guidance	Calculation Based on Measured Soil Gas			Measured Emission Rate from Surface of Ground (g/s/m ²)	Concentration within Building (mg/m ³)	Outdoor Air Concentration Q/C (mg/m ³)
	Vapour Phase Concentration at Source (ug/m ³)	Vapour Phase Concentration at Source (g/cm ³)	Calculated Emission Rate from Surface of Ground (g/s/m ²)			
benzene				1.6E-08	9.7E-04	1.7E-04
toluene				7.3E-09	4.4E-04	7.9E-05
ethylbenzene				4.2E-10	2.5E-05	4.5E-06
xylenes				2.2E-09	1.3E-04	2.3E-05
vinyl chloride (adult exposures)	200	2.0E-10	2.3E-10	4.0E-08	2.4E-03	4.3E-04
trans-1,2-dichloroethene				3.0E-09	1.8E-04	3.2E-05
cis-1,2-dichloroethene				1.1E-08	6.3E-04	1.1E-04
1,2-dichloroethane (EDC)				4.7E-08	2.8E-03	5.0E-04
trichloroethene (TCE)	91	9.1E-11	7.9E-11	8.3E-08	5.0E-03	9.0E-04
1,1,2-trichloroethane				3.2E-09	1.9E-04	3.4E-05
tetrachloroethene (PCE)	4.9	4.9E-12	3.9E-12	6.5E-07	3.9E-02	7.0E-03
1,1,1,2-tetrachloroethane				6.3E-09	3.8E-04	6.8E-05
chloroform	160	1.6E-10	1.8E-10	4.2E-08	2.5E-03	4.5E-04
carbon tetrachloride				2.2E-07	1.3E-02	2.3E-03
dichloromethane	5.1	5.1E-12	5.7E-12	1.8E-07	1.1E-02	1.9E-03
1,2,4-trimethylbenzene				1.5E-09	8.8E-05	1.6E-05
naphthalene				5.3E-08	3.2E-03	5.7E-04

↑
Maximum of detected concentrations
(refer to Table 3-9)

↑
Maximum measured
(refer to Table 3-8)

↑
Calculated based on the maximum of emission rate derived from soil gas or measured from flux emissions

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS

Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 2 Southlands - Average

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.5	Calculated from layers
Soil Temperature	[C]	T	17	Assumed
Vadose Zone Layer 1 Characteristics				
Depth of Layer 1	[m]	vd1	0.5	Fill
Moisture Content	[g/g]	mocon	0.08	Assume soil gas data represents what may be beneath fill
Organic Carbon Fraction	-	foc	0.003	Conservative value for soil materials
Soil Bulk Density	[g/ml]	rhob	1.7	Assumed for fill
Density of Solids	[g/ml]	sd	2.65	Assumed for fill
Total Soil Porosity	[ml/ml]	theta	0.36	default
Volumetric Water Content	[ml/ml]	wacon	0.136	1 - (rhob/sd)
Volumetric Air Content	[ml/ml]	acon	0.222	mocon*rhob
				theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	emarea	625.0	Assumed size of building/room in area (25mx25m)
Height of Room	[m]	boxh	3	Assumed for commercial/industrial building
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum Australian Std for Commercial Bldgs
Air Exchange Rate	[exch/s]	er	0.00056	Calculated
Floor Attenuation Factor	-	faf	0.1	Conservative value for range of commercial bldgs
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	Assumed area contributing to outdoor air concentration
Width of Contaminated Area	[m]	width	20	Assumed area contributing to outdoor air concentration
Outdoor Area	[m ²]	oarea	400	calculated
Q/C Dispersion term	[g/m ² /s per kg/m ³]	qc	9.28E+01	Calculated for climate similar to East coast of Australia

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
benzene	1780	78.12	58.9	0.088	9.8E-06	95.2	0.228
TPH C6-C9 aliphatic	5.4	100	3160	0.067	6.9E-06	48	50
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
1,1-dichloroethene	2420	96.95	35.04	0.09	1.0E-05	634	1.07
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384
1,1-dichloroethane	5040	98.96	35.04	0.0742	1.1E-05	227	0.23
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133
1,2,4-trimethylbenzene	57	120.2	717.6	0.064	7.9E-06	2.1	0.252
1,3,5-trimethylbenzene	48.2	120.2	703	0.0602	8.7E-06	2.1	0.316

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Capillary Fringe (cm ² /s)	Total Effective Diffusion (GW to surface) (cm ² /s)
benzene	4.57E-3			4.57E-3
TPH C6-C9 aliphatic	3.48E-3			3.48E-3
vinyl chloride (adult exposures)	5.50E-3			5.50E-3
1,1-dichloroethene	4.67E-3			4.67E-3
trans-1,2-dichloroethene	3.67E-3			3.67E-3
1,1-dichloroethane	3.85E-3			3.85E-3
cis-1,2-dichloroethene	3.82E-3			3.82E-3
1,2-dichloroethane (EDC)	5.40E-3			5.40E-3
trichloroethene (TCE)	4.10E-3			4.10E-3
tetrachloroethene (PCE)	3.74E-3			3.74E-3
chloroform	5.40E-3			5.40E-3
dichloromethane	5.25E-3			5.25E-3
1,2,4-trimethylbenzene	3.32E-3			3.32E-3
1,3,5-trimethylbenzene	3.13E-3			3.13E-3

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS
Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)
Assessment of Vapour Migration - Block 2 Southlands - Average

Calculated Air Concentrations ASTM Guidance	Calculation Based on Measured Soil Gas			Measured Emission Rate from Surface of Ground (g/s/m ²)	Concentration within Building (mg/m ³)	Outdoor Air Concentration Q/C (mg/m ³)
	Vapour Phase Concentration at Source (ug/m ³)	Vapour Phase Concentration at Source (g/cm ³)	Calculated Emission Rate from Surface of Ground (g/s/m ²)			
benzene	9.65	9.7E-12	8.8E-12	2.0E-09	1.2E-04	2.2E-05
toluene				9.8E-09	5.9E-04	1.1E-04
ethylbenzene				1.0E-09	6.2E-05	1.1E-05
xylenes				5.2E-09	3.1E-04	5.6E-05
TPH C6-C9 aliphatic	310	3.1E-10	2.2E-10		1.3E-05	2.3E-06
TPH C10-C14 aromatic				2.7E-10	1.6E-05	2.9E-06
vinyl chloride (adult exposures)	205	2.1E-10	2.3E-10	9.5E-10	5.7E-05	1.0E-05
1,1-dichloroethene	852	8.5E-10	8.0E-10	2.0E-09	1.2E-04	2.2E-05
trans-1,2-dichloroethene	282	2.8E-10	2.1E-10		1.2E-05	2.2E-06
1,1-dichloroethane				4.5E-09	2.7E-04	4.8E-05
cis-1,2-dichloroethene	602	6.0E-10	4.6E-10		2.8E-05	5.0E-06
1,2-dichloroethane (EDC)	184.5	1.8E-10	2.0E-10	5.0E-09	3.0E-04	5.4E-05
trichloroethene (TCE)	46	4.6E-11	3.8E-11	5.7E-10	3.4E-05	6.1E-06
1,1,2-trichloroethane				3.3E-10	2.0E-05	3.6E-06
tetrachloroethene (PCE)				1.3E-08	7.9E-04	1.4E-04
1,1,2,2-tetrachloroethane				6.7E-10	4.0E-05	7.2E-06
chloroform	11.7	1.2E-11	1.3E-11	1.4E-08	8.4E-04	1.5E-04
carbon tetrachloride				4.2E-10	2.5E-05	4.5E-06
dichloromethane	32	3.2E-11	3.4E-11	2.0E-08	1.2E-03	2.2E-04
1,2,4-trimethylbenzene	150	1.5E-10	1.0E-10	2.3E-09	1.4E-04	2.5E-05
1,3,5-trimethylbenzene	25.15	2.5E-11	1.6E-11	6.3E-10	3.8E-05	6.8E-06
naphthalene				8.2E-09	4.9E-04	8.8E-05
trichlorofluoromethane				9.2E-09	5.5E-04	9.9E-05

Arithmetic average of detected concentrations (maximum where only detected once, refer to Tables 3-7 and 3-9)

Calculated based on the maximum of emission rate derived from soil gas or measured from flux emissions

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS

Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 2 Southlands - Maximum

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.5	Calculated from layers
Soil Temperature	[C]	T	17	Assumed
Vadose Zone Layer 1 Characteristics				
Depth of Layer 1	[m]	vd1	0.5	Fill
Moisture Content	[g/g]	mocon	0.08	Assume soil gas data represents what may be beneath fill
Organic Carbon Fraction	-	foc	0.003	Conservative value for soil materials
Soil Bulk Density	[g/ml]	rhob	1.7	Assumed for fill
Density of Solids	[g/ml]	sd	2.65	Assumed for fill
Total Soil Porosity	[ml/ml]	theta	0.36	default
Volumetric Water Content	[ml/ml]	wacon	0.136	1 - (rhob/sd)
Volumetric Air Content	[ml/ml]	acon	0.222	mocon*rhob
				theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	emarea	625.0	Assumed size of building/room in area (25mx25m)
Height of Room	[m]	boxh	3	Assumed for commercial/industrial building
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum Australian Std for Commercial Bldgs
Air Exchange Rate	[exch/s]	er	0.00056	Calculated
Floor Attenuation Factor	-	faf	0.1	Conservative value for range of commercial bldgs
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	Assumed area contributing to outdoor air concentration
Width of Contaminated Area	[m]	width	20	Assumed area contributing to outdoor air concentration
Outdoor Area	[m ²]	oarea	400	calculated
Q/C Dispersion term	[g/m ² /s per kg/m ³]	qc	9.28E+01	Calculated for climate similar to East coast of Australia

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ² /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
benzene	1780	78.12	58.9	0.088	9.8E-06	95.2	0.228
TPH C6-C9 aliphatic	5.4	100	3160	0.067	6.9E-06	48	50
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
1,1-dichloroethene	2420	96.95	35.04	0.09	1.0E-05	634	1.07
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
carbon tetrachloride	800	153.8	170	0.078	8.8E-06	91.3	1.2
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133
1,2,4-trimethylbenzene	57	120.2	717.6	0.064	7.9E-06	2.1	0.252
1,3,5-trimethylbenzene	48.2	120.2	703	0.0602	8.7E-06	2.1	0.316

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Capillary Fringe (cm ² /s)	Total Effective Diffusion (GW to surface) (cm ² /s)
benzene	4.57E-3			4.57E-3
TPH C6-C9 aliphatic	3.48E-3			3.48E-3
vinyl chloride (adult exposures)	5.50E-3			5.50E-3
1,1-dichloroethene	4.67E-3			4.67E-3
trans-1,2-dichloroethene	3.67E-3			3.67E-3
cis-1,2-dichloroethene	3.82E-3			3.82E-3
1,2-dichloroethane (EDC)	5.40E-3			5.40E-3
trichloroethene (TCE)	4.10E-3			4.10E-3
tetrachloroethene (PCE)	3.74E-3			3.74E-3
chloroform	5.40E-3			5.40E-3
dichloromethane	5.25E-3			5.25E-3
1,2,4-trimethylbenzene	3.32E-3			3.32E-3
1,3,5-trimethylbenzene	3.13E-3			3.13E-3

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS

Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 2 Southlands - Maximum

Calculated Air Concentrations ASTM Guidance	Calculation Based on Measured Soil Gas			Measured Emission Rate from Surface of Ground (g/s/m ²)	Concentration within Building (mg/m ³)	Outdoor Air Concentration Q/C (mg/m ³)
	Vapour Phase Concentration at Source (ug/m ³)	Vapour Phase Concentration at Source (g/cm ³)	Calculated Emission Rate from Surface of Ground (g/s/m ²)			
benzene	15	1.5E-11	1.4E-11	2.5E-09	1.5E-04	2.7E-05
toluene				1.6E-08	9.5E-04	1.7E-04
ethylbenzene				1.5E-09	9.1E-05	1.6E-05
xylenes				7.3E-09	4.4E-04	7.9E-05
TPH C6-C9 aliphatic	310	3.1E-10	2.2E-10	0.0E+00	1.3E-05	2.3E-06
TPH C10-C14 aromatic				2.7E-10	1.6E-05	2.9E-06
vinyl chloride (adult exposures)	205	2.1E-10	2.3E-10	9.5E-10	5.7E-05	1.0E-05
1,1-dichloroethene	852	8.5E-10	8.0E-10	2.0E-09	1.2E-04	2.2E-05
trans-1,2-dichloroethene	282	2.8E-10	2.1E-10	0.0E+00	1.2E-05	2.2E-06
1,1-dichloroethane				4.5E-09	2.7E-04	4.8E-05
cis-1,2-dichloroethene	602	6.0E-10	4.6E-10	0.0E+00	2.8E-05	5.0E-06
1,2-dichloroethane (EDC)	343	3.4E-10	3.7E-10	2.3E-08	1.4E-03	2.5E-04
trichloroethene (TCE)	46	4.6E-11	3.8E-11	1.2E-09	6.9E-05	1.2E-05
1,1,2-trichloroethane				4.5E-10	2.7E-05	4.8E-06
tetrachloroethene (PCE)				1.3E-07	7.7E-03	1.4E-03
1,1,2,2-tetrachloroethane				6.7E-10	4.0E-05	7.2E-06
chloroform	20	2.0E-11	2.2E-11	3.2E-08	1.9E-03	3.4E-04
carbon tetrachloride				9.2E-10	5.5E-05	9.9E-06
dichloromethane	32	3.2E-11	3.4E-11	1.3E-07	7.7E-03	1.4E-03
1,2,4-trimethylbenzene	150	1.5E-10	1.0E-10	3.2E-09	1.9E-04	3.4E-05
1,3,5-trimethylbenzene	49	4.9E-11	3.1E-11	6.3E-10	3.8E-05	6.8E-06
naphthalene				1.5E-08	9.2E-04	1.7E-04
trichlorofluoromethane				9.2E-09	5.5E-04	9.9E-05

Maximum of detected concentrations
(Table 3-9)

Maximum measured
(Table 3-7)

Calculated based on the maximum of emission rate derived from soil gas or measured from flux emissions

Inhalation of Volatile Chemicals above Groundwater (and some soil impacts)

Stage 1 Development

General Data/ Equations		Exposure Calculations (Average)		Exposure Calculations (RME)	
Units		Indoors on Block 2 (Table 5-2, Scenario 2)		Indoors on Block 2 (Table 5-2, Scenario 2)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.17	Inhalation rate indoors	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	9	Assume 9 hours per day spent indoors	9	Assume 9 hours per day spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time spent at inside building located above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	4.2E-02	NonThreshold	4.2E-02	NonThreshold
		9.9E-02	Threshold	9.9E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	3.4E-7	9.5E-2			TOTAL	8.9E-7	1.9E-1
benzene	2.1E-02	7.7E-03	1.20E-04	5.1E-06	1.2E-05	1.1E-7	1.5E-3	1.50E-04	6.4E-06	1.5E-05	1.3E-7	1.9E-3
toluene		6.7E-02	5.90E-04	2.5E-05	5.8E-05	--	8.7E-4	9.50E-04	4.0E-05	9.4E-05	--	1.4E-3
ethylbenzene		6.3E+00	6.20E-05	2.6E-06	6.1E-06	--	9.8E-7	9.10E-05	3.9E-06	9.0E-06	--	1.4E-6
xylenes		2.4E-01	3.10E-04	1.3E-05	3.1E-05	--	1.3E-4	4.40E-04	1.9E-05	4.4E-05	--	1.8E-4
TPH C6-C9 aliphatic		1.8E-01	1.29E-05	5.5E-07	1.3E-06	--	7.1E-6	1.29E-05	5.5E-07	1.3E-06	--	7.1E-6
TPH C10-C14 aromatic		5.1E-02	1.60E-05	6.8E-07	1.6E-06	--	3.1E-5	1.60E-05	6.8E-07	1.6E-06	--	3.1E-5
vinyl chloride (adult exposures)	1.5E-02		5.70E-05	2.4E-06	5.6E-06	3.7E-8	--	5.70E-05	2.4E-06	5.6E-06	3.7E-8	--
1,1-dichloroethene		5.7E-02	1.20E-04	5.1E-06	1.2E-05	--	2.1E-4	1.20E-04	5.1E-06	1.2E-05	--	2.1E-4
trans-1,2-dichloroethene		1.7E-02	1.24E-05	5.3E-07	1.2E-06	--	7.2E-5	1.24E-05	5.3E-07	1.2E-06	--	7.2E-5
1,1-dichloroethane		1.4E-01	2.70E-04	1.1E-05	2.7E-05	--	1.9E-4	2.70E-04	1.1E-05	2.7E-05	--	1.9E-4
cis-1,2-dichloroethene		1.0E-02	2.76E-05	1.2E-06	2.7E-06	--	2.7E-4	2.76E-05	1.2E-06	2.7E-06	--	2.7E-4
1,2-dichloroethane (EDC)	9.8E-03		3.00E-04	1.3E-05	3.0E-05	1.2E-7	--	1.40E-03	5.9E-05	1.4E-04	5.8E-7	--
trichloroethene (TCE)	1.5E-03		3.40E-05	1.4E-06	3.4E-06	2.2E-9	--	6.90E-05	2.9E-06	6.8E-06	4.4E-9	--
1,1,2-trichloroethane		4.0E-03	2.00E-05	8.5E-07	2.0E-06	--	4.9E-4	2.70E-05	1.1E-06	2.7E-06	--	6.7E-4
tetrachloroethene (PCE)		3.8E-02	7.90E-04	3.3E-05	7.8E-05	--	2.1E-3	7.72E-03	3.3E-04	7.6E-04	--	2.0E-2
1,1,2,2-tetrachloroethane	1.1E-02		4.00E-05	1.7E-06	4.0E-06	1.8E-8	--	4.00E-05	1.7E-06	4.0E-06	1.8E-8	--
chloroform	1.5E-03		8.40E-04	3.6E-05	8.3E-05	5.2E-8	4.2E-3	1.90E-03	8.1E-05	1.9E-04	1.2E-7	9.4E-3
carbon tetrachloride		6.1E-04	2.50E-05	1.1E-06	2.5E-06	--	4.1E-3	5.50E-05	2.3E-06	5.4E-06	--	8.9E-3
dichloromethane		2.3E-01	1.20E-03	5.1E-05	1.2E-04	--	5.2E-4	7.70E-03	3.3E-04	7.6E-04	--	3.3E-3
1,2,4-trimethylbenzene		8.6E-04	1.40E-04	5.9E-06	1.4E-05	--	1.6E-2	1.90E-04	8.1E-06	1.9E-05	--	2.2E-2
1,3,5-trimethylbenzene		8.6E-04	3.80E-05	1.6E-06	3.8E-06	--	4.4E-3	3.80E-05	1.6E-06	3.8E-06	--	4.4E-3
naphthalene		8.1E-04	4.90E-04	2.1E-05	4.8E-05	--	6.0E-2	9.20E-04	3.9E-05	9.1E-05	--	1.1E-1
trichlorofluoromethane		2.0E-01	5.50E-04	2.3E-05	5.4E-05	--	2.7E-4	5.50E-04	2.3E-05	5.4E-05	--	2.7E-4

↑
Calculated from average of measured data for Block 2

↑
Calculated from maximum of measured data for Block 2

Inhalation of Volatile Chemicals above Groundwater (and some soil impacts)

Stage 1 Development

General Data/ Equations	Units	Exposure Calculations (RME)	
		Outdoors on Block 1 or 2 (Table 5-2, Scenario 2)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	1	Assume 1 hour outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	8.9E-03	NonThreshold
		2.1E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air (mg/m ³)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	1.6E-7	1.0E-1
benzene	2.1E-02	7.7E-03	1.74E-04	1.5E-06	3.6E-06	3.2E-8	4.7E-4
toluene		6.7E-02	1.71E-04	1.5E-06	3.5E-06	--	5.3E-5
ethylbenzene		6.3E+00	1.63E-05	1.4E-07	3.4E-07	--	5.4E-8
xylenes		2.4E-01	7.90E-05	7.0E-07	1.6E-06	--	6.7E-6
TPH C6-C9 aliphatic		1.8E-01	2.32E-06	2.1E-08	4.8E-08	--	2.7E-7
TPH C10-C14 aromatic		5.1E-02	2.87E-06	2.5E-08	5.9E-08	--	1.2E-6
vinyl chloride (adult exposures)	1.5E-02		4.27E-04	3.8E-06	8.8E-06	5.8E-8	--
1,1-dichloroethene		5.7E-02	2.16E-05	1.9E-07	4.5E-07	--	7.8E-6
trans-1,2-dichloroethene		1.7E-02	3.23E-05	2.9E-07	6.7E-07	--	3.9E-5
1,1-dichloroethane		1.4E-01	4.85E-05	4.3E-07	1.0E-06	--	7.0E-6
cis-1,2-dichloroethene		1.0E-02	1.13E-04	1.0E-06	2.3E-06	--	2.3E-4
1,2-dichloroethane (EDC)	9.8E-03		5.03E-04	4.5E-06	1.0E-05	4.4E-8	--
trichloroethene (TCE)	1.5E-03		8.98E-04	8.0E-06	1.9E-05	1.2E-8	--
1,1,2-trichloroethane		4.0E-03	3.41E-05	3.0E-07	7.1E-07	--	1.8E-4
tetrachloroethene (PCE)		3.8E-02	7.00E-03	6.2E-05	1.4E-04	--	3.8E-3
1,1,2,2-tetrachloroethane	1.1E-02		6.82E-05	6.0E-07	1.4E-06	6.3E-9	--
chloroform	1.5E-03	2.0E-02	4.49E-04	4.0E-06	9.3E-06	5.8E-9	4.6E-4
carbon tetrachloride		6.1E-04	2.33E-03	2.1E-05	4.8E-05	--	7.9E-2
dichloromethane		2.3E-01	1.89E-03	1.7E-05	3.9E-05	--	1.7E-4
1,2,4-trimethylbenzene		8.6E-04	3.41E-05	3.0E-07	7.1E-07	--	8.2E-4
1,3,5-trimethylbenzene		8.6E-04	6.82E-06	6.0E-08	1.4E-07	--	1.6E-4
naphthalene		8.1E-04	5.75E-04	5.1E-06	1.2E-05	--	1.5E-2
trichlorofluoromethane		2.0E-01	9.88E-05	8.7E-07	2.0E-06	--	1.0E-5

↑
Calculated from maximum of measured data
Maximum outdoor air concentration from Block 1 or Block 2

Inhalation of Volatile Chemicals above Groundwater (and some soil impacts) - using Occupational Inhalation Exposure Guidance

Stage 1 Development

General Data/ Equations		Exposure Calculations (RME)			
		Indoors on Block 2 (Table 5-2, Scenario 2)		Outdoors on Block 1 or 2 (Table 5-2, Scenario 2)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.17	Inhalation rate indoors	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	9	Assume 9 hours per day spent indoors	1	Assume 1 hour outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time spent at inside building located above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	4.2E-02 9.9E-02	NonThreshold Threshold	8.9E-03 2.1E-02	NonThreshold Threshold

Daily Intake = Concentration in Air x Intake Factor
NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWAs	in Air	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	
					TOTAL		8.9E-7 3.3E-4				TOTAL		1.6E-7 5.7E-4	
benzene	2.1E-02	4.6E-01	1.50E-04	6.4E-06	1.5E-05	1.3E-7	3.2E-5	1.74E-04	1.5E-06	3.6E-06	3.2E-8	7.9E-6		
toluene		2.7E+01	9.50E-04	4.0E-05	9.4E-05	--	3.4E-6	1.71E-04	1.5E-06	3.5E-06	--	1.3E-7		
ethylbenzene		6.2E+01	9.10E-05	3.9E-06	9.0E-06	--	1.5E-7	1.63E-05	1.4E-07	3.4E-07	--	5.4E-9		
xylenes		5.0E+01	4.40E-04	1.9E-05	4.4E-05	--	8.7E-7	7.90E-05	7.0E-07	1.6E-06	--	3.3E-8		
TPH C6-C9 aliphatic		1.0E+01	1.29E-05	5.5E-07	1.3E-06	--	1.2E-7	2.32E-06	2.1E-08	4.8E-08	--	4.7E-9		
TPH C10-C14 aromatic		5.0E+01	1.60E-05	6.8E-07	1.6E-06	--	3.2E-8	2.87E-06	2.5E-08	5.9E-08	--	1.2E-9		
vinyl chloride (adult exposures)	1.5E-02	1.9E+00	5.70E-05	2.4E-06	5.6E-06	3.7E-8	3.0E-6	4.27E-04	3.8E-06	8.8E-06	5.8E-8	4.8E-6		
1,1-dichloroethene		2.9E+00	1.20E-04	5.1E-06	1.2E-05	--	4.2E-6	2.16E-05	1.9E-07	4.5E-07	--	1.6E-7		
trans-1,2-dichloroethene		1.1E+02	1.24E-05	5.3E-07	1.2E-06	--	1.1E-8	3.23E-05	2.9E-07	6.7E-07	--	5.9E-9		
1,1-dichloroethane		5.9E+01	2.70E-04	1.1E-05	2.7E-05	--	4.5E-7	4.85E-05	4.3E-07	1.0E-06	--	1.7E-8		
cis-1,2-dichloroethene		1.1E+02	2.76E-05	1.2E-06	2.7E-06	--	2.4E-8	1.13E-04	1.0E-06	2.3E-06	--	2.1E-8		
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.40E-03	5.9E-05	1.4E-04	5.8E-7	2.4E-5	5.03E-04	4.5E-06	1.0E-05	4.4E-8	1.8E-6		
trichloroethene (TCE)	1.5E-03	7.7E+00	6.90E-05	2.9E-06	6.8E-06	4.4E-9	8.8E-7	8.98E-04	8.0E-06	1.9E-05	1.2E-8	2.4E-6		
1,1,2-trichloroethane		7.9E+00	2.70E-05	1.1E-06	2.7E-06	--	3.4E-7	3.41E-05	3.0E-07	7.1E-07	--	9.0E-8		
tetrachloroethene (PCE)		4.8E+01	7.72E-03	3.3E-04	7.6E-04	--	1.6E-5	7.00E-03	6.2E-05	1.4E-04	--	3.0E-6		
1,1,2,2-tetrachloroethane	1.1E-02	9.9E-01	4.00E-05	1.7E-06	4.0E-06	1.8E-8	4.0E-6	6.82E-05	6.0E-07	1.4E-06	6.3E-9	1.4E-6		
chloroform	1.5E-03	1.4E+00	1.90E-03	8.1E-05	1.9E-04	1.2E-7	1.3E-4	4.49E-04	4.0E-06	9.3E-06	5.8E-9	6.5E-6		
carbon tetrachloride		9.0E-02	5.50E-05	2.3E-06	5.4E-06	--	6.0E-5	2.33E-03	2.1E-05	4.8E-05	--	5.4E-4		
dichloromethane		2.5E+01	7.70E-03	3.3E-04	7.6E-04	--	3.1E-5	1.89E-03	1.7E-05	3.9E-05	--	1.6E-6		
1,2,4-trimethylbenzene		1.8E+01	1.90E-04	8.1E-06	1.9E-05	--	1.1E-6	3.41E-05	3.0E-07	7.1E-07	--	4.0E-8		
1,3,5-trimethylbenzene		1.8E+01	3.80E-05	1.6E-06	3.8E-06	--	2.1E-7	6.82E-06	6.0E-08	1.4E-07	--	8.0E-9		
naphthalene		7.4E+00	9.20E-04	3.9E-05	9.1E-05	--	1.2E-5	5.75E-04	5.1E-06	1.2E-05	--	1.6E-6		
trichlorofluoromethane		8.0E+02	5.50E-04	2.3E-05	5.4E-05	--	6.8E-8	9.88E-05	8.7E-07	2.0E-06	--	2.5E-9		

↑
Calculated from maximum of measured data

↑
Calculated from maximum of measured data

**VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR CONTAMINATED SOILS
UTILISING ASTM GUIDANCE E 1739-95^F (2002)**

Vapour Migration from Impacted Sediments - Surface

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Surface Contamination	[m]	surfd	2	Estimated for soil types
Soil Temperature	[C]	T	17	assumed for area
Vadose Zone Characteristics of Top Layer				
Depth of Layer 1	[m]	vd1	0.5	Fill Assume impacted soils at surface
Moisture Content	[g/g]	mocon	0.08	Conservative value for soil materials
Organic Carbon Fraction	-	foc	0.003	Assumed for fill
Soil Bulk Density	[g/cm ³]	rhob	1.7	Assumed for fill
Density of Solids	[g/cm ³]	sd	2.65	Default
Total Soil Porosity	[cm ³ /cm ³]	theta	0.36	1 - (rhob/sd)
Volumetric Water Content	[cm ³ /cm ³]	wacon	0.136	mocon*rhob
Volumetric Air Content	[cm ³ /cm ³]	acon	0.222	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	area	100.0	assumed area of the room
Percentage of area above contamination	[%]	Pem	100%	assume all of building above impact
Foundation/wall thickness	[m]	ftthick	0.15	Default Value
Height of Room	[m]	boxh	3	site-specific assumption
Ratio of enclosed space volume:infiltration area	[m]	LB	3	Calculated
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum exchange rate as per AS 1668.2
Fraction of Cracks in Walls and foundation	-	cracks	0.001	Representative of sound concrete
Volumetric Water Content in foundation/wall cracks	[cm ³ /cm ³]	fwacon	0.12	Default Value
Volumetric Air Content in foundation/wall cracks	[cm ³ /cm ³]	facon	0.260	Default Value
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	assumed area impacted
Width of Contaminated Area	[m]	width	20	assumed area impacted
Wind Speed Outdoors	[m/s]	wspd	4.9	Average 9am and 3pm winds from Sydney Airport
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Default Value
Averaging Time for Vapour Flux	[yrs]	avtime	7.80E+08	Default Value - use for surface soil calculations only

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
TPH C6-C9 aliphatic	5.4	100	3160	0.067	6.9E-06	48	50
TPH C10-C14 aromatic	25	130	2510	0.048	7.7E-06	0.48	0.14
TPH C10-C14 aliphatic	0.034	160	316000	0.046	5.2E-06	0.48	130
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167
1,2-dichloroethane (EDC)	8690	98.96	17.4	0.104	9.9E-06	79.1	0.0401
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
1,1,2,2-tetrachloroethane	2870	167.85	106.8	0.071	7.9E-06	13.3	0.0141
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
carbon tetrachloride	800	153.8	170	0.078	8.8E-06	91.3	1.2
1,4-dichlorobenzene	81.3	147	434	0.069	7.9E-06	1.74	0.0996

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Foundations and Cracks (cm ² /s)	Total Effective Diffusion (Subsurface soil to surface) (cm ² /s)
TPH C6-C9 aliphatic	3.48E-3	3.48E-3	5.85E-3	3.48E-3
TPH C10-C14 aromatic	2.49E-3	2.49E-3	4.19E-3	2.49E-3
TPH C10-C14 aliphatic	2.39E-3	2.39E-3	4.02E-3	2.39E-3
vinyl chloride (adult exposures)	5.50E-3	5.50E-3	9.25E-3	5.50E-3
trans-1,2-dichloroethene	3.67E-3	3.67E-3	6.17E-3	3.67E-3
cis-1,2-dichloroethene	3.82E-3	3.82E-3	6.43E-3	3.82E-3
1,2-dichloroethane (EDC)	5.40E-3	5.40E-3	9.08E-3	5.40E-3
trichloroethene (TCE)	4.10E-3	4.10E-3	6.90E-3	4.10E-3
1,1,2-trichloroethane	4.05E-3	4.05E-3	6.81E-3	4.05E-3
tetrachloroethene (PCE)	3.74E-3	3.74E-3	6.29E-3	3.74E-3
1,1,2,2-tetrachloroethane	3.69E-3	3.69E-3	6.20E-3	3.69E-3
chloroform	5.40E-3	5.40E-3	9.08E-3	5.40E-3
carbon tetrachloride	4.05E-3	4.05E-3	6.81E-3	4.05E-3
1,4-dichlorobenzene	3.58E-3	3.58E-3	6.02E-3	3.58E-3

**VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR CONTAMINATED SOILS
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Vapour Migration from Impacted Sediments - Surface

Subsurface Soils Phase Partitioning Results	Soil Concentration (mg/kg)	Vapour Phase Concentration (g/cm ³)	Saturated Soil Concentration (mg/kg)	Saturated Vapour Concentration (g/cm ³)	Free Phase Mole Fraction (mol/mol)	Concentration above Free Phase (g/cm ³)	Calculation Vapour Phase used in Calculation (g/cm ³)
TPH C6-C9 aliphatic	113	3.5E-04	8.7E+01	2.7E-04	0	0.0E+00	3.5E-04
TPH C10-C14 aromatic	249	4.6E-06	1.9E+02	3.5E-06	0	0.0E+00	4.6E-06
aliphatic	249	3.4E-05	3.3E+01	4.2E-06	0	0.0E+00	3.4E-05
vinyl chloride (adult exposures)	384	1.5E-03	7.7E+02	9.0E-03	0	0.0E+00	1.5E-03
trans-1,2-dichloroethene	13.4	1.8E-05	1.8E+03	1.4E-03	0	0.0E+00	1.8E-05
cis-1,2-dichloroethene	302	2.4E-04	7.3E+02	9.6E-04	0	0.0E+00	2.4E-04
1,2-dichloroethane (EDC)	29.8	8.7E-06	1.2E+03	4.3E-04	0	0.0E+00	8.7E-06
trichloroethene (TCE)	1050	7.0E-04	7.0E+02	5.4E-04	0	0.0E+00	5.4E-04
1,1,2-trichloroethane	115	1.5E-05	3.2E+02	1.7E-04	0	0.0E+00	1.5E-05
tetrachloroethene (PCE)	7130	8.4E-03	1.3E+02	1.7E-04	0	0.0E+00	1.7E-04
1,1,2,2-tetrachloroethane	42.7	1.5E-06	1.2E+03	1.2E-04	0	0.0E+00	1.5E-06
chloroform	213	1.5E-04	1.7E+03	1.1E-03	0	0.0E+00	1.5E-04
carbon tetrachloride	0.07	1.1E-07	6.0E+02	7.8E-04	0	0.0E+00	1.1E-07
1,4-dichlorobenzene	1.1	7.9E-08	1.1E+02	1.4E-05	0	0.0E+00	7.9E-08

Calculated Air Concentrations ASTM Guidance for Subsurface Soils	Vapour Phase Concentration at Source (g/cm ³)	Emission Rate from Surface of Ground (g/s)	Concentration in Building (mg/m ³)	Outdoor Air Concentration (mg/m ³)	TWA (mg/m ³)
TPH C6-C9 aliphatic	3.5E-04	2.4E-02	8.2E-01		72
TPH C10-C14 aromatic	4.6E-06	2.3E-04	7.6E-03		350
aliphatic	3.4E-05	1.6E-03	5.4E-02		350
vinyl chloride (adult exposures)	1.5E-03	1.7E-01	5.6E+00		13
trans-1,2-dichloroethene	1.8E-05	1.3E-03	4.4E-02		793
cis-1,2-dichloroethene	2.4E-04	1.9E-02	6.2E-01		793
1,2-dichloroethane (EDC)	8.7E-06	9.4E-04	3.1E-02		40
trichloroethene (TCE)	5.4E-04	4.4E-02	1.5E+00		54
1,1,2-trichloroethane	1.5E-05	1.2E-03	4.0E-02		55
tetrachloroethene (PCE)	1.7E-04	1.3E-02	4.2E-01		335
1,1,2,2-tetrachloroethane	1.5E-06	1.1E-04	3.7E-03		6.9
chloroform	1.5E-04	1.6E-02	5.3E-01		10
carbon tetrachloride	1.1E-07	9.1E-06	3.0E-04		0.63
1,4-dichlorobenzene	7.9E-08	5.6E-06	1.9E-04		150

Surface Soils Phase Partitioning Results	Soil Concentration (mg/kg)	Vapour Phase Concentration (g/cm ³)	Saturated Soil Concentration (mg/kg)	Saturated Vapour Concentration (g/cm ³)
TPH C6-C9 aliphatic	113	3.5E-04	8.7E+01	2.7E-04
TPH C10-C14 aromatic	249	4.6E-06	1.9E+02	3.5E-06
aliphatic	249	3.4E-05	3.3E+01	4.2E-06
vinyl chloride (adult exposures)	384	1.5E-03	7.7E+02	9.0E-03
trans-1,2-dichloroethene	13.4	1.8E-05	1.8E+03	1.4E-03
cis-1,2-dichloroethene	302	2.4E-04	7.3E+02	9.6E-04
1,2-dichloroethane (EDC)	29.8	8.7E-06	1.2E+03	4.3E-04
trichloroethene (TCE)	1050	7.0E-04	7.0E+02	5.4E-04
1,1,2-trichloroethane	115	1.5E-05	3.2E+02	1.7E-04
tetrachloroethene (PCE)	7130	8.4E-03	1.3E+02	1.7E-04
1,1,2,2-tetrachloroethane	42.7	1.5E-06	1.2E+03	1.2E-04
chloroform	213	1.5E-04	1.7E+03	1.1E-03
carbon tetrachloride	0.07	1.1E-07	6.0E+02	7.8E-04
1,4-dichlorobenzene	1.1	7.9E-08	1.1E+02	1.4E-05

Calculated Air Emissions and Concentrations ASTM Guidance for Surface Soils	Outdoor Air Concentration from Volatiles (mg/m ³)	TWA (mg/m ³)
TPH C6-C9 aliphatic	1.3E-03	72
TPH C10-C14 aromatic	2.4E-03	350
aliphatic	2.9E-03	350
vinyl chloride (adult exposures)	4.5E-03	13
trans-1,2-dichloroethene	1.6E-04	793
cis-1,2-dichloroethene	3.6E-03	793
1,2-dichloroethane (EDC)	3.5E-04	40
trichloroethene (TCE)	1.2E-02	54
1,1,2-trichloroethane	1.4E-03	55
tetrachloroethene (PCE)	8.4E-02	335
1,1,2,2-tetrachloroethane	5.0E-04	6.9
chloroform	2.5E-03	10
carbon tetrachloride	8.3E-07	0.63
1,4-dichlorobenzene	1.3E-05	150

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Vapour Migration from Impacted Soils - Surface

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Depth of Top of Contaminated Soil (BGS)	[m]	d	0.5	Calculated from layers
Thickness of Surface Contamination	[m]	surfd	2	Estimated for soil types
Soil Temperature	[C]	T	17	assumed for area
Vadose Zone Characteristics of Top Layer				<i>Fill</i>
Depth of Layer 1	[m]	vd1	0.5	Assume impacted soils at surface
Moisture Content	[g/g]	mocon	0.08	Conservative value for soil materials
Organic Carbon Fraction	-	foc	0.003	Assumed for fill
Soil Bulk Density	[g/cm ³]	rhob	1.7	Assumed for fill
Density of Solids	[g/cm ³]	sd	2.65	Default
Total Soil Porosity	[cm ³ /cm ³]	theta	0.36	1 - (rhob/sd)
Volumetric Water Content	[cm ³ /cm ³]	wacon	0.136	mocon*rhob
Volumetric Air Content	[cm ³ /cm ³]	acon	0.222	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	area	100.0	assumed area of the room
Percentage of area above contamination	[%]	Pem	100%	assume all of building above impact
Foundation/wall thickness	[m]	fthick	0.15	Default Value
Height of Room	[m]	boxh	3	site-specific assumption
Ratio of enclosed space volume:infiltration area	[m]	LB	3	Calculated
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum exchange rate as per AS 1668.2
Fraction of Cracks in Walls and foundation	-	cracks	0.001	Representative of sound concrete
Volumetric Water Content in foundation/wall cracks	[cm ³ /cm ³]	fwacon	0.12	Default Value
Volumetric Air Content in foundation/wall cracks	[cm ³ /cm ³]	facon	0.260	Default Value
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	assumed area impacted
Width of Contaminated Area	[m]	width	20	assumed area impacted
Wind Speed Outdoors	[m/s]	wspd	4.9	Average 9am and 3pm winds from Sydney Airport
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Default Value
Averaging Time for Vapour Flux	[yrs]	avtime	7.80E+08	Default Value - use for surface soil calculations only

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384
1,1-dichloroethane	5040	98.96	35.04	0.0742	1.1E-05	227	0.23
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167
1,2-dichloroethane (EDC)	8690	98.96	17.4	0.104	9.9E-06	79.1	0.0401
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
1,1,2,2-tetrachloroethane	2870	167.85	106.8	0.071	7.9E-06	13.3	0.0141
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
1,3,5-trichlorobenzene	4	181.45	2670	0.03	8.2E-06	0.24	0.078

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Foundations and Cracks (cm ² /s)	Total Effective Diffusion (Subsurface soil to surface) (cm ² /s)
vinyl chloride (adult exposures)	5.50E-3	5.50E-3	9.25E-3	5.50E-3
trans-1,2-dichloroethene	3.67E-3	3.67E-3	6.17E-3	3.67E-3
1,1-dichloroethane	3.85E-3	3.85E-3	6.48E-3	3.85E-3
cis-1,2-dichloroethene	3.82E-3	3.82E-3	6.43E-3	3.82E-3
1,2-dichloroethane (EDC)	5.40E-3	5.40E-3	9.08E-3	5.40E-3
trichloroethene (TCE)	4.10E-3	4.10E-3	6.90E-3	4.10E-3
1,1,2-trichloroethane	4.05E-3	4.05E-3	6.81E-3	4.05E-3
tetrachloroethene (PCE)	3.74E-3	3.74E-3	6.29E-3	3.74E-3
1,1,2,2-tetrachloroethane	3.69E-3	3.69E-3	6.20E-3	3.69E-3
chloroform	5.40E-3	5.40E-3	9.08E-3	5.40E-3
1,3,5-trichlorobenzene	1.56E-3	1.56E-3	2.62E-3	1.56E-3

Subsurface Soils Phase Partitioning Results	Soil Concentration (mg/kg)	Vapour Phase Concentration (g/cm ³)	Saturated Soil Concentration (mg/kg)	Saturated Vapour Concentration (g/cm ³)	Free Phase Mole Fraction (mol/mol)	Concentration above Free Phase (g/cm ³)	Calculation Vapour Phase used in Calculation (g/cm ³)
vinyl chloride (adult exposures)	53	2.1E-04	7.7E+02	9.0E-03	0	0.0E+00	2.1E-04
trans-1,2-dichloroethene	106	1.4E-04	1.8E+03	1.4E-03	0	0.0E+00	1.4E-04
1,1-dichloroethane	22.9	2.4E-05	1.1E+03	1.2E-03	0	0.0E+00	2.4E-05
cis-1,2-dichloroethene	165	1.3E-04	7.3E+02	9.6E-04	0	0.0E+00	1.3E-04
1,2-dichloroethane (EDC)	4.5	1.3E-06	1.2E+03	4.3E-04	0	0.0E+00	1.3E-06
trichloroethene (TCE)	95.1	6.3E-05	7.0E+02	5.4E-04	0	0.0E+00	6.3E-05
1,1,2-trichloroethane	1	1.3E-07	3.2E+02	1.7E-04	0	0.0E+00	1.3E-07
tetrachloroethene (PCE)	90.8	1.1E-04	1.3E+02	1.7E-04	0	0.0E+00	1.1E-04
1,1,2,2-tetrachloroethane	10.2	3.6E-07	1.2E+03	1.2E-04	0	0.0E+00	3.6E-07
chloroform	3.9	2.7E-06	1.7E+03	1.1E-03	0	0.0E+00	2.7E-06
1,3,5-trichlorobenzene	15	1.4E-07	3.2E+01	2.4E-06	0	0.0E+00	1.4E-07

**VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR CONTAMINATED SOILS
UTILISING ASTM GUIDANCE E 1739-95^E (2002)**

Vapour Migration from Impacted Soils - Surface

Calculated Air Concentrations ASTM Guidance for Subsurface Soils	Vapour Phase Concentration at Source (g/cm ³)	Emission Rate from Surface of Ground (g/s)	Concentration in Building (mg/m ³)	Outdoor Air Concentration (mg/m ³)	TWA (mg/m ³)
vinyl chloride (adult exposures)	2.1E-04	2.3E-02	7.7E-01		13
trans-1,2-dichloroethene	1.4E-04	1.0E-02	3.5E-01		793
1,1-dichloroethane	2.4E-05	1.9E-03	6.3E-02		412
cis-1,2-dichloroethene	1.3E-04	1.0E-02	3.4E-01		793
1,2-dichloroethane (EDC)	1.3E-06	1.4E-04	4.7E-03		40
trichloroethene (TCE)	6.3E-05	5.2E-03	1.7E-01		54
1,1,2-trichloroethane	1.3E-07	1.1E-05	3.5E-04		55
tetrachloroethene (PCE)	1.1E-04	8.0E-03	2.7E-01		335
1,1,2,2-tetrachloroethane	3.6E-07	2.6E-05	8.8E-04		6.9
chloroform	2.7E-06	2.9E-04	9.6E-03		10
1,3,5-trichlorobenzene	1.4E-07	4.5E-06	1.5E-04		37

Surface Soils Phase Partitioning Results	Soil Concentration (mg/kg)	Vapour Phase Concentration (g/cm ³)	Saturated Soil Concentration (mg/kg)	Saturated Vapour Concentration (g/cm ³)
vinyl chloride (adult exposures)	53	2.1E-04	7.7E+02	9.0E-03
trans-1,2-dichloroethene	106	1.4E-04	1.8E+03	1.4E-03
1,1-dichloroethane	22.9	2.4E-05	1.1E+03	1.2E-03
cis-1,2-dichloroethene	165	1.3E-04	7.3E+02	9.6E-04
1,2-dichloroethane (EDC)	4.5	1.3E-06	1.2E+03	4.3E-04
trichloroethene (TCE)	95.1	6.3E-05	7.0E+02	5.4E-04
1,1,2-trichloroethane	1	1.3E-07	3.2E+02	1.7E-04
tetrachloroethene (PCE)	90.8	1.1E-04	1.3E+02	1.7E-04
1,1,2,2-tetrachloroethane	10.2	3.6E-07	1.2E+03	1.2E-04
chloroform	3.9	2.7E-06	1.7E+03	1.1E-03
1,3,5-trichlorobenzene	15	1.4E-07	3.2E+01	2.4E-06

Calculated Air Emissions and Concentrations ASTM Guidance for Surface Soils	Outdoor Air Concentration from Volatiles (mg/m ³)	TWA (mg/m ³)
vinyl chloride (adult exposures)	6.3E-04	13
trans-1,2-dichloroethene	1.3E-03	793
1,1-dichloroethane	2.7E-04	412
cis-1,2-dichloroethene	2.0E-03	793
1,2-dichloroethane (EDC)	5.3E-05	40
trichloroethene (TCE)	1.1E-03	54
1,1,2-trichloroethane	1.2E-05	55
tetrachloroethene (PCE)	1.1E-03	335
1,1,2,2-tetrachloroethane	1.2E-04	7
chloroform	4.6E-05	10
1,3,5-trichlorobenzene	8.3E-05	37

Inhalation of Volatile Chemicals above Soil Hot-Spots (where not addressed by measured soil gas or flux data)

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME) - Soils		Exposure Calculations (RME) - Sediments	
		Indoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)		Indoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.17	Inhalation rate indoors	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	9	Assume 9 hours per day spent indoors	9	Assume 9 hours per day spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	4.2E-02 9.9E-02	NonThreshold Threshold	4.2E-02 9.9E-02	NonThreshold Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	5.2E-4	6.2E+0			TOTAL	3.8E-3	1.2E+1
TPH C6-C9 aliphatic		1.8E-01				--	--	8.16E-01	3.5E-02	8.1E-02	--	4.5E-1
TPH C10-C14 aromatic		5.1E-02				--	--	7.62E-03	3.2E-04	7.5E-04	--	1.5E-2
TPH C10-C14 aliphatic		2.6E-01				--	--	5.36E-02	2.3E-03	5.3E-03	--	2.1E-2
vinyl chloride (adult exposures)	1.5E-02		7.69E-01	3.3E-02	7.6E-02	5.0E-4	--	5.57E+00	2.4E-01	5.5E-01	3.6E-3	--
trans-1,2-dichloroethene		1.7E-02	3.47E-01	1.5E-02	3.4E-02	--	2.0E+0	4.39E-02	1.9E-03	4.3E-03	--	2.6E-1
1,1-dichloroethane		1.4E-01	6.31E-02	2.7E-03	6.2E-03	--	4.4E-2				--	--
cis-1,2-dichloroethene		1.0E-02	3.38E-01	1.4E-02	3.3E-02	--	3.3E+0	6.19E-01	2.6E-02	6.1E-02	--	6.1E+0
1,2-dichloroethane (EDC)	9.8E-03		4.74E-03	2.0E-04	4.7E-04	2.0E-6	--	3.14E-02	1.3E-03	3.1E-03	1.3E-5	--
trichloroethene (TCE)	1.5E-03		1.74E-01	7.4E-03	1.7E-02	1.1E-5	--	1.47E+00	6.3E-02	1.5E-01	9.4E-5	--
1,1,2-trichloroethane		4.0E-03	3.52E-04	1.5E-05	3.5E-05	--	8.7E-3	4.05E-02	1.7E-03	4.0E-03	--	1.0E+0
tetrachloroethene (PCE)		3.8E-02	2.66E-01	1.1E-02	2.6E-02	--	7.0E-1	4.24E-01	1.8E-02	4.2E-02	--	1.1E+0
1,1,2,2-tetrachloroethane	1.1E-02		8.82E-04	3.7E-05	8.7E-05	3.9E-7	--	3.69E-03	1.6E-04	3.7E-04	1.6E-6	--
chloroform	1.5E-03	2.0E-02	9.65E-03	4.1E-04	9.5E-04	6.0E-7	4.8E-2	5.27E-01	2.2E-02	5.2E-02	3.3E-5	2.6E+0
carbon tetrachloride		6.1E-04				--	--	3.05E-04	1.3E-05	3.0E-05	--	4.9E-2
1,3,5-trichlorobenzene		4.6E-04	1.50E-04	6.4E-06	1.5E-05	--	3.2E-2				--	--
1,4-dichlorobenzene		2.9E-01				--	--	1.88E-04	8.0E-06	1.9E-05	--	6.5E-5

↑
Modelled from maximum measured soil concentrations in areas where soil gas or flux data have not been collected (Table 3-5)

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Modelled from maximum measured sediment concentrations (refer to Table 3-6)

Inhalation of Volatile Chemicals above Soil Hot-Spots (where not addressed by measured soil gas or flux data)

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME) - Soils				Exposure Calculations (RME)- Sediments			
		Outdoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)				Outdoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)			
Exposure Parameters									
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks		240	Work for 5 days per week for 48 weeks			
Exposure Duration (ED)	years	30	Working life at the one location		30	Working life at the one location			
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996		70	USEPA 1989 and CSMS 1996			
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996		25550	USEPA 1989 and CSMS 1996			
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996		10950	USEPA 1989 and CSMS 1996			
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003		2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003			
Exposure Time (ET)	hours/day	1	Assume 1 hour outdoors		1	Assume 1 hour outdoors			
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation		100%	Assume 100% bioavailability via inhalation			
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume		100%	Assume all time outdoors spent in area above plume			
Intake Factor = InhR*ET*B*FI*EF*ED BW*AT	m ³ /kg/day	8.9E-03	NonThreshold		8.9E-03	NonThreshold			
		2.1E-02	Threshold		2.1E-02	Threshold			

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	1.2E-7	1.0E-2			TOTAL	8.9E-7	6.5E-2
TPH C6-C9 aliphatic		1.8E-01				--	--	1.34E-03	1.2E-05	2.8E-05	--	1.5E-4
TPH C10-C14 aromatic		5.1E-02				--	--	2.41E-03	2.1E-05	5.0E-05	--	9.7E-4
TPH C10-C14 aliphatic		2.6E-01				--	--	2.94E-03	2.6E-05	6.1E-05	--	2.4E-4
vinyl chloride (adult exposures)	1.5E-02		6.27E-04	5.5E-06	1.3E-05	8.5E-8	--	4.54E-03	4.0E-05	9.4E-05	6.2E-7	--
1,1-dichloroethene		5.7E-02				--	--				--	--
trans-1,2-dichloroethene		1.7E-02	1.25E-03	1.1E-05	2.6E-05	--	1.5E-3	1.58E-04	1.4E-06	3.3E-06	--	1.9E-4
1,1-dichloroethane		1.4E-01	2.71E-04	2.4E-06	5.6E-06	--	3.9E-5				--	--
cis-1,2-dichloroethene		1.0E-02	1.95E-03	1.7E-05	4.0E-05	--	4.0E-3	3.57E-03	3.2E-05	7.4E-05	--	7.4E-3
1,2-dichloroethane (EDC)	9.8E-03		5.32E-05	4.7E-07	1.1E-06	4.6E-9	--	3.52E-04	3.1E-06	7.3E-06	3.1E-8	--
trichloroethene (TCE)	1.5E-03		1.12E-03	1.0E-05	2.3E-05	1.5E-8	--	1.24E-02	1.1E-04	2.6E-04	1.7E-7	--
1,1,2-trichloroethane		4.0E-03	1.18E-05	1.0E-07	2.4E-07	--	6.1E-5	1.36E-03	1.2E-05	2.8E-05	--	7.0E-3
tetrachloroethene (PCE)		3.8E-02	1.07E-03	9.5E-06	2.2E-05	--	5.9E-4	8.43E-02	7.5E-04	1.7E-03	--	4.6E-2
1,1,2,2-tetrachloroethane	1.1E-02		1.21E-04	1.1E-06	2.5E-06	1.1E-8	--	5.05E-04	4.5E-06	1.0E-05	4.7E-8	--
chloroform	1.5E-03		4.61E-05	4.1E-07	9.5E-07	6.0E-10	4.8E-5	2.52E-03	2.2E-05	5.2E-05	3.3E-8	2.6E-3
carbon tetrachloride		6.1E-04				--	--	8.27E-07	7.3E-09	1.7E-08	--	2.8E-5
1,3,5-trichlorobenzene		4.6E-04	8.30E-05	7.4E-07	1.7E-06	--	3.7E-3				--	--
1,4-dichlorobenzene		2.9E-01				--	--	1.30E-05	1.2E-07	2.7E-07	--	9.4E-7

↑
Modelled from maximum measured soil concentrations in areas
where soil gas or flux data have not been collected (Table 3-5)

↑
Modelled from maximum measured sediment concentrations
(refer to Table 3-6)

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Floodvale Drain

Stage 1 and 2 Development

General Data/ Equations		Units	Exposure Calculations (RME)				
			Indoor/Outdoor Workers (Table 5-2, Scenario 4)				
Exposure Parameters							
Exposure Frequency (EF)		days/year	240	Work for 5 days per week for 48 weeks			
Exposure Duration (ED)		years	30	Working life at the one location			
Body Weight (BW)		kg	70	USEPA 1989 and CSMS 1996			
Averaging Time - NonThreshold (ATc)		days	25550	USEPA 1989 and CSMS 1996			
Averaging Time - Threshold (ATn)		days	10950	USEPA 1989 and CSMS 1996			
Inhalation Rate (InhR)		m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)			
Exposure Time (ET)		hours/day	10	Assume 9 hours indoors and 1 hr outdoors			
Bioavailability (B)		-	100%	Assume 100% bioavailability via inhalation			
Fraction Inhaled (FI)		-	100%	Assume all time spent at inside building located above plume			
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$		m ³ /kg/day	5.1E-02	NonThreshold			
			1.2E-01	Threshold			
<i>Daily Intake = Concentration in Air x Intake Factor</i>							
<i>NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor</i>							
<i>Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)</i>							
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		3.9E-6	
vinyl chloride (adult exposures)	1.5E-02		4.40E-04	2.3E-05	5.3E-05	3.5E-7	--
1,2-dichloroethane (EDC)	9.8E-03		7.00E-03	3.6E-04	8.4E-04	3.5E-6	--

↑
Measured air concentrations
Maximum measured adjacent to Floodvale Drain (Table 3-10)

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Springvale Drain

Stage 1 and 2 Development

General Data/ Equations		Exposure Calculations (Average)		Exposure Calculations (Average)	
Units		Indoor/Outdoor Work (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)		Outdoor Worker (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	4	Assume exposure by workers of up to 4 hours (trucks waiting)
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.1E-02	NonThreshold	3.5E-02	NonThreshold
		1.2E-01	Threshold	8.3E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	6.4E-4	8.0E+0			TOTAL	4.4E-4	5.5E+0		
vinyl chloride (adult exposures)	1.5E-02		4.30E-02	2.2E-03	5.1E-03	3.4E-5	--	4.30E-02	1.5E-03	3.6E-03	2.3E-5	--		
cis-1,2-dichloroethene		1.0E-02	4.00E-02	2.0E-03	4.8E-03	--	4.8E-1	4.00E-02	1.4E-03	3.3E-03	--	3.3E-1		
1,2-dichloroethane (EDC)	9.8E-03		1.19E+00	6.1E-02	1.4E-01	6.0E-4	--	1.19E+00	4.2E-02	9.9E-02	4.1E-4	--		
trichloroethene (TCE)	1.5E-03		8.90E-02	4.6E-03	1.1E-02	6.9E-6	--	8.90E-02	3.2E-03	7.4E-03	4.7E-6	--		
tetrachloroethene (PCE)		3.8E-02	8.60E-02	4.4E-03	1.0E-02	--	2.7E-1	8.60E-02	3.0E-03	7.1E-03	--	1.9E-1		
carbon tetrachloride		6.1E-04	3.70E-02	1.9E-03	4.4E-03	--	7.3E+0	3.70E-02	1.3E-03	3.1E-03	--	5.0E+0		

↑
Measured air concentrations
Average (of detected COPC) adjacent to Springvale Drain
Pre GTP (up to end 2006), refer to Table 3-10 in report

↑
Measured air concentrations
Average (of detected COPC) adjacent to Springvale Drain
Pre GTP (up to end 2006), refer to Table 3-10 in report

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Springvale Drain

Stage 1 and 2 Development

General Data/ Equations		Exposure Calculations (RME)		Exposure Calculations (RME)	
Units		Indoor/Outdoor Work (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)		Outdoor Worker (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	4	Assume exposure by workers of up to 4 hours (trucks waiting)
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.1E-02	NonThreshold	3.5E-02	NonThreshold
		1.2E-01	Threshold	8.3E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	1.3E-3	1.7E+1			TOTAL	9.0E-4	1.1E+1		
vinyl chloride (adult exposures)	1.5E-02		9.90E-02	5.1E-03	1.2E-02	7.8E-5	--	9.90E-02	3.5E-03	8.2E-03	5.4E-5	--		
cis-1,2-dichloroethene		1.0E-02	8.00E-02	4.1E-03	9.6E-03	--	9.6E-1	8.00E-02	2.8E-03	6.6E-03	--	6.6E-1		
1,2-dichloroethane (EDC)	9.8E-03		2.40E+00	1.2E-01	2.9E-01	1.2E-3	--	2.40E+00	8.5E-02	2.0E-01	8.3E-4	--		
trichloroethene (TCE)	1.5E-03		1.80E-01	9.2E-03	2.2E-02	1.4E-5	--	1.80E-01	6.4E-03	1.5E-02	9.6E-6	--		
tetrachloroethene (PCE)		3.8E-02	1.60E-01	8.2E-03	1.9E-02	--	5.1E-1	1.60E-01	5.7E-03	1.3E-02	--	3.5E-1		
carbon tetrachloride		6.1E-04	7.70E-02	3.9E-03	9.2E-03	--	1.5E+1	7.70E-02	2.7E-03	6.4E-03	--	1.0E+1		

↑
Measured air concentrations
Maximum measured adjacent to Springvale or Floodvale Drains
Pre GTP (up to end 2006), refer to Table 3-10 in report

↑
Measured air concentrations
Maximum measured adjacent to Springvale or Floodvale Drains
Pre GTP (up to end 2006), refer to Table 3-10 in report

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Springvale Drain

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME post GTP)		Exposure Calculations (RME post GTP)	
		Indoor/Outdoor Work (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)		Outdoor Worker (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	4	Assume exposure by workers of up to 4 hours (trucks waiting)
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.1E-02	NonThreshold	3.5E-02	NonThreshold
		1.2E-01	Threshold	8.3E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	5.3E-4	6.8E+0			TOTAL	3.6E-4	4.7E+0		
vinyl chloride (adult exposures)	1.5E-02		4.30E-02	2.2E-03	5.1E-03	3.4E-5	--	4.30E-02	1.5E-03	3.6E-03	2.3E-5	--		
cis-1,2-dichloroethene		1.0E-02	1.00E-01	5.1E-03	1.2E-02	--	1.2E+0	1.00E-01	3.5E-03	8.3E-03	--	8.3E-1		
1,2-dichloroethane (EDC)	9.8E-03		9.30E-01	4.8E-02	1.1E-01	4.7E-4	--	9.30E-01	3.3E-02	7.7E-02	3.2E-4	--		
trichloroethene (TCE)	1.5E-03		1.30E-01	6.7E-03	1.6E-02	1.0E-5	--	1.30E-01	4.6E-03	1.1E-02	6.9E-6	--		
tetrachloroethene (PCE)		3.8E-02	3.80E-02	1.9E-03	4.5E-03	--	1.2E-1	3.80E-02	1.3E-03	3.1E-03	--	8.3E-2		
carbon tetrachloride		6.1E-04	2.80E-02	1.4E-03	3.3E-03	--	5.5E+0	2.80E-02	9.9E-04	2.3E-03	--	3.8E+0		
1,4-dioxane	2.7E-02	8.6E-01	1.00E-03	5.1E-05	1.2E-04	1.4E-6	1.4E-4	4.20E-04	1.5E-05	3.5E-05	4.0E-7	4.1E-5		
1,3-butadiene	6.0E-01	5.7E-03	4.20E-04	2.2E-05	5.0E-05	1.3E-5	8.8E-3	4.20E-04	1.5E-05	3.5E-05	8.9E-6	6.1E-3		

↑
Measured air concentrations
Maximum reported following commissioning of GTP
Post GTP (2007 onwards), refer to Table 3-10 in report

↑
Measured air concentrations
Maximum reported following commissioning of GTP
Post GTP (2007 onwards), refer to Table 3-10 in report

Concentrations in Air from Flood Storage Area with GW Discharge

(Reference: Air Emissions Models for Waste and Wastewater, USEPA 1994)

General Parameters

U =	Windspeed (m/s)
depth =	Depth of water body (m)
d =	Diameter of water body (m)
A =	Area of water body (m ²)
F/D =	Fetch to depth ratio (unitless)
Q =	Flow (m ³ /s)
H =	Height of mixing zone (m)
Ws	Windspeed at surface (m/s)
T =	Absolute Temperature (K)
R =	Universal gas constant (atm.m ³ /g.mol.K)
De =	Diffusivity of ether in water (cm ² /s)
ug =	Viscosity of air (g/cm.s)
pg =	Density of air (g/cm ³)

All Calculations	
	5
	0.01
	170
	8500.0
	17000
	0.015
	10.0
	2
	293
	8.21E-05
	8.50E-06
	1.81E-04
	1.20E-03

estimated to grass slope 170m long and 1:10 slope

Chemical Parameters

Cw =	Concentration in water (mg/L)
C =	Concentration in water (g/m ³)
H =	Henry's Law (atm.m ³ /g.mol)
MW =	Molecular Weight
Da =	Diffusivity in air (cm ² /s)
Dw =	Diffusivity in water (cm ² /s)
log Kow =	log octanol/water partition coeff. (L/kg)

	vinyl chloride	1,1-dichloroethene	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	chloroform	carbon tetrachloride	dichloromethane	HCBD
Cw	6.8	0.056	0.54	4.8	9.9	1.2	0.21	1.8	0.95	2.3	0.056	0.036
C	6.80E+00	5.60E-02	5.40E-01	4.80E+00	9.90E+00	1.20E+00	2.10E-01	1.80E+00	9.50E-01	2.30E+00	5.60E-02	3.60E-02
H	0.027	0.0261	0.00938	0.00408	0.0011	0.0103	0.000913	0.0184	0.00367	0.03	0.00219	0.0103
MW	62.5	96.95	96.95	96.95	78.9	131.4	133.4	165.83	119.38	153.8	84.93	260.76
Da	0.106	0.09	0.0707	0.0736	0.104	0.079	0.078	0.072	0.104	0.078	0.101	0.0561
Dw	1.20E-06	1.04E-05	1.20E-05	1.10E-05	9.90E-06	9.10E-06	0.0000088	8.20E-06	1.00E-05	8.80E-06	1.17E-05	6.16E-06
log Kow	1.62	2.13	2.09	2.1	1.5	2.42	1.9	3.4	2.0	2.8	1.3	4.8

Calculations

SCg =	Schmidt number on gas side
Kl =	Liquid Phase Mass Transfer Coefficient (m/s)
Kg =	Gas Phase Mass Transfer Coefficient (m/s)
Keq =	Equilibrium constant (unitless)
K =	Overall Mass Transfer Coefficient (m/s)
CL =	Equilibrium Concentration in basin (g/m ³)
E =	Emission rate from water surface (g/s)
Cb =	Concentration in breathing zone (g/m ³)

	vinyl chloride	1,1-dichloroethene	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	chloroform	carbon tetrachloride	dichloromethane	HCBD
SCg	1.42	1.68	2.13	2.05	1.45	1.91	1.93	2.09	1.45	1.93	1.49	2.69
Kl	3.01E-04	1.27E-03	1.40E-03	1.32E-03	1.23E-03	1.16E-03	1.14E-03	1.08E-03	1.24E-03	1.14E-03	1.37E-03	8.96E-04
Kg	4.42E-03	3.96E-03	3.37E-03	3.46E-03	4.36E-03	3.63E-03	3.60E-03	3.41E-03	4.36E-03	3.60E-03	4.28E-03	2.89E-03
Keq	1.12E+00	1.08E+00	3.90E-01	1.70E-01	4.57E-02	4.28E-01	3.80E-02	7.65E-01	1.53E-01	1.25E+00	9.10E-02	4.28E-01
K	2.84E-04	9.80E-04	6.77E-04	4.06E-04	1.72E-04	6.65E-04	1.22E-04	7.66E-04	4.33E-04	9.07E-04	3.04E-04	5.19E-04
CL	4.20E-02	1.01E-04	1.40E-03	2.08E-02	1.01E-01	3.18E-03	3.00E-03	4.14E-03	3.86E-03	4.47E-03	3.24E-04	1.22E-04
E	1.01E-01	8.38E-04	8.08E-03	7.17E-02	1.47E-01	1.80E-02	3.11E-03	2.69E-02	1.42E-02	3.44E-02	8.35E-04	5.38E-04
Cb	1.01E-04	8.38E-07	8.08E-06	7.17E-05	1.47E-04	1.80E-05	3.11E-06	2.69E-05	1.42E-05	3.44E-05	8.35E-07	5.38E-07

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS
Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)

Assessment of Vapour Migration - Block 1 Southlands - Flood Storage Area

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Depth of Top of Contaminated Aquifer (BGS)	[m]	d	0.5	Calculated from layers
Thickness of Capillary Fringe	[m]	cd	0.02	Estimated for soil types
Thickness of Vadose Zone	[m]	vd	0.48	Calculated from layers
Soil Temperature	[C]	T	17	Assumed
Vadose Zone Layer 1 Characteristics				Fill
Depth of Layer 1	[m]	vd1	0.48	Assume average depth to GW is 50cm
Moisture Content	[g/g]	mocon	0.08	Conservative value for soil materials
Organic Carbon Fraction	-	foc	0.003	Assumed for fill
Soil Bulk Density	[g/ml]	rhob	1.7	Assumed for fill
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.36	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.136	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.222	theta-wacon
Capillary Fringe				
Volumetric Water Content	[ml/ml]	cfwacon	0.34	estimated
Volumetric Air Content	[ml/ml]	cfacon	0.02	theta2-cfwacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	20	area assumed to contribute to outdoor concentration
Width of Contaminated Area	[m]	width	20	area assumed to contribute to outdoor concentration
Outdoor Area	[m ²]	oarea	400	calculated
Q/C Dispersion term	[g/m ² /s per kg/m ³]	qc	9.28E+01	Calculated for climate similar to East coast of Australia

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
1,1-dichloroethene	2420	96.95	35.04	0.09	1.0E-05	634	1.07
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
carbon tetrachloride	800	153.8	48.64	0.078	8.8E-06	115	1.2
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133
hexachlorobutadiene (HCBD)	3.2	260.76	993.5	0.0561	6.2E-06	0.22	0.421

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SUBSURFACE IMPACTS
Using Vapour Migration Model Presented by Farmer (1980) and DEH (2000)
Assessment of Vapour Migration - Block 1 Southlands - Flood Storage Area

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Capillary Fringe (cm ² /s)	Total Effective Diffusion (GW to surface) (cm ² /s)
vinyl chloride (adult exposures)	5.50E-3		2.02E-6	5.00E-5
1,1-dichloroethene	4.67E-3		3.57E-6	8.75E-5
trans-1,2-dichloroethene	3.67E-3		7.77E-6	1.85E-4
cis-1,2-dichloroethene	3.82E-3		1.51E-5	3.45E-4
1,2-dichloroethane (EDC)	5.40E-3		4.50E-5	9.37E-4
trichloroethene (TCE)	4.10E-3		5.87E-6	1.42E-4
1,1,2-trichloroethane	4.05E-3		5.08E-5	9.76E-4
tetrachloroethene (PCE)	3.74E-3		3.50E-6	8.57E-5
chloroform	5.40E-3		1.58E-5	3.69E-4
carbon tetrachloride	4.05E-3		2.86E-6	7.03E-5
dichloromethane	5.25E-3		2.02E-5	4.62E-4
hexachlorobutadiene (HCBD)	2.91E-3		4.03E-6	9.74E-5

Phase Partitioning Results	Dissolved Phase Concentration (mg/L)	Vapour Phase Concentration (g/cm ³)	Saturated Vapour Concentration (g/cm ³)	Free Phase Mole Fraction (mol/mol)	Concentration above Free Phase (g/cm ³)	Calculation Vapour Phase used in Calculation (g/cm ³)
vinyl chloride (adult exposures)	7.3	8.1E-06	9.0E-03	0	0.0E+00	8.1E-06
1,1-dichloroethene	0.058	6.2E-08	3.4E-03	0	0.0E+00	6.2E-08
trans-1,2-dichloroethene	0.78	3.0E-07	1.4E-03	0	0.0E+00	3.0E-07
cis-1,2-dichloroethene	5.2	8.7E-07	9.6E-04	0	0.0E+00	8.7E-07
1,2-dichloroethane (EDC)	7.2	3.5E-07	4.3E-04	0	0.0E+00	3.5E-07
trichloroethene (TCE)	1.2	5.1E-07	5.4E-04	0	0.0E+00	5.1E-07
1,1,2-trichloroethane	0.16	6.0E-09	1.7E-04	0	0.0E+00	6.0E-09
tetrachloroethene (PCE)	1.3	9.8E-07	1.7E-04	0	0.0E+00	9.8E-07
chloroform	0.75	1.1E-07	1.1E-03	0	0.0E+00	1.1E-07
carbon tetrachloride	1.8	2.2E-06	9.8E-04	0	0.0E+00	2.2E-06
dichloromethane	0.055	7.3E-09	2.0E-03	0	0.0E+00	7.3E-09
hexachlorobutadiene (HCBD)	0.023	9.7E-09	3.2E-06	0	0.0E+00	9.7E-09

Calculated Air Concentrations ASTM Guidance	Vapour Phase Concentration at Source - Modelled from GW (g/cm ³)	Calculated Emission Rate from Surface of Ground - Modelled from GW (g/s/m ²)	Outdoor Air Concentration Q/C (mg/m ³)	TWA (mg/m ³)
vinyl chloride (adult exposures)		8.1E-08		13
1,1-dichloroethene	8.1E-06	1.1E-09	8.7E-04	20
trans-1,2-dichloroethene	6.2E-08	1.1E-08	1.2E-05	793
cis-1,2-dichloroethene	3.0E-07	6.0E-08	1.2E-04	793
1,2-dichloroethane (EDC)	8.7E-07	6.5E-08	6.5E-04	40
trichloroethene (TCE)	3.5E-07	1.4E-08	7.0E-04	54
1,1,2-trichloroethane	5.1E-07	1.2E-09	1.5E-04	55
tetrachloroethene (PCE)	6.0E-09	1.7E-08	1.3E-05	335
chloroform	9.8E-07	8.3E-09	1.8E-04	10
carbon tetrachloride	1.1E-07	3.0E-08	8.9E-05	0.63
dichloromethane	2.2E-06	6.8E-10	3.3E-04	174
hexachlorobutadiene (HCBD)	7.3E-09	1.9E-10	7.3E-06	0.21

Inhalation of Volatile Chemicals in Areas Adjacent to Flood Storage Area

Stage 1 Development

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Outdoors - With GW Discharge to SW (Table 5-2, Scenario 7)		Outdoors - No SW Discharge (Table 5-2, Scenario 8)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate for adult walking at 6.4 km/hr, enHealth 2003	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	8	Assume 8 hours outdoors	8	Assume 8 hours outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	18%	Assume GW only discharges 18% of the time	82%	Remaining 82% of time, no GW discharge or saturates soils overlying
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	1.3E-02	NonThreshold	5.8E-02	NonThreshold
		3.0E-02	Threshold	1.4E-01	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting)	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	3.9E-5	2.1E+0			TOTAL	1.2E-6	8.6E-2
vinyl chloride (adult exposures)	1.5E-02		1.01E-01	1.3E-03	3.0E-03	2.0E-5	--	8.72E-04	5.1E-05	1.2E-04	7.8E-7	--
1,1-dichloroethene		5.7E-02	8.38E-04	1.1E-05	2.5E-05	--	4.4E-4	1.17E-05	6.8E-07	1.6E-06	--	2.8E-5
trans-1,2-dichloroethene		1.7E-02	8.08E-03	1.0E-04	2.4E-04	--	1.4E-2	1.19E-04	6.9E-06	1.6E-05	--	9.5E-4
cis-1,2-dichloroethene		1.0E-02	7.17E-02	9.1E-04	2.1E-03	--	2.1E-1	6.45E-04	3.7E-05	8.7E-05	--	8.7E-3
1,2-dichloroethane (EDC)	9.8E-03		1.47E-01	1.9E-03	4.4E-03	1.8E-5	--	7.01E-04	4.1E-05	9.5E-05	4.0E-7	--
trichloroethene (TCE)	1.5E-03		1.80E-02	2.3E-04	5.3E-04	3.4E-7	--	1.55E-04	9.0E-06	2.1E-05	1.4E-8	--
1,1,2-trichloroethane		4.0E-03	3.11E-03	4.0E-05	9.2E-05	--	2.3E-2	1.26E-05	7.3E-07	1.7E-06	--	4.3E-4
tetrachloroethene (PCE)		3.8E-02	2.69E-02	3.4E-04	8.0E-04	--	2.1E-2	1.81E-04	1.1E-05	2.5E-05	--	6.5E-4
chloroform	1.5E-03		1.42E-02	1.8E-04	4.2E-04	2.7E-7	2.1E-2	8.94E-05	5.2E-06	1.2E-05	7.6E-9	6.1E-4
carbon tetrachloride		6.1E-04	3.44E-02	4.4E-04	1.0E-03	--	1.7E+0	3.27E-04	1.9E-05	4.4E-05	--	7.3E-2
dichloromethane		2.3E-01	8.35E-04	1.1E-05	2.5E-05	--	1.1E-4	7.29E-06	4.2E-07	9.9E-07	--	4.3E-6
hexachlorobutadiene (HCBd)		2.0E-04	5.38E-04	6.9E-06	1.6E-05	--	8.0E-2	2.03E-06	1.2E-07	2.8E-07	--	1.4E-3

↑
Modelled assuming discharge of shallow GW

↑
Modelled assuming shallow groundwater remains, on average, 50cm below drainage surface

Inhalation of Volatile Chemicals in Adjacent to Flood Storage Area (Evaluated using Occupational TWA Values)

Stage 1 Development

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Outdoors - With GW Discharge to SW (Table 5-2, Scenario 7)		Outdoors - No SW Discharge (Table 5-2, Scenario 8)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	8	Assume 8 hours outdoors	8	Assume 8 hours outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	18%	Assume GW only discharges 18% of the time	82%	Assume overlying soils dry for 82% of the year
Intake Factor = InhR*ET*B*FI*EF*ED BW*AT	m ³ /kg/day	1.3E-02 3.0E-02	NonThreshold Threshold	5.8E-02 1.4E-01	NonThreshold Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	
					TOTAL		3.9E-5	1.5E-2			TOTAL		1.2E-6	6.0E-4
vinyl chloride (adult exposures)	1.5E-02	1.9E+00	1.01E-01		3.0E-03		2.0E-5	1.6E-3	8.72E-04		1.2E-04		7.8E-7	6.4E-5
1,1-dichloroethene		2.9E+00	8.38E-04		2.5E-05		--	8.7E-6	1.17E-05		1.6E-06		--	5.6E-7
trans-1,2-dichloroethene		1.1E+02	8.08E-03		2.4E-04		--	2.1E-6	1.19E-04		1.6E-05		--	1.4E-7
cis-1,2-dichloroethene		1.1E+02	7.17E-02		2.1E-03		--	1.9E-5	6.45E-04		8.7E-05		--	7.7E-7
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.47E-01		4.4E-03		1.8E-5	7.7E-4	7.01E-04		9.5E-05		4.0E-7	1.7E-5
trichloroethene (TCE)	1.5E-03	7.7E+00	1.80E-02		5.3E-04		3.4E-7	6.9E-5	1.55E-04		2.1E-05		1.4E-8	2.7E-6
1,1,2-trichloroethane		7.9E+00	3.11E-03		9.2E-05		--	1.2E-5	1.26E-05		1.7E-06		--	2.2E-7
tetrachloroethene (PCE)		4.8E+01	2.69E-02		8.0E-04		--	1.7E-5	1.81E-04		2.5E-05		--	5.1E-7
chloroform	1.5E-03	1.4E+00	1.42E-02		4.2E-04		2.7E-7	3.0E-4	8.94E-05		1.2E-05		7.6E-9	8.5E-6
carbon tetrachloride		9.0E-02	3.44E-02		1.0E-03		--	1.1E-2	3.27E-04		4.4E-05		--	4.9E-4
dichloromethane		2.5E+01	8.35E-04		2.5E-05		--	1.0E-6	7.29E-06		9.9E-07		--	4.0E-8
hexachlorobutadiene (HCBD)		3.0E-02	5.38E-04		1.6E-05		--	5.3E-4	2.03E-06		2.8E-07		--	9.2E-6

Modelled assuming discharge of shallow GW

Modelled assuming shallow groundwater remains, on average, 50cm below drainage surface

Exposure to Chemicals via Ingestion of Soils

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Ingestion of Soils by Workers (Table 5-2, Scenario 9)		Ingestion of Soils by Gardeners (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Ingestion Rate (IRs)	mg/day	25	Soil intake for adults as per CSMS 1996	25	Soil intake for adults as per CSMS 1996
Fraction Ingested (FI)	-	100%	All soil ingested from site	100%	All soil ingested from site
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in soil	100%	Assume 100% bioavailability via ingestion of chemicals in soil
Conversion factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Intake Factor = IRs*FI*B*CF*EF*ED	kg/kg/day	1.0E-07	NonThreshold	1.1E-08	NonThreshold
BW*AT		2.3E-07	Threshold	2.5E-08	Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		7.2E-6	3.8E+0		TOTAL		7.8E-7	4.2E-1
mercury (as total, inorganic)		5.4E-04	1233	1.2E-04	2.9E-04	--	5.4E-1	1233	1.3E-05	3.1E-05	--	5.8E-2
chromium (assumed Cr(VI))		2.1E-03	1500	1.5E-04	3.5E-04	--	1.7E-1	1500	1.6E-05	3.8E-05	--	1.8E-2
lead		1.9E-03	7670	7.7E-04	1.8E-03	--	9.7E-1	7670	8.4E-05	2.0E-04	--	1.1E-1
benzene	3.5E-02		31.3	3.2E-06	7.4E-06	1.1E-7	--	31.3	3.4E-07	8.0E-07	1.2E-8	--
ethylbenzene		9.7E-02	141	1.4E-05	3.3E-05	--	3.4E-4	141	1.5E-06	3.6E-06	--	3.7E-5
xylenes		1.8E-01	41.4	4.2E-06	9.7E-06	--	5.5E-5	41.4	4.5E-07	1.1E-06	--	6.0E-6
TPH C6-C9 aliphatic		4.5E+00	12100	1.2E-03	2.8E-03	--	6.3E-4	12100	1.3E-04	3.1E-04	--	6.8E-5
TPH C10-C14 aromatic		4.0E-02	1220	1.2E-04	2.9E-04	--	7.2E-3	1220	1.3E-05	3.1E-05	--	7.8E-4
TPH C10-C14 aliphatic		9.0E-02	1220	1.2E-04	2.9E-04	--	3.2E-3	1220	1.3E-05	3.1E-05	--	3.4E-4
TPH C15+ aromatic		2.7E-02	2795	2.8E-04	6.6E-04	--	2.4E-2	2795	3.0E-05	7.1E-05	--	2.6E-3
TPH C15+ aliphatic		1.8E+00	2795	2.8E-04	6.6E-04	--	3.6E-4	2795	3.0E-05	7.1E-05	--	4.0E-5
vinyl chloride (adult exposures)	1.2E+00		53	5.3E-06	1.2E-05	6.1E-6	--	53	5.8E-07	1.3E-06	6.6E-7	--
cis-1,2-dichloroethene		1.0E-02	165	1.7E-05	3.9E-05	--	3.9E-3	165	1.8E-06	4.2E-06	--	4.2E-4
1,2-dichloroethane (EDC)	1.2E-02		4.5	4.5E-07	1.1E-06	5.4E-9	--	4.5	4.9E-08	1.1E-07	5.9E-10	--
trichloroethene (TCE)		1.5E-03	95.1	9.6E-06	2.2E-05	--	1.5E-2	95.1	1.0E-06	2.4E-06	--	1.7E-3
1,1,2-trichloroethane		4.0E-03	2	2.0E-07	4.7E-07	--	1.2E-4	2	2.2E-08	5.1E-08	--	1.3E-5
tetrachloroethene (PCE)		9.2E-03	90.8	9.1E-06	2.1E-05	--	2.3E-3	90.8	9.9E-07	2.3E-06	--	2.5E-4
1,1,2,2-tetrachloroethane		4.0E-02	10.2	1.0E-06	2.4E-06	--	6.0E-5	10.2	1.1E-07	2.6E-07	--	6.5E-6
chloroform		6.5E-03	3.9	3.9E-07	9.2E-07	--	1.4E-4	3.9	4.3E-08	9.9E-08	--	1.5E-5
PAHs (BaP equivalent)	5.0E-01		14.1	1.4E-06	3.3E-06	7.1E-7	--	14.1	1.5E-07	3.6E-07	7.7E-8	--
hexachlorobutadiene (HCBd)		2.0E-04	326	3.3E-05	7.7E-05	--	3.8E-1	326	3.6E-06	8.3E-06	--	4.1E-2
hexachlorobenzene (HCB)		1.6E-04	1170	1.2E-04	2.7E-04	--	1.7E+0	1170	1.3E-05	3.0E-05	--	1.9E-1
bis(2-chloroethyl)ether	1.1E+00		2.1	2.1E-07	4.9E-07	2.3E-7	--	2.1	2.3E-08	5.3E-08	2.5E-8	--

↑
Maximum concentration reported in Soils from Block 1 and Block 2
Maximum reported from Tables 3-3 and 3-4 in report

↑
Maximum concentration reported in Soils from Block 1 and Block 2
Maximum reported from Tables 3-3 and 3-4 in report

Dermal Exposure to Chemicals via Contact with Soil

Stage 1 and 2 Development (Hawley Method)

General Data/ Equations		Exposure Calculations (RME)		Exposure Calculations (RME)	
Units		Dermal Contact with Soils by Workers (Table 5-2, Scenario 9)		Dermal Contact with Soils by Gardeners (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forarms and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Dermal Absorption Rate (Abs)	per hour	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996
Exposure Time (ET)	hr/day	12	Assume that soil remains on skin for 12 hours before washing	12	Assume that soil remains on skin for 12 hours before washing
Matrix Effect (ME)	-	15%	Absorption of chemicals from soil matrix is 15% from pure compound	15%	Absorption of chemicals from soil matrix is 15% from pure compound
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -		1.00	Not used for intrusive works	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	4.2E-08 9.9E-08	NonThreshold Threshold	9.7E-09 2.3E-08	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor
 NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
 Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Soil	Daily Intake		Calculated Risk		Concentration in Soil	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		3.0E-6	9.1E-1		TOTAL		7.0E-7	2.1E-1
benzene	3.5E-02		31.3	1.3E-06	3.1E-06	4.7E-8	--	31.3	3.0E-07	7.1E-07	1.1E-8	--
ethylbenzene		9.7E-02	141	6.0E-06	1.4E-05	--	1.4E-4	141	1.4E-06	3.2E-06	--	3.3E-5
xylenes		1.8E-01	41.4	1.8E-06	4.1E-06	--	2.3E-5	41.4	4.0E-07	9.4E-07	--	5.4E-6
TPH C6-C9 aliphatic		4.5E+00	12100	5.1E-04	1.2E-03	--	2.7E-4	12100	1.2E-04	2.7E-04	--	6.1E-5
TPH C10-C14 aromatic		4.0E-02	1220	5.2E-05	1.2E-04	--	3.0E-3	1220	1.2E-05	2.8E-05	--	6.9E-4
TPH C10-C14 aliphatic		9.0E-02	1220	5.2E-05	1.2E-04	--	1.3E-3	1220	1.2E-05	2.8E-05	--	3.1E-4
TPH C15+ aromatic		3.0E-02	2795	1.2E-04	2.8E-04	--	9.2E-3	2795	2.7E-05	6.3E-05	--	2.1E-3
TPH C15+ aliphatic		1.8E+00	2795	1.2E-04	2.8E-04	--	1.5E-4	2795	2.7E-05	6.3E-05	--	3.5E-5
vinyl chloride (adult exposures)	1.2E+00		53	2.3E-06	5.3E-06	2.6E-6	--	53	5.2E-07	1.2E-06	5.9E-7	--
cis-1,2-dichloroethene		1.0E-02	165	7.0E-06	1.6E-05	--	1.6E-3	165	1.6E-06	3.7E-06	--	3.7E-4
1,2-dichloroethane (EDC)	1.2E-02		4.5	1.9E-07	4.5E-07	2.3E-9	--	4.5	4.4E-08	1.0E-07	5.3E-10	--
trichloroethene (TCE)		1.5E-03	95.1	4.0E-06	9.4E-06	--	6.5E-3	95.1	9.3E-07	2.2E-06	--	1.5E-3
1,1,2-trichloroethane		4.0E-03	2	8.5E-08	2.0E-07	--	5.0E-5	2	1.9E-08	4.5E-08	--	1.1E-5
tetrachloroethene (PCE)		1.4E-02	90.8	3.9E-06	9.0E-06	--	6.4E-4	90.8	8.8E-07	2.1E-06	--	1.5E-4
1,1,2,2-tetrachloroethane		4.0E-02	10.2	4.3E-07	1.0E-06	--	2.5E-5	10.2	9.9E-08	2.3E-07	--	5.8E-6
chloroform		1.3E-02	3.9	1.7E-07	3.9E-07	--	3.0E-5	3.9	3.9E-08	8.9E-08	--	6.8E-6
PAHs (BaP equivalent)	5.0E-01		14.1	6.0E-07	1.4E-06	3.0E-7	--	14.1	1.4E-07	3.2E-07	6.9E-8	--
hexachlorobutadiene (HCBBD)		2.0E-04	326	1.4E-05	3.2E-05	--	1.6E-1	326	3.2E-06	7.4E-06	--	3.7E-2
hexachlorobenzene (HCB)		1.6E-04	1170	5.0E-05	1.2E-04	--	7.3E-1	1170	1.1E-05	2.7E-05	--	1.7E-1
bis(2-chloroethyl)ether	1.1E+00		2.1	8.9E-08	2.1E-07	--	9.8E-8	2.1	2.0E-08	4.8E-08	2.2E-8	--

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

Dermal Exposure to Chemicals via Contact with Soil
Stage 1 and 2 Development (USEPA Method)

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Dermal Contact with Soils by Workers (Table 5-2, Scenario 9)		Dermal Contact with Soils by Gardeners (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forarms and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Fraction of Day Exposed (FE)	-	0.5	12 hour exposure, CSMS 1991, 1993 and 1998	0.5	12 hour exposure, CSMS 1991, 1993 and 1998
Matrix Effect (ME)	-	100%	Assume 100% bioavailability, CSMS 1996	100%	Assume 100% bioavailability, CSMS 1996
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -	-	1.00	Not used	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot FE \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	2.4E-06 5.5E-06	NonThreshold Threshold	5.4E-07 1.3E-06	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Absorption x Intake Factor
 NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
 Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity and Dermal Data			Concentration in Soil (mg/kg)	Daily Intake (mg/kg/day)		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake (mg/kg/day)		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	Dermal Absorption		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(unitless)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	3.7E-6	5.9E-1			TOTAL	8.4E-7	1.3E-1
benzene	3.5E-02		0.01	31.3	7.4E-07	1.7E-06	2.6E-8	--	31.3	1.7E-07	3.9E-07	5.9E-9	--
ethylbenzene		9.7E-02	0.01	141	3.3E-06	7.8E-06	--	8.0E-5	141	7.6E-07	1.8E-06	--	1.8E-5
xylenes		1.8E-01	0.01	41.4	9.8E-07	2.3E-06	--	1.3E-5	41.4	2.2E-07	5.2E-07	--	3.0E-6
TPH C6-C9 aliphatic		4.5E+00	0.01	12100	2.9E-04	6.7E-04	--	1.5E-4	12100	6.5E-05	1.5E-04	--	3.4E-5
TPH C10-C14 aromatic		4.0E-02	0.13	1220	3.7E-04	8.7E-04	--	2.2E-2	1220	8.6E-05	2.0E-04	--	5.0E-3
TPH C10-C14 aliphatic		9.0E-02	0.01	1220	2.9E-05	6.7E-05	--	7.5E-4	1220	6.6E-06	1.5E-05	--	1.7E-4
TPH C15+ aromatic		3.0E-02	0.13	2795	8.6E-04	2.0E-03	--	6.7E-2	2795	2.0E-04	4.6E-04	--	1.5E-2
TPH C15+ aliphatic		1.8E+00	0.01	2795	6.6E-05	1.5E-04	--	8.6E-5	2795	1.5E-05	3.5E-05	--	2.0E-5
vinyl chloride (adult exposures)	1.2E+00		0.01	53	1.3E-06	2.9E-06	1.4E-6	--	53	2.9E-07	6.7E-07	3.3E-7	--
cis-1,2-dichloroethene		1.0E-02	0.01	165	3.9E-06	9.1E-06	--	9.1E-4	165	8.9E-07	2.1E-06	--	2.1E-4
1,2-dichloroethane (EDC)	1.2E-02		0.01	4.5	1.1E-07	2.5E-07	1.3E-9	--	4.5	2.4E-08	5.7E-08	2.9E-10	--
trichloroethene (TCE)		1.5E-03	0.01	95.1	2.2E-06	5.2E-06	--	3.6E-3	95.1	5.1E-07	1.2E-06	--	8.2E-4
1,1,2-trichloroethane		4.0E-03	0.01	2	4.7E-08	1.1E-07	--	2.8E-5	2	1.1E-08	2.5E-08	--	6.3E-6
tetrachloroethene (PCE)		9.2E-03	0.01	90.8	2.1E-06	5.0E-06	--	5.4E-4	90.8	4.9E-07	1.1E-06	--	1.2E-4
1,1,2,2-tetrachloroethane		4.0E-02	0.01	10.2	2.4E-07	5.6E-07	--	1.4E-5	10.2	5.5E-08	1.3E-07	--	3.2E-6
chloroform		6.5E-03	0.01	3.9	9.2E-08	2.1E-07	--	3.3E-5	3.9	2.1E-08	4.9E-08	--	7.6E-6
PAHs (BaP equivalent)	5.0E-01		0.13	14.1	4.3E-06	1.0E-05	2.2E-6	--	14.1	9.9E-07	2.3E-06	5.0E-7	--
hexachlorobutadiene (HCBD)		2.0E-04	0.01	326	7.7E-06	1.8E-05	--	9.0E-2	326	1.8E-06	4.1E-06	--	2.1E-2
hexachlorobenzene (HCB)		1.6E-04	0.01	1170	2.8E-05	6.4E-05	--	4.0E-1	1170	6.3E-06	1.5E-05	--	9.2E-2
bis(2-chloroethyl)ether	1.1E+00		0.01	2.1	5.0E-08	1.2E-07	5.5E-8	--	2.1	1.1E-08	2.6E-08	1.2E-8	--

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

Exposure to Chemicals via Ingestion of Sediments

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Ingestion of Sediments by Workers (Table 5-2, Scenario 9)		Ingestion of Sediments during Maintenance of Ponds (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	20	Assume 1 months work in pond areas
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	6	Assume maintenance works occur once every 5 years for 30 years
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	2190	USEPA 1989 and CSMS 1996
Ingestion Rate (IRs)	mg/day	25	Soil intake for adults as per CSMS 1996	25	Soil intake for adults as per CSMS 1996
Fraction Ingested (FI)	-	100%	All soil ingested from site	100%	All soil ingested from site
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in soil	100%	Assume 100% bioavailability via ingestion of chemicals in soil
Conversion factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Intake Factor = $\frac{IRs \cdot FI \cdot B \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg/kg/day	1.0E-07	NonThreshold	1.7E-09	NonThreshold
		2.3E-07	Threshold	2.0E-08	Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	4.4E-5	5.4E-1			TOTAL	7.4E-7	4.5E-2
chromium (assumed Cr(VI))		2.1E-03	188	1.9E-05	4.4E-05	--	2.1E-2	188	3.2E-07	3.7E-06	--	1.8E-3
TPH C6-C9 aliphatic		4.5E+00	113	1.1E-05	2.7E-05	--	5.9E-6	113	1.9E-07	2.2E-06	--	4.9E-7
TPH C10-C14 aromatic		4.0E-02	249	2.5E-05	5.8E-05	--	1.5E-3	249	4.2E-07	4.9E-06	--	1.2E-4
TPH C10-C14 aliphatic		9.0E-02	249	2.5E-05	5.8E-05	--	6.5E-4	249	4.2E-07	4.9E-06	--	5.4E-5
TPH C15+ aromatic		2.7E-02	1730	1.7E-04	4.1E-04	--	1.5E-2	1730	2.9E-06	3.4E-05	--	1.3E-3
TPH C15+ aliphatic		1.8E+00	1730	1.7E-04	4.1E-04	--	2.3E-4	1730	2.9E-06	3.4E-05	--	1.9E-5
vinyl chloride (adult exposures)	1.2E+00		384	3.9E-05	9.0E-05	4.4E-5	--	384	6.4E-07	7.5E-06	7.4E-7	--
cis-1,2-dichloroethene		1.0E-02	302	3.0E-05	7.1E-05	--	7.1E-3	302	5.1E-07	5.9E-06	--	5.9E-4
1,2-dichloroethane (EDC)	1.2E-02		29.8	3.0E-06	7.0E-06	3.6E-8	--	29.8	5.0E-08	5.8E-07	6.0E-10	--
trichloroethene (TCE)		1.5E-03	1050	1.1E-04	2.5E-04	--	1.7E-1	1050	1.8E-06	2.1E-05	--	1.4E-2
1,1,2-trichloroethane		4.0E-03	115	1.2E-05	2.7E-05	--	6.8E-3	115	1.9E-07	2.3E-06	--	5.6E-4
tetrachloroethene (PCE)		9.2E-03	7130	7.2E-04	1.7E-03	--	1.8E-1	7130	1.2E-05	1.4E-04	--	1.6E-2
1,1,2,2-tetrachloroethane		4.0E-02	42.7	4.3E-06	1.0E-05	--	2.5E-4	42.7	7.2E-08	8.4E-07	--	2.1E-5
chloroform		6.5E-03	213	2.1E-05	5.0E-05	--	7.7E-3	213	3.6E-07	4.2E-06	--	6.4E-4
hexachlorobutadiene (HCBD)		2.0E-04	112	1.1E-05	2.6E-05	--	1.3E-1	112	1.9E-07	2.2E-06	--	1.1E-2

↑
Maximum concentration in sediments from Table 3-6 in report

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Maximum concentration in sediments from Table 3-6 in report

Dermal Exposure to Chemicals via Contact with Sediments

Stage 1 and 2 Development (Hawley Method)

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Dermal Contact with Sediments by Workers (Table 5-2, Scenario 9)		Dermal Contact with Sediments during Maintenance of Ponds (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	20	Assume 1 months work in pond areas
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	6	Assume maintenance works occur once every 5 years for 30 years
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	2190	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forams and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Dermal Absorption Rate (Abs)	per hour	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996
Exposure Time (ET)	hr/day	12	Assume that soil remains on skin for 12 hours before washing	12	Assume that soil remains on skin for 12 hours before washing
Matrix Effect (ME)	-	15%	Absorption of chemicals from soil matrix is 15% from pure compound	15%	Absorption of chemicals from soil matrix is 15% from pure compound
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -		1.00	Not used for intrusive works	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	4.2E-08 9.9E-08	NonThreshold Threshold	1.5E-09 1.7E-08	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (accounting for background) (mg/kg/day)		NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)		NonThreshold Risk (unitless)	Hazard Quotient (unitless)		
					TOTAL	1.9E-5	1.9E-1			TOTAL	6.6E-7	3.4E-2
TPH C6-C9 aliphatic		4.5E+00	113	4.8E-06	1.1E-05	--	2.5E-6	113	1.7E-07	2.0E-06	--	4.4E-7
TPH C10-C14 aromatic		4.0E-02	249	1.1E-05	2.5E-05	--	6.2E-4	249	3.7E-07	4.3E-06	--	1.1E-4
TPH C10-C14 aliphatic		9.0E-02	249	1.1E-05	2.5E-05	--	2.7E-4	249	3.7E-07	4.3E-06	--	4.8E-5
TPH C15+ aromatic		3.0E-02	1730	7.4E-05	1.7E-04	--	5.7E-3	1730	2.6E-06	3.0E-05	--	1.0E-3
TPH C15+ aliphatic		1.8E+00	1730	7.4E-05	1.7E-04	--	9.5E-5	1730	2.6E-06	3.0E-05	--	1.7E-5
vinyl chloride (adult exposures)	1.2E+00		384	1.6E-05	3.8E-05	1.9E-5	--	384	5.7E-07	6.7E-06	6.6E-7	--
cis-1,2-dichloroethene		1.0E-02	302	1.3E-05	3.0E-05	--	3.0E-3	302	4.5E-07	5.3E-06	--	5.3E-4
1,2-dichloroethane (EDC)	1.2E-02		29.8	1.3E-06	3.0E-06	1.5E-8	--	29.8	4.5E-08	5.2E-07	5.4E-10	--
trichloroethene (TCE)		1.5E-03	1050	4.5E-05	1.0E-04	--	7.1E-2	1050	1.6E-06	1.8E-05	--	1.3E-2
1,1,2-trichloroethane		4.0E-03	115	4.9E-06	1.1E-05	--	2.9E-3	115	1.7E-07	2.0E-06	--	5.0E-4
tetrachloroethene (PCE)		1.4E-02	7130	3.0E-04	7.1E-04	--	5.1E-2	7130	1.1E-05	1.2E-04	--	8.9E-3
1,1,2,2-tetrachloroethane		4.0E-02	42.7	1.8E-06	4.2E-06	--	1.1E-4	42.7	6.4E-08	7.5E-07	--	1.9E-5
chloroform		1.3E-02	213	9.1E-06	2.1E-05	--	1.6E-3	213	3.2E-07	3.7E-06	--	2.9E-4
hexachlorobutadiene (HCBd)		2.0E-04	112	4.8E-06	1.1E-05	--	5.6E-2	112	1.7E-07	2.0E-06	--	9.8E-3

↑
Maximum concentration in sediments from Table 3-6 in report

↑
Maximum concentration in sediments from Table 3-6 in report

Dermal Exposure to Chemicals via Contact with Sediment

Stage 1 and 2 Development (USEPA Method)

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Dermal Contact with Sediments by Workers (Table 5-2, Scenario 9)		Dermal Contact with Sediments during Maintenance of Ponds (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forearms and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Fraction of Day Exposed (FE)	-	0.5	12 hour exposure, CSMS 1991, 1993 and 1998	0.5	12 hour exposure, CSMS 1991, 1993 and 1998
Matrix Effect (ME)	-	100%	Assume 100% bioavailability, CSMS 1996	100%	Assume 100% bioavailability, CSMS 1996
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -	-	1.00	Not used	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot FE \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	2.4E-06 5.5E-06	NonThreshold Threshold	5.4E-07 1.3E-06	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Absorption x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity and Dermal Data			Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (accounting for background) (mg/kg/day)	Dermal Absorption (unitless)		NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)		NonThreshold Risk (unitless)	Hazard Quotient (unitless)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)
TPH C6-C9 aliphatic		4.5E+00	0.01	113	2.7E-06	6.2E-06	--	1.4E-6	113	6.1E-07	1.4E-06	--	3.2E-7
TPH C10-C14 aromatic		4.0E-02	0.13	249	7.6E-05	1.8E-04	--	4.5E-3	249	1.7E-05	4.1E-05	--	1.0E-3
TPH C10-C14 aliphatic		9.0E-02	0.01	249	5.9E-06	1.4E-05	--	1.5E-4	249	1.3E-06	3.1E-06	--	3.5E-5
TPH C15+ aromatic		3.0E-02	0.13	1730	5.3E-04	1.2E-03	--	4.1E-2	1730	1.2E-04	2.8E-04	--	9.5E-3
TPH C15+ aliphatic		1.8E+00	0.01	1730	4.1E-05	9.5E-05	--	5.3E-5	1730	9.4E-06	2.2E-05	--	1.2E-5
vinyl chloride (adult exposures)	1.2E+00		0.01	384	9.1E-06	2.1E-05	1.0E-5	--	384	2.1E-06	4.8E-06	2.4E-6	--
cis-1,2-dichloroethene		1.0E-02	0.01	302	7.1E-06	1.7E-05	--	1.7E-3	302	1.6E-06	3.8E-06	--	3.8E-4
1,2-dichloroethane (EDC)	1.2E-02		0.01	29.8	7.0E-07	1.6E-06	8.4E-9	--	29.8	1.6E-07	3.8E-07	1.9E-9	--
trichloroethene (TCE)		1.5E-03	0.01	1050	2.5E-05	5.8E-05	--	4.0E-2	1050	5.7E-06	1.3E-05	--	9.1E-3
1,1,2-trichloroethane		4.0E-03	0.01	115	2.7E-06	6.3E-06	--	1.6E-3	115	6.2E-07	1.5E-06	--	3.6E-4
tetrachloroethene (PCE)		9.2E-03	0.01	7130	1.7E-04	3.9E-04	--	4.3E-2	7130	3.9E-05	9.0E-05	--	9.7E-3
1,1,2,2-tetrachloroethane		4.0E-02	0.01	42.7	1.0E-06	2.4E-06	--	5.9E-5	42.7	2.3E-07	5.4E-07	--	1.3E-5
chloroform		6.5E-03	0.01	213	5.0E-06	1.2E-05	--	1.8E-3	213	1.2E-06	2.7E-06	--	4.1E-4
hexachlorobutadiene (HCBD)		2.0E-04	0.01	112	2.6E-06	6.2E-06	--	3.1E-2	112	6.1E-07	1.4E-06	--	7.1E-3

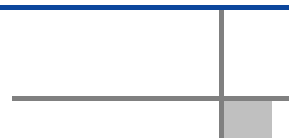
↑
Maximum concentration in sediments from Table 3-6 in report

↑
Maximum concentration in sediments from Table 3-6 in report

Appendix D

Risk Calculations

A.2 Calculations Relevant for Table 5-3 in Main Report



Inhalation of Volatile Chemicals - Emissions from Other Sources on BIP

Stage 1 and Stage 2 Development

General Data/ Equations		Units	Exposure Calculations (RME)				
			Outdoors - Gardeners etc (Table 5-3, Scenario 1)				
Exposure Parameters							
Exposure Frequency (EF)		days/year	26	Assume gardening activities once every 2 weeks			
Exposure Duration (ED)		years	30	Assume gardener works on the site for duration of working life			
Body Weight (BW)		kg	70	USEPA 1989 and CSMS 1996			
Averaging Time - NonThreshold (ATc)		days	25550	USEPA 1989 and CSMS 1996			
Averaging Time - Threshold (ATn)		days	10950	USEPA 1989 and CSMS 1996			
Inhalation Rate (InhR)		m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003			
Exposure Time (ET)		hours/day	10	Assume 10 hours spent on the site			
Bioavailability (B)		-	100%	Assume 100% bioavailability via inhalation			
Fraction Inhaled (FI)		-	100%	Assume all time spent on the site			
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$		m ³ /kg/day	9.6E-03 2.2E-02	NonThreshold Threshold			
<p><i>Daily Intake = Concentration in Air x Intake Factor</i> <i>NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)</i></p>							
Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		2.9E-10	8.8E-4
mercury (as total, inorganic)		2.2E-04	1.60E-06	1.5E-08	3.6E-08	--	1.6E-4
vinyl chloride (adult exposures)	1.5E-02		1.10E-06	1.1E-08	2.5E-08	1.6E-10	--
1,1-dichloroethene		5.7E-02	1.10E-07	1.1E-09	2.5E-09	--	4.3E-8
trans-1,2-dichloroethene		1.7E-02	7.40E-09	7.1E-11	1.7E-10	--	9.7E-9
1,1-dichloroethane		1.4E-01	5.50E-09	5.3E-11	1.2E-10	--	8.6E-10
cis-1,2-dichloroethene		1.0E-02	6.10E-08	5.9E-10	1.4E-09	--	1.4E-7
1,2-dichloroethane (EDC)	9.8E-03		8.40E-07	8.1E-09	1.9E-08	7.9E-11	--
trichloroethene (TCE)	1.5E-03		2.60E-06	2.5E-08	5.8E-08	3.8E-11	--
1,1,2-trichloroethane		4.0E-03	1.40E-08	1.3E-10	3.1E-10	--	7.8E-8
tetrachloroethene (PCE)		3.8E-02	4.10E-04	3.9E-06	9.2E-06	--	2.4E-4
chloroform	1.5E-03	2.0E-02	5.40E-07	5.2E-09	1.2E-08	7.6E-12	6.0E-7
dichloromethane		2.3E-01	4.60E-08	4.4E-10	1.0E-09	--	4.5E-9
hexachlorobutadiene (HCBD)		2.0E-04	1.00E-06	9.6E-09	2.2E-08	--	1.1E-4
hexachlorobenzene (HCB)		1.6E-04	6.10E-09	5.9E-11	1.4E-10	--	8.5E-7
hexachloroethane (HCE)		1.0E-03	1.60E-05	1.5E-07	3.6E-07	--	3.6E-4

↑
Modelled ground level concentrations - emissions from BIP including CPWE remediation
Concentrations considered presented in Table 3-11

Inhalation of Volatile Chemicals - Southlands

Stage 1 and Stage 2

General Data/ Equations	Units	Exposure Calculations (RME) - Emissions from Subsurface		Exposure Calculations (RME) - Emissions from Drain	
		Inhalation Outdoors during Gardening and Maintenance (Table 5-3, Scenario 2)		Inhalation Outdoors during Gardening and Maintenance (Table 5-3, Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	26	Assume gardening activities once every 2 weeks	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Assume gardener works on the site for duration of working life	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 10 hours spent on the site	10	Assume 10 hours spent on the site
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	9.6E-03	NonThreshold	9.6E-03	NonThreshold
		2.2E-02	Threshold	2.2E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air (mg/m ³)	Daily Intake		Calculated Risk		Concentration in Air (mg/m ³)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (mg/kg/day)		NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)		NonThreshold Risk (unitless)	Hazard Quotient (unitless)		
					TOTAL					TOTAL		
benzene	2.1E-02	7.7E-03	1.74E-04	1.7E-06	3.9E-06	5.1E-4	3.5E-8				--	--
toluene		6.7E-02	1.71E-04	1.6E-06	3.8E-06	--	--				--	--
ethylbenzene		6.3E+00	1.63E-05	1.6E-07	3.7E-07	--	--				--	--
xylenes		2.4E-01	7.90E-05	7.6E-07	1.8E-06	--	--				--	--
TPH C6-C9 aliphatic		1.8E-01	2.32E-06	2.2E-08	5.2E-08	--	--				--	--
TPH C10-C14 aromatic		5.1E-02	2.87E-06	2.8E-08	6.4E-08	--	--				--	--
vinyl chloride (adult exposures)	1.5E-02		4.27E-04	4.1E-06	9.6E-06	6.3E-8	--	9.90E-02	9.5E-04	2.2E-03	1.5E-5	--
1,1-dichloroethene		5.7E-02	2.16E-05	2.1E-07	4.8E-07	--	--				--	--
trans-1,2-dichloroethene		1.7E-02	3.23E-05	3.1E-07	7.2E-07	--	--				--	--
1,1-dichloroethane		1.4E-01	4.85E-05	4.7E-07	1.1E-06	--	--				--	--
cis-1,2-dichloroethene		1.0E-02	1.13E-04	1.1E-06	2.5E-06	--	2.5E-4	8.00E-02	7.7E-04	1.8E-03	--	1.8E-1
1,2-dichloroethane (EDC)	9.8E-03		5.03E-04	4.8E-06	1.1E-05	4.7E-8	--	2.40E+00	2.3E-02	5.4E-02	2.3E-4	--
trichloroethene (TCE)	1.5E-03		8.98E-04	8.6E-06	2.0E-05	1.3E-8	--	1.80E-01	1.7E-03	4.0E-03	2.6E-6	--
1,1,2-trichloroethane		4.0E-03	3.41E-05	3.3E-07	7.6E-07	--	1.9E-4				--	--
tetrachloroethene (PCE)		3.8E-02	7.00E-03	6.7E-05	1.6E-04	--	4.2E-3	1.60E-01	1.5E-03	3.6E-03	--	9.5E-2
1,1,2,2-tetrachloroethane	1.1E-02		6.82E-05	6.5E-07	1.5E-06	6.9E-9	--				--	--
chloroform	1.5E-03	2.0E-02	4.49E-04	4.3E-06	1.0E-05	6.3E-9	--				--	--
carbon tetrachloride		6.1E-04	2.33E-03	2.2E-05	5.2E-05	--	8.6E-2	7.70E-02	7.4E-04	1.7E-03	--	2.8E+0
dichloromethane		2.3E-01	1.89E-03	1.8E-05	4.2E-05	--	1.8E-4				--	--
1,2,4-trimethylbenzene		8.6E-04	3.41E-05	3.3E-07	7.6E-07	--	8.9E-4				--	--
1,3,5-trimethylbenzene		8.6E-04	6.82E-06	6.5E-08	1.5E-07	--	1.8E-4				--	--
naphthalene		8.1E-04	5.75E-04	5.5E-06	1.3E-05	--	1.6E-2				--	--
trichlorofluoromethane		2.0E-01	9.88E-05	9.5E-07	2.2E-06	--	1.1E-5				--	--

↑ Calculated from maximum measured data from Block 1 or Block 2

↑ Maximum measured concentrations adjacent to drains prior to GTP operation (Table 3-10)

Inhalation of Volatile Chemicals - Southlands

Stage 1 and Stage 2

General Data/ Equations		Units	Exposure Calculations (RME) - Emissions from Soil/Sediment Inhalation Outdoors during Gardening and Maintenance (Table 5-3, Scenario 3)	
Exposure Parameters				
Exposure Frequency (EF)	days/year	26	Assume gardening activities once every 2 weeks	
Exposure Duration (ED)	years	30	Assume gardener works on the site for duration of working life	
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003	
Exposure Time (ET)	hours/day	10	Assume 10 hours spent on the site	
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	9.6E-03 2.2E-02	NonThreshold Threshold	

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air (mg/m ³)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		9.7E-7	7.6E-2
TPH C6-C9 aliphatic		1.8E-01	1.34E-03	1.3E-05	3.0E-05	--	1.7E-4
TPH C10-C14 aromatic		5.1E-02	2.41E-03	2.3E-05	5.4E-05	--	1.0E-3
TPH C10-C14 aliphatic		2.6E-01	2.94E-03	2.8E-05	6.6E-05	--	2.6E-4
vinyl chloride (adult exposures)	1.5E-02		4.54E-03	4.4E-05	1.0E-04	6.7E-7	--
trans-1,2-dichloroethene		1.7E-02	1.25E-03	1.2E-05	2.8E-05	--	1.7E-3
1,1-dichloroethane		1.4E-01	2.71E-04	2.6E-06	6.1E-06	--	4.2E-5
cis-1,2-dichloroethene		1.0E-02	3.57E-03	3.4E-05	8.0E-05	--	8.0E-3
1,2-dichloroethane (EDC)	9.8E-03		3.52E-04	3.4E-06	7.9E-06	3.3E-8	--
trichloroethene (TCE)	1.5E-03		1.24E-02	1.2E-04	2.8E-04	1.8E-7	--
1,1,2-trichloroethane		4.0E-03	1.36E-03	1.3E-05	3.0E-05	--	7.6E-3
tetrachloroethene (PCE)		3.8E-02	8.43E-02	8.1E-04	1.9E-03	--	5.0E-2
1,1,2,2-tetrachloroethane	1.1E-02		5.05E-04	4.8E-06	1.1E-05	5.1E-8	--
chloroform	1.5E-03	2.0E-02	2.52E-03	2.4E-05	5.6E-05	3.6E-8	2.8E-3
carbon tetrachloride		6.1E-04	8.27E-07	7.9E-09	1.9E-08	--	3.0E-5
1,3,5-trichlorobenzene		4.6E-04	8.30E-05	8.0E-07	1.9E-06	--	4.0E-3
1,4-dichlorobenzene		2.9E-01	1.30E-05	1.2E-07	2.9E-07	--	1.0E-6

↑
Maximum modelled outdoor air concentration from soil or sediments

Exposure to Chemicals via Ingestion of Soils

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Ingestion of Soils by Workers (Table 5-2, Scenario 9)		Ingestion of Soils by Gardeners (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Ingestion Rate (IRs)	mg/day	25	Soil intake for adults as per CSMS 1996	25	Soil intake for adults as per CSMS 1996
Fraction Ingested (FI)	-	100%	All soil ingested from site	100%	All soil ingested from site
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in soil	100%	Assume 100% bioavailability via ingestion of chemicals in soil
Conversion factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Intake Factor = IRs*FI*B*CF*EF*ED	kg/kg/day	1.0E-07	NonThreshold	1.1E-08	NonThreshold
BW*AT		2.3E-07	Threshold	2.5E-08	Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		7.2E-6	3.8E+0		TOTAL		7.8E-7	4.2E-1
mercury (as total, inorganic)		5.4E-04	1233	1.2E-04	2.9E-04	--	5.4E-1	1233	1.3E-05	3.1E-05	--	5.8E-2
chromium (assumed Cr(VI))		2.1E-03	1500	1.5E-04	3.5E-04	--	1.7E-1	1500	1.6E-05	3.8E-05	--	1.8E-2
lead		1.9E-03	7670	7.7E-04	1.8E-03	--	9.7E-1	7670	8.4E-05	2.0E-04	--	1.1E-1
benzene	3.5E-02		31.3	3.2E-06	7.4E-06	1.1E-7	--	31.3	3.4E-07	8.0E-07	1.2E-8	--
ethylbenzene		9.7E-02	141	1.4E-05	3.3E-05	--	3.4E-4	141	1.5E-06	3.6E-06	--	3.7E-5
xylenes		1.8E-01	41.4	4.2E-06	9.7E-06	--	5.5E-5	41.4	4.5E-07	1.1E-06	--	6.0E-6
TPH C6-C9 aliphatic		4.5E+00	12100	1.2E-03	2.8E-03	--	6.3E-4	12100	1.3E-04	3.1E-04	--	6.8E-5
TPH C10-C14 aromatic		4.0E-02	1220	1.2E-04	2.9E-04	--	7.2E-3	1220	1.3E-05	3.1E-05	--	7.8E-4
TPH C10-C14 aliphatic		9.0E-02	1220	1.2E-04	2.9E-04	--	3.2E-3	1220	1.3E-05	3.1E-05	--	3.4E-4
TPH C15+ aromatic		2.7E-02	2795	2.8E-04	6.6E-04	--	2.4E-2	2795	3.0E-05	7.1E-05	--	2.6E-3
TPH C15+ aliphatic		1.8E+00	2795	2.8E-04	6.6E-04	--	3.6E-4	2795	3.0E-05	7.1E-05	--	4.0E-5
vinyl chloride (adult exposures)	1.2E+00		53	5.3E-06	1.2E-05	6.1E-6	--	53	5.8E-07	1.3E-06	6.6E-7	--
cis-1,2-dichloroethene		1.0E-02	165	1.7E-05	3.9E-05	--	3.9E-3	165	1.8E-06	4.2E-06	--	4.2E-4
1,2-dichloroethane (EDC)	1.2E-02		4.5	4.5E-07	1.1E-06	5.4E-9	--	4.5	4.9E-08	1.1E-07	5.9E-10	--
trichloroethene (TCE)		1.5E-03	95.1	9.6E-06	2.2E-05	--	1.5E-2	95.1	1.0E-06	2.4E-06	--	1.7E-3
1,1,2-trichloroethane		4.0E-03	2	2.0E-07	4.7E-07	--	1.2E-4	2	2.2E-08	5.1E-08	--	1.3E-5
tetrachloroethene (PCE)		9.2E-03	90.8	9.1E-06	2.1E-05	--	2.3E-3	90.8	9.9E-07	2.3E-06	--	2.5E-4
1,1,2,2-tetrachloroethane		4.0E-02	10.2	1.0E-06	2.4E-06	--	6.0E-5	10.2	1.1E-07	2.6E-07	--	6.5E-6
chloroform		6.5E-03	3.9	3.9E-07	9.2E-07	--	1.4E-4	3.9	4.3E-08	9.9E-08	--	1.5E-5
PAHs (BaP equivalent)	5.0E-01		14.1	1.4E-06	3.3E-06	7.1E-7	--	14.1	1.5E-07	3.6E-07	7.7E-8	--
hexachlorobutadiene (HCBd)		2.0E-04	326	3.3E-05	7.7E-05	--	3.8E-1	326	3.6E-06	8.3E-06	--	4.1E-2
hexachlorobenzene (HCB)		1.6E-04	1170	1.2E-04	2.7E-04	--	1.7E+0	1170	1.3E-05	3.0E-05	--	1.9E-1
bis(2-chloroethyl)ether	1.1E+00		2.1	2.1E-07	4.9E-07	2.3E-7	--	2.1	2.3E-08	5.3E-08	2.5E-8	--

↑
Maximum concentration reported in Soils from Block 1 and Block 2
Maximum reported from Tables 3-3 and 3-4 in report

↑
Maximum concentration reported in Soils from Block 1 and Block 2
Maximum reported from Tables 3-3 and 3-4 in report

Dermal Exposure to Chemicals via Contact with Soil

Stage 1 and 2 Development (Hawley Method)

General Data/ Equations		Exposure Calculations (RME)		Exposure Calculations (RME)	
Units		Dermal Contact with Soils by Workers (Table 5-2, Scenario 9)		Dermal Contact with Soils by Gardeners (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forarms and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Dermal Absorption Rate (Abs)	per hour	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996
Exposure Time (ET)	hr/day	12	Assume that soil remains on skin for 12 hours before washing	12	Assume that soil remains on skin for 12 hours before washing
Matrix Effect (ME)	-	15%	Absorption of chemicals from soil matrix is 15% from pure compound	15%	Absorption of chemicals from soil matrix is 15% from pure compound
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -		1.00	Not used for intrusive works	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	4.2E-08 9.9E-08	NonThreshold Threshold	9.7E-09 2.3E-08	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor
 NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
 Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	3.0E-6	9.1E-1			TOTAL	7.0E-7	2.1E-1		
benzene	3.5E-02		31.3	1.3E-06	3.1E-06	4.7E-8	--	31.3	3.0E-07	7.1E-07	1.1E-8	--		
ethylbenzene		9.7E-02	141	6.0E-06	1.4E-05	--	1.4E-4	141	1.4E-06	3.2E-06	--	3.3E-5		
xylenes		1.8E-01	41.4	1.8E-06	4.1E-06	--	2.3E-5	41.4	4.0E-07	9.4E-07	--	5.4E-6		
TPH C6-C9 aliphatic		4.5E+00	12100	5.1E-04	1.2E-03	--	2.7E-4	12100	1.2E-04	2.7E-04	--	6.1E-5		
TPH C10-C14 aromatic		4.0E-02	1220	5.2E-05	1.2E-04	--	3.0E-3	1220	1.2E-05	2.8E-05	--	6.9E-4		
TPH C10-C14 aliphatic		9.0E-02	1220	5.2E-05	1.2E-04	--	1.3E-3	1220	1.2E-05	2.8E-05	--	3.1E-4		
TPH C15+ aromatic		3.0E-02	2795	1.2E-04	2.8E-04	--	9.2E-3	2795	2.7E-05	6.3E-05	--	2.1E-3		
TPH C15+ aliphatic		1.8E+00	2795	1.2E-04	2.8E-04	--	1.5E-4	2795	2.7E-05	6.3E-05	--	3.5E-5		
vinyl chloride (adult exposures)	1.2E+00		53	2.3E-06	5.3E-06	2.6E-6	--	53	5.2E-07	1.2E-06	5.9E-7	--		
cis-1,2-dichloroethene		1.0E-02	165	7.0E-06	1.6E-05	--	1.6E-3	165	1.6E-06	3.7E-06	--	3.7E-4		
1,2-dichloroethane (EDC)	1.2E-02		4.5	1.9E-07	4.5E-07	2.3E-9	--	4.5	4.4E-08	1.0E-07	5.3E-10	--		
trichloroethene (TCE)		1.5E-03	95.1	4.0E-06	9.4E-06	--	6.5E-3	95.1	9.3E-07	2.2E-06	--	1.5E-3		
1,1,2-trichloroethane		4.0E-03	2	8.5E-08	2.0E-07	--	5.0E-5	2	1.9E-08	4.5E-08	--	1.1E-5		
tetrachloroethene (PCE)		1.4E-02	90.8	3.9E-06	9.0E-06	--	6.4E-4	90.8	8.8E-07	2.1E-06	--	1.5E-4		
1,1,2,2-tetrachloroethane		4.0E-02	10.2	4.3E-07	1.0E-06	--	2.5E-5	10.2	9.9E-08	2.3E-07	--	5.8E-6		
chloroform		1.3E-02	3.9	1.7E-07	3.9E-07	--	3.0E-5	3.9	3.9E-08	8.9E-08	--	6.8E-6		
PAHs (BaP equivalent)	5.0E-01		14.1	6.0E-07	1.4E-06	3.0E-7	--	14.1	1.4E-07	3.2E-07	6.9E-8	--		
hexachlorobutadiene (HCBD)		2.0E-04	326	1.4E-05	3.2E-05	--	1.6E-1	326	3.2E-06	7.4E-06	--	3.7E-2		
hexachlorobenzene (HCB)		1.6E-04	1170	5.0E-05	1.2E-04	--	7.3E-1	1170	1.1E-05	2.7E-05	--	1.7E-1		
bis(2-chloroethyl)ether	1.1E+00		2.1	8.9E-08	2.1E-07	--	9.8E-8	2.1	2.0E-08	4.8E-08	2.2E-8	--		

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

Dermal Exposure to Chemicals via Contact with Soil
Stage 1 and 2 Development (USEPA Method)

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Dermal Contact with Soils by Workers (Table 5-2, Scenario 9)		Dermal Contact with Soils by Gardeners (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forarms and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Fraction of Day Exposed (FE)	-	0.5	12 hour exposure, CSMS 1991, 1993 and 1998	0.5	12 hour exposure, CSMS 1991, 1993 and 1998
Matrix Effect (ME)	-	100%	Assume 100% bioavailability, CSMS 1996	100%	Assume 100% bioavailability, CSMS 1996
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -	-	1.00	Not used	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot FE \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	2.4E-06 5.5E-06	NonThreshold Threshold	5.4E-07 1.3E-06	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Absorption x Intake Factor
 NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
 Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity and Dermal Data			Concentration in Soil (mg/kg)	Daily Intake (mg/kg/day)		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake (mg/kg/day)		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	Dermal Absorption		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(unitless)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	3.7E-6	5.9E-1			TOTAL	8.4E-7	1.3E-1
benzene	3.5E-02		0.01	31.3	7.4E-07	1.7E-06	2.6E-8	--	31.3	1.7E-07	3.9E-07	5.9E-9	--
ethylbenzene		9.7E-02	0.01	141	3.3E-06	7.8E-06	--	8.0E-5	141	7.6E-07	1.8E-06	--	1.8E-5
xylenes		1.8E-01	0.01	41.4	9.8E-07	2.3E-06	--	1.3E-5	41.4	2.2E-07	5.2E-07	--	3.0E-6
TPH C6-C9 aliphatic		4.5E+00	0.01	12100	2.9E-04	6.7E-04	--	1.5E-4	12100	6.5E-05	1.5E-04	--	3.4E-5
TPH C10-C14 aromatic		4.0E-02	0.13	1220	3.7E-04	8.7E-04	--	2.2E-2	1220	8.6E-05	2.0E-04	--	5.0E-3
TPH C10-C14 aliphatic		9.0E-02	0.01	1220	2.9E-05	6.7E-05	--	7.5E-4	1220	6.6E-06	1.5E-05	--	1.7E-4
TPH C15+ aromatic		3.0E-02	0.13	2795	8.6E-04	2.0E-03	--	6.7E-2	2795	2.0E-04	4.6E-04	--	1.5E-2
TPH C15+ aliphatic		1.8E+00	0.01	2795	6.6E-05	1.5E-04	--	8.6E-5	2795	1.5E-05	3.5E-05	--	2.0E-5
vinyl chloride (adult exposures)	1.2E+00		0.01	53	1.3E-06	2.9E-06	1.4E-6	--	53	2.9E-07	6.7E-07	3.3E-7	--
cis-1,2-dichloroethene		1.0E-02	0.01	165	3.9E-06	9.1E-06	--	9.1E-4	165	8.9E-07	2.1E-06	--	2.1E-4
1,2-dichloroethane (EDC)	1.2E-02		0.01	4.5	1.1E-07	2.5E-07	1.3E-9	--	4.5	2.4E-08	5.7E-08	2.9E-10	--
trichloroethene (TCE)		1.5E-03	0.01	95.1	2.2E-06	5.2E-06	--	3.6E-3	95.1	5.1E-07	1.2E-06	--	8.2E-4
1,1,2-trichloroethane		4.0E-03	0.01	2	4.7E-08	1.1E-07	--	2.8E-5	2	1.1E-08	2.5E-08	--	6.3E-6
tetrachloroethene (PCE)		9.2E-03	0.01	90.8	2.1E-06	5.0E-06	--	5.4E-4	90.8	4.9E-07	1.1E-06	--	1.2E-4
1,1,2,2-tetrachloroethane		4.0E-02	0.01	10.2	2.4E-07	5.6E-07	--	1.4E-5	10.2	5.5E-08	1.3E-07	--	3.2E-6
chloroform		6.5E-03	0.01	3.9	9.2E-08	2.1E-07	--	3.3E-5	3.9	2.1E-08	4.9E-08	--	7.6E-6
PAHs (BaP equivalent)	5.0E-01		0.13	14.1	4.3E-06	1.0E-05	2.2E-6	--	14.1	9.9E-07	2.3E-06	5.0E-7	--
hexachlorobutadiene (HCBD)		2.0E-04	0.01	326	7.7E-06	1.8E-05	--	9.0E-2	326	1.8E-06	4.1E-06	--	2.1E-2
hexachlorobenzene (HCB)		1.6E-04	0.01	1170	2.8E-05	6.4E-05	--	4.0E-1	1170	6.3E-06	1.5E-05	--	9.2E-2
bis(2-chloroethyl)ether	1.1E+00		0.01	2.1	5.0E-08	1.2E-07	5.5E-8	--	2.1	1.1E-08	2.6E-08	1.2E-8	--

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

Exposure to Chemicals via Ingestion of Sediments

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Ingestion of Sediments by Workers (Table 5-2, Scenario 9)		Ingestion of Sediments during Maintenance of Ponds (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	20	Assume 1 months work in pond areas
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	6	Assume maintenance works occur once every 5 years for 30 years
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	2190	USEPA 1989 and CSMS 1996
Ingestion Rate (IRs)	mg/day	25	Soil intake for adults as per CSMS 1996	25	Soil intake for adults as per CSMS 1996
Fraction Ingested (FI)	-	100%	All soil ingested from site	100%	All soil ingested from site
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in soil	100%	Assume 100% bioavailability via ingestion of chemicals in soil
Conversion factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Intake Factor = $\frac{IRs \cdot FI \cdot B \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg/kg/day	1.0E-07	NonThreshold	1.7E-09	NonThreshold
		2.3E-07	Threshold	2.0E-08	Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	4.4E-5	5.4E-1			TOTAL	7.4E-7	4.5E-2
chromium (assumed Cr(VI))		2.1E-03	188	1.9E-05	4.4E-05	--	2.1E-2	188	3.2E-07	3.7E-06	--	1.8E-3
TPH C6-C9 aliphatic		4.5E+00	113	1.1E-05	2.7E-05	--	5.9E-6	113	1.9E-07	2.2E-06	--	4.9E-7
TPH C10-C14 aromatic		4.0E-02	249	2.5E-05	5.8E-05	--	1.5E-3	249	4.2E-07	4.9E-06	--	1.2E-4
TPH C10-C14 aliphatic		9.0E-02	249	2.5E-05	5.8E-05	--	6.5E-4	249	4.2E-07	4.9E-06	--	5.4E-5
TPH C15+ aromatic		2.7E-02	1730	1.7E-04	4.1E-04	--	1.5E-2	1730	2.9E-06	3.4E-05	--	1.3E-3
TPH C15+ aliphatic		1.8E+00	1730	1.7E-04	4.1E-04	--	2.3E-4	1730	2.9E-06	3.4E-05	--	1.9E-5
vinyl chloride (adult exposures)	1.2E+00		384	3.9E-05	9.0E-05	4.4E-5	--	384	6.4E-07	7.5E-06	7.4E-7	--
cis-1,2-dichloroethene		1.0E-02	302	3.0E-05	7.1E-05	--	7.1E-3	302	5.1E-07	5.9E-06	--	5.9E-4
1,2-dichloroethane (EDC)	1.2E-02		29.8	3.0E-06	7.0E-06	3.6E-8	--	29.8	5.0E-08	5.8E-07	6.0E-10	--
trichloroethene (TCE)		1.5E-03	1050	1.1E-04	2.5E-04	--	1.7E-1	1050	1.8E-06	2.1E-05	--	1.4E-2
1,1,2-trichloroethane		4.0E-03	115	1.2E-05	2.7E-05	--	6.8E-3	115	1.9E-07	2.3E-06	--	5.6E-4
tetrachloroethene (PCE)		9.2E-03	7130	7.2E-04	1.7E-03	--	1.8E-1	7130	1.2E-05	1.4E-04	--	1.6E-2
1,1,2,2-tetrachloroethane		4.0E-02	42.7	4.3E-06	1.0E-05	--	2.5E-4	42.7	7.2E-08	8.4E-07	--	2.1E-5
chloroform		6.5E-03	213	2.1E-05	5.0E-05	--	7.7E-3	213	3.6E-07	4.2E-06	--	6.4E-4
hexachlorobutadiene (HCBD)		2.0E-04	112	1.1E-05	2.6E-05	--	1.3E-1	112	1.9E-07	2.2E-06	--	1.1E-2

↑ Maximum concentration in sediments from Table 3-6 in report

↑ Maximum concentration in sediments from Table 3-6 in report

Dermal Exposure to Chemicals via Contact with Sediments

Stage 1 and 2 Development (Hawley Method)

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Dermal Contact with Sediments by Workers (Table 5-2, Scenario 9)		Dermal Contact with Sediments during Maintenance of Ponds (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	20	Assume 1 months work in pond areas
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	6	Assume maintenance works occur once every 5 years for 30 years
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	2190	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forams and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Dermal Absorption Rate (Abs)	per hour	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996
Exposure Time (ET)	hr/day	12	Assume that soil remains on skin for 12 hours before washing	12	Assume that soil remains on skin for 12 hours before washing
Matrix Effect (ME)	-	15%	Absorption of chemicals from soil matrix is 15% from pure compound	15%	Absorption of chemicals from soil matrix is 15% from pure compound
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -		1.00	Not used for intrusive works	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	4.2E-08 9.9E-08	NonThreshold Threshold	1.5E-09 1.7E-08	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (accounting for background) (mg/kg/day)		NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)		NonThreshold Risk (unitless)	Hazard Quotient (unitless)		
					TOTAL	1.9E-5	1.9E-1			TOTAL	6.6E-7	3.4E-2
TPH C6-C9 aliphatic		4.5E+00	113	4.8E-06	1.1E-05	--	2.5E-6	113	1.7E-07	2.0E-06	--	4.4E-7
TPH C10-C14 aromatic		4.0E-02	249	1.1E-05	2.5E-05	--	6.2E-4	249	3.7E-07	4.3E-06	--	1.1E-4
TPH C10-C14 aliphatic		9.0E-02	249	1.1E-05	2.5E-05	--	2.7E-4	249	3.7E-07	4.3E-06	--	4.8E-5
TPH C15+ aromatic		3.0E-02	1730	7.4E-05	1.7E-04	--	5.7E-3	1730	2.6E-06	3.0E-05	--	1.0E-3
TPH C15+ aliphatic		1.8E+00	1730	7.4E-05	1.7E-04	--	9.5E-5	1730	2.6E-06	3.0E-05	--	1.7E-5
vinyl chloride (adult exposures)	1.2E+00		384	1.6E-05	3.8E-05	1.9E-5	--	384	5.7E-07	6.7E-06	6.6E-7	--
cis-1,2-dichloroethene		1.0E-02	302	1.3E-05	3.0E-05	--	3.0E-3	302	4.5E-07	5.3E-06	--	5.3E-4
1,2-dichloroethane (EDC)	1.2E-02		29.8	1.3E-06	3.0E-06	1.5E-8	--	29.8	4.5E-08	5.2E-07	5.4E-10	--
trichloroethene (TCE)		1.5E-03	1050	4.5E-05	1.0E-04	--	7.1E-2	1050	1.6E-06	1.8E-05	--	1.3E-2
1,1,2-trichloroethane		4.0E-03	115	4.9E-06	1.1E-05	--	2.9E-3	115	1.7E-07	2.0E-06	--	5.0E-4
tetrachloroethene (PCE)		1.4E-02	7130	3.0E-04	7.1E-04	--	5.1E-2	7130	1.1E-05	1.2E-04	--	8.9E-3
1,1,2,2-tetrachloroethane		4.0E-02	42.7	1.8E-06	4.2E-06	--	1.1E-4	42.7	6.4E-08	7.5E-07	--	1.9E-5
chloroform		1.3E-02	213	9.1E-06	2.1E-05	--	1.6E-3	213	3.2E-07	3.7E-06	--	2.9E-4
hexachlorobutadiene (HCBd)		2.0E-04	112	4.8E-06	1.1E-05	--	5.6E-2	112	1.7E-07	2.0E-06	--	9.8E-3

↑
Maximum concentration in sediments from Table 3-6 in report

↑
Maximum concentration in sediments from Table 3-6 in report

Dermal Exposure to Chemicals via Contact with Sediment

Stage 1 and 2 Development (USEPA Method)

General Data/ Equations	Units	Exposure Calculations (RME)		Exposure Calculations (RME)	
		Dermal Contact with Sediments by Workers (Table 5-2, Scenario 9)		Dermal Contact with Sediments during Maintenance of Ponds (Table 5-3, Scenario 6)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	26	Assume gardening activities once every 2 weeks
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	30	Assume gardener works on the site for duration of working life
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)	4860	Hands, forearms and lower legs for adult male (50th percentile, USEPA 1997 and 2004)
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996	0.51	Default value as per CSMS 1996
Fraction of Day Exposed (FE)	-	0.5	12 hour exposure, CSMS 1991, 1993 and 1998	0.5	12 hour exposure, CSMS 1991, 1993 and 1998
Matrix Effect (ME)	-	100%	Assume 100% bioavailability, CSMS 1996	100%	Assume 100% bioavailability, CSMS 1996
Conversion Factor (CF)	mg to kg	1.E-06	Conversion	1.E-06	Conversion
Site-Specific Factor (SS), -	-	1.00	Not used	1.00	Not used for intrusive works
Intake Factor = $\frac{SAs \cdot AF \cdot FE \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg-hr/kg/day	2.4E-06 5.5E-06	NonThreshold Threshold	5.4E-07 1.3E-06	NonThreshold Threshold

Daily Intake from Soil = Concentration in Soil x Absorption x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity and Dermal Data			Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (accounting for background) (mg/kg/day)	Dermal Absorption (unitless)		NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)		NonThreshold Risk (unitless)	Hazard Quotient (unitless)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)
TPH C6-C9 aliphatic		4.5E+00	0.01	113	2.7E-06	6.2E-06	--	1.4E-6	113	6.1E-07	1.4E-06	--	3.2E-7
TPH C10-C14 aromatic		4.0E-02	0.13	249	7.6E-05	1.8E-04	--	4.5E-3	249	1.7E-05	4.1E-05	--	1.0E-3
TPH C10-C14 aliphatic		9.0E-02	0.01	249	5.9E-06	1.4E-05	--	1.5E-4	249	1.3E-06	3.1E-06	--	3.5E-5
TPH C15+ aromatic		3.0E-02	0.13	1730	5.3E-04	1.2E-03	--	4.1E-2	1730	1.2E-04	2.8E-04	--	9.5E-3
TPH C15+ aliphatic		1.8E+00	0.01	1730	4.1E-05	9.5E-05	--	5.3E-5	1730	9.4E-06	2.2E-05	--	1.2E-5
vinyl chloride (adult exposures)	1.2E+00		0.01	384	9.1E-06	2.1E-05	1.0E-5	--	384	2.1E-06	4.8E-06	2.4E-6	--
cis-1,2-dichloroethene		1.0E-02	0.01	302	7.1E-06	1.7E-05	--	1.7E-3	302	1.6E-06	3.8E-06	--	3.8E-4
1,2-dichloroethane (EDC)	1.2E-02		0.01	29.8	7.0E-07	1.6E-06	8.4E-9	--	29.8	1.6E-07	3.8E-07	1.9E-9	--
trichloroethene (TCE)		1.5E-03	0.01	1050	2.5E-05	5.8E-05	--	4.0E-2	1050	5.7E-06	1.3E-05	--	9.1E-3
1,1,2-trichloroethane		4.0E-03	0.01	115	2.7E-06	6.3E-06	--	1.6E-3	115	6.2E-07	1.5E-06	--	3.6E-4
tetrachloroethene (PCE)		9.2E-03	0.01	7130	1.7E-04	3.9E-04	--	4.3E-2	7130	3.9E-05	9.0E-05	--	9.7E-3
1,1,2,2-tetrachloroethane		4.0E-02	0.01	42.7	1.0E-06	2.4E-06	--	5.9E-5	42.7	2.3E-07	5.4E-07	--	1.3E-5
chloroform		6.5E-03	0.01	213	5.0E-06	1.2E-05	--	1.8E-3	213	1.2E-06	2.7E-06	--	4.1E-4
hexachlorobutadiene (HCBD)		2.0E-04	0.01	112	2.6E-06	6.2E-06	--	3.1E-2	112	6.1E-07	1.4E-06	--	7.1E-3

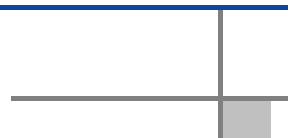
↑
Maximum concentration in sediments from Table 3-6 in report

↑
Maximum concentration in sediments from Table 3-6 in report

Appendix D

Risk Calculations

A.3 Calculations Relevant for Table 5-4 in Main Report



Inhalation of Volatile Chemicals - Southlands

Stage 1 and Stage 2

General Data/ Equations	Units	Exposure Calculations (RME) - Emissions from Soil and GW Inhalation Outdoors during Intrusive Works Table 5-4, Scenario 1)		Exposure Calculations (RME) - Excavations into Shallow GW Inhalation in Excavation during Intrusive Works (Table 5-4, Scenario 1)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	30	Assume intrusive works occur over 30 days	30	Assume intrusive works occur over 30 days
Exposure Duration (ED)	years	1	Assume works occur over a 1 year period	1	Assume works occur over a 1 year period
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	365	USEPA 1989 and CSMS 1996	365	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	6	Assume 6 hours each day spent outdoors on the site	4	Assume 4 hours spent in or at the top of excavations
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	2.2E-04 1.5E-02	NonThreshold Threshold	1.5E-04 1.0E-02	NonThreshold Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	2.6E-8	1.3E-1			TOTAL	4.0E-6	6.1E+0
benzene	2.1E-02	7.7E-03	1.74E-04	3.9E-08	2.7E-06	8.1E-10	3.5E-4				--	--
toluene		6.7E-02	1.71E-04	3.8E-08	2.6E-06	--	4.0E-5				--	--
ethylbenzene		6.3E+00	1.63E-05	3.6E-09	2.5E-07	--	4.0E-8				--	--
xylenes		2.4E-01	7.90E-05	1.7E-08	1.2E-06	--	5.0E-6				--	--
TPH C6-C9 aliphatic		1.8E-01	1.34E-03	3.0E-07	2.1E-05	--	1.2E-4				--	--
TPH C10-C14 aromatic		5.1E-02	2.41E-03	5.3E-07	3.7E-05	--	7.3E-4				--	--
vinyl chloride (adult exposures)	1.5E-02		4.97E-03	1.1E-06	7.7E-05	1.7E-8	--	9.04E-01	1.3E-04	9.3E-03	2.1E-6	--
1,1-dichloroethene		5.7E-02	2.16E-05	4.8E-09	3.3E-07	--	5.8E-6	7.45E-03	1.1E-06	7.7E-05	--	1.3E-3
trans-1,2-dichloroethene		1.7E-02	1.29E-03	2.8E-07	2.0E-05	--	1.2E-3	7.18E-02	1.1E-05	7.4E-04	--	4.4E-2
1,1-dichloroethane		1.4E-01	3.19E-04	7.1E-08	4.9E-06	--	3.5E-5				--	--
cis-1,2-dichloroethene		1.0E-02	3.68E-03	8.2E-07	5.7E-05	--	5.7E-3	6.38E-01	9.4E-05	6.6E-03	--	6.6E-1
1,2-dichloroethane (EDC)	9.8E-03		8.55E-04	1.9E-07	1.3E-05	1.9E-9	--	1.32E+00	1.9E-04	1.4E-02	1.9E-6	--
trichloroethene (TCE)	1.5E-03		1.33E-02	2.9E-06	2.1E-04	4.4E-9	--	1.60E-01	2.4E-05	1.6E-03	3.5E-8	--
1,1,2-trichloroethane		4.0E-03	1.39E-03	3.1E-07	2.2E-05	--	5.4E-3	2.79E-02	4.1E-06	2.9E-04	--	7.2E-2
tetrachloroethene (PCE)		3.8E-02	9.13E-02	2.0E-05	1.4E-03	--	3.8E-2	2.39E-01	3.5E-05	2.5E-03	--	6.6E-2
1,1,2,2-tetrachloroethane	1.1E-02		5.73E-04	1.3E-07	8.9E-06	1.3E-9	--				--	--
chloroform	1.5E-03		2.97E-03	6.6E-07	4.6E-05	9.7E-10	2.3E-3	1.26E-01	1.9E-05	1.3E-03	2.7E-8	6.5E-2
carbon tetrachloride		6.1E-04	2.34E-03	5.2E-07	3.6E-05	--	5.9E-2	3.06E-01	4.5E-05	3.2E-03	--	5.2E+0
dichloromethane		2.3E-01	1.89E-03	4.2E-07	2.9E-05	--	1.3E-4	7.45E-03	1.1E-06	7.7E-05	--	3.4E-4
1,2,4-trimethylbenzene		8.6E-04	3.41E-05	7.6E-09	5.3E-07	--	6.2E-4				--	--
1,3,5-trimethylbenzene		8.6E-04	6.82E-06	1.5E-09	1.1E-07	--	1.2E-4				--	--
naphthalene		8.1E-04	5.75E-04	1.3E-07	8.9E-06	--	1.1E-2				--	--
1,3,5-trichlorobenzene		4.6E-04	8.30E-05	1.8E-08	1.3E-06	--	2.8E-3				--	--
1,4-dichlorobenzene		2.9E-01	1.30E-05	2.9E-09	2.0E-07	--	7.1E-7				--	--
trichlorofluoromethane		2.0E-01	9.88E-05	2.2E-08	1.5E-06	--	7.7E-6				--	--

↑
Sum of outdoor air concentrations from soil/sediment and groundwater sources
Outdoor air concentrations only

↑
Calculated based on shallow groundwater concentrations assumed in excavation

Exposure to Chemicals via Ingestion of Soils

Stage 1 and Stage 2 Development

General Data/ Equations		Units	Exposure Calculations (RME)	
			Ingestion of Soils by Intrusive Workers (Table 5-4, Scenario 2)	
Exposure Parameters				
Exposure Frequency (EF)	days/year	30	Assume intrusive works for 30 days of the year	
Exposure Duration (ED)	years	1	Assume works occur over a 1 year period	
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	
Averaging Time - Threshold (ATn)	days	365	USEPA 1989 and CSMS 1996	
Ingestion Rate (IRs)	mg/day	25	Soil intake for adults as per CSMS 1996	
Fraction Ingested (FI)	-	100%	All soil ingested from site	
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in soil	
Conversion factor (CF)	mg to kg	1.E-06	Conversion	
Intake Factor = $\frac{IRs \cdot FI \cdot B \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$	kg/kg/day	4.2E-10	NonThreshold	
		2.9E-08	Threshold	

Daily Intake from Soil = Concentration in Soil x Intake Factor

NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Soil (mg/kg)	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient Risk
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL	1.9E-7	5.2E-1	
mercury (as total, inorganic)		5.4E-04	1233	5.2E-07	3.6E-05	--	6.7E-2
chromium (assumed Cr(VI))		2.1E-03	1500	6.3E-07	4.4E-05	--	2.1E-2
lead		1.9E-03	7670	3.2E-06	2.3E-04	--	1.2E-1
benzene	3.5E-02		31.3	1.3E-08	9.2E-07	4.6E-10	--
ethylbenzene		9.7E-02	141	5.9E-08	4.1E-06	--	4.3E-5
xylenes		1.8E-01	41.4	1.7E-08	1.2E-06	--	6.9E-6
TPH C6-C9 aliphatic		4.5E+00	12100	5.1E-06	3.6E-04	--	7.9E-5
TPH C10-C14 aromatic		4.0E-02	1220	5.1E-07	3.6E-05	--	9.0E-4
TPH C10-C14 aliphatic		9.0E-02	1220	5.1E-07	3.6E-05	--	4.0E-4
TPH C15+ aromatic		2.7E-02	2795	1.2E-06	8.2E-05	--	3.0E-3
TPH C15+ aliphatic		1.8E+00	2795	1.2E-06	8.2E-05	--	4.6E-5
vinyl chloride (adult exposures)	1.2E+00		384	1.6E-07	1.1E-05	1.9E-7	--
cis-1,2-dichloroethene		1.0E-02	302	1.3E-07	8.9E-06	--	8.9E-4
1,2-dichloroethane (EDC)	1.2E-02		29.8	1.2E-08	8.7E-07	1.5E-10	--
trichloroethene (TCE)		1.5E-03	1050	4.4E-07	3.1E-05	--	2.1E-2
1,1,2-trichloroethane		4.0E-03	115	4.8E-08	3.4E-06	--	8.4E-4
tetrachloroethene (PCE)		9.2E-03	7130	3.0E-06	2.1E-04	--	2.3E-2
1,1,2,2-tetrachloroethane		4.0E-02	42.7	1.8E-08	1.3E-06	--	3.1E-5
chloroform		6.5E-03	213	8.9E-08	6.3E-06	--	9.6E-4
PAHs (BaP equivalent)	5.0E-01		14.1	5.9E-09	4.1E-07	3.0E-9	--
hexachlorobutadiene (HCBD)		2.0E-04	326	1.4E-07	9.6E-06	--	4.8E-2
hexachlorobenzene (HCB)		1.6E-04	1170	4.9E-07	3.4E-05	--	2.1E-1
bis(2-chloroethyl)ether	1.1E+00		2.1	8.8E-10	6.2E-08	9.7E-10	--

↑
Maximum concentration reported in Soils and Sediments from Block 1 and Block 2
Maximum reported from Tables 3-3 and 3-4 in report

Dermal Exposure to Chemicals via Contact with Soil

Stage 1 and Stage 2 Development (Hawley Method)

General Data/ Equations		Units	Exposure Calculations (RME)				
Exposure Parameters			Dermal Contact with Soils by Intrusive Workers (Table 5-4, Scenario 2)				
Exposure Frequency (EF)		days/year	30	Assume intrusive works for 30 days of the year			
Exposure Duration (ED)		years	1	Assume works occur over a 1 year period			
Body Weight (BW)		kg	70	USEPA 1989 and CSMS 1996			
Averaging Time - NonThreshold (ATc)		days	25550	USEPA 1989 and CSMS 1996			
Averaging Time - Threshold (ATn)		days	365	USEPA 1989 and CSMS 1996			
Surface Area (SAs)		cm ²	3300	Exposed surface area for construction workers as per USEPA 2001			
Adherence Factor (AF)		mg/cm ²	0.51	Default value as per CSMS 1996			
Dermal Absorption Rate (Abs)		per hour	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996			
Exposure Time (ET)		hr/day	12	Assume that soil remains on skin for 12 hours before washing			
Matrix Effect (ME)		-	15%	Absorption of chemicals from soil matrix is 15% from pure compound			
Conversion Factor (CF)		mg to kg	1.E-06	Conversion			
Site-Specific Factor (SS), -			1.00	Not used for intrusive works			
Intake Factor = $\frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$		kg-hr/kg/day	2.5E-10	NonThreshold			
			1.8E-08	Threshold			
<i>Daily Intake from Soil = Concentration in Soil x Intake Factor</i> <i>NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake for Threshold Effects/ADI)</i>							
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	1.1E-7	1.8E-1
benzene	3.5E-02		31.3	8.0E-09	5.6E-07	2.8E-10	--
ethylbenzene		9.7E-02	141	3.6E-08	2.5E-06	--	2.6E-5
xylenes		1.8E-01	41.4	1.1E-08	7.4E-07	--	4.2E-6
TPH C6-C9 aliphatic		4.5E+00	12100	3.1E-06	2.2E-04	--	4.8E-5
TPH C10-C14 aromatic		4.0E-02	1220	3.1E-07	2.2E-05	--	5.4E-4
TPH C10-C14 aliphatic		9.0E-02	1220	3.1E-07	2.2E-05	--	2.4E-4
TPH C15+ aromatic		3.0E-02	2795	7.1E-07	5.0E-05	--	1.7E-3
TPH C15+ aliphatic		1.8E+00	2795	7.1E-07	5.0E-05	--	2.8E-5
vinyl chloride (adult exposures)	1.2E+00		384	9.8E-08	6.8E-06	1.1E-7	--
cis-1,2-dichloroethene		1.0E-02	302	7.7E-08	5.4E-06	--	5.4E-4
1,2-dichloroethane (EDC)	1.2E-02		29.8	7.6E-09	5.3E-07	9.1E-11	--
trichloroethene (TCE)		1.5E-03	1050	2.7E-07	1.9E-05	--	1.3E-2
1,1,2-trichloroethane		4.0E-03	115	2.9E-08	2.0E-06	--	5.1E-4
tetrachloroethene (PCE)		1.4E-02	7130	1.8E-06	1.3E-04	--	9.1E-3
1,1,2,2-tetrachloroethane		4.0E-02	42.7	1.1E-08	7.6E-07	--	1.9E-5
chloroform		1.3E-02	213	5.4E-08	3.8E-06	--	2.9E-4
PAHs (BaP equivalent)	5.0E-01		14.1	3.6E-09	2.5E-07	1.8E-9	--
hexachlorobutadiene (HCBD)		2.0E-04	326	8.3E-08	5.8E-06	--	2.9E-2
hexachlorobenzene (HCB)		1.6E-04	1170	3.0E-07	2.1E-05	--	1.3E-1
bis(2-chloroethyl)ether	1.1E+00		2.1	5.3E-10	3.7E-08	5.9E-10	--

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

Dermal Exposure to Chemicals via Contact with Soil

Stage 1 and Stage 2 Development (USEPA Method)

General Data/ Equations		Units	Exposure Calculations (RME)					
Exposure Parameters			Dermal Contact with Soils by Intrusive Workers					
Exposure Frequency (EF)	days/year	30	Assume intrusive works for 30 days of the year					
Exposure Duration (ED)	years	1	Assume works occur over a 1 year period					
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996					
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996					
Averaging Time - Threshold (ATn)	days	365	USEPA 1989 and CSMS 1996					
Surface Area (SAs)	cm ²	3300	Exposed surface area for construction workers as per USEPA 2001					
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996					
Fraction of Day Exposed (FE)	-	0.5	12 hour exposure, CSMS 1991, 1993 and 1998					
Matrix Effect (ME)	-	100%	Assume 100% bioavailability, CSMS 1996					
Conversion Factor (CF)	mg to kg	1.E-06	Conversion					
Site-Specific Factor (SS), -		1.00	Not used for intrusive works					
Intake Factor = $\frac{SAs \cdot AF \cdot FE \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$		kg-hr/kg/day	1.4E-08	NonThreshold				
			9.9E-07	Threshold				
<i>Daily Intake from Soil = Concentration in Soil x Absorption x Intake Factor</i> <i>NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake for Threshold Effects/ADI)</i>								
Chemical	Toxicity and Dermal Data			Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL		7.6E-8	1.2E-1
benzene	3.5E-02		0.01	31.3	4.4E-09	3.1E-07	1.5E-10	--
ethylbenzene		9.7E-02	0.01	141	2.0E-08	1.4E-06	--	1.4E-5
xylenes		1.8E-01	0.01	41.4	5.8E-09	4.1E-07	--	2.3E-6
TPH C6-C9 aliphatic		4.5E+00	0.01	12100	1.7E-06	1.2E-04	--	2.7E-5
TPH C10-C14 aromatic		4.0E-02	0.13	1220	2.2E-06	1.6E-04	--	3.9E-3
TPH C10-C14 aliphatic		9.0E-02	0.01	1220	1.7E-07	1.2E-05	--	1.3E-4
TPH C15+ aromatic		3.0E-02	0.13	2795	5.1E-06	3.6E-04	--	1.2E-2
TPH C15+ aliphatic		1.8E+00	0.01	2795	3.9E-07	2.8E-05	--	1.5E-5
vinyl chloride (adult exposures)	1.2E+00		0.01	384	5.4E-08	3.8E-06	6.2E-8	--
cis-1,2-dichloroethene		1.0E-02	0.01	302	4.3E-08	3.0E-06	--	3.0E-4
1,2-dichloroethane (EDC)	1.2E-02		0.01	29.8	4.2E-09	2.9E-07	5.0E-11	--
trichloroethene (TCE)		1.5E-03	0.01	1050	1.5E-07	1.0E-05	--	7.1E-3
1,1,2-trichloroethane		4.0E-03	0.01	115	1.6E-08	1.1E-06	--	2.8E-4
tetrachloroethene (PCE)		9.2E-03	0.01	7130	1.0E-06	7.0E-05	--	7.6E-3
1,1,2,2-tetrachloroethane		4.0E-02	0.01	42.7	6.0E-09	4.2E-07	--	1.1E-5
chloroform		6.5E-03	0.01	213	3.0E-08	2.1E-06	--	3.2E-4
PAHs (BaP equivalent)	5.0E-01		0.13	14.1	2.6E-08	1.8E-06	1.3E-8	--
hexachlorobutadiene (HCBd)		2.0E-04	0.01	326	4.6E-08	3.2E-06	--	1.6E-2
hexachlorobenzene (HCB)		1.6E-04	0.01	1170	1.7E-07	1.2E-05	--	7.2E-2
bis(2-chloroethyl)ether	1.1E+00		0.01	2.1	3.0E-10	2.1E-08	3.3E-10	--

↑
 Maximum concentration reported in Soils from Block 1 and Block 2
 Maximum reported from Tables 3-3 and 3-4 in report

Exposure to Chemicals via Incidental Ingestion of Water

Stage 1 and Stage 2 Development

General Data/ Equations		Units	Exposure Calculations (RME)				
Exposure Parameters			Ingestion of Shallow Groundwater during Intrusive Works (Table 5-4, Scenario 3)				
Exposure Frequency (EF)	days/year	30	Assume intrusive works occur over 30 days				
Exposure Duration (ED)	years	1	Assume works occur over a 1 year period				
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996				
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996				
Averaging Time - Threshold (ATn)	days	365	USEPA 1989 and CSMS 1996				
Ingestion Rate (IRw)	L/day or L/hr	0.005	Incidental ingestion of 5 ml (1 teaspoon) of water per day while working				
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.				
Intake Factor = $\frac{IRw \cdot ET \cdot B \cdot EF \cdot ED}{BW \cdot AT}$		L/kg/day	8.4E-08	NonThreshold			
			5.9E-06	Threshold			
<i>Daily Intake from Water = Concentration in Water x Intake Factor</i> <i>NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake for Threshold Effects/ADI)</i>							
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Water	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	6.7E-7	3.9E-2
vinyl chloride (adult exposures)	1.2E+00		6.8	5.7E-07	4.0E-05	6.6E-7	--
1,1-dichloroethene		4.6E-02	0.056	4.7E-09	3.3E-07	--	7.1E-6
trans-1,2-dichloroethene		1.7E-02	0.54	4.5E-08	3.2E-06	--	1.9E-4
cis-1,2-dichloroethene		1.0E-02	4.8	4.0E-07	2.8E-05	--	2.8E-3
1,2-dichloroethane (EDC)	1.2E-02		9.9	8.3E-07	5.8E-05	1.0E-8	--
trichloroethene (TCE)		1.5E-03	1.2	1.0E-07	7.0E-06	--	4.8E-3
1,1,2-trichloroethane		4.0E-03	0.21	1.8E-08	1.2E-06	--	3.1E-4
tetrachloroethene (PCE)		9.2E-03	1.8	1.5E-07	1.1E-05	--	1.1E-3
chloroform		6.5E-03	0.95	8.0E-08	5.6E-06	--	8.6E-4
carbon tetrachloride		5.0E-04	2.3	1.9E-07	1.4E-05	--	2.7E-2
dichloromethane		9.6E-04	0.056	4.7E-09	3.3E-07	--	3.4E-4
hexachlorobutadiene (HCBd)		2.0E-04	0.036	3.0E-09	2.1E-07	--	1.1E-3

↑
Concentrations considered representative of Shallow Groundwater (refer to Table 3-2 in report)

Dermal Exposure to Chemicals via Contact with Water

Stage 1 and Stage 2 Development

General Data/ Equations				Exposure Calculations (RME)					
Units		Dermal Contact with Groundwater during Intrusive Works (Table 5-4, Scenario 3)							
Exposure Parameters									
Exposure Frequency (EF)	days/year		30	Assume intrusive works occur over 30 days					
Exposure Duration (ED)	years		1	Assume works occur over a 1 year period					
Body Weight (BW)	kg		70	USEPA 1989 and CSMS 1996					
Averaging Time - NonThreshold (ATc)	days		25550	USEPA 1989 and CSMS 1996					
Averaging Time - Threshold (ATn)	days		365	USEPA 1989 and CSMS 1996					
Surface Area (SAw)	cm ²		3300	Exposed surface area of construction worker as per USEPA 2001					
Exposure Time (ET)	hr/day		2	Assume contact with water for 2 hours per day					
Conversion Factor (CF)	L/cm ³		1.E-03	Conversion of units					
Intake Factor = $\frac{SAw \cdot ET \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$		L-hr/(cm ³ -kg-day)	1.1E-04	NonThreshold					
			7.7E-03	Threshold					
<i>Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor</i> <i>NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)</i>									
Chemical	Toxicity Data			Concentration in Water (mg/L)	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (mg/kg/day)	Dermal Permeability (cm/hr)		NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)	
						TOTAL	9.9E-6	1.2E+0	
vinyl chloride (adult exposures)	1.2E+00		1.1E-2	6.8	8.5E-06	6.0E-04	9.8E-6	--	
1,1-dichloroethene		4.6E-02	1.6E-2	0.056	9.9E-08	6.9E-06	--	1.5E-4	
trans-1,2-dichloroethene		1.7E-02	1.5E-2	0.54	8.9E-07	6.2E-05	--	3.7E-3	
cis-1,2-dichloroethene		1.0E-02	1.5E-2	4.8	7.9E-06	5.5E-04	--	5.5E-2	
1,2-dichloroethane (EDC)	1.2E-02		5.3E-3	9.9	5.8E-06	4.1E-04	7.0E-8	--	
trichloroethene (TCE)		1.5E-03	1.6E-2	1.2	2.1E-06	1.5E-04	--	1.0E-1	
1,1,2-trichloroethane		4.0E-03	6.4E-3	0.21	1.5E-07	1.0E-05	--	2.6E-3	
tetrachloroethene (PCE)		9.2E-03	4.8E-2	1.8	9.6E-06	6.7E-04	--	7.2E-2	
chloroform		6.5E-03	8.9E-3	0.95	9.4E-07	6.6E-05	--	1.0E-2	
carbon tetrachloride		5.0E-04	2.2E-2	2.3	5.6E-06	3.9E-04	--	7.9E-1	
dichloromethane		9.6E-04	4.5E-3	0.056	2.8E-08	2.0E-06	--	2.0E-3	
hexachlorobutadiene (HCBD)		2.0E-04	1.2E-1	0.036	4.8E-07	3.3E-05	--	1.7E-1	

↑
Concentrations considered representative of Shallow Groundwater (refer to Table 3-2 in report)

Appendix D

Risk Calculations

A.4 Calculations Relevant for Table 5-5 in Main Report



Inhalation of Volatile Chemicals - Emissions from Other Sources on BIP

Stage 1 and Stage 2 Development

General Data/ Equations		Units	Exposure Calculations					
			Indoors/Outdoors - Workers on Site (Tables 5-2 and 5-5, Scenario 1)					
Exposure Parameters								
Exposure Frequency (EF)		days/year	240	Work for 5 days per week for 48 weeks				
Exposure Duration (ED)		years	30	Working life at the one location				
Body Weight (BW)		kg	70	USEPA 1989 and CSMS 1996				
Averaging Time - NonThreshold (ATc)		days	25550	USEPA 1989 and CSMS 1996				
Averaging Time - Threshold (ATn)		days	10950	USEPA 1989 and CSMS 1996				
Inhalation Rate (InhR)		m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)				
Exposure Time (ET)		hours/day	10	Assume 9 hours indoors and 1 hr outdoors				
Bioavailability (B)		-	100%	Assume 100% bioavailability via inhalation				
Fraction Inhaled (FI)		-	100%	Assume all time spent on the site				
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$		m ³ /kg/day	5.1E-02	NonThreshold				
			1.2E-01	Threshold				
<i>Daily Intake = Concentration in Air x Intake Factor</i> <i>NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)</i>								
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	
				TOTAL		1.5E-9	4.7E-3	
mercury (as total, inorganic)		2.2E-04	1.60E-06	8.2E-08	1.9E-07	--	8.8E-4	
vinyl chloride (adult exposures)	1.5E-02		1.10E-06	5.6E-08	1.3E-07	8.7E-10	--	
1,1-dichloroethene		5.7E-02	1.10E-07	5.6E-09	1.3E-08	--	2.3E-7	
trans-1,2-dichloroethene		1.7E-02	7.40E-09	3.8E-10	8.8E-10	--	5.2E-8	
1,1-dichloroethane		1.4E-01	5.50E-09	2.8E-10	6.6E-10	--	4.6E-9	
cis-1,2-dichloroethene		1.0E-02	6.10E-08	3.1E-09	7.3E-09	--	7.3E-7	
1,2-dichloroethane (EDC)	9.8E-03		8.40E-07	4.3E-08	1.0E-07	4.2E-10	--	
trichloroethene (TCE)	1.5E-03		2.60E-06	1.3E-07	3.1E-07	2.0E-10	--	
1,1,2-trichloroethane		4.0E-03	1.40E-08	7.2E-10	1.7E-09	--	4.2E-7	
tetrachloroethene (PCE)		3.8E-02	4.10E-04	2.1E-05	4.9E-05	--	1.3E-3	
chloroform	1.5E-03	2.0E-02	5.40E-07	2.8E-08	6.5E-08	4.1E-11	3.2E-6	
dichloromethane		2.3E-01	4.60E-08	2.4E-09	5.5E-09	--	2.4E-8	
hexachlorobutadiene (HCBD)		2.0E-04	1.00E-06	5.1E-08	1.2E-07	--	6.0E-4	
hexachlorobenzene (HCB)		1.6E-04	6.10E-09	3.1E-10	7.3E-10	--	4.6E-6	
hexachloroethane (HCE)		1.0E-03	1.60E-05	8.2E-07	1.9E-06	--	1.9E-3	

↑
 Modelled ground level concentrations - emissions from BIP including CPWE remediation
 Concentrations considered presented in Table 3-11

Inhalation of Volatile Chemicals above Groundwater (and some soil impacts)

Stage 2 Development

General Data/ Equations		Exposure Calculations (Average)				Exposure Calculations (RME)			
		Indoors on Block 1 (Table 5-5, Scenario 2)				Indoors on Block 1 (Table 5-5, Scenario 2)			
Units									
Exposure Parameters									
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks			240	Work for 5 days per week for 48 weeks		
Exposure Duration (ED)	years	30	Working life at the one location			30	Working life at the one location		
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996			70	USEPA 1989 and CSMS 1996		
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996			25550	USEPA 1989 and CSMS 1996		
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996			10950	USEPA 1989 and CSMS 1996		
Inhalation Rate (InhR)	m ³ /hr	1.17	Inhalation rate indoors			1.17	Inhalation rate indoors		
Exposure Time (ET)	hours/day	9	Assume 9 hours per day spent indoors			9	Assume 9 hours per day spent indoors		
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation			100%	Assume 100% bioavailability via inhalation		
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume			100%	Assume all time spent at inside building located above plume		
Intake Factor = InhR*ET*B*FI*EF*ED	m ³ /kg/day	4.2E-02	NonThreshold			4.2E-02	NonThreshold		
BW*AT		9.9E-02	Threshold			9.9E-02	Threshold		

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	1.3E-6	8.9E-1			TOTAL	4.2E-6	2.7E+0
benzene	2.1E-02	7.7E-03	5.70E-05	2.4E-06	5.6E-06	5.1E-8	7.3E-4	9.70E-04	4.1E-05	9.6E-05	8.6E-7	1.2E-2
toluene		6.7E-02	2.65E-04	1.1E-05	2.6E-05	--	3.9E-4	4.40E-04	1.9E-05	4.4E-05	--	6.5E-4
ethylbenzene		6.3E+00	2.05E-05	8.7E-07	2.0E-06	--	3.2E-7	2.50E-05	1.1E-06	2.5E-06	--	3.9E-7
xylenes		2.4E-01	1.01E-04	4.3E-06	1.0E-05	--	4.1E-5	1.30E-04	5.5E-06	1.3E-05	--	5.3E-5
TPH C6-C9 aliphatic		1.8E-01				--	--				--	--
TPH C10-C14 aromatic		5.1E-02				--	--				--	--
vinyl chloride (adult exposures)	1.5E-02		1.20E-03	5.1E-05	1.2E-04	7.8E-7	--	2.38E-03	1.0E-04	2.4E-04	1.6E-6	--
1,1-dichloroethene		5.7E-02				--	--				--	--
trans-1,2-dichloroethene		1.7E-02	1.80E-04	7.6E-06	1.8E-05	--	1.0E-3	1.80E-04	7.6E-06	1.8E-05	--	1.0E-3
1,1-dichloroethane		1.4E-01				--	--				--	--
cis-1,2-dichloroethene		1.0E-02	6.30E-04	2.7E-05	6.2E-05	--	6.2E-3	6.30E-04	2.7E-05	6.2E-05	--	6.2E-3
1,2-dichloroethane (EDC)	9.8E-03		7.27E-04	3.1E-05	7.2E-05	3.0E-7	--	2.80E-03	1.2E-04	2.8E-04	1.2E-6	--
trichloroethene (TCE)	1.5E-03		7.29E-04	3.1E-05	7.2E-05	4.7E-8	--	5.00E-03	2.1E-04	4.9E-04	3.2E-7	--
1,1,2-trichloroethane		4.0E-03	8.57E-05	3.6E-06	8.5E-06	--	2.1E-3	1.90E-04	8.1E-06	1.9E-05	--	4.7E-3
tetrachloroethene (PCE)		3.8E-02	3.33E-03	1.4E-04	3.3E-04	--	8.7E-3	3.90E-02	1.7E-03	3.9E-03	--	1.0E-1
1,1,2,2-tetrachloroethane	1.1E-02		1.46E-04	6.2E-06	1.4E-05	6.5E-8	--	3.80E-04	1.6E-05	3.8E-05	1.7E-7	--
chloroform	1.5E-03	2.0E-02	6.67E-04	2.8E-05	6.6E-05	4.2E-8	3.3E-3	2.50E-03	1.1E-04	2.5E-04	1.6E-7	1.2E-2
carbon tetrachloride		6.1E-04	4.36E-03	1.8E-04	4.3E-04	--	7.1E-1	1.30E-02	5.5E-04	1.3E-03	--	2.1E+0
dichloromethane		2.3E-01	2.33E-03	9.9E-05	2.3E-04	--	1.0E-3	1.05E-02	4.5E-04	1.0E-03	--	4.5E-3
1,2,4-trimethylbenzene		8.6E-04	6.80E-05	2.9E-06	6.7E-06	--	7.8E-3	8.80E-05	3.7E-06	8.7E-06	--	1.0E-2
1,3,5-trimethylbenzene		8.6E-04				--	--				--	--
naphthalene		8.1E-04	1.25E-03	5.3E-05	1.2E-04	--	1.5E-1	3.20E-03	1.4E-04	3.2E-04	--	3.9E-1
trichlorofluoromethane		2.0E-01				--	--				--	--

↑
Calculated from average of measured data for Block 1

↑
Calculated from maximum of measured data for Block 1

Inhalation of Volatile Chemicals above Groundwater (and some soil impacts)

Stage 2 Development

General Data/ Equations		Units	Exposure Calculations (RME)	
			Outdoors on Block 1 or 2 (Table 5-5, Scenario 2)	
Exposure Parameters				
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	
Exposure Duration (ED)	years	30	Working life at the one location	
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003	
Exposure Time (ET)	hours/day	1	Assume 1 hour outdoors	
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	
Fraction Inhaled (FI)	-	100%	Assume all time outdoors spent in area above plume	
Intake Factor = InhR*ET*B*FI*EF*ED		m³/kg/day	8.9E-03	NonThreshold
BW*AT			2.1E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	1.6E-7	1.0E-1
benzene	2.1E-02	7.7E-03	1.74E-04	1.5E-06	3.6E-06	3.2E-8	4.7E-4
toluene		6.7E-02	1.71E-04	1.5E-06	3.5E-06	--	5.3E-5
ethylbenzene		6.3E+00	1.63E-05	1.4E-07	3.4E-07	--	5.4E-8
xylenes		2.4E-01	7.90E-05	7.0E-07	1.6E-06	--	6.7E-6
TPH C6-C9 aliphatic		1.8E-01	2.32E-06	2.1E-08	4.8E-08	--	2.7E-7
TPH C10-C14 aromatic		5.1E-02	2.87E-06	2.5E-08	5.9E-08	--	1.2E-6
vinyl chloride (adult exposures)	1.5E-02		4.27E-04	3.8E-06	8.8E-06	5.8E-8	--
1,1-dichloroethene		5.7E-02	2.16E-05	1.9E-07	4.5E-07	--	7.8E-6
trans-1,2-dichloroethene		1.7E-02	3.23E-05	2.9E-07	6.7E-07	--	3.9E-5
1,1-dichloroethane		1.4E-01	4.85E-05	4.3E-07	1.0E-06	--	7.0E-6
cis-1,2-dichloroethene		1.0E-02	1.13E-04	1.0E-06	2.3E-06	--	2.3E-4
1,2-dichloroethane (EDC)	9.8E-03		5.03E-04	4.5E-06	1.0E-05	4.4E-8	--
trichloroethene (TCE)	1.5E-03		8.98E-04	8.0E-06	1.9E-05	1.2E-8	--
1,1,2-trichloroethane		4.0E-03	3.41E-05	3.0E-07	7.1E-07	--	1.8E-4
tetrachloroethene (PCE)		3.8E-02	7.00E-03	6.2E-05	1.4E-04	--	3.8E-3
1,1,2,2-tetrachloroethane	1.1E-02		6.82E-05	6.0E-07	1.4E-06	6.3E-9	--
chloroform	1.5E-03	2.0E-02	4.49E-04	4.0E-06	9.3E-06	5.8E-9	4.6E-4
carbon tetrachloride		6.1E-04	2.33E-03	2.1E-05	4.8E-05	--	7.9E-2
dichloromethane		2.3E-01	1.89E-03	1.7E-05	3.9E-05	--	1.7E-4
1,2,4-trimethylbenzene		8.6E-04	3.41E-05	3.0E-07	7.1E-07	--	8.2E-4
1,3,5-trimethylbenzene		8.6E-04	6.82E-06	6.0E-08	1.4E-07	--	1.6E-4
naphthalene		8.1E-04	5.75E-04	5.1E-06	1.2E-05	--	1.5E-2
trichlorofluoromethane		2.0E-01	9.88E-05	8.7E-07	2.0E-06	--	1.0E-5

↑
Calculated from maximum of measured data

Inhalation of Volatile Chemicals above Groundwater (and some soil impacts) - using Occupational Inhalation Exposure Guidance

Stage 2 Development

General Data/ Equations		Exposure Calculations (RME)		Exposure Calculations (RME)	
Units		Indoors on Block 1 (Table 5-5, Scenario 2)		Outdoors on Block 1 or 2 (Table 5-5, Scenario 2)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.17	Inhalation rate indoors	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	9	Assume 9 hours per day spent indoors	1	Assume 1 hour outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time spent at inside building located above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	4.2E-02	NonThreshold	8.9E-03	NonThreshold
		9.9E-02	Threshold	2.1E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor
NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	4.2E-6	1.5E-2			TOTAL	1.6E-7	5.7E-4		
benzene	2.1E-02	4.6E-01	9.70E-04	4.1E-05	9.6E-05	8.6E-7	2.1E-4	1.74E-04	1.5E-06	3.6E-06	3.2E-8	7.9E-6		
toluene		2.7E+01	4.40E-04	1.9E-05	4.4E-05	--	1.6E-6	1.71E-04	1.5E-06	3.5E-06	--	1.3E-7		
ethylbenzene		6.2E+01	2.50E-05	1.1E-06	2.5E-06	--	4.0E-8	1.63E-05	1.4E-07	3.4E-07	--	5.4E-9		
xylenes		5.0E+01	1.30E-04	5.5E-06	1.3E-05	--	2.6E-7	7.90E-05	7.0E-07	1.6E-06	--	3.3E-8		
TPH C6-C9 aliphatic		1.0E+01				--	--	2.32E-06	2.1E-08	4.8E-08	--	4.7E-9		
TPH C10-C14 aromatic		5.0E+01				--	--	2.87E-06	2.5E-08	5.9E-08	--	1.2E-9		
vinyl chloride (adult exposures)	1.5E-02	1.9E+00	2.38E-03	1.0E-04	2.4E-04	1.6E-6	1.3E-4	4.27E-04	3.8E-06	8.8E-06	5.8E-8	4.8E-6		
1,1-dichloroethene		2.9E+00				--	--	2.16E-05	1.9E-07	4.5E-07	--	1.6E-7		
trans-1,2-dichloroethene		1.1E+02	1.80E-04	7.6E-06	1.8E-05	--	1.6E-7	3.23E-05	2.9E-07	6.7E-07	--	5.9E-9		
1,1-dichloroethane		5.9E+01				--	--	4.85E-05	4.3E-07	1.0E-06	--	1.7E-8		
cis-1,2-dichloroethene		1.1E+02	6.30E-04	2.7E-05	6.2E-05	--	5.5E-7	1.13E-04	1.0E-06	2.3E-06	--	2.1E-8		
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.80E-03	1.2E-04	2.8E-04	1.2E-6	4.8E-5	5.03E-04	4.5E-06	1.0E-05	4.4E-8	1.8E-6		
trichloroethene (TCE)	1.5E-03	7.7E+00	5.00E-03	2.1E-04	4.9E-04	3.2E-7	6.4E-5	8.98E-04	8.0E-06	1.9E-05	1.2E-8	2.4E-6		
1,1,2-trichloroethane		7.9E+00	1.90E-04	8.1E-06	1.9E-05	--	2.4E-6	3.41E-05	3.0E-07	7.1E-07	--	9.0E-8		
tetrachloroethene (PCE)		4.8E+01	3.90E-02	1.7E-03	3.9E-03	--	8.1E-5	7.00E-03	6.2E-05	1.4E-04	--	3.0E-6		
1,1,2,2-tetrachloroethane	1.1E-02	9.9E-01	3.80E-04	1.6E-05	3.8E-05	1.7E-7	3.8E-5	6.82E-05	6.0E-07	1.4E-06	6.3E-9	1.4E-6		
chloroform	1.5E-03	1.4E+00	2.50E-03	1.1E-04	2.5E-04	1.6E-7	1.7E-4	4.49E-04	4.0E-06	9.3E-06	5.8E-9	6.5E-6		
carbon tetrachloride		9.0E-02	1.30E-02	5.5E-04	1.3E-03	--	1.4E-2	2.33E-03	2.1E-05	4.8E-05	--	5.4E-4		
dichloromethane		2.5E+01	1.05E-02	4.5E-04	1.0E-03	--	4.2E-5	1.89E-03	1.7E-05	3.9E-05	--	1.6E-6		
1,2,4-trimethylbenzene		1.8E+01	8.80E-05	3.7E-06	8.7E-06	--	5.0E-7	3.41E-05	3.0E-07	7.1E-07	--	4.0E-8		
1,3,5-trimethylbenzene		1.8E+01				--	--	6.82E-06	6.0E-08	1.4E-07	--	8.0E-9		
naphthalene		7.4E+00	3.20E-03	1.4E-04	3.2E-04	--	4.3E-5	5.75E-04	5.1E-06	1.2E-05	--	1.6E-6		
trichlorofluoromethane		8.0E+02				--	--	9.88E-05	2.0E-06	2.0E-06	--	2.5E-9		

↑
Calculated from maximum of measured data

↑
Calculated from maximum of measured data

Inhalation of Volatile Chemicals above Soil Hot-Spots (where not addressed by measured soil gas or flux data)

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME) - Soils		Exposure Calculations (RME) - Sediments	
		Indoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)		Indoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.17	Inhalation rate indoors	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	9	Assume 9 hours per day spent indoors	9	Assume 9 hours per day spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	4.2E-02 9.9E-02	NonThreshold Threshold	4.2E-02 9.9E-02	NonThreshold Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	5.2E-4	6.2E+0			TOTAL	3.8E-3	1.2E+1
TPH C6-C9 aliphatic		1.8E-01				--	--	8.16E-01	3.5E-02	8.1E-02	--	4.5E-1
TPH C10-C14 aromatic		5.1E-02				--	--	7.62E-03	3.2E-04	7.5E-04	--	1.5E-2
TPH C10-C14 aliphatic		2.6E-01				--	--	5.36E-02	2.3E-03	5.3E-03	--	2.1E-2
vinyl chloride (adult exposures)	1.5E-02		7.69E-01	3.3E-02	7.6E-02	5.0E-4	--	5.57E+00	2.4E-01	5.5E-01	3.6E-3	--
trans-1,2-dichloroethene		1.7E-02	3.47E-01	1.5E-02	3.4E-02	--	2.0E+0	4.39E-02	1.9E-03	4.3E-03	--	2.6E-1
1,1-dichloroethane		1.4E-01	6.31E-02	2.7E-03	6.2E-03	--	4.4E-2				--	--
cis-1,2-dichloroethene		1.0E-02	3.38E-01	1.4E-02	3.3E-02	--	3.3E+0	6.19E-01	2.6E-02	6.1E-02	--	6.1E+0
1,2-dichloroethane (EDC)	9.8E-03		4.74E-03	2.0E-04	4.7E-04	2.0E-6	--	3.14E-02	1.3E-03	3.1E-03	1.3E-5	--
trichloroethene (TCE)	1.5E-03		1.74E-01	7.4E-03	1.7E-02	1.1E-5	--	1.47E+00	6.3E-02	1.5E-01	9.4E-5	--
1,1,2-trichloroethane		4.0E-03	3.52E-04	1.5E-05	3.5E-05	--	8.7E-3	4.05E-02	1.7E-03	4.0E-03	--	1.0E+0
tetrachloroethene (PCE)		3.8E-02	2.66E-01	1.1E-02	2.6E-02	--	7.0E-1	4.24E-01	1.8E-02	4.2E-02	--	1.1E+0
1,1,2,2-tetrachloroethane	1.1E-02		8.82E-04	3.7E-05	8.7E-05	3.9E-7	--	3.69E-03	1.6E-04	3.7E-04	1.6E-6	--
chloroform	1.5E-03	2.0E-02	9.65E-03	4.1E-04	9.5E-04	6.0E-7	4.8E-2	5.27E-01	2.2E-02	5.2E-02	3.3E-5	2.6E+0
carbon tetrachloride		6.1E-04				--	--	3.05E-04	1.3E-05	3.0E-05	--	4.9E-2
1,3,5-trichlorobenzene		4.6E-04	1.50E-04	6.4E-06	1.5E-05	--	3.2E-2				--	--
1,4-dichlorobenzene		2.9E-01				--	--	1.88E-04	8.0E-06	1.9E-05	--	6.5E-5

↑
Modelled from maximum measured soil concentrations in areas where soil gas or flux data have not been collected (Table 3-5)

↑
Modelled from maximum measured sediment concentrations (refer to Table 3-6)

Inhalation of Volatile Chemicals above Soil Hot-Spots (where not addressed by measured soil gas or flux data)

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME) - Soils				Exposure Calculations (RME)- Sediments			
		Outdoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)				Outdoors on Block 1 or 2 (Tables 5-2 and 5-5, Scenario 3)			
Exposure Parameters									
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks		240	Work for 5 days per week for 48 weeks			
Exposure Duration (ED)	years	30	Working life at the one location		30	Working life at the one location			
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996		70	USEPA 1989 and CSMS 1996			
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996		25550	USEPA 1989 and CSMS 1996			
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996		10950	USEPA 1989 and CSMS 1996			
Inhalation Rate (InhR)	m ³ /hr	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003		2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003			
Exposure Time (ET)	hours/day	1	Assume 1 hour outdoors		1	Assume 1 hour outdoors			
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation		100%	Assume 100% bioavailability via inhalation			
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume		100%	Assume all time outdoors spent in area above plume			
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	8.9E-03	NonThreshold		8.9E-03	NonThreshold			
		2.1E-02	Threshold		2.1E-02	Threshold			

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	1.2E-7	1.0E-2			TOTAL	8.9E-7	6.5E-2
TPH C6-C9 aliphatic		1.8E-01				--	--	1.34E-03	1.2E-05	2.8E-05	--	1.5E-4
TPH C10-C14 aromatic		5.1E-02				--	--	2.41E-03	2.1E-05	5.0E-05	--	9.7E-4
TPH C10-C14 aliphatic		2.6E-01				--	--	2.94E-03	2.6E-05	6.1E-05	--	2.4E-4
vinyl chloride (adult exposures)	1.5E-02		6.27E-04	5.5E-06	1.3E-05	8.5E-8	--	4.54E-03	4.0E-05	9.4E-05	6.2E-7	--
1,1-dichloroethene		5.7E-02				--	--				--	--
trans-1,2-dichloroethene		1.7E-02	1.25E-03	1.1E-05	2.6E-05	--	1.5E-3	1.58E-04	1.4E-06	3.3E-06	--	1.9E-4
1,1-dichloroethane		1.4E-01	2.71E-04	2.4E-06	5.6E-06	--	3.9E-5				--	--
cis-1,2-dichloroethene		1.0E-02	1.95E-03	1.7E-05	4.0E-05	--	4.0E-3	3.57E-03	3.2E-05	7.4E-05	--	7.4E-3
1,2-dichloroethane (EDC)	9.8E-03		5.32E-05	4.7E-07	1.1E-06	4.6E-9	--	3.52E-04	3.1E-06	7.3E-06	3.1E-8	--
trichloroethene (TCE)	1.5E-03		1.12E-03	1.0E-05	2.3E-05	1.5E-8	--	1.24E-02	1.1E-04	2.6E-04	1.7E-7	--
1,1,2-trichloroethane		4.0E-03	1.18E-05	1.0E-07	2.4E-07	--	6.1E-5	1.36E-03	1.2E-05	2.8E-05	--	7.0E-3
tetrachloroethene (PCE)		3.8E-02	1.07E-03	9.5E-06	2.2E-05	--	5.9E-4	8.43E-02	7.5E-04	1.7E-03	--	4.6E-2
1,1,2,2-tetrachloroethane	1.1E-02		1.21E-04	1.1E-06	2.5E-06	1.1E-8	--	5.05E-04	4.5E-06	1.0E-05	4.7E-8	--
chloroform	1.5E-03		4.61E-05	4.1E-07	9.5E-07	6.0E-10	4.8E-5	2.52E-03	2.2E-05	5.2E-05	3.3E-8	2.6E-3
carbon tetrachloride		6.1E-04				--	--	8.27E-07	7.3E-09	1.7E-08	--	2.8E-5
1,3,5-trichlorobenzene		4.6E-04	8.30E-05	7.4E-07	1.7E-06	--	3.7E-3				--	--
1,4-dichlorobenzene		2.9E-01				--	--	1.30E-05	1.2E-07	2.7E-07	--	9.4E-7

↑
Modelled from maximum measured soil concentrations in areas where soil gas or flux data have not been collected (Table 3-5)

↑
Modelled from maximum measured sediment concentrations (refer to Table 3-6)

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Springvale Drain

Stage 1 and 2 Development

General Data/ Equations		Exposure Calculations (Average)		Exposure Calculations (Average)	
Units		Indoor/Outdoor Work (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)		Outdoor Worker (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	4	Assume exposure by workers of up to 4 hours (trucks waiting)
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.1E-02	NonThreshold	3.5E-02	NonThreshold
		1.2E-01	Threshold	8.3E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	6.4E-4	8.0E+0			TOTAL	4.4E-4	5.5E+0		
vinyl chloride (adult exposures)	1.5E-02		4.30E-02	2.2E-03	5.1E-03	3.4E-5	--	4.30E-02	1.5E-03	3.6E-03	2.3E-5	--		
cis-1,2-dichloroethene		1.0E-02	4.00E-02	2.0E-03	4.8E-03	--	4.8E-1	4.00E-02	1.4E-03	3.3E-03	--	3.3E-1		
1,2-dichloroethane (EDC)	9.8E-03		1.19E+00	6.1E-02	1.4E-01	6.0E-4	--	1.19E+00	4.2E-02	9.9E-02	4.1E-4	--		
trichloroethene (TCE)	1.5E-03		8.90E-02	4.6E-03	1.1E-02	6.9E-6	--	8.90E-02	3.2E-03	7.4E-03	4.7E-6	--		
tetrachloroethene (PCE)		3.8E-02	8.60E-02	4.4E-03	1.0E-02	--	2.7E-1	8.60E-02	3.0E-03	7.1E-03	--	1.9E-1		
carbon tetrachloride		6.1E-04	3.70E-02	1.9E-03	4.4E-03	--	7.3E+0	3.70E-02	1.3E-03	3.1E-03	--	5.0E+0		

↑
Measured air concentrations
Average (of detected COPC) adjacent to Springvale Drain
Pre GTP (up to end 2006), refer to Table 3-10 in report

↑
Measured air concentrations
Average (of detected COPC) adjacent to Springvale Drain
Pre GTP (up to end 2006), refer to Table 3-10 in report

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Springvale Drain

Stage 1 and 2 Development

General Data/ Equations		Exposure Calculations (RME)		Exposure Calculations (RME)	
Units		Indoor/Outdoor Work (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)		Outdoor Worker (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	4	Assume exposure by workers of up to 4 hours (trucks waiting)
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.1E-02	NonThreshold	3.5E-02	NonThreshold
		1.2E-01	Threshold	8.3E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	1.3E-3	1.7E+1			TOTAL	9.0E-4	1.1E+1		
vinyl chloride (adult exposures)	1.5E-02		9.90E-02	5.1E-03	1.2E-02	7.8E-5	--	9.90E-02	3.5E-03	8.2E-03	5.4E-5	--		
cis-1,2-dichloroethene		1.0E-02	8.00E-02	4.1E-03	9.6E-03	--	9.6E-1	8.00E-02	2.8E-03	6.6E-03	--	6.6E-1		
1,2-dichloroethane (EDC)	9.8E-03		2.40E+00	1.2E-01	2.9E-01	1.2E-3	--	2.40E+00	8.5E-02	2.0E-01	8.3E-4	--		
trichloroethene (TCE)	1.5E-03		1.80E-01	9.2E-03	2.2E-02	1.4E-5	--	1.80E-01	6.4E-03	1.5E-02	9.6E-6	--		
tetrachloroethene (PCE)		3.8E-02	1.60E-01	8.2E-03	1.9E-02	--	5.1E-1	1.60E-01	5.7E-03	1.3E-02	--	3.5E-1		
carbon tetrachloride		6.1E-04	7.70E-02	3.9E-03	9.2E-03	--	1.5E+1	7.70E-02	2.7E-03	6.4E-03	--	1.0E+1		

↑
Measured air concentrations
Maximum measured adjacent to Springvale or Floodvale Drains
Pre GTP (up to end 2006), refer to Table 3-10 in report

↑
Measured air concentrations
Maximum measured adjacent to Springvale or Floodvale Drains
Pre GTP (up to end 2006), refer to Table 3-10 in report

Inhalation of Volatile Chemicals in Areas Above Main Plumes and Near Springvale Drain

Stage 1 and 2 Development

General Data/ Equations	Units	Exposure Calculations (RME post GTP)		Exposure Calculations (RME post GTP)	
		Indoor/Outdoor Work (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)		Outdoor Worker (Table 5-2 Scenario 5 and Table 5-5 Scenario 4)	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Work for 5 days per week for 48 weeks	240	Work for 5 days per week for 48 weeks
Exposure Duration (ED)	years	30	Working life at the one location	30	Working life at the one location
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	10950	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	2.20	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	4	Assume exposure by workers of up to 4 hours (trucks waiting)
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume	100%	Assume all time outdoors spent in area above plume
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.1E-02	NonThreshold	3.5E-02	NonThreshold
		1.2E-01	Threshold	8.3E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration		Daily Intake		Calculated Risk		Concentration		Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)		
					TOTAL	5.3E-4	6.8E+0			TOTAL	3.6E-4	4.7E+0		
vinyl chloride (adult exposures)	1.5E-02		4.30E-02	2.2E-03	5.1E-03	3.4E-5	--	4.30E-02	1.5E-03	3.6E-03	2.3E-5	--		
cis-1,2-dichloroethene		1.0E-02	1.00E-01	5.1E-03	1.2E-02	--	1.2E+0	1.00E-01	3.5E-03	8.3E-03	--	8.3E-1		
1,2-dichloroethane (EDC)	9.8E-03		9.30E-01	4.8E-02	1.1E-01	4.7E-4	--	9.30E-01	3.3E-02	7.7E-02	3.2E-4	--		
trichloroethene (TCE)	1.5E-03		1.30E-01	6.7E-03	1.6E-02	1.0E-5	--	1.30E-01	4.6E-03	1.1E-02	6.9E-6	--		
tetrachloroethene (PCE)		3.8E-02	3.80E-02	1.9E-03	4.5E-03	--	1.2E-1	3.80E-02	1.3E-03	3.1E-03	--	8.3E-2		
carbon tetrachloride		6.1E-04	2.80E-02	1.4E-03	3.3E-03	--	5.5E+0	2.80E-02	9.9E-04	2.3E-03	--	3.8E+0		
1,4-dioxane	2.7E-02	8.6E-01	1.00E-03	5.1E-05	1.2E-04	1.4E-6	1.4E-4	4.20E-04	1.5E-05	3.5E-05	4.0E-7	4.1E-5		
1,3-butadiene	6.0E-01	5.7E-03	4.20E-04	2.2E-05	5.0E-05	1.3E-5	8.8E-3	4.20E-04	1.5E-05	3.5E-05	8.9E-6	6.1E-3		

↑
Measured air concentrations
Maximum reported following commissioning of GTP
Post GTP (2007 onwards), refer to Table 3-10 in report

↑
Measured air concentrations
Maximum reported following commissioning of GTP
Post GTP (2007 onwards), refer to Table 3-10 in report

Appendix E

Review of Risk Issues - Flood Storage Area

E.1 Introduction

This appendix presents further review of risks to human health associated with specific scenarios related to the proposed design of the Southlands development and proposed risk management measures for Springvale Drain. This appendix relates to the construction of a flood storage area (as presented in The Flood Management Design prepared by Connell Wagner) on the southern portion of Block 1 as part of the Stage 1 development. The proposed storage area extends below the top of the groundwater table and hence the potential for shallow groundwater to seep into the area (basin) at the ground surface requires assessment.

This scenario has been further assessed with respect to inhalation exposures only. Direct contact with water in the flood storage area is expected to be managed such that regular exposures by workers or visitors would not occur. Maintenance of these areas is expected to be undertaken in accordance with a long term environmental management plan that addresses the potential for direct contact with shallow groundwater.

Exposures associated with the discharge of shallow groundwater (contaminated with chlorinated hydrocarbons) into the proposed flood storage area will be greater compared with the scenario where no discharge to the surface occurs. Hence further assessment of potential risks to human health has been undertaken with the aim of:

- Quantifying risks to human health associated with the potential discharge of shallow groundwater into the basin. It is noted that the Preliminary HHRA (URS, 2006) identified that this exposure scenario would be considered unacceptable;
- Quantify risks to human health associated with the potential for groundwater discharges to be managed (via a drainage system) such that it does not discharge directly to the basin, but remains at least 30-50cm below the ground surface.

The assessment of exposure and risk requires the consideration of a number of issues. These include the potential for shallow groundwater to discharge into the flood storage area (with the quantification of likely duration and frequency of discharge required), the concentrations in shallow groundwater likely to be discharged, potential routes of exposure and quantification of exposure concentrations, and calculation of risk. These are discussed further in the following sections.

E.2 Potential for Discharge and Estimation of Duration and Frequency of Discharge

An assessment of the potential for shallow groundwater to discharge into the proposed flood storage area on the southern portion of Block 1 has been undertaken by JBS Environmental Pty Ltd (12 November 2007). This assessment is attached to the end of this appendix.

The assessment presented considered the proposed level of the flood storage area in relation to historical groundwater levels on Block 1 as well as issues associated with rainfall events that are expected to result in the shallow groundwater table rising. With respect to the assessment of potential exposure to contaminated groundwater that may discharge into the flood storage area the following is of particular note:

- Water levels in the area regularly exceed the proposed excavation surface level;
- The water level response to rainfall events is expected to be similar over the majority of the flood storage area;

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Review of Risk Issues – Flood Storage Area

- Conservative modelling of potential discharges into the flood storage area indicate that shallow groundwater levels exceed the proposed excavation surface level for an average of 18% of the time each year; and
- Groundwater levels remain within 30cm (0.3m, below) of the proposed excavation level approximately 40% of the time each year. It is assumed that groundwater will be deeper than 30cm for the remainder of the year (when not discharging).

The above estimates of likely frequency of discharge each year have been assumed to be conservative estimates relevant to long term average exposure. It is expected that there may be some years where a lower or higher frequency of exposure occurs (due to varying rainfall patterns), however for the consideration of long term exposures the average estimated frequency is considered appropriate. It is also noted that significant ponding of shallow groundwater within the flood storage area is not expected to occur as discharges are expected to runoff to the collection and discharge system (with discharge of water expected to be to the GTP but yet to be confirmed (not to Springvale Drain)) rather than remain at the surface for any significant period of time.

E.3 Concentrations in Shallow Groundwater Discharging to the Basin

A review of groundwater concentrations in the vicinity of Block 1 that are relevant to the quantification of current concentrations has been presented in **Section 3.3.3** of the main report. While limited data is available regarding the quality of groundwater in the top metre of the groundwater table, the average¹ groundwater concentration reported (in the data set identified by URS) in wells screened from 2-3m and those installed as shallow (S) wells is considered representative of concentrations that may discharge to the proposed flood storage area. These concentrations have been further reviewed and COPC identified, as presented in **Table 3-2** in the main report.

Table 1 presents a summary of the COPC identified and the exposure concentrations considered relevant for the assessment of shallow groundwater seepage into the proposed flood storage area.

Table 1 COPC and Exposure Concentrations for Assessment of Flood Storage Area

COPC Identified	Exposure Concentration in Shallow Groundwater ¹ (mg/L)
carbon tetrachloride	2.3
chloroform	0.95
methylene chloride	0.056
1,1,2-trichloroethane	0.21
1,2-dichloroethane (EDC)	9.9
tetrachloroethene (PCE)	1.8
trichloroethene (TCE)	1.2
1,1-dichloroethene	0.056
cis-1,2-dichloroethene	4.8
trans-1,2-dichloroethene	0.54
vinyl chloride	6.8
hexachlorobutadiene (HCBD)	0.036

¹ Arithmetic average of concentrations reported. Where a concentration was not reported above the analytical limit of reporting, the reporting limit has been used in the calculation.

Appendix E

Review of Risk Issues – Flood Storage Area

Notes:

- 1 Concentrations in shallow groundwater that may discharge into the detention basin has been calculated as average concentration from wells screened at depths of 2-3m and shallow wells.

E.4 Calculation of Exposure and Risk

The assessment of exposure has focused on inhalation exposures by workers adjacent to the flood storage area. These workers may be predominantly in outdoors areas as there are no buildings proposed to be located adjacent to (or within 50m) of the proposed flood storage area. Hence exposures assessed include consideration of 8 hours spent outdoors close to the area.

Modelling of Shallow Groundwater Seepage to the Surface of the Flood Storage Area

The concentration of COPC in air above and close to the flood storage area has been estimated using a volatilisation model (USEPA 1994) relevant for the scenario where groundwater discharges into the flood storage area as well as the scenario where groundwater remains just below the surface. The water in the flood storage area is assumed to flow (quiescent flow) across the surface to the collection point. The area is assumed to be a grass slope approximately 170m long with a 1:10 gradient. The depth of the water has been assumed to be 0.01m (relevant for groundwater seepage, rather than flood situations where any groundwater seepage will be diluted). On this basis the volumetric flow rate of water in the flood area has been estimated to be 0.015 m³/s with a temperature of 18 °C. The mixing zone height has been set to 10m to assess potential exposures 0-10m from the flood storage area, the closest distance where workers may be present in outdoor areas.

Modelling of Emissions Associated with No Groundwater Seepage

The concentration of COPC in air above and adjacent to the proposed flood storage area, assuming that a drainage system is installed to prevent shallow groundwater discharging to the surface, have been estimated using the outdoor air vapour model presented by USEPA (1996). This model has been set up assuming the average depth to shallow groundwater is 0.5m, with groundwater rising to more shallow depths captured by the proposed drainage system.

Risks to workers have then been calculated following the methodology presented in the main HHRA report. **Table 2** presents a summary of the exposures assessed, models and assumptions considered and calculated risks. It is noted that these exposures and risks are in addition to the exposures presented in the main report with respect to other exposures during the Stage 1 development.

Calculations associated with the assessment of the flood storage area are presented in **Appendix D**.

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Review of Risk Issues – Flood Storage Area

Table 2 Summary of Exposure Pathways, Assumptions and Calculated Risks – Flood Storage Area

Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
			Non-Threshold Risk	Threshold HI
		<p>Parameters relevant for all pathways Body weight of 70 kg. Exposure for 240 days/year (5 days per week for 48 weeks) for 30 years</p>		
Inhalation of vapours derived from Detention Basin – Stage 1 development	<p>Discharge of shallow groundwater into basin: Air concentrations estimated using volatilisation model for estimating emissions from water with queiscent flow (USEPA 1994). Assumed diameter of basin of 170m, depth of surface flow of 1cm and a flow rate of 0.015 m³/s. Concentrations in surface water discharge as in Table 1. Concentrations in air estimated for workers 10m from the basin.</p>	<p>Inhalation of 2.2 m³ air per hour outdoors (8 hours) in work areas directly adjacent to the detention basin (where no buildings are constructed). Assume groundwater discharges to basin 18% of the total work time (i.e. 18% of time spent at work)</p>	3.9x10 ⁻⁵	2.1
	<p>No discharge of shallow groundwater into basin, however shallow groundwater remains just below surface of the basin. Assumed shallow groundwater with concentrations equal to those presented in Table 1 remain 30cm below ground surface. Concentrations in breathing zone estimated using an outdoor air model (USEPA, 1996 and 2002) assuming the detention basin is 170m long and wide and overlying soils are similar to fill and not saturated.</p>	<p>Inhalation of 2.2 m³ air per hour outdoors (8 hours) in work areas directly adjacent to the detention basin (where no buildings are constructed). Assume groundwater remains at least 30cm below surface of basin for remaining 82% of the year (i.e. when not discharging).</p>	1.2x10 ⁻⁶	0.086

Appendix E

Review of Risk Issues – Flood Storage Area

The following can be noted from the above table:

- Calculated risks to workers associated with emissions to air from the proposed flood storage area when shallow groundwater is not discharging to the surface are low and considered to be acceptable.
- Calculated risks to workers associated with emissions to air from the proposed flood storage area when shallow groundwater discharges to the surface 18% of the time are above the risk levels considered representative of acceptable risk (i.e non-threshold risk of 1×10^{-5} and threshold HI of 1). The calculated risks are dominated by the potential presence of vinyl chloride, EDC and carbon tetrachloride in air adjacent to the area.

Review of the above indicates that risks to workers are considered unacceptable based on the scenario where groundwater discharges directly to the flood storage area. If an appropriate drainage system were installed beneath the flood storage area (at least 30-50cm) to capture and drain shallow groundwater before it discharged to the surface, risks would be sufficiently low to ensure risks to all workers in areas adjacent to the area are protected.



JBS40341-12195

19 February 2008

Jeffrey Lord
DBL Property Pty Ltd
Level 6, 432 Kent Street, Sydney NSW 2000

(Via email: jlord@dblproperty.com)

Southlands Development - Groundwater Level Assessment Proposed Stage 2 Flood Storage Area

Dear Jeffrey,

1. Introduction and Objectives

JBS Environmental Pty Ltd has been engaged to assess the potential interaction between groundwater and the proposed Flood Storage basin to be constructed as part of the Southlands Development. The proposed basin is located on the southern portion of Block 1 Southlands and encompasses the majority of the Stage 2 development area. The basin is required to provide compensatory flood storage for the part of the flood plain that will be filled during Stage 1 of the development. Subsequent development of Southlands (i.e. Stages 2 and 3) will require additional flood mitigation works that are not considered in this assessment.

The objectives of the assessment are as follows:

- Assess whether groundwater is likely to discharge into the proposed flood storage basin; and
- In the case that discharge does occur, assess its likely duration and frequency.

2. Reviewed Data

The following data have been reviewed as part of this assessment:

- Historic standing water level measurements on Block 1 Southlands, since October 2004 (data collected and reported by URS Australia as part of the Quarterly GTP Surface and Groundwater Monitoring Program);
- Hydrographs (water levels monitored hourly) for monitoring wells (MWB12S/I/D, WG73S/D, MWB01S/I/D, MWB02S/I/D and MWB03S/I/D) located on Block 1 Southlands (data collected and reported by URS Australia as part of the Quarterly GTP Surface and Groundwater Monitoring Program); and
- The proposed finished levels of the flood detention basin (Connell Wagner drawing supplied by DBL Property).

3. Assessment of Potential for Groundwater Discharge to Basin

The following table presents a summary of measured water levels for shallow monitoring wells in the Stage 2 development area. The table also includes the approximate finished level for the flood storage basin. A figure showing the well locations is provided as Attachment A and the available hydrographs are presented in Attachment B.

Table 1
Measured Shallow Monitoring Well Water Levels

Well Name	Oct-04	Jun-05	Sep-05	Dec-05	Mar-06	Jun-06	Sep-06	Dec-06	Mar-07	Jun-07	Maximum from Hydrograph	Maximum Measured	Proposed Excavation Surface
BP24-2			3.91	4.15	3.76	4.25	4.07	3.73	3.16	4.15	NA	4.25	4.17
WG141	3.95	3.72	3.58	3.77		3.80	3.53	3.20	3.09	2.76	NA	3.95	4.17
BP36-2			3.58	3.56	2.93	3.54	3.15				NA	3.58	4.00
BP40-2			3.56	3.65	3.81	3.48	3.77	2.66	2.09	3.92	NA	3.92	3.85
BP49-2			2.67			3.09					NA	3.09	3.20
WG20	2.89	2.76	2.31	2.82	2.26	2.96	2.70	2.06			NA	2.96	3.30
BP50-2			2.52	2.86	2.30	3.07	2.87				NA	3.07	3.00
MWB01S		2.83	2.82	2.89	2.77	2.74	2.32	1.95	1.90	1.95	3.00	3.00	3.42
MWB02S		2.62	2.66	2.67	2.25	2.78	2.55	2.10	2.07	1.92	3.10	3.10	3.25
MWB03S		2.40	2.42	2.39	2.02	2.56	2.30	1.88	1.79	1.77	2.90	2.90	2.95
MWB12S				3.70	3.23	3.79	3.53	3.15	3.37	3.23	4.10	4.10	3.68
WG21	3.09	2.76	2.61	2.85	2.23	2.89	2.60	2.15	2.15	1.88	NA	3.09	3.75
WG73S	3.34	3.02	2.83	3.07	2.49	3.09	2.83	2.38	2.36	2.10	3.50	3.50	3.47

Notes: All levels reported to metres Australian Height Datum (mAHD)

The data presented in **Table 1** indicates that measured water levels have historically exceeded the proposed base of the flood storage basin at a number of locations (BP24, BP40, BP50, MWB12S and WG73S). As a result there is potential for groundwater to discharge into the proposed storage basin.

4. Potential Duration and Frequency of Groundwater Discharge to Basin

Assessment of the direction and frequency of groundwater discharge to the proposed flood storage basin requires an understanding of the mechanisms of recharge and groundwater flow in the shallow aquifer. While point water level measurements are useful in developing a general understanding of groundwater flow they do not allow detailed analysis of the dynamic water level changes related to rainfall infiltration and groundwater flow. As a result the following analysis is primarily based on the hydrographs collected since the start of GTP operation (January 2006).

Groundwater levels in the shallow aquifer at Southlands Block 1 are influenced by a number of factors:

- High rainfall infiltration across Block 1 and in adjacent upgradient areas. The high infiltration rates are due to poor surface drainage and highly permeable surface soils;
- Interaction with the deep aquifer. The available hydrographs indicate that natural groundwater flow conditions near Springvale Drain include upward leakage of groundwater from the deep to shallow aquifer. Conversely at MWB12S there is a significant downward vertical hydraulic gradient. This downward gradient highlights the presence of shallow low permeability layers that restrict vertical groundwater flow.
- When groundwater extraction is occurring from the PCA the vertical hydraulic gradient reverses and groundwater leaks downward from the shallow to deep aquifer; and
- Interaction with Springvale Drain. Previous studies have demonstrated that shallow groundwater on Block 1 discharges to Springvale Drain.

The importance of rainfall in determining groundwater levels on Block 1 is clearly demonstrated by the response of MWB12S to rainfall in June 2007 (Attachment B). Following an extended dry period 134 mm of rainfall increased groundwater levels by more than 0.6 m. Groundwater levels then receded by approximately 0.2 m over four days until further precipitation of 141 mm occurred over five days. This rainfall event pushed groundwater levels over 4.1 mAHD which was more than 0.9 m higher than at the start of June 2007.

The month's rainfall in June 2007 represented a greater than 9 decile event (i.e. on average its chance of occurring is less than 10%). **Table 2** below presents statistics for monthly rainfall at the Sydney Airport weather station. **Tables 3** and **4** present the maximum reported monthly shallow groundwater level since January 2006.

Table 2
Sydney Airport Rainfall Statistics and Actual Rainfall in 2006 and 2007

	Mean rainfall (mm)	Decile 1 monthly	Decile 5 (median) monthly	Decile 9 monthly	Actual 2006	Actual 2007
January	97	27	74	188	74	47
February	112	22	79	245	29	105
March	117	32	84	271	35	70
April	104	21	79	223	11	109
May	101	22	81	188	28	11
June	122	25	93	278	207	319
July	69	12	51	157	135	37
August	79	8	48	182	87	87
September	62	10	47	135	145	41
October	72	12	47	176	16	12
November	80	16	68	134	29	
December	74	19	54	160	72	
Annual	1087	729	1073	1564	867	838

Notes: Bold indicates actual rainfall exceeded the monthly median
 Bold and shaded indicates actual rainfall exceeded the Decile 9 monthly

Table 3
Maximum Measured Shallow Water Levels 2006

	Actual Rainfall 2006	MWB01S	MWB02S	MWB03S	WG73S	MWB12S
January	74	2.9	2.7	2.7	3	3.6
February	29	2.9	2.5	2.6	2.9	3.5
March	35	2.9	2.3	2.4	2.7	3.4
April	11	3	2.3	2	2.5	3.25
May	28	2.9	2.4	2	2.5	3.3
June	207	2.9	3.1	2.75	3.4	4.1
July	135	2.9	3	2.75	3.5	4.1
August	87	3	3.1	2.8	3.5	4.1
September	145	2.8	3	2.75	3.3	4
October	16	2.3	2.5	2.3	2.8	3.5
November	29	2.1	2.5	2	2.8	3.25
December	72	2.2	2.5	2	2.8	3.2
Annual	867	3	3.1	2.8	3.5	4.1
	Proposed Excavation Surface	3.4	3.25	3.2	3.45	3.65

Notes: Bold indicates groundwater level within 300 mm of proposed excavation surface
 Bold and shaded indicates groundwater level higher than proposed excavation surface
 All water levels presented as mAHD

**Table 4
Measured Shallow Monitoring Well Water Levels 2007**

	Actual Rainfall 2007	MWB01S	MWB02S	MWB03S	WG73S	MWB12S
January	47	1.9	2	1.8	2.3	3.2
February	105	1.9	2	1.8	2.4	3.25
March	70	2	2.1	1.8	2.4	3.5
April	109	2.1	2.3	2	2.55	3.6
May	11	2	2.2	1.9	2.5	3.5
June	319	2.1	3.1	2.9	3.5	4.1
July	37					3.95
August	87					3.65
September	41					3.55
October	12					
November						
December						
Annual	838	2.1	3.1	2.9	3.5	4.1
	Proposed Excavation Surface	3.4	3.25	3.2	3.45	3.65

Notes: Bold indicates groundwater level within 300 mm of proposed excavation surface
 Bold and shaded indicates groundwater level higher than proposed excavation surface
 All water levels presented as mAHD

Based on the data presented in **Tables 3 and 4** water levels at monitoring wells WG73S and MWB12S regularly exceeded the proposed excavation surface. Levels at other continuously logged monitoring wells were always below the proposed excavation surface. This may be partly explained by the locations of the monitoring wells relative to the PCA extraction wells with MWB01S, MWB02S and MWB03S being located directly adjacent to extraction wells. While WG73S is not directly adjacent to the containment line MWB12S is located a significant distance from the eastern end of the PCA.

Although the following analysis of water level response to rainfall is primarily derived from the observed water levels at MWB12S it is expected that water level response to rainfall will be similar over the majority of the proposed flood storage basin.

The water level response at MWB12S is characterised as follows:

- For rainfall events following relatively dry conditions (June 2006) recharge to the shallow aquifer is nearly 100% of rainfall. This calculation is based on the assumption that the aquifer porosity is between 0.15-0.20 and the observation groundwater levels rose 0.75 m after that during the June 2006 event of 114 mm. It should be noted that the total rainfall in the June 2006 event was 141 mm over five days but inspection of the hydrograph at MWB12S shows that water level rise peaked after the first three days of rainfall;
- Calculations for rainfall events in May and June 2007 yield recharge rates of between 90 and 110%, respectively. The variation in recharge rates is likely to be due to small variations in rainfall between the local weather station (Sydney Airport) and Block 1 Southlands;
- In assessing the potential for rainfall to cause shallow groundwater discharge to the proposed flood storage basin the assumption that recharge rates are high is conservative. As a result, a recharge rate of 100% has been adopted for this assessment;
- The maximum observed groundwater level at MWB12S was 4.1 mAHD. This level has occurred several times since continuous logging began in January 2006. A range of rainfall events (including extreme 9 decile events) and as a result it is considered that an external control dictates maximum levels at MWB12S. Given that the ground level at MWB12S is approximately 4.3 mAHD it is likely that the maximum groundwater levels is controlled by ponding and overland flow of water; and

- Where rainfall events are followed by dry periods water levels gradually decline at a decreasing rate. For the case of moderate to high rainfall events (May 2007) water levels initially declined at a rate of approximately 0.025 m per day for the first 8 days and then at around 0.01 m per day until the high rainfall event in early June 2007.

Based on the observed recharge and water level decline characteristics at MWB12S a model of groundwater recharge and decline has been developed using a spreadsheet. The model is based on the following data and assumptions:

- Daily rainfall at Sydney Airport (since 1929) is representative of rainfall at Southlands; and
- The recharge characteristics (100%) and water level decline (0.025 m when levels greater than 3.5 m and 0.01 m when below 3.5 m) observed at MWB12S since January 2006 are representative of site conditions.

The results of the model indicate that levels exceed the proposed excavation surface at MWB12S for an average of 18% of the time each year. In addition, groundwater levels remain within 300 mm of the proposed excavation surface approximately 40% of the time each year.

5. Comments on Post Development Hydrogeological Conditions

The analysis presented above assumes that hydrogeological conditions at Southlands remain the same after development of Stage 1. The construction of the flood storage however, will result in significant changes to groundwater flow as summarised below:

- The maximum groundwater level in the storage area will be at the excavation surface. As a result, groundwater discharge to the storage area will be of significantly shorter duration; and
- Infiltration rates over the storage area will be lower as runoff is increased by regrading of the ground surface (i.e. ponding will be prevented).

The combined effects of lower maximum groundwater levels and decreased infiltration rates will result in lower frequencies of shorter durations for groundwater discharge to the flood storage area. However, quantifying the change in frequency and duration is impractical and as a conservative approach it is recommended that risk analysis is based on the assumption that groundwater discharges to the storage area for an average of 18% of the time each year.

Should you have any queries or require further clarification, please feel free to contact me on 8338 1011 or by email gdasey@jbsgroup.com.au.

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Attachments: (A) Monitoring Well Locations
(B) Hydrographs

Appendix F

Review of Risk Issues - Springvale Drain

Appendix F**Review of Risk Issues – Springvale Drain****F.1 Introduction**

This appendix presents further review of risks to human health associated with specific scenarios related to the proposed design of the Southlands development and proposed risk management measures for Springvale Drain. In particular this appendix presents further assessment of the following:

- The proposed risk management measure considered for the reduction in concentrations of volatile chlorinated hydrocarbons in Springvale Drain is the installation of a shallow groundwater extraction system along the eastern side of the drain to intersect shallow groundwater prior to entry into the drain. The shallow groundwater extraction system is proposed to be connected to the Groundwater Treatment Plant (GTP) for treatment. If the GTP is offline, groundwater levels in Southlands are expected to rise and potentially discharge directly into Springvale Drain. The potential short term impact of such discharges requires further assessment.

This review has focused on inhalation exposures only. Direct contact with water in Springvale Drain is expected to be managed such that regular exposures by workers or visitors would not occur. Maintenance of these areas is expected to be undertaken in accordance with a long term environmental management plan that addresses the potential for direct contact with surface water in the drain.

It is noted that the approach adopted for the assessment of exposure and risk is consistent with the approach presented in the main HHRA report.

The calculation of risk presented in the main report identified emissions to air from Springvale Drain as an issue that requires risk management or remediation. As part of the ongoing assessment of measures that may be implemented to mitigate the discharge of shallow groundwater into the drain, further assessment of potential discharges into the drain are required. In particular, the potential for short duration exposures associated with the discharge of shallow groundwater into the drain during periods of time when the preferred risk management measure may be offline. Hence further assessment of potential risks to human health has been undertaken with the aim of quantifying risks to human health associated with the potential discharge of shallow groundwater into the Springvale Drain intermittently over a short period of time. The assessment has particularly focused on identifying the maximum number of days that discharge may occur before risks become unacceptable.

As with the assessment of the detention basin, the assessment of exposure and risk requires the consideration of a number of issues. These include the potential for shallow groundwater to discharge into the drain, the concentrations in shallow groundwater likely to be discharged, quantification of air concentrations, identification of appropriate toxicity values relevant for short-duration exposures and calculation of risk. These are discussed further in the following sections.

F.2 Potential for Discharge into Springvale Drain

The proposed approach to addressing potential discharge of shallow groundwater into Springvale Drain is the installation of a shallow groundwater extraction system along the eastern side of the drain. The shallow groundwater extraction system is proposed to be connected into the existing GTP. It is noted, however that the GTP needs to be offline during some short periods of time for maintenance. The period of time the GTP is offline will not affect containment of the groundwater plumes, however it has the potential to cause groundwater levels in the vicinity of the drain to rise and discharge directly into the drain where emissions to air of volatile chemicals in groundwater would occur. It is unknown how many times the GTP will be offline. In addition it is currently unknown how long after shut-down of the GTP the shallow groundwater will discharge into the drain as this is expected to be dependant of rainfall during the period when the GTP is offline.

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Review of Risk Issues – Springvale Drain

F.3 Concentrations in Shallow Groundwater Discharging to the Drain

The assessment of exposures to emissions to air of volatile chemicals derived from surface water in Springvale Drain can be undertaken using a number of approaches. These include:

- Use of measured air concentrations collected adjacent to Springvale Drain during periods of time when the GTP was not operating. This was presented in the assessment of risks to human health (refer to main report). The data collected included locations adjacent to the current realignment channel where maximum concentrations were typically reported. This is discussed further in **Section 3.8** in the main report;
- Use of measured surface water concentrations collected in Springvale Drain within Southlands during periods of time when the GTP was not operating. As noted above the available data also includes locations within the realignment channel. The measured surface water concentrations can be used in a volatilisation model to estimate potential concentrations in air next to the drain; and
- Use of measured groundwater concentrations in the vicinity of the drain to identify potential concentrations that may discharge into the drain should the GTP not be operating. This would assume that groundwater from depths up to 7-8m below ground surface could discharge into the drain (as identified in the conceptual model referenced in the Stage 2 Assessment (Woodward-Clyde 1996)) and surface water concentrations would be similar to the shallow groundwater concentrations. The groundwater concentrations that may discharge into the drain can be used in a volatilisation model to estimate potential concentrations in air next to the drain.

A review of relevant surface water and shallow groundwater concentrations in the vicinity of Block 1 that are relevant to the quantification of current concentrations has been undertaken and is presented in **Section 3.3.1** in the main report.

Table 1 presents a summary of the COPC identified in the main report (**Table3-1**), exposure concentrations considered in surface water and groundwater and the relevant screening level guideline adopted.

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Review of Risk Issues – Springvale Drain

Table 1 COPC and Exposure Concentrations for Assessment of Springvale Drain

COPC Identified	Concentration in Surface Water ¹ (mg/L)		Concentration in Shallow Groundwater ² (mg/L)	Screening Level Guideline ³ (mg/L)
	Average	Maximum		
carbon tetrachloride	0.78	5.1	17.8	0.003 (ADWG)
chloroform	0.74	4.5	4.8	0.25 (ADWG)
methylene chloride	0.021	0.027	0.14	0.004 (ADWG)
1,1,2,2-tetrachloroethane	0.069	0.311	0.35	0.37 (PRG) ^C
1,1,2-trichloroethane	0.13	0.42	0.92	0.024 (PRG)
1,2-dichloroethane (EDC)	24.7	83.1	34.9	0.003 (ADWG)
tetrachloroethene (PCE)	1.8	10.5	13.3	0.05 (ADWG)
trichloroethene (TCE)	0.87	6.6	3.3	0.02 (WHO)
1,1-dichloroethene	0.063	0.43	0.15	0.03 (ADWG)
cis-1,2-dichloroethene	0.60	2.7	4.3	0.06 (ADWG)
trans-1,2-dichloroethene	0.036	0.18	0.38	0.06 (ADWG)
vinyl chloride	1.9	7.6	11.5	0.0003 (ADWG)
hexachlorobutadiene (HCBd)	NA	NA	0.064	0.0007 (ADWG)
hexachloroethane (HCE)	NA	NA	0.099	0.036 (PRG)

Notes:

- 1 Concentrations reported in Springvale Drain at locations SW046, SW049 and SW005 from data collected from March 2004 to December 2005. Concentrations reported based on detected concentrations as an average and maximum during the period of time considered.
- 2 Concentrations in shallow groundwater that may discharge into Springvale Drain calculated as average concentration from wells screened at depths of 2-8m and shallow and intermediate wells.
- 3 Screening level guidelines adopted based on drinking water guidelines (as referenced)
- C Maximum concentration reported in water close to the adopted guideline. Based on information available about the chemical, there is the potential for cumulative risks to be of concern as the health endpoints are similar to a range of other chlorinated compounds also identified as COPC (refer to toxicity summaries in **Appendix B**). On this basis this chemical has also been selected as a COPC for further assessment. Note that the assessment of inhalation exposures adjacent to Springvale Drain has been based on measured air data where this compound was not reported above the LOR, therefore it is not considered a COPC in air adjacent to the drain.

F.4 Estimation of Relevant Exposure Concentrations

Only inhalation exposures are considered relevant and hence the concentrations of volatile COPC need to be estimated in the breathing zone in areas adjacent to the drain. The air concentrations in this zone can be based on measured concentrations prior to the GTP operating or estimated using a volatilisation model based on estimated surface water concentrations.

Concentrations in air adjacent to the drain have been estimated using an emissions model (USEPA 1994) based on quiescent flow through a drain. The water in the drain has been estimated to be 0.8m wide and 0.5m deep with emissions occurring from a 20 m segment as representative of those emissions that are likely to contribute to a breathing zone concentrations adjacent to the drain. In addition the flow rate of water in the drain has been estimated to be 5.56 m/s with a temperature of 18 °C. The same model (and assumptions) has been used in the assessment of data collected in 2006 where both surface water and air samples (adjacent to the drain) were collected at the same time. Review of data from 2006 showed reasonable agreement between the modelled and measured concentrations that are either close to the drain (2m mixing zone) or further from the drain (10m mixing zone). Hence the use of this model for the purpose of estimating concentrations in air based on surface water concentrations is considered to be reasonable.

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Review of Risk Issues – Springvale Drain

Air concentrations adjacent to (close to) the drain have also been directly measured prior to the GTP becoming operational. These air concentrations within Southlands are variable, however they indicate that concentrations in air derived from the drain when shallow groundwater is discharging can be elevated (as assessed in the main report). For the purpose of comparison the measured concentrations in southlands have also been reviewed.

Table 2 presents a summary of the estimated air concentrations 10m from the drain based on assumed concentrations of COPC in the surface water of the drain. The calculations have been presented for the average measured surface water concentration as well as concentrations estimated from the shallow groundwater discharge concentrations as outlined in **Table 1**. In addition the average and maximum measured air concentrations in Southlands has been included for comparison. Calculations are presented at the end of this appendix.

Table 2 Estimated Air Concentrations Adjacent to Springvale Drain

COPC Identified	Estimated Air Concentration Modelled from Surface Water (mg/m ³)		Measured Air Concentrations Adjacent to Springvale Drain (mg/m ³) ³	
	Average ¹	Estimated Maximum ²	Average	Maximum
carbon tetrachloride	0.038	0.88	0.026	0.077
chloroform	0.037	0.24	0.034	0.094
methylene chloride	0.0011	0.0073	0.005	0.011
1,1,2,2-tetrachloroethane	0.0012	0.0055	ND	ND
1,1,2-trichloroethane	0.0041	0.029	NA	NA
1,2-dichloroethane (EDC)	0.95	1.3	0.45	2.4
tetrachloroethene (PCE)	0.082	0.60	0.069	0.16
trichloroethene (TCE)	0.043	0.16	0.045	0.18
1,1-dichloroethene	0.0037	0.0087	NA	NA
cis-1,2-dichloroethene	0.032	0.23	0.08	0.28
trans-1,2-dichloroethene	0.0023	0.024	NA	NA
vinyl chloride	0.013	0.078	0.027	0.099
hexachlorobutadiene (HCBD)	NA	0.0021	NA	NA
hexachloroethane (HCE)	NA	0.0015	NA	NA

Notes:

- 1 Air concentrations estimated based on average measured surface water concentrations reported in Springvale Drain at locations SW046, SW049 and SW005 from data collected March 2004 to December 2005. Average calculated based on detected concentrations during the period of time considered.
 - 2 Air concentrations estimated based on concentrations in shallow groundwater that may discharge into the drain when the GTP is offline assuming no dilution. Water concentrations calculated based on the average concentration from wells screened at depths of 2-8m and shallow and intermediate wells.
 - 3 Measured air concentrations in Southlands adjacent to Springvale Drain prior to GTP being operational.
- ND – Not detected above the analytical LOR
 NA – Not assessed as not analysed (HCBD and HCE in surface water) or not detected in air data collected.

Review of the above table indicates reasonable agreement between the range of modelled air concentrations and measured air concentrations considered representative of average and maximum exposures. Hence the modelled average and maximum (estimated maximum) concentrations have been used in this assessment.

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Review of Risk Issues – Springvale Drain

F.5 Identification of Appropriate Toxicity Values for Hazard Assessment

The calculation of risk requires the quantification of exposure as well as quantification of the hazards associated with the COPC identified. This involves identifying the most appropriate toxicity values for the COPC and exposure period considered. The assessment of risk presented in the main report has focused on long-term chronic risks and has therefore utilised toxicity values relevant for chronic exposures. These are exposures that may occur every day for a working lifetime. The assessment of chronic risks, however, is not considered relevant to the assessment of short duration peaks of exposure as might occur during down time of the GTP. If the proposed risk mitigation measure is implemented such that exposures during the operation of the GTP are considered negligible, then the only exposures that require assessment are associated with shorter duration events associated with the GTP being offline. These events do not occur every day over a working life time as assumed in chronic health risk assessments. However, these events will occur for periods longer than would be defined as acute (e.g. an hour or a day). They should therefore be considered to be representative of intermediate duration exposures which, by definition from the ATSDR, involves exposures that may be continuous for 14 to 364 days per event (which may be during one year). On this basis toxicity values have been adopted in this assessment that are associated with the assessment of intermediate duration exposures as outlined in **Section 4.4** in the main report.

With respect to overall exposure, consideration has been included for contributions from the flood storage area (as designed to prevent shallow groundwater discharge to the surface) and emissions from groundwater to indoor (average) and outdoor air. Consideration of inhalation exposures associated with emissions to air from groundwater has been undertaken for the Stage 1 development only assuming no vapour mitigation measures are implemented to lower exposure. It is expected that the Stage 2 development will involve vapour mitigation measures reducing exposure to at least that calculated for Stage 1 (likely to be much lower if designed appropriately). In addition the contribution from direct contact and inhalation exposures associated with soils/sediments has not been considered as it is assumed that these hot-spots are adequately remediated prior to development. Risks associated with emissions to air from Floodvale drain have not been considered as these exposures will not coincide with maximum exposures associated with Springvale Drain due to the location of the two drains in relation to the development (ie Floodvale and Springvale Drains are on opposite sides of Block 2). Contributions from other sources on the GTP are essentially negligible. Hence cumulative issues result in a reduction in the target risk levels to 8×10^{-6} (non-threshold) and 0.7 (threshold HI).

F.6 Calculation of Exposure Frequency

The assessment of exposure has focused on inhalation by workers adjacent to the drain during the peak events considered. These workers may be in areas outdoors or in buildings close to the drain where ventilation air (or open windows) for the building is drawn from the side of the building closest to the drain. Exposures by workers adjacent to the drain are consistent with those assessed in the main report, namely exposures for 10 hours per day (9 hours indoors and 1 hour outdoors) or 4 hours per day outdoors (waiting in trucks or loading vehicles). For the purpose of considering maximum exposures the most significant exposure, namely works indoors/outdoors for 10 hours each day have been considered further.

Based on the assumptions presented on the previous sections with respect to toxicity and air concentrations adjacent to the drain the maximum duration of exposure (as a frequency (days per year) and duration (years)) has been calculated such that the total non-threshold risk (i.e. for all non-threshold COPC) is equal to 8×10^{-6} and/or the total threshold HI (i.e. for all threshold COPC) is equal to 0.7 (accounting for cumulative exposures). These risk levels are considered representative of acceptable risks to human health. This has been undertaken for average and reasonable maximum air concentrations as modelled and presented in **Table 2**.

Based on the assessment undertaken, noting that the assessment of risks to human health associated with non-threshold carcinogenic effects needs to consider exposures averaged over a working life,

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Review of Risk Issues – Springvale Drain

whereas threshold effects requires an assessment of exposure on a yearly basis, the following can be noted with respect to the maximum frequency of exposure:

- Under the average exposure scenario, the maximum number of days where peak exposure results in the target risk values being reached is 120 days. This may occur at any time over a 30 year working life (eg 4 days per year over 30 years, or 8 days per year over 15 years). Alternatively, this can occur all within one year, however no further peak exposures would then be allowable in relation to meeting target risk values.
- Under the maximum exposure scenario, the maximum number of days where peak exposures results in the target risk values being reached is 66 days. Again this may occur over a 30 year working life (based on the calculated non-threshold risk), however it is noted that during any one year no more than 30 days is allowable (based on the calculated threshold HI).

It is noted that the assessment presented has considered exposures 0 to 10m from the drain. However current development plans have allowed for a minimum distance of 20m from Springvale Drain to the nearest building or work area (including outdoor work areas), hence further assessment of the maximum exposure frequency has been undertaken based on distance from the drain.

Under some stable meteorological conditions it is possible that the concentrations estimated 10m from the drain will be representative of concentrations in air further downwind in the proposed development. However under most conditions it is likely that concentrations in buildings located further from the drain will be lower.

It is difficult to estimate downwind concentrations in the near field of a source (i.e. less than 100m from the source), however the dispersion model AUSPLUME has been used to estimate reasonable worst-case dispersion at 10m intervals from the source.

Assuming distances up to 50m from the drain the following table presents a summary of the allowable days of peak frequency calculated to be protective of workers. It also includes the calculated exposure frequency as presented above for 0 to 10m from the drain.

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Review of Risk Issues – Springvale Drain

Table 3 Estimated Allowable Exposure Frequency for Emissions from Drain (GTP or other Technology offline)

Distance from Springvale Drain (Workplace as building or outdoors)	Estimated Exposure Frequency Allowable based on Protection of Human Health	
	Average ¹	Estimated Maximum ²
0-10m (worst case)	120 days over 30 years (eg 4 days each year for 30 years)	66 days over 30 years (eg 2.2 days each year for 30 years) with no more than 30 days during any one year
20m (current proposal) 31% of concentration from 10m	360 days over 30 years (eg 12 days each year for 30 years)	225 days over 30 years (eg 7.5 days each year for 30 years) with no more than 90 days during any one year
30m 15% of concentration from 10m	720 days over 30 years (eg 24 days each year for 30 years)	450 days over 30 years (eg 15 days each year for 30 years) with no more than 200 days during any one year
40m 9% of concentration from 10m	1200 days over 30 years (eg 40 days each year for 30 years)	750 days over 30 years (eg 25 days each year for 30 years) with no more than 300 days during any one year
50m 6% of concentration from 10m	1950 days over 30 years (eg 65 days each year for 30 years)	1200 days over 30 years (eg 40 days each year for 30 years) with no more than 500 days during any one year
Average for 20-50m from Drain (considered relevant for exposures onsite)	1060 days over 30 years	650 days over 30 years with no more than 270 days during any one year

Notes:

- 1 Estimated exposure frequency for the average scenario presented as a total number of days over a working lifetime as the calculated risks are dominated by non-threshold carcinogenic risks. In the event that all the days of exposure occurred in only one year then the threshold HI would remain acceptable and hence no additional limit on maximum number of days per year has been set.
- 2 Estimated exposure frequency for the maximum scenario presented as a total number of days over a working lifetime as the calculated risks are dominated by non-threshold carcinogenic risks. In addition for distances up to 30m from the drain the total number of days during any one year is also limited by the calculated threshold HI. Hence an additional limit on the maximum number of days each year where peak exposures occur has been set.

The approach adopted for the quantification of exposure and health impacts is considered adequately conservative that it would be considered reasonable that an exposure frequency less than that estimated for the average scenario would be considered reasonable. In particular it is noted that in reality, no one worker would be likely to spend 10 hours every day of their working life 20m from the drain and hence it is considered reasonable that the frequency of exposure relevant to the GTP being offline (relevant to acceptable risks) is an average of all scenarios presented above for 20-50m (presented in the table) reflecting exposures in a work area 20-50m from the drain which is more realistic.

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SURFACE WATER IMPACTS

Using Equations presented in model WATER9 and USEPA 1994

Assessment of Vapour Emissions from Drain - Average measured 2004 and 2005

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Width of drain	[m]	dwidth	0.8	Estimated for Springvale Drain
Length of drain	[m]	dlength	20	Assumed length of river contributing to exposure
Depth of drain	[m]	ddepth	0.5	Estimated average depth (though variable over seasons)
Effective diameter of water	[m]	deffd	4.51	Calculated
Flow rater of water in drain	[m/s]	dfr	5.56	Estimated based on old data for drain
Temperature of water	[oC]		18	Estimated
Temperature of water (°C+273)	[K]	dtemp	291	Calculated
Wind Velocity (=0.5 * wind speed @ 10m height)	[m/s]	dvw	2.5	Based on average 9am and 3pm winds for Airport

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	0.8	Assumed area likely to contribute to emissions
Width of Contaminated Area	[m]	width	20	Assumed area likely to contribute to emissions
Wind Speed Outdoors	[m/s]	wspd	4.9	Average 9am and 3pm winds from Airport
Height of Outdoor Mixing Zone	[m]	outboxh	10	Mixing zone to areas near drain

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)	Henry's Law Constant (atm.m ³ /g.mol)
vinyl chloride	2760	62.5	18.6	0.106	1.2E-06	2600	1.107	0.027
1,1-dichloroethene	2420	96.95	35.04	0.09	1.0E-05	634	1.07	0.0261
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384	0.00938
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167	0.00408
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482	0.0011
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422	0.0103
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374	0.000913
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754	0.0184
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15	0.00367
carbon tetrachloride	800	153.8	48.64	0.078	8.8E-06	115	1.2	0.03
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133	0.00219
PAHs (BaP equivalent)								
hexachlorobutadiene (HCBD)	3.2	260.76	993.5	0.0561	6.2E-06	0.22	0.421	0.0103
hexachloroethane (HCE)	50	236.74	224.7	0.0025	6.8E-06	0.4	0.114	0.0028

Calculations	Schmidt Number on Gas Side	Gas Phase Mass Transfer Coefficient (m/s)	Liquid Phase Mass Transfer Coefficient (m/s)	Overall Mass Transfer Coefficient (m/s)	Water Concentration (mg/L)	Emission Rate From Water Surface (g/s)	Concentration in Breathing Zone (mg/m ³)
vinyl chloride	1.422955975	6.5E-03	1.7E-05	1.7E-05	1.9	5.1E-04	1.3E-02
1,1-dichloroethene	1.675925926	5.8E-03	1.5E-04	1.4E-04	0.063	1.4E-04	3.6E-03
trans-1,2-dichloroethene	2.133427628	5.0E-03	1.7E-04	1.5E-04	0.036	8.9E-05	2.3E-03
cis-1,2-dichloroethene	2.049365942	5.1E-03	1.5E-04	1.3E-04	0.6	1.3E-03	3.2E-02
1,2-dichloroethane (EDC)	1.450320513	6.4E-03	1.4E-04	9.4E-05	24.7	3.7E-02	9.5E-01
trichloroethene (TCE)	1.9092827	5.3E-03	1.3E-04	1.2E-04	0.87	1.7E-03	4.3E-02
1,1,2-trichloroethane	1.933760684	5.3E-03	1.2E-04	7.7E-05	0.13	1.6E-04	4.1E-03
tetrachloroethene (PCE)	2.094907407	5.0E-03	1.1E-04	1.1E-04	1.8	3.2E-03	8.2E-02
chloroform	1.450320513	6.4E-03	1.4E-04	1.2E-04	0.74	1.5E-03	3.7E-02
carbon tetrachloride	1.933760684	5.3E-03	1.2E-04	1.2E-04	0.78	1.5E-03	3.8E-02
dichloromethane	1.49339934	6.3E-03	1.6E-04	1.3E-04	0.021	4.3E-05	1.1E-03
hexachlorobutadiene (HCBD)	2.688651218	4.2E-03	8.6E-05	8.2E-05		0.0E+00	0.0E+00
hexachloroethane (HCE)	60.33333333	5.3E-04	9.5E-05	3.8E-05		0.0E+00	0.0E+00

Inhalation of Volatile Chemicals in Areas Near Springvale Drain (Indoors/Outdoors)

Peak Exposure - 14-364 days

General Data/ Equations	Units	Exposure Calculations (Average)		Exposure Calculations (Average)	
		Review of Carcinogenic Exposure Frequency		Review of Threshold (non-carcinogenic) frequency	
Exposure Parameters					
Exposure Frequency (EF)	days/year	12	Allowable for peak exposure	360	Allowable for peak exposure
Exposure Duration (ED)	years	30	Assume event occurs once during working time at site	1	Assume event occurs once during working time at site
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	365	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	10	Assume 9 hours indoors and 1 hr outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	31%	Concentration 31% of that at 10m, at 20m distance	31%	Concentration 31% of that at 10m, at 20m distance
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	7.9E-04	NonThreshold	7.9E-04	NonThreshold
		1.9E-03	Threshold	5.6E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Intermediate Threshold ADI, TDI or RfD		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	8E-6	1E-2			TOTAL	8E-6	3E-1
vinyl chloride	3.1E-02	2.2E-02	1.30E-02	1.0E-05	2.4E-05	3.2E-7	1.1E-3	1.30E-02	1.0E-05	7.2E-04	3.2E-7	3.3E-2
1,1-dichloroethene		2.3E-02	3.65E-03	2.9E-06	6.8E-06	--	3.0E-4	3.65E-03	2.9E-06	2.0E-04	--	9.0E-3
trans-1,2-dichloroethene		2.3E-01	2.27E-03	1.8E-06	4.2E-06	--	1.9E-5	2.27E-03	1.8E-06	1.3E-04	--	5.6E-4
cis-1,2-dichloroethene		3.4E-01	3.19E-02	2.5E-05	5.9E-05	--	1.7E-4	3.19E-02	2.5E-05	1.8E-03	--	5.2E-3
1,2-dichloroethane (EDC)	9.8E-03	6.9E-01	9.48E-01	7.5E-04	1.8E-03	7.4E-6	2.5E-3	9.48E-01	7.5E-04	5.3E-02	7.4E-6	7.6E-2
trichloroethene (TCE)	1.5E-03	1.5E-01	4.27E-02	3.4E-05	7.9E-05	5.1E-8	5.1E-4	4.27E-02	3.4E-05	2.4E-03	5.1E-8	1.5E-2
1,1,2-trichloroethane		4.0E-02	4.05E-03	3.2E-06	7.5E-06	--	1.9E-4	4.05E-03	3.2E-06	2.3E-04	--	5.6E-3
tetrachloroethene (PCE)		5.7E-02	8.17E-02	6.5E-05	1.5E-04	--	2.6E-3	8.17E-02	6.5E-05	4.5E-03	--	7.9E-2
chloroform	1.5E-03	6.9E-02	3.69E-02	2.9E-05	6.8E-05	4.3E-8	1.0E-3	3.69E-02	2.9E-05	2.1E-03	4.3E-8	3.0E-2
carbon tetrachloride		5.4E-02	3.84E-02	3.0E-05	7.1E-05	--	1.3E-3	3.84E-02	3.0E-05	2.1E-03	--	3.9E-2
dichloromethane		3.0E-01	1.09E-03	8.7E-07	2.0E-06	--	6.8E-6	1.09E-03	8.7E-07	6.1E-05	--	2.0E-4
hexachlorobutadiene (HCBD)		2.0E-04				--	--				--	--
hexachloroethane (HCE)		1.7E+01				--	--				--	--

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Modelled Air Concentrations
Based on maximum measured SW concentrations pre GTP

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Modelled Air Concentrations
Based on maximum measured SW concentrations pre GTP

VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR SURFACE WATER IMPACTS

Using Equations presented in model WATER9 and USEPA 1994

Assessment of Vapour Emissions from Drain - Max from Estimated GW discharge

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Width of drain	[m]	dwidth	0.8	Estimated for Springvale Drain
Length of drain	[m]	dlength	20	Assumed length of river contributing to exposure
Depth of drain	[m]	ddepth	0.5	Estimated average depth (though variable over seasons)
Effective diameter of water	[m]	ddeff	4.51	Calculated
Flow rate of water in drain	[m/s]	dfr	5.56	Estimated based on old data for drain
Temperature of water	[°C]		18	Estimated
Temperature of water (°C+273)	[K]	dtemp	291	Calculated
Wind Velocity (=0.5 * wind speed @ 10m height)	[m/s]	dvw	2.5	Based on average 9am and 3pm winds for Airport

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	0.8	Assumed area likely to contribute to emissions
Width of Contaminated Area	[m]	width	20	Assumed area likely to contribute to emissions
Wind Speed Outdoors	[m/s]	wspd	4.9	Average 9am and 3pm winds from Airport
Height of Outdoor Mixing Zone	[m]	outboxh	10	Mixing zone to areas near drain

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)	Henry's Law Constant (atm.m ³ /g.mol)
vinyl chloride	2760	62.5	18.6	0.106	1.2E-06	2600	1.107	0.027
1,1-dichloroethene	2420	96.95	35.04	0.09	1.0E-05	634	1.07	0.0261
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384	0.00938
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167	0.00408
1,2-dichloroethane (EDC)	5100	98.96	43.79	0.104	9.9E-06	78.9	0.0482	0.0011
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422	0.0103
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374	0.000913
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754	0.0184
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15	0.00367
carbon tetrachloride	800	153.8	48.64	0.078	8.8E-06	115	1.2	0.03
dichloromethane	13200	84.93	23.74	0.101	1.2E-05	435	0.133	0.00219
hexachlorobutadiene (HCBD)	3.2	260.76	993.5	0.0561	6.2E-06	0.22	0.421	0.0103
hexachloroethane (HCE)	50	236.74	224.7	0.0025	6.8E-06	0.4	0.114	0.0028

Calculations	Schmidt Number on Gas Side	Gas Phase Mass Transfer Coefficient (m/s)	Liquid Phase Mass Transfer Coefficient (m/s)	Overall Mass Transfer Coefficient (m/s)	Water Concentration (mg/L)	Emission Rate From Water Surface (g/s)	Concentration in Breathing Zone (mg/m ³)
vinyl chloride	1.422955975	6.5E-03	1.7E-05	1.7E-05	11.5	3.1E-03	7.8E-02
1,1-dichloroethene	1.675925926	5.8E-03	1.5E-04	1.4E-04	0.15	3.4E-04	8.7E-03
trans-1,2-dichloroethene	2.133427628	5.0E-03	1.7E-04	1.5E-04	0.38	9.4E-04	2.4E-02
cis-1,2-dichloroethene	2.049365942	5.1E-03	1.5E-04	1.3E-04	4.3	9.0E-03	2.3E-01
1,2-dichloroethane (EDC)	1.450320513	6.4E-03	1.4E-04	9.4E-05	34.9	5.3E-02	1.3E+00
trichloroethene (TCE)	1.9092827	5.3E-03	1.3E-04	1.2E-04	3.3	6.4E-03	1.6E-01
1,1,2-trichloroethane	1.933760684	5.3E-03	1.2E-04	7.7E-05	0.92	1.1E-03	2.9E-02
tetrachloroethene (PCE)	2.094907407	5.0E-03	1.1E-04	1.1E-04	13.3	2.4E-02	6.0E-01
chloroform	1.450320513	6.4E-03	1.4E-04	1.2E-04	4.8	9.4E-03	2.4E-01
carbon tetrachloride	1.933760684	5.3E-03	1.2E-04	1.2E-04	17.8	3.4E-02	8.8E-01
dichloromethane	1.49339934	6.3E-03	1.6E-04	1.3E-04	0.14	2.9E-04	7.3E-03
hexachlorobutadiene (HCBD)	2.688651218	4.2E-03	8.6E-05	8.2E-05	0.064	8.4E-05	2.1E-03
hexachloroethane (HCE)	60.33333333	5.3E-04	9.5E-05	3.8E-05	0.099	5.9E-05	1.5E-03

Inhalation of Volatile Chemicals in Areas Near Springvale Drain (Indoors/Outdoors)

Peak Exposure - 14-364 days

General Data/ Equations		Exposure Calculations (RME)		Exposure Calculations (RME)	
Units		Review of Carcinogenic Exposure Frequency		Review of Threshold (non-carcinogenic) frequency	
Exposure Parameters					
Exposure Frequency (EF)	days/year	7.5	Allowable for peak exposure	90	Allowable for peak exposure
Exposure Duration (ED)	years	30	Assume event occurs once during working time at site	1	Assume event occurs once during working time at site
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996	70	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996	365	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m ³ /hr	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)	1.27	Inhalation rate indoors (9 hrs) and outdoors (1hr)
Exposure Time (ET)	hours/day	10	Assume 9 hours indoors and 1 hr outdoors	10	Assume 9 hours indoors and 1 hr outdoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	31%	Concentration 31% of that at 10m, at 20m distance	31%	Concentration 31% of that at 10m, at 20m distance
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$	m ³ /kg/day	5.0E-04	NonThreshold	2.0E-04	NonThreshold
		1.2E-03	Threshold	1.4E-02	Threshold

Daily Intake = Concentration in Air x Intake Factor

NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor

Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)

Chemical	Toxicity Data		Concentration in Air	Daily Intake		Calculated Risk		Concentration in Air	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Intermediate Threshold ADI, TDI or RfD		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient		NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	8E-6	6E-2			TOTAL	3E-6	7E-1
vinyl chloride	3.1E-02	2.2E-02	7.84E-02	3.9E-05	9.1E-05	1.2E-6	4.1E-3	7.84E-02	1.6E-05	1.1E-03	4.8E-7	5.0E-2
1,1-dichloroethene		2.3E-02	8.69E-03	4.3E-06	1.0E-05	--	4.5E-4	8.69E-03	1.7E-06	1.2E-04	--	5.4E-3
trans-1,2-dichloroethene		2.3E-01	2.39E-02	1.2E-05	2.8E-05	--	1.2E-4	2.39E-02	4.7E-06	3.3E-04	--	1.5E-3
cis-1,2-dichloroethene		3.4E-01	2.29E-01	1.1E-04	2.7E-04	--	7.8E-4	2.29E-01	4.5E-05	3.2E-03	--	9.4E-3
1,2-dichloroethane (EDC)	9.8E-03	6.9E-01	1.34E+00	6.7E-04	1.6E-03	6.5E-6	2.2E-3	1.34E+00	2.7E-04	1.9E-02	2.6E-6	2.7E-2
trichloroethene (TCE)	1.5E-03	1.5E-01	1.62E-01	8.0E-05	1.9E-04	1.2E-7	1.2E-3	1.62E-01	3.2E-05	2.3E-03	4.8E-8	1.5E-2
1,1,2-trichloroethane		4.0E-02	2.87E-02	1.4E-05	3.3E-05	--	8.3E-4	2.87E-02	5.7E-06	4.0E-04	--	1.0E-2
tetrachloroethene (PCE)		5.7E-02	6.03E-01	3.0E-04	7.0E-04	--	1.2E-2	6.03E-01	1.2E-04	8.4E-03	--	1.5E-1
chloroform	1.5E-03	6.9E-02	2.39E-01	1.2E-04	2.8E-04	1.7E-7	4.0E-3	2.39E-01	4.8E-05	3.3E-03	7.0E-8	4.9E-2
carbon tetrachloride		5.4E-02	8.76E-01	4.3E-04	1.0E-03	--	1.9E-2	8.76E-01	1.7E-04	1.2E-02	--	2.2E-1
dichloromethane		3.0E-01	7.27E-03	3.6E-06	8.4E-06	--	2.8E-5	7.27E-03	1.4E-06	1.0E-04	--	3.4E-4
hexachlorobutadiene (HCBD)		2.0E-04	2.14E-03	1.1E-06	2.5E-06	--	1.2E-2	2.14E-03	4.3E-07	3.0E-05	--	1.5E-1
hexachloroethane (HCE)		1.7E+01	1.51E-03	7.5E-07	1.8E-06	--	1.1E-7	1.51E-03	3.0E-07	2.1E-05	--	1.3E-6

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Modelled Air Concentrations
Based on potential shallow GW discharge

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Modelled Air Concentrations
Based on potential shallow GW discharge

Appendix G

Derivation of Preliminary RBSC

**Derived Risk Based Soil Concentrations
Southlands HHRA**

COPC	RBC (mg/kg)	Calculated Risks - Commercial Worker							
		Inhalation		Ingestion		Dermal Contact		Total Risk	
		Non-threshold risk	HI	Non-threshold risk	HI	Non-threshold risk	HI	Non-threshold risk	HI
TPH C6-C9 aliphatic	100		0.20		0.000052		0.000022		0.20
TPH C10-C14 aromatic	1000		0.030		0.0059		0.0025		0.038
TPH C10-C14 aliphatic	1000		0.042		0.0026		0.0011		0.045
TPH C15+ aromatic	2500				0.022		0.0083		0.030
TPH C15+ aliphatic	2500				0.00033		0.00014		0.00046
vinyl chloride (adult exposures)	0.3	1.4E-06		3.5E-08		1.5E-08		1.5E-06	
trans-1,2-dichloroethene	5		0.048		0.000069		0.000029		0.048
1,1-dichloroethane	45		0.043		0.00011		0.000045		0.043
cis-1,2-dichloroethene	4		0.041		0.000094		0.000040		0.041
1,2-dichloroethane (EDC)	4	8.8E-07		4.8E-09	--	2.0E-09	--	8.9E-07	
trichloroethene (TCE)	12	7.0E-07			0.0019		0.00082	7.0E-07	0.0027
1,1,2-trichloroethane	9		0.039		0.00053		0.00022		0.040
tetrachloroethene (PCE)	12		0.046		0.00030		0.000085		0.047
1,1,2,2-tetrachloroethane	20	3.9E-07			0.00012		0.000050	3.9E-07	0.00017
chloroform	8	6.2E-07	0.049		0.00029		0.000061	6.2E-07	0.050
carbon tetrachloride	0.2		0.071		0.000095		0.000014		0.071
1,3,5-trichlorobenzene	15		0.044		0.00046		0.00019		0.044
Total Risk								4E-06	0.7

Notes:

RBC derived to ensure total risks to workers from inhalation, ingestion and dermal contact exposures equal to the target identified in the main report (total risk noted above)

RBC reflect nature of contamination identified on the site, with higher allowance from more significant contributors to total risk (such as vinyl chloride and TPH)

RBC rounded to practical values

If remediation identifies the presence of other contaminants that require the derivation of RBC, these can be provided, however the total risk is remain unchanged.

In addition, concentrations may be adjusted to better reflect distribution of contamination in some areas.

TPH conservatively assumed to be consistent with a petrol type product with 50:50 aromatic:aliphatic split. Total TPH for each fraction equal to the sum of aromatic and aliphatic

**VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR CONTAMINATED SOILS
UTILISING ASTM GUIDANCE E 1739-95^E (2002)**

Vapour Migration from Impacted Soils - RBC Calculations

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Depth of Top of Contaminated Soil (BGS)	[m]	d	0.01	Calculated from layers
Thickness of Surface Contamination	[m]	surfd	1	Estimated for soil types
Soil Temperature	[C]	T	17	assumed for area
Vadose Zone Characteristics of Top Layer				Fill
Depth of Layer 1	[m]	vd1	0.01	Assumed impacted soils beneath building
Moisture Content	[g/g]	mocon	0.08	Conservative value for soil materials
Organic Carbon Fraction	-	foc	0.003	Assumed for fill
Soil Bulk Density	[g/cm ³]	rhob	1.7	Assumed for fill
Density of Solids	[g/cm ³]	sd	2.65	Default
Total Soil Porosity	[cm ³ /cm ³]	theta	0.36	1 - (rhob/sd)
Volumetric Water Content	[cm ³ /cm ³]	wacon	0.136	mocon*rhob
Volumetric Air Content	[cm ³ /cm ³]	acon	0.222	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m ²]	area	100.0	Assumed size of building or room
Percentage of area above contamination	[%]	Pem	50%	Assumed area of residual impacts beneath building
Foundation/wall thickness	[m]	fthick	0.15	Default Value
Height of Room	[m]	boxh	3	site-specific assumption
Ratio of enclosed space volume:infiltration area	[m]	LB	6	Calculated
Hourly Volume Exchange of Fresh Air	[exch/hr]	exchanges	2	Minimum exchange rate as per AS 1668.2
Fraction of Cracks in Walls and foundation	-	cracks	0.001	Representative of sound concrete
Volumetric Water Content in foundation/wall cracks	[cm ³ /cm ³]	fwacon	0.12	Default Value
Volumetric Air Content in foundation/wall cracks	[cm ³ /cm ³]	facon	0.260	Default Value

Chemical Specific Parameters	Water Solubility (mg/L)	MW (g/mol)	Koc (cm ³ /g)	Air Diffusion Coefficient (cm ² /s)	Water Diffusion Coefficient (cm ² /s)	Vapour Pressure (mmHg)	Henry's Law Constant (unitless)
TPH C6-C9							
aromatic	520	120	251	0.086	8.6E-06	29	0.27
aliphatic	5.4	100	3160	0.067	6.9E-06	48	50
TPH C10-C14							
aromatic	25	130	2510	0.048	7.7E-06	0.48	0.14
aliphatic	0.034	160	316000	0.046	5.2E-06	0.48	130
vinyl chloride (adult exposures)	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
trans-1,2-dichloroethene	6300	96.95	52.5	0.0707	1.2E-05	265	0.384
1,1-dichloroethane	5040	98.96	35.04	0.0742	1.1E-05	227	0.23
cis-1,2-dichloroethene	3500	96.95	35.5	0.0736	1.1E-05	180	0.167
1,2-dichloroethane (EDC)	8690	98.96	17.4	0.104	9.9E-06	79.1	0.0401
trichloroethene (TCE)	1100	131.4	166	0.079	9.1E-06	74	0.422
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374
tetrachloroethene (PCE)	200	165.83	155	0.072	8.2E-06	18.5	0.754
1,1,2,2-tetrachloroethane	2870	167.85	106.8	0.071	7.9E-06	13.3	0.0141
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
carbon tetrachloride	800	153.8	170	0.078	8.8E-06	91.3	1.2
1,3,5-trichlorobenzene	49	181.45	717.6	0.03	8.2E-06	0.46	0.0581

Vapour Transport Calculations	Deff Layer 1 (cm ² /s)	Deff Layer 2 (cm ² /s)	Deff Foundations and Cracks (cm ² /s)	Total Effective Diffusion (Subsurface soil to surface) (cm ² /s)
TPH C6-C9				
aromatic	4.47E-3		7.51E-3	4.47E-3
aliphatic	3.48E-3		5.85E-3	3.48E-3
TPH C10-C14				
aromatic	2.49E-3		4.19E-3	2.49E-3
aliphatic	2.39E-3		4.02E-3	2.39E-3
vinyl chloride (adult exposures)	5.50E-3		9.25E-3	5.50E-3
trans-1,2-dichloroethene	3.67E-3		6.17E-3	3.67E-3
1,1-dichloroethane	3.85E-3		6.48E-3	3.85E-3
cis-1,2-dichloroethene	3.82E-3		6.43E-3	3.82E-3
1,2-dichloroethane (EDC)	5.40E-3		9.08E-3	5.40E-3
trichloroethene (TCE)	4.10E-3		6.90E-3	4.10E-3
1,1,2-trichloroethane	4.05E-3		6.81E-3	4.05E-3
tetrachloroethene (PCE)	3.74E-3		6.29E-3	3.74E-3
1,1,2,2-tetrachloroethane	3.69E-3		6.20E-3	3.69E-3
chloroform	5.40E-3		9.08E-3	5.40E-3
carbon tetrachloride	4.05E-3		6.81E-3	4.05E-3
1,3,5-trichlorobenzene	1.56E-3		2.62E-3	1.56E-3

**VAPOUR PARTITIONING, EMISSION AND AIR DISPERSION MODEL FOR CONTAMINATED SOILS
UTILISING ASTM GUIDANCE E 1739-95^F (2002)**

Vapour Migration from Impacted Soils - RBC Calculations

Subsurface Soils Phase Partitioning Results	Soil Concentration - RBC (mg/kg)	Vapour Phase Concentration (g/cm ³)	Saturated Soil Concentration (mg/kg)	Saturated Vapour Concentration (g/cm ³)	Free Phase Mole Fraction (mol/mol)	Concentration above Free Phase (g/cm ³)	Calculation Vapour Phase used in Calculation (g/cm ³)
TPH C6-C9							
aromatic		0.0E+00	4.5E+02	1.9E-04	0	0.0E+00	0.0E+00
aliphatic	100	3.1E-04	8.7E+01	2.7E-04	0	0.0E+00	3.1E-04
TPH C10-C14							
aromatic	1000	1.8E-05	1.9E+02	3.5E-06	0	0.0E+00	1.8E-05
aliphatic	1000	1.3E-04	3.3E+01	4.2E-06	0	0.0E+00	1.3E-04
vinyl chloride (adult exposures)	0.3	1.2E-06	7.7E+02	9.0E-03	0	0.0E+00	1.2E-06
trans-1,2-dichloroethene	5	6.7E-06	1.8E+03	1.4E-03	0	0.0E+00	6.7E-06
1,1-dichloroethane	45	4.8E-05	1.1E+03	1.2E-03	0	0.0E+00	4.8E-05
cis-1,2-dichloroethene	4	3.2E-06	7.3E+02	9.6E-04	0	0.0E+00	3.2E-06
1,2-dichloroethane (EDC)	4	1.2E-06	1.2E+03	4.3E-04	0	0.0E+00	1.2E-06
trichloroethene (TCE)	12	8.0E-06	7.0E+02	5.4E-04	0	0.0E+00	8.0E-06
1,1,2-trichloroethane	9	1.2E-06	3.2E+02	1.7E-04	0	0.0E+00	1.2E-06
tetrachloroethene (PCE)	12	1.4E-05	1.3E+02	1.7E-04	0	0.0E+00	1.4E-05
1,1,2,2-tetrachloroethane	20	7.0E-07	1.2E+03	1.2E-04	0	0.0E+00	7.0E-07
chloroform	8	5.5E-06	1.7E+03	1.1E-03	0	0.0E+00	5.5E-06
carbon tetrachloride	0.2	3.2E-07	6.0E+02	7.8E-04	0	0.0E+00	3.2E-07
1,3,5-trichlorobenzene	15	3.9E-07	1.1E+02	4.6E-06	0	0.0E+00	3.9E-07

Calculated Air Concentrations ASTM Guidance for Subsurface Soils	Vapour Phase Concentration at Source (g/cm ³)	Emission Rate from Surface of Ground (g/s)	Concentration in Building (mg/m ³)	Outdoor Air Concentration (mg/m ³)
TPH C6-C9				
aliphatic	3.1E-04	1.1E+00	3.6E-01	
TPH C10-C14				
aromatic	1.8E-05	4.6E-02	1.5E-02	
aliphatic	1.3E-04	3.2E-01	1.1E-01	
vinyl chloride (adult exposures)	1.2E-06	6.5E-03	2.2E-03	
trans-1,2-dichloroethene	6.7E-06	2.5E-02	8.2E-03	
1,1-dichloroethane	4.8E-05	1.9E-01	6.2E-02	
cis-1,2-dichloroethene	3.2E-06	1.2E-02	4.1E-03	
1,2-dichloroethane (EDC)	1.2E-06	6.3E-03	2.1E-03	
trichloroethene (TCE)	8.0E-06	3.3E-02	1.1E-02	
1,1,2-trichloroethane	1.2E-06	4.7E-03	1.6E-03	
tetrachloroethene (PCE)	1.4E-05	5.3E-02	1.8E-02	
1,1,2,2-tetrachloroethane	7.0E-07	2.6E-03	8.7E-04	
chloroform	5.5E-06	3.0E-02	9.9E-03	
carbon tetrachloride	3.2E-07	1.3E-03	4.4E-04	
1,3,5-trichlorobenzene	3.9E-07	6.1E-04	2.0E-04	

Inhalation of Volatile Chemicals above Soil Hot-Spots (RBC)

General Data/ Equations		Units		Exposure Calculations (RME) - Soils Indoors			
Exposure Parameters							
Exposure Frequency (EF)		days/year	240	240	Work for 5 days per week for 48 weeks		
Exposure Duration (ED)		years	30	30	Working life at the one location		
Body Weight (BW)		kg	70	70	USEPA 1989 and CSMS 1996		
Averaging Time - NonThreshold (ATc)		days	25550	25550	USEPA 1989 and CSMS 1996		
Averaging Time - Threshold (ATn)		days	10950	10950	USEPA 1989 and CSMS 1996		
Inhalation Rate (InhR)		m ³ /hr	1.17	1.17	Inhalation rate indoors		
Exposure Time (ET)		hours/day	9	9	Assume 9 hours per day spent indoors		
Bioavailability (B)		-	100%	100%	Assume 100% bioavailability via inhalation		
Fraction Inhaled (FI)		-	100%	100%	Assume all time spent at inside building located above plume		
Intake Factor = $\frac{InhR \cdot ET \cdot B \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$		m ³ /kg/day	4.2E-02	9.9E-02	NonThreshold		
					Threshold		
<i>Daily Intake = Concentration in Air x Intake Factor</i>							
<i>NonThreshold Risk = Daily Intake from Air for NonThreshold Effects x Slope Factor</i>							
<i>Hazard Quotients = (Daily Intake from Air for Threshold Effects/ADI)</i>							
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD (accounting for background)	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/m ³)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		4.0E-6	6.5E-1
TPH C6-C9 aliphatic		1.8E-01	3.63E-01	1.5E-02	3.6E-02	--	2.0E-1
TPH C10-C14 aromatic		5.1E-02	1.54E-02	6.5E-04	1.5E-03	--	3.0E-2
TPH C10-C14 aliphatic		2.6E-01	1.08E-01	4.6E-03	1.1E-02	--	4.2E-2
vinyl chloride (adult exposures)	1.5E-02		2.19E-03	9.3E-05	2.2E-04	1.4E-6	--
trans-1,2-dichloroethene		1.7E-02	8.24E-03	3.5E-04	8.1E-04	--	4.8E-2
1,1-dichloroethane		1.4E-01	6.23E-02	2.6E-03	6.2E-03	--	4.3E-2
cis-1,2-dichloroethene		1.0E-02	4.12E-03	1.7E-04	4.1E-04	--	4.1E-2
1,2-dichloroethane (EDC)	9.8E-03		2.12E-03	9.0E-05	2.1E-04	8.8E-7	--
trichloroethene (TCE)	1.5E-03		1.10E-02	4.7E-04	1.1E-03	7.0E-7	--
1,1,2-trichloroethane		4.0E-03	1.59E-03	6.7E-05	1.6E-04	--	3.9E-2
tetrachloroethene (PCE)		3.8E-02	1.77E-02	7.5E-04	1.7E-03	--	4.6E-2
1,1,2,2-tetrachloroethane	1.1E-02		8.69E-04	3.7E-05	8.6E-05	3.9E-7	--
chloroform	1.5E-03	2.0E-02	9.95E-03	4.2E-04	9.8E-04	6.2E-7	4.9E-2
carbon tetrachloride		6.1E-04	4.37E-04	1.9E-05	4.3E-05	--	7.1E-2
1,3,5-trichlorobenzene		4.6E-04	2.04E-04	8.6E-06	2.0E-05	--	4.4E-2

↑
Modelled from RBC

Exposure to Chemicals via Ingestion of Soils (RBC)

General Data/ Equations		Units	Exposure Calculations (RME)				
Exposure Parameters			Ingestion of Soils by Workers				
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays				
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989				
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996				
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996				
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996				
Ingestion Rate (IRs)	mg/day	25	Soil intake for adults as per CSMS 1996				
Fraction Ingested (FI)	-	100%	All soil ingested from site				
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in soil				
Conversion factor (CF)	mg to kg	1.E-06	Conversion				
Intake Factor = $\frac{IRs \cdot FI \cdot B \cdot CFEF \cdot ED}{BW \cdot AT}$		kg/kg/day	1.0E-07	NonThreshold			
			2.3E-07	Threshold			
<i>Daily Intake from Soil = Concentration in Soil x Intake Factor</i>							
<i>NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor</i>							
<i>Hazard Quotients = (Daily Intake for Threshold Effects/ADI)</i>							
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
				TOTAL		4.4E-8	3.5E-2
TPH C6-C9 aliphatic		4.5E+00	100	1.0E-05	2.3E-05	--	5.2E-6
TPH C10-C14 aromatic		4.0E-02	1000	1.0E-04	2.3E-04	--	5.9E-3
TPH C10-C14 aliphatic		9.0E-02	1000	1.0E-04	2.3E-04	--	2.6E-3
TPH C15+ aromatic		2.7E-02	2500	2.5E-04	5.9E-04	--	2.2E-2
TPH C15+ aliphatic		1.8E+00	2500	2.5E-04	5.9E-04	--	3.3E-4
vinyl chloride (adult exposures)	1.2E+00		0.3	3.0E-08	7.0E-08	3.5E-8	--
trans-1,2-dichloroethene		1.7E-02	5	5.0E-07	1.2E-06	--	6.9E-5
1,1-dichloroethane		1.0E-01	45	4.5E-06	1.1E-05	--	1.1E-4
cis-1,2-dichloroethene		1.0E-02	4	4.0E-07	9.4E-07	--	9.4E-5
1,2-dichloroethane (EDC)	1.2E-02		4	4.0E-07	9.4E-07	4.8E-9	--
trichloroethene (TCE)		1.5E-03	12	1.2E-06	2.8E-06	--	1.9E-3
1,1,2-trichloroethane		4.0E-03	9	9.1E-07	2.1E-06	--	5.3E-4
tetrachloroethene (PCE)		9.2E-03	12	1.2E-06	2.8E-06	--	3.0E-4
1,1,2,2-tetrachloroethane		4.0E-02	20	2.0E-06	4.7E-06	--	1.2E-4
chloroform		6.5E-03	8	8.1E-07	1.9E-06	--	2.9E-4
carbon tetrachloride		5.0E-04	0.2	2.0E-08	4.7E-08	--	9.5E-5
1,3,5-trichlorobenzene		7.7E-03	15	1.5E-06	3.5E-06	--	4.6E-4

↑
RBC derived

Dermal Exposure to Chemicals via Contact with Soil (RBC)

General Data/ Equations		Units	Exposure Calculations (RME)					
			Dermal Contact with Soils by Workers					
Exposure Parameters								
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays					
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989					
Body Weight (BW)	kg	70	USEPA 1989 and CSMS 1996					
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996					
Averaging Time - Threshold (ATn)	days	10950	USEPA 1989 and CSMS 1996					
Surface Area (SAs)	cm ²	2300	Hands and forearms for adult male (50th percentile, USEPA 1997 and 2004)					
Adherence Factor (AF)	mg/cm ²	0.51	Default value as per CSMS 1996					
Dermal Absorption Rate (Abs)	per hour	0.005	Rate per hour for adults as per Hawley 1985, CSOIL 1995 and 2001 and SEDISOIL 1996					
Exposure Time (ET)	hr/day	12	Assume that soil remains on skin for 12 hours before washing					
Matrix Effect (ME)	-	15%	Absorption of chemicals from soil matrix is 15% from pure compound					
Conversion Factor (CF)	mg to kg	1.E-06	Conversion					
Site-Specific Factor (SS), -		1.00	Not used for intrusive works					
Intake Factor = $\frac{SAs \cdot AF \cdot Abs \cdot ET \cdot ME \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$		kg-hr/kg/day	4.2E-08	NonThreshold				
			9.9E-08	Threshold				
<i>Daily Intake from Soil = Concentration in Soil x Intake Factor</i> <i>NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor</i> <i>Hazard Quotients = (Daily Intake for Threshold Effects/ADI)</i>								
Chemical	Toxicity Data		Concentration	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold ADI, TDI or RfD (mg/kg/day)	in Soil (mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	NonThreshold Risk (unitless)	Hazard Quotient (unitless)	
						TOTAL	1.7E-8	1.4E-2
TPH C6-C9 aliphatic		4.5E+00	100	4.2E-06	9.9E-06	--	2.2E-6	
TPH C10-C14 aromatic		4.0E-02	1000	4.2E-05	9.9E-05	--	2.5E-3	
TPH C10-C14 aliphatic		9.0E-02	1000	4.2E-05	9.9E-05	--	1.1E-3	
TPH C15+ aromatic		3.0E-02	2500	1.1E-04	2.5E-04	--	8.3E-3	
TPH C15+ aliphatic		1.8E+00	2500	1.1E-04	2.5E-04	--	1.4E-4	
vinyl chloride (adult exposures)	1.2E+00		0.3	1.3E-08	3.0E-08	1.5E-8	--	
trans-1,2-dichloroethene		1.7E-02	5	2.1E-07	5.0E-07	--	2.9E-5	
1,1-dichloroethane		1.0E-01	45	1.9E-06	4.5E-06	--	4.5E-5	
cis-1,2-dichloroethene		1.0E-02	4	1.7E-07	4.0E-07	--	4.0E-5	
1,2-dichloroethane (EDC)	1.2E-02		4	1.7E-07	4.0E-07	2.0E-9	--	
trichloroethene (TCE)		1.5E-03	12	5.1E-07	1.2E-06	--	8.2E-4	
1,1,2-trichloroethane		4.0E-03	9	3.8E-07	8.9E-07	--	2.2E-4	
tetrachloroethene (PCE)		1.4E-02	12	5.1E-07	1.2E-06	--	8.5E-5	
1,1,2,2-tetrachloroethane		4.0E-02	20	8.5E-07	2.0E-06	--	5.0E-5	
chloroform		1.3E-02	8	3.4E-07	7.9E-07	--	6.1E-5	
carbon tetrachloride		1.4E-03	0.2	8.5E-09	2.0E-08	--	1.4E-5	
1,3,5-trichlorobenzene		7.7E-03	15	6.4E-07	1.5E-06	--	1.9E-4	

↑
RBC derived

Appendix H

Preliminary HHRA



27 November 2006
Project No. 43217543

DBL Property
Level 6, 432 Kent Street
Sydney NSW 2000

Attention: Jeff Lord
Director

Dear Jeff,

Subject: Preliminary QRA for Proposed Southlands Development

1. Introduction

URS has been commissioned by Orica Australia Pty Ltd (Orica) and Macquarie Goodman (jointly the proponent) to prepare a quantitative human health risk assessment (HHRA) as part of the Environmental Assessment (EA) being prepared for the proposed development of the Southlands property. As the final design of the proposed development has not yet been determined, a preliminary HHRA has been undertaken to provide a review of the available data and an assessment of risks to human health assuming no risk mitigation measures are adopted. The aim of the preliminary HHRA is to provide an initial assessment of risks to human health to identify key issues for consideration within the Remediation Action Plan (RAP) to ensure that all issues identified with respect to risks to human health are addressed appropriately as part of the remediation and development of the site. The results may also be relevant to the finalisation of the development layout and design.

Once the RAP and other design aspects of the project have been finalised, the HHRA can be completed as a detailed report that addresses all issues identified in the preliminary HHRA and the RAP.

The preliminary HHRA can also be used to facilitate further discussion with the Department of Environment and Conservation (DEC) and NSW Department of Health (NSW Health) such that they have the opportunity to provide comment and input on key aspects before completion of the final HHRA.

URS Australia Pty Ltd (ABN 46 000 691 690)
Level 3, 116 Miller Street North Sydney
NSW 2060, Australia
Tel: 61 2 8925 5500
Fax: 61 2 8925 5555





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2. Objectives and Methodology

2.1 Objectives of the Preliminary HHRA

Based on the available information, the overall objectives of the preliminary HHRA are:

- To provide a quantitative assessment of potential risks to human health associated with the presence of chemicals in soil, groundwater, surface water and air within the Southlands property; and
- Depending on the outcome of the preliminary HHRA, identify issues that require risk mitigation measures to be incorporated into the RAP with the development risk based soil, groundwater and/or vapour concentrations that can be used at a screening level for further investigation or remediation end-points (if required).

2.2 Approach to Human Health Risk Assessment

The approach taken to the assessment of human health risks is generally in accordance with the protocols/ guidelines recommended by enHealth (Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards, June 2002). These guidelines draw on and are supplemented by those provided by ANZECC and NH&MRC and detailed in the documents:

- “The Health Risk Assessment and Management of Contaminated Sites” (CSMS 1991, 1993, 1996 and 1998 and enHealth 2002b);
- ANZECC/NH&MRC (1992); and
- The NEPM (Schedule B(4), Guideline on Health Risk Assessment Methodology, 1999).

ANZECC and NH&MRC currently provide only general guidance for the completion of these tasks and, as such, the more detailed protocols and guidelines developed by the US EPA (1989 and 2001) have been used to provide supplementary guidance.

In addition the HHRA will be undertaken to be consistent with other key risk assessment undertaken in the area, in particular, the Consolidated Human Health Risk Assessment (Consolidated HHRA, URS 2005) that provided an assessment of risks to human health associated with issues derived from chlorinated groundwater plumes in off-site areas, including commercial areas surrounding Southlands.

Human health risk assessment can be divided into the following four prime tasks:

- Issue Identification/Data Evaluation;



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- Exposure Assessment;
- Toxicity/Hazard Assessment; and
- Risk characterisation.

This letter presents a preliminary HHRA and as such, has not presented all of the details associated with the completion of each of the above tasks. These will be presented within the final HHRA report.

3. Identification of Issues

3.1 General

The site is zoned commercial/industrial and it is expected that future use/development of the site would be consistent with this zoning. It is understood that the site is proposed to be developed in stages, with Block 2 to be developed first (Stages 1 and 2), the southern portion of Block 1 (Stage 3) to be developed at a later stage and the northern portion of Block 1 (Stage 4) to be further investigated. It is noted that the Stage 4 areas is associated with a number of DNAPL sources currently being investigated. It is unsure whether any development will be able to be undertaken in this area due to the presence of these sources and the need to provide an area that may be used to remove and treat DNAPL in this area.

On this basis, the preliminary HHRA has focused on 2 key areas:

- Block 2 – the preliminary assessment has considered the potential for commercial development in all areas of Block 2; and
- Block 1 – while the extent of development in this area is uncertain at this stage, for the purpose of this preliminary assessment it has been assumed that all of Block 1 may be developed for commercial purposes.

The assessment presented does not address off-site issues as these have been addressed separately in the Consolidated HHRA (URS, 2005) and subsequent monitoring reports.

3.2 Review of Available Data

Review of the available data and identification of chemicals of potential concern is presented in Appendix A. In summary the available data suggests the following:

- Soil impacts have been identified on Block 1 and to a lesser extent Block 2;
- Groundwater (shallow and deep plumes) impacts have been identified beneath the site, with shallow groundwater interacting with and discharging into the ponds on Block 1 and Springvale Drain;



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- Emission to air of volatile chemicals present in shallow groundwater;
- Impacted surface water and sediments have been identified in the ponds in Block 1. While samples collected at one ephemeral pond (SW06) appears to be representative of shallow groundwater discharge, the concentrations reported within the permanent ponds are lower;
- Impacted surface water and sediments have been identified in Springvale Drain (that intersects shallow groundwater plumes), and to a lesser extent Floodvale Drain. Ambient air quality in the vicinity of Springvale Drain is affected by emissions of volatile contaminants present in the drain; and
- Impacts on ambient air quality associated with current and proposed remediation activities on the adjoining BIP, such as the on going operation of the groundwater treatment plant (GTP), operation of the HCB repackaging plant and proposed car park waste encapsulation (CPWE) remediation project.

3.3 Summary of Chemicals of Potential Concern

A review of data and identification of chemicals of potential concern (COPCs) is presented in Appendix A. The following presents a summary of the COPCs identified in soil, surface water, sediment and air (as relevant) within Block 1 and Block 2.



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Table 1 – Summary of COPC Identified on Block 1 and Block 2

Chemical	COPC – Block 1					COPC – Block 2		
	Soil	Sediment	Surface Water	Emissions to Air	Emissions from Drains	Soil	Emissions to Air	Emissions from Drains
Asbestos						•		
Arsenic			•					
Cadmium			•					
Chromium	•	•	•			•		
Lead	•		•			•		
Nickel			•					
Zinc			•					
Mercury	•					•		
TPH	•	•					•	
Benzene	•			•			•	
Toluene				•			•	
Ethylbenzene	•			•			•	
Xylenes	•			•			•	
1,2,4-trimethylbenzene				•			•	
1,3,5-trimethylbenzene							•	
vinyl chloride	•	•	•	•	•		•	•
1,1-dichloroethene			•				•	
trans-1,2-dichloroethene			•	•			•	
cis-1,2-dichloroethene	•	•	•	•	•		•	•
trichloroethene (TCE)	•	•	•	•	•		•	•
tetrachloroethene (PCE)	•	•	•	•			•	
1,1-dichloroethane							•	
1,2-dichloroethane (EDC)	•	•	•	•	•		•	•
1,1,2-trichloroethane	•	•	•	•			•	
1,1,2,2-tetrachloroethane	•	•	•	•				
Chloroform	•	•	•	•	•		•	•
carbon tetrachloride			•	•	•		•	•
dichloromethane				•			•	
Naphthalene				•			•	
PAHs (non-threshold)	•							
hexachlorobutadiene (HCBD)	•	•	•					
hexachlorobenzene (HCB)	•					•		
bis(2-chloroethyl)ether						•		
tetrahydrofuran					•			•
trichlorofluoromethane (freon 11)							•	

3.4 Summary of Key Exposures

Groundwater will not to be extracted for use on the site as it is located within the Groundwater Extraction Exclusion Area (GEEA). Groundwater extraction wells and associated pipe-work associated with the primary containment lines for the GTP are located along the McPherson Street boundary of the property. These are to remain with the pipe-work to be redirected (once designs are completed). Orica is to retain ownership of the easements used for this infrastructure. It is also understood that other easements may be required on the Southlands property for the purpose of ongoing bioremediation trials (and other trials as needed). As the location of these areas is currently unknown, the presence of such easement have not been accounted for in this assessment.

Based on the landuse of the site and the nature and extent of impacts identified on the property, the following exposure pathways are considered to be complete and warrant further assessment.

Table 2 – Summary of Key Exposure Pathways

Receptor	Exposure Pathway	Activity
Adult workers – within Block 2 (Stages 1 and 2)	Inhalation of volatile chemicals derived from impacts in shallow groundwater, emissions from Floodvale and Springvale Drains and emissions from the BIP Direct contact (incidental ingestion and dermal contact) with impacted soil	Working on the site following redevelopment as well as gardeners and non-intrusive maintenance workers (such as maintenance of GTP aboveground infrastructure)
Adult workers – within Block 1	Inhalation of volatile chemicals derived from impacts in shallow groundwater, emissions from Springvale Drain, emissions from ponds and emissions from the BIP Direct contact (incidental ingestion and dermal contact) with impacted soil and sediment in ephemeral ponds by workers on-site and all pond areas by gardeners and workers involved in maintaining ponds Incidental contact (ingestion and dermal contact) with water in ephemeral and permanent ponds during maintenance of weeds etc*	Working on the site following redevelopment including the maintenance of GTP aboveground infrastructure Workers on the site following redevelopment as well as gardeners and non-intrusive maintenance workers Maintenance of visual amenity of area following development

* It has been assumed that workers within Block 1 may come into contact with sediments associated with ephemeral ponds when these areas are dry and accessible. It has been assumed that when these areas are wet, workers on the site would not come into contact with the water or sediments during normal workday activities. However it has been assumed that maintenance workers may access all ponds (ephemeral and permanent) during gardening and maintenance of visual amenity.

Exposures by visitors would be lower than long term workers due to the shorter duration of time spent on site. Hence quantification of risks for visitors is not required. The



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proposed road adjacent to Springvale Drain is to be a private road and hence will not be accessed by the general public. No consideration of more sensitive uses of the area has been considered such as a childcare facility (which is permitted under the Industrial 4(a) zoning). As such, only adult commercial and industrial workers are relevant to the assessment.

Exposures by intrusive workers on the site (including intrusive works associated with the maintenance of GTP infrastructure) and workers involved in the maintenance of Springvale and Floodvale Drains have not been assessed as it is considered appropriate that such works be managed under a specific management plan that includes health and safety requirements to minimise contact with groundwater (and surface water) and address issues associated with inhalation of vapours (particularly in confined space areas).

4. Quantification of Risk – Block 1

4.1 Exposure and Calculation of Risk

The quantification of risk associated with exposures that may be relevant to development of Block 1 has adopted the overall methodology presented within the Consolidated HHRA (URS, 2005). The following presents a summary of the exposure parameters and calculated risks for Block 1, with details of the calculations presented in Appendix B.

Exposure parameters that are considered representative of reasonable maximum exposure (RME) have been selected for the receptor groups and exposure pathways identified for Block 1. Where available, exposure data has been obtained from Australian sources (CSMS, 1991, 1993, 1996 and 1998, ANZECC 1992, NEPM 1999 and enHealth 2002 and 2003). The exposure parameters identified for each key receptor and exposure pathway are presented below.

It is noted that risk levels considered representative of acceptable risk are:

- non-threshold risk (sum over all pathways) = 1×10^{-5} ; and
- threshold hazard index (HI) (sum over all pathways) = 1.



Table 3 - Summary of Preliminary Calculation of Exposure and Risk – Southlands Block 1

Receptor	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
				Non-Threshold Risk	Threshold HI
Worker On-site (Adult)	Inhalation of COPC in emissions derived from sources within the BIP	Modelled ground level concentrations based on emissions from the BIP that include GTP, HCB repackaging and CPWE remediation and other sources	<p><u>Parameters relevant for all pathways</u></p> <p>Body weight of 70 kg. Exposure for 12 hours per day (i.e. long work shifts) for 240 days/year (5 days per week for 48 weeks) for 30 years</p> <p>Inhalation of 1.17 m³ air per hour indoors (9 hours) and 2.2 m³ air per hour outdoors (1 hours).</p>	2.4x10 ⁻⁹	0.0024
	Inhalation of volatile chemicals derived from soil and groundwater	Measured soil gas and flux emission data (maximum and average) collected in areas relevant to the assessment of Block 1	Inhalation of 1.17 m ³ air per hour indoors (9 hours) and 2.2 m ³ air per hour outdoors (1 hours).	2.4x10 ⁻⁶ (av) 6.1x10 ⁻⁶ (max)	1.0 (av) 2.7 (max)
	Inhalation of volatile emissions derived from open drains and ponds	Measured air concentrations (average and maximum) adjacent to the drains. Potential emissions to air from ponds on Block 1 are assumed to be at most equal to emissions from Springvale Drain – hence no additional modelling has been undertaken	Inhalation of 1.27 m ³ air per hour indoors/outdoors (10 hours) (note risks associated with 4 hrs per day outdoors lower and not presented in preliminary summary)	6.2x10 ⁻⁴ (av) 1.4x10 ⁻³ (max)	7.4 (av) 17 (max)
	Ingestion and dermal contact with chemicals in soil and sediment (assuming excavation and movement across the site during construction)	Soil concentrations from available data. Maximum value from available data from soil and sediments (ephemeral ponds).	Ingestion of 25 mg of soil per day by adults. Once ingested it is assumed that 100% is absorbed into the body. When outdoors it is assumed that the hands and forearms get dirty each day (2300 cm ² of skin). Once dirty it is assumed that 0.51 mg of soil adheres to each cm ² of skin. Assume an adult will wash at the end of each day resulting in up to 12 hours of the day dirty.	1.3x10 ⁻⁴	5.4
TOTAL RISK (based on average and maximum exposures presented)				8x10⁻⁴ (av) 1x10⁻³ (max)	14 (av) 25 (max)

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Table 4 - Summary of Preliminary Calculation of Exposure and Risk – Southlands Block 1

Receptor	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
				Non-Threshold Risk	Threshold HI
Gardener / Maintenance Worker (Adult)*	Inhalation of COPC in emissions derived from sources within the BIP	Modelled ground level concentrations based on emissions from the BIP that include GTP, HCB repackaging and CPWE remediation and other sources	<p><u>Parameters relevant for all pathways</u></p> Body weight of 70 kg. Exposure for 10 hours per day (i.e. long work day) once every 2 weeks (gardening) and 1 month every 5 years (weeding ponds) over 30 years 2.2 m ³ air per hour outdoors (assuming all 10 hours is spent outdoors).	4.5x10 ⁻¹⁰	0.00044
	Inhalation of volatile chemicals present in soil and groundwater	Measured soil gas and flux emission data (maximum) collected in areas relevant to the assessment of Block 1	2.2 m ³ air per hour outdoors (assuming all 10 hours is spent outdoors).	3.3x10 ⁻⁷	0.15
	Inhalation of volatile emissions derived from open drains and ponds	Measured air concentrations adjacent to the drains. Potential emissions to air from ponds on Block 1 are assumed to be at most equal to emissions from Springvale Drain – hence no additional modelling has been undertaken	2.2 m ³ air per hour outdoors (assuming all 10 hours is spent adjacent to drains or pond areas).	2.6x10 ⁻⁴	3.3
	Ingestion and dermal contact with chemicals in soil and sediment	Soil concentrations from available data. Maximum value from available data from soil (gardening) and sediment (all ponds during maintenance of ponds).	Ingestion of 25 mg of soil per day by adults. Once ingested it is assumed that 100% is absorbed. When outdoors it is assumed that the hands, forearms and lower legs get dirty each day (4580 cm ² of skin). Once dirty it is assumed that 0.51 mg of soil adheres to each cm ² of skin. Assume an adult will wash at the end of each day with 12 hours of the day dirty.	Soils: 3.6x10 ⁻⁶ Sediments: 2.8x10 ⁻⁶	Soil: 0.77 Sediments: 0.063
	Ingestion and dermal contact with water in ponds	Water concentration from ponds, both ephemeral and permanent ponds	Incidental ingestion of 5 mL (teaspoon) each day where 100% is absorbed. Assume the hands, forearms and lower legs get wet (4580 cm ² of skin) for 2 hours each time.	Ephemeral: 2.5x10 ⁻⁴ Permanent: 5.3x10 ⁻⁶	Ephemeral: 2.7 Permanent: 1.4



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Receptor	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
			TOTAL RISK (all exposures with average risk from ephemeral and permanent ponds)	4x10 ⁻⁴	6

4.2 Identification of Risk Issues

Based on the preliminary calculation of risk presented in Tables 3 and 4 the following issues have been identified that warrant specific consideration within the RAP to reduce the level of exposure and thus risk.

4.2.1 Soil and Sediment

Potential risks to workers associated with direct exposure (ingestion and dermal contact) to maximum concentrations of COPC identified in soil and sediment (ephemeral ponds only) assuming development within Block 1 with no removal of hot-spots or capping are above acceptable risk targets.

With respect to the calculations presented and the potential for exposure, the following is noted:

- Calculated non-threshold risk is dominated by exposure to vinyl chloride (>90% total risk) identified in soil and sediment (ephemeral pond location);
 - Elevated concentrations (above adopted screening criteria) of vinyl chloride in soil are present at TP37 (1.3-1.4m depth, 53 mg/kg), TP29 (0.8-0.9m depth, 33 mg/kg) and SS057 (0.7-0.8m depth, 3.5 mg/kg). All these are located in the south-eastern portion of Block 1 in an area where further delineation works were undertaken by URS in 2006. No vinyl chloride was reported above the laboratory LOR in samples collected from this area suggesting the elevated concentrations of vinyl chloride are associated with a limited area (hot-spot).
 - Elevated concentrations (above adopted screening criteria) of vinyl chloride in sediment were identified at the ephemeral pond location SED06 (384 mg/kg reported at 0.5-0.6m depth and 285 mg/kg reported at 0.2-0.3m depth) and SED05 (16 mg/kg reported at 0.2-0.3m depth). Both of these areas are located in drainage hollows within the eastern portion of Block 1 in the vicinity of the pond areas. There is the potential for these elevated concentrations to be associated with periodic discharge of shallow groundwater into ephemeral drainage hollows and are expected to be limited to these types of areas within Block 1.
 - If the areas impacted with vinyl chloride were removed from Block 1 (to a level less than 5 mg/kg), then non-threshold risks would be reduced to a level that would be considered acceptable (less than 1×10^{-5}).
- Calculated threshold risk (HI) is dominated by exposure to HCB (45%), lead (26%), chromium (14%) and HCB (10%) identified in soil and PCE (42%) and HCB (31%) in sediment (ephemeral ponds);



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- Elevated concentrations of HCB and HCBD have been identified in soil within the south-eastern portion of Block 1 (HA01-HA03 and TP29) with elevated concentrations of HCB also reported adjacent to the southern portion of Nant St (TP01 and TP02). These areas were subject to further delineation investigations by URS in 2006 that suggested that the elevated concentrations reported in these areas are not widespread and are likely to be associated with hot-spots.
- Elevated concentrations of lead and chromium were reported in the southern portion of Block 1 (TP31) and south-eastern portion of Block 1 (TP127). Additional data collected by URS in 2006 indicates that elevated concentrations of lead and chromium are not widespread across Block 1.
- Elevated concentrations of PCE and HCBD were reported in SED06, ephemeral drainage depression located within the eastern portion of Block 1. There is the potential for these elevated concentrations to be associated with periodic discharge of shallow groundwater into ephemeral drainage hollows and are expected to be limited to these types of areas within Block 1.
- If the areas impacted with HCB, HCBD, lead, chromium and PCE were removed from Block 1 (to a level less than the adopted criteria), then the total threshold risk (HI) would be reduced to a level that would be considered acceptable (i.e. less than 1).

While elevated exposures have been identified to some key COPC identified in soil and sediment within Block 1, exposures may be reduced by the removal of hot-spots. In addition, should soil removal not be undertaken the potential for exposure can be minimised by the placement of clean fill or capping across the site. This would essentially eliminate exposures to non-volatile COPC (subject to implementation of a site management plan) and reduce exposures to volatile COPC. Note additional requirements for capping within Block 1 are presented below in Section 4.2.2.

4.2.2 Vapour Migration from Subsurface Sources

Potential risks to workers associated with inhalation of volatile COPC that may be present in emissions to air from the surface of the ground, overlying shallow groundwater and/or soil impacts, assuming development within Block 1 with no additional capping are above acceptable target risk values.

With respect to the calculations presented and the potential for exposure, the following is noted:

- The quantification of exposure has relied on a limited number of flux emissions and soil gas samples collected from Block 1 and the adjacent rail corridor. While the data is considered to provide a reasonable estimate of potential emissions to air

that may be derived from shallow groundwater, no data has been collected above hot-spots associated with volatile and semi-volatile COPCs in soil and sediment within Block 1. Hence the data used may not be representative of site conditions should these hot-spots remain in place (rather than removal).

- Calculated non-threshold risks, based on both maximum (6×10^{-6}) and the average (2×10^{-6}) of all data reported are considered acceptable ($< 1 \times 10^{-5}$), however when considered in the calculation of total risk, the contribution associated with the inhalation of the volatile non-threshold COPCs is significant and would warrant reduction.
- Calculated threshold risk (HI), based on the maximum (2.7) and average (1.0) of all data reported are greater than or equal to 1, with the total HI from all exposure pathways in excess of 1 and thus unacceptable. The calculated HI is dominated by exposure to carbon tetrachloride (80%) and naphthalene (16%) within buildings that may be constructed on Block 1..

In the event Block 1 were to be developed with commercial buildings then risk mitigation measures would need to be adopted to ensure vapour migration and accumulation within these buildings was minimised such that risks to human health are below acceptable target values. Risk mitigation measures could include removal of volatile soil and sediment sources (not characterised at present), sub floor vapour extraction and/or provision of a suitable capping or vapour barrier.

If capping was to be considered across Block 1, the capping would need to be designed such that the vapour attenuation provided was at least 0.1. Based on preliminary calculations (undertaken using Johnson and Ettinger equations, USEPA 2003), a clay capping of 0.5 to 1m thickness would provide this level of attenuation.

4.2.3 Emissions to Air from Springvale Drain

Potential risks to workers associated with inhalation of volatile COPC in emissions to air from Springvale Drain (and potentially ephemeral ponds within Block 1) are above target risk values and therefore unacceptable.

With respect to the calculations presented and the potential for exposure, the following is noted:

- Calculated non-threshold risks are dominated by exposure to measured concentrations of EDC (88%), vinyl chloride (10%) and TCE (1%) in air adjacent to Springvale Drain.
- Calculated threshold risk (HI) are dominated by exposure to measured concentrations of carbon tetrachloride (89%), cis-1,2-dichloroethene (6%), chloroform (3%) and PCE (2%) in air adjacent to Springvale Drain.

Calculated risks to human health associated with emissions to air from Springvale Drain based on the maximum and average concentrations reported are significantly greater than the risk levels considered representative of acceptable risk (non-threshold 1×10^{-5} and threshold HI of 1). Concentrations of COPC in air would need to decrease by a factor of 100 to 1000 for calculated risks to fall below the target values. As such the RAP will need to consider measures to reduce the concentrations of volatile COPC within Springvale Drain. It is expected that the operation of the GTP may affect the concentrations present within the drain. However as the magnitude of this effect is an unknown, alternate measure that specifically targets the potential for shallow groundwater to discharge into the drain should be considered. Details on the proposed remediation option are presented in the RAP

4.2.4 Water in Ponds

Potential risks to workers associated with direct contact with water in the ponds (and potential inhalation exposures) during maintenance of ephemeral ponds within Block 1 are above target risk values and unacceptable.

In the event that these ponds remain on the site in the present form, and they require regular maintenance, then these works must be undertaken in accordance with an appropriate management plan to ensure adoption of appropriate work methods to minimise exposure to the COPC.

If the ponds are to be filled in, the potential for exposure will be essentially removed. If, however the ponds were to be lined and used for stormwater management, exposures would be expected to be lower as a consequence of reduced concentrations of COPC, however further testing of water would be required to confirm the water concentrations and exposure.

5. Quantification of Risk – Block 2

5.1 Exposure and Calculation of Risk

The quantification of risk associated with exposures that may be relevant to the proposed development of Block 2 has adopted the methodology presented within the Consolidated HHRA (URS, 2005). The following presents a summary of the exposure parameters and calculated risks for Block 2, with details of the calculations undertaken presented in Appendix C.

Exposure parameters that are considered representative of reasonable maximum exposure (RME) have been selected for the receptor groups and exposure pathways identified within Block 2. Where available, exposure data has been obtained from Australian sources (CSMS, 1991, 1993, 1996 and 1998, ANZECC 1992, NEPM 1999 and enHealth 2002 and 2003). The exposure parameters identified for each key receptor and exposure pathway are presented below.



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It is noted that risk levels considered representative of acceptable risk are:

- non-threshold risk (sum over all pathways) = 1×10^{-5} ; and
- threshold hazard index (HI) (sum over all pathways) = 1.



Table 5 - Summary of Preliminary Calculation of Exposure and Risk – Southlands Block 2

Receptor	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk		
				Non-Threshold Risk	Threshold HI	
Worker On-site (Adult)	<p>Inhalation of COPC in emissions derived from sources within the BIP</p> <p>Inhalation of volatile chemicals derived from soil and groundwater</p> <p>Inhalation of volatile emissions derived from open drains and ponds</p> <p>Ingestion and dermal contact with chemicals in soil (assuming excavation and movement across the site during construction)</p>	<p>Modelled ground level concentrations based on emissions from the BIP that include GTP, HCB repackaging and CPWE remediation and other sources</p> <p>Measured soil gas and flux emission data (maximum and average) collected in areas relevant to the assessment of Block 2</p> <p>Measured air concentrations (average and maximum) adjacent to Springvale and Floodvale Drains.</p> <p>Soil concentrations from available data. Maximum value from available data from soil within Block 2</p>	<p><u>Parameters relevant for all pathways</u></p> <p>Body weight of 70 kg. Exposure for 12 hours per day (i.e. long work shifts) for 240 days/year (5 days per week for 48 weeks) for 30 years</p>			
			<p>Inhalation of 1.17 m³ air per hour indoors (9 hours) and 2.2 m³ air per hour outdoors (1 hour).</p>	→	2.4x10 ⁻⁹	0.0024
			<p>Inhalation of 1.17 m³ air per hour indoors (9 hours) and 2.2 m³ air per hour outdoors (1 hour).</p>	→	4.3x10 ⁻⁷ (av) 9.6x10 ⁻⁷ (max)	0.097 (av) 0.16 (max)
			<p>Inhalation of 1.27 m³ air per hour indoors/outdoors (10 hours) (note risks associated with 4 hrs per day outdoors lower and not presented in preliminary summary)</p>	→	5.1x10 ⁻⁴ (av) 1.4x10 ⁻³ (max)	9.8 (av) 17 (max)
			<p>Ingestion of 25 mg of soil per day by adults. Once ingested it is assumed that 100% is absorbed into the body. When outdoors it is assumed that the hands and forearms get dirty each day (2300 cm² of skin). Once dirty it is assumed that 0.51 mg of soil adheres to each cm² of skin. Assume an adult will wash at the end of each day resulting in up to 12 hours of the day dirty.</p>	→	3.3x10 ⁻⁷	1.4
TOTAL RISK (based on average and maximum exposures presented)						
				→	5x10 ⁻⁴ (av) 1x10 ⁻³ (max)	11 (av) 18 (max)

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Table 6 - Summary of Preliminary Calculation of Exposure and Risk – Southlands Block 2

Receptor	Exposure Pathways	Chemical Concentrations	Exposure Parameters	Calculated Risk	
				Non-Threshold Risk	Threshold HI
			<p><u>Parameters relevant for all pathways</u></p> <p>Body weight of 70 kg. Exposure for 10 hours per day (i.e. long work day) once every 2 weeks (gardening) over 30 years</p>		
Gardener / Maintenance Worker (Adult)*	Inhalation of COPC in emissions derived from sources within the BIP	Modelled ground level concentrations based on emissions from the BIP that include GTP, HCB repackaging and CPWE remediation and other sources	→ 2.2 m ³ air per hour outdoors (assuming all 10 hours is spent outdoors).	→ 4.5x10 ⁻¹⁰	0.00044
	Inhalation of volatile chemicals present in soil and groundwater	Measured soil gas and flux emission data (maximum) collected in areas relevant to the assessment of Block 2	→ 2.2 m ³ air per hour outdoors (assuming all 10 hours is spent outdoors).	→ 5.2x10 ⁻⁸	0.0089
	Inhalation of volatile emissions derived from open drains	Measured air concentrations adjacent to the drains.	→ 2.2 m ³ air per hour outdoors (assuming all 10 hours is spent adjacent to drains).	→ 2.6x10 ⁻⁴	3.2
	Ingestion and dermal contact with chemicals in soil	Soil concentrations from available data. Maximum value from available data from soil (gardening).	→ Ingestion of 25 mg of soil per day by adults. Once ingested it is assumed that 100% is absorbed. When outdoors it is assumed that the hands, forearms and lower legs get dirty each day (4580 cm ² of skin). Once dirty it is assumed that 0.51 mg of soil adheres to each cm ² of skin. Assume an adult will wash at the end of each day with 12 hours of the day dirty.	→ 4.8x10 ⁻⁸	0.21
TOTAL RISK (all exposures with average risk from ephemeral and permanent ponds)				3x10⁻⁴	3

5.2 Identification of Risk Issues

Based on the preliminary calculation of risk relevant to the potential development of Block 2 presented in Tables 5 and 6 the following issues have been identified that warrant consideration within the RAP to reduce the level of exposure and risk:

5.2.1 Soil

Potential risks to workers associated with direct exposure (ingestion and dermal contact) to maximum concentrations of COPC identified in soil assuming development within Block 2 occurs with no removal of hot-spots or capping are unacceptable.

With respect to the calculations presented and potential for exposure, the following is noted:

- Calculated non-threshold risks (3×10^{-7}) are considered acceptable ($< 1 \times 10^{-5}$).
- Calculated threshold risk (HI = 1) is equal to the adopted HI considered to be acceptable (HI=1). While it equals the target HI, the contribution of risk derived from exposures to soil is high and should be reduced. The calculated HI is dominated by exposure to mercury (82%) and lead (15%), where the following can be noted:
 - Elevated concentrations of mercury have been identified in soil within the central eastern portion of Block 2 at location TP50 (1233 mg/kg). No other samples located within Block 2 have reported concentrations of mercury that exceed the adopted NEPM HIL (75 mg/kg). The area in the vicinity of TP50 were subject to further delineation investigations by URS in 2006 that suggest that the elevated concentration reported is not widespread (maximum concentration reported in 2006 adjacent to TP50 was 5.3 mg/kg) and are likely to be associated with a localised hot-spot.
 - Elevated concentrations of lead were reported within a stockpile sample located in the central portion of the Block 2, SP50 (2220 mg/kg). No other samples located within Block 2 have reported concentrations of lead that exceed the adopted NEPM HIL (1500 mg/kg).
- In addition to the calculated risks to human health, asbestos has been identified in soil located within Block 2. The presence of asbestos fibres in the soil within Block 2 requires risk management measures to be undertaken.

While the potential for elevated exposures have been identified to some key COPC identified in soil (mercury, lead and asbestos) within Block 2, exposures may be reduced by the removal of hot-spots (including soil affected by asbestos fibres). In addition, should soil removal not be undertaken the potential for exposure could be addressed by the placement of clean fill or capping across the area (sufficiently deep to ensure appropriate management of asbestos). This would essentially eliminate exposures to the key COPC identified in soil within Block 2 subject to the implementation of an appropriate site management plan.



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5.2.2 Vapour Migration from Subsurface Sources

Potential risks to workers associated with inhalation of volatile COPC that may be present in emissions to air from the surface of the ground, overlying shallow groundwater and/or soil impacts, assuming development within Block 2 with no additional capping are considered to be below target risk values and acceptable. The contribution to the total risk calculated is sufficiently low that risk mitigation measures are not warranted.

5.2.3 Emissions to Air from Springvale Drain

Potential risks to workers associated with inhalation of volatile COPC that may be present in emissions to air derived from Springvale Drain are above target risk values. Potential exposures derived from emissions from Floodvale Drain are lower and below target risk values.

With respect to the emissions from Springvale Drain the relative contributions of COPC and risk mitigation measures are as discussed for Block 1 in Section 4.2.3.

6. Summary of Risk Issues

The following table presents a summary of the risk issues associated with the development of Block 1 and Block 2 of the Southlands property. The table presents the risk issues identified and mitigation measures to be addressed within the RAP or other aspects of the proposed development (details not yet complete).

Potential for Exposure	Issues Identified	Risk Mitigation Measures
All Areas		
Intrusive workers in all areas including the maintenance of GTP infrastructure	Potential for elevated exposures to chemicals in shallow groundwater and inhalation of volatile chlorinated chemicals (particularly within any confined spaces)	All works to be undertaken under site management plan that ensures appropriate safe work practices
Workers involved in maintenance of Springvale and Floodvale Drains	Potential for elevated exposures to chemicals in surface water and inhalation of volatile emissions	All works to be undertaken under site management plan ensures appropriate safe work practices
Block 1		
Elevated concentrations identified in soil and sediment (ephemeral ponds)	Potential for elevated exposure by workers on the site, including gardening activities (ingestion and dermal contact)	Removal of hot-spots and/or capping of area (also required to address vapour migration issues)
Inhalation of volatile chemicals in outdoor air (emissions from subsurface soil and groundwater)	No issues identified for outdoor air	
Inhalation of volatile chemicals within buildings (emissions from subsurface soil and groundwater)	Potential for elevated exposure to volatile chlorinated chemicals, present in shallow groundwater, that may accumulate in buildings (including buildings associated with GTP infrastructure)	If buildings are constructed in the area sub-floor venting, use of a vapour barrier (beneath the building foundations) or placement of capping (0.5-1m clay) required
Exposure to chemicals identified in ponds (permanent and ephemeral)	Potential for elevated exposures during maintenance of the existing ponds	If ponds are to remain on the site then works must be undertaken in accordance with a site management plan that ensures appropriate safe work practices. If ponds are lined or filled in then exposure should be effectively eliminated. If other changes (further excavation or increasing size) are to occur, then implications with respect to risk should be reviewed.
Inhalation of volatile chlorinated chemicals associated with emissions to air from Springvale Drain	Potential for significant exposures by all users of the site	Surface water concentrations within the drain to be reduced such that air concentrations reduce by 100 to 1000 fold. This may require a reduction in the discharge of shallow groundwater into Springvale Drain.
Block2		
Elevated concentrations (including asbestos) identified in soil	Potential for elevated exposure by workers on the site(ingestion and dermal contact)	Removal of hot-spots and/or capping of area (appropriate capping required to address asbestos issues). Where capping is used a site management plan will need to be developed.
Inhalation of volatile chemicals in outdoor air and within buildings (emissions from subsurface soil and groundwater)	No issues identified	
Inhalation of volatile chlorinated chemicals associated with emissions to air from Springvale Drain	Potential for significant exposures by all users of the site	Surface water concentrations within the drain to be reduced such that air concentrations reduce by 100 to 1000 fold. This may require a reduction in the discharge of shallow groundwater into Springvale Drain.



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Yours sincerely,
URS AUSTRALIA PTY LTD

Jackie Wright
Principal Environmental Scientist

Martin Howell
Senior Principal

Attachments

Appendix A
Summary of Data and Identification of COPC

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Appendix A

Summary of Data and Identification of COPC

A1.1 Introduction

This Appendix presents a summary of the available data relevant to the assessment of potential risks to human health on the Southlands site. The data has been reviewed to identify chemicals of potential concern (COPCs) that require detailed consideration within the HHRA.

A2.1 Identification of COPCs

A2.1.1 Soil and Sediment

Soil and sediment concentrations have been screened against the following guidelines to identify COPC for evaluation in the HHRA:

- *National Environment Protection Measure (NEPM)*, Assessment of Site Contamination, 1999, *Schedule B1, Health Based Soil Investigation Levels*, Level F - Industrial Sites (Table 5A); and
- *NSW EPA Guidelines for Assessing Service Station Sites*, 1994, Table 3 - Human Health Levels for BTEX and TPH compounds in soils (no HILs defined in NEPM, 1999).

Where guidelines are not available from the above sources for specific chemicals, screening levels have been obtained from the following sources. These sources are from the United States and while not recognised in Australia, the values provide a screening level for a wide range of chemicals in soils for the purpose of identifying COPC:

- *Region IX Preliminary Remediation Goals, 2004*. US EPA Region IX Preliminary Remediation Goals (PRGs) are conservative risk-based values for soil, tap water and air. The values presented for industrial soil (considering exposure via ingestion, dermal contact, inhalation of particulates and inhalation of volatiles, based on target risk levels of 10^{-6} for carcinogenic effects and 1 for non-carcinogenic effects) have been considered within this assessment.
- *Region III Risk Based Concentrations, 2006*. US EPA Region III Risk Based Concentrations (RBC) have been used where there are no values available from the above sources. The RBC are conservative risk-based values for soil, tap water and air. The values presented for industrial soil (considering exposure via ingestion, dermal contact, inhalation of particulates and inhalation of volatiles, based on target risk levels of 10^{-6} for carcinogenic effects and 1 for non-carcinogenic effects) have been considered within this assessment.

For some PAH compounds, no guidelines are available and hence soil screening levels have been adopted on the basis of considering other PAH compounds as surrogates. The surrogate compounds have been selected based on consideration of toxicity equivalent factors (TEFs) discussed and presented by

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Fitzgerald (1998)¹. These are noted where considered. Some other chemicals also have no data available and hence surrogate compounds have also been identified based on the similarity of the compounds and likelihood that the surrogate selected is more toxic than the chemical for which no data is available.

It is noted that the adopted screening levels are relevant for commercial/industrial use of the site, consistent with proposed development and land use of the site.

A2.1.2 Groundwater and Surface Water

The screening level guidelines adopted for the assessment of groundwater (where relevant) and surface water data are:

- ***Australian Drinking Water Guidelines (ADWG), 2004.*** The National Health and Medical Research Council (NHMRC) and the Agriculture and Resource Management Council of Australia and New Zealand have developed the Australian Drinking Water Guidelines. The guidelines provide health-based and aesthetic values for a range of micro-organisms, physical quality, inorganic chemicals, organic chemicals, radiological quality and pesticides. The health-based guideline values, which have been used to identify COPC in the groundwater, are concentrations, which based on present knowledge, do not result in any significant risk to the health of a consumer of the water over a lifetime. This approach is conservative for the assessment of groundwater, as groundwater in the area is not used as a source of drinking water. These guidelines are more extensive than the ANZECC 2000 Guidelines for Recreational Water Quality and Aesthetics (relevant to lower levels of exposure than drinking water) and hence have been used in preference to the recreational guidelines.
- ***World Health Organisation Drinking Water Guidelines (WHO DWG), 2004 (and rolling revisions).*** The WHO have also developed drinking water guidelines using the same approach as in the ADWG. The health-based guideline values, which have been used to identify COPC in the groundwater, are concentrations, which based on present knowledge, do not result in any significant risk to the health of a consumer of the water over a lifetime.
- ***Region IX Preliminary Remediation Goals (PRGs), 2004.*** Where no guideline value was available from the above sources, the US EPA Region IX Preliminary Remediation Goals (PRGs) have been used. The PRGs are conservative risk-based values for soil, tap water and air. It should be noted that the PRGs are currently not recognised in Australia. However, the PRGs have been used in this assessment to provide a screening level for the purpose of identifying the COPC within groundwater for further assessment.

¹ Fitzgerald J., 1998. *The benchmark dose approach and health-based investigation level for polycyclic aromatic hydrocarbons (PAHs)*, presented in Contaminated Sites Monograph Series, No. 7, 1998.

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A2.1.3 Ambient Air

Numerous guidelines exist for VOCs in air. Some guidelines are designed to protect individuals from chronic or long term exposure, whilst others are designed to protect workers during an 8 hour working day. For the purpose of this assessment, the focus is on long term exposure, as this is considered to best represent potential exposures by residents.

The guidelines relevant for VOCs in ambient air are contained in the National Environment Protection Measure (NEPM) Air Investigation Levels (NEPC, 2004), however, these investigation levels are limited with respect to the specific compounds included. Where a NEPM investigation level was not available for individual VOCs detected, reference has been made in the first instance to World Health Organisation (WHO) ambient air guidelines and then the US EPA Region IX (2004) Preliminary Remediation Goals (PRG) for ambient air followed by alternate US guidelines such as those established by the Californian EPA. A discussion of these guidelines is provided below.

NEPC Air Toxics Measure

In June 1998, the National Environment Protection Council (NEPC) released a National Environment Protection Measure (NEPM) for Ambient Air Quality, setting out national standards and investigation levels for criteria pollutants. In addition, air investigation levels based on a number of averaging times for high priority VOCs were established in 2004. It should be noted however, that these guidelines are designed for use as regional goals and are not intended to be used as near-source or site boundary criteria from operating facilities with known air emissions.

WHO Air Quality Guidelines

Where individual VOCs detected during the air sampling did not have NEPC Air guidelines, the WHO air quality guidelines 2000 and 2000b have been referenced. In some cases air quality goals have been updated by the WHO in relevant review documents (available from the WHO). The WHO Air Quality Guidelines provides guidelines for non cancer effects and carcinogenic effects for a specific averaging time. The selection of the WHO guidelines following those published by the NEPC is in accordance with the NEPM guideline hierarchy.

US EPA Region IX Preliminary Remediation Goals

Where individual VOCs detected during the air sampling have neither NEPM guidelines or WHO guidelines, US EPA Region IX (2004) Preliminary Remediation Goals (PRG) values have been used. The PRGs are conservative human health risk-based values for soil, tap water and ambient air. The values available for air represent concentrations that the general public may inhale every day for a lifetime without adverse health effects. It should be noted that the PRGs are currently not endorsed in Australia, however, the PRGs have been used in this assessment to provide a screening level for the purpose of identifying the presence of any individual VOCs in air that may be of concern with respect to human health.

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Californian Office of Environmental Health Hazard Assessment (OEHHA)

The Californian Office of Environmental Health Hazard Assessment (COEHHA) Chronic and Acute Reference levels have been adopted in the absence of NEPM, WHO, USEPA and TNRCC sources.

Use of Air Criteria

For the purpose of identifying COPC an annual average has been adopted. It is noted that the NEPM investigation levels based on an annual average consists of the mean of 24-hour data collected from monitoring locations and hence use of the annual average levels is considered relevant in comparison with the mean or average 24-hour concentrations. For samples collected over a shorter period of time such as an 8 hour period, the data collected over the 8 hours (considered representative of a workday) has been assumed to be representative of concentrations that may be present over the whole day. This approach has been adopted for consideration of other guidelines that are presented as annual averages such that they can be directly compared against the air samples collected. As a limited amount of data has been collected, the ambient air concentrations have not been averaged, with each concentration and the maximum reported compared directly against the annual average screening criteria. This provides a conservative approach to screening the ambient air data.

A2.2 Soil

Soil data have been collected by Woodward-Clyde (now URS) during the Stage 2 Survey in 1994 and by HLA in 2005. Additional soil data have been collected by URS in 2006 as part of further delineation works undertaken on the site.

Tables 1 and 2 present a listing of the maximum concentrations of chemicals detected in soils on the site associated with Block 1 (Table 2.1) and Block 2 (Table 2.2) of the property based on all data collected in 1994, 2005 and 2006 and comparison with the screening levels. Only chemicals detected above the laboratory limit of reporting (LOR) in at least one sample have been included in the following Tables.

Table 1 – Review of Soil Data and Identification of Chemicals of Potential Concern – Southlands Block 1

Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comments
arsenic	62	500 ¹	
cadmium	11	100 ¹	
chromium	1500	60 ¹	Maximum reported at TP31 with the only other exceedances reported at SS058, SS059 and SS051
copper	1380	5000 ¹	
lead	7670	1500 ¹	Maximum reported at TP127 with the only other exceedance reported at TP31
nickel	373	3000 ¹	
zinc	4200	35000 ¹	
mercury	176	75 ¹	Maximum reported at TP06, with five other

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Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comments
			sample locations report concentrations in excess of the adopted criteria
TPH and MAH			
TPH C ₆ -C ₉	12100	65 ²	TPH not assessed by Woodward Clyde. Exceedances identified within the south western corner of Block 1 in the vicinity of TP1, TP81 and TP82 and in the area of HA02
TPH C ₁₀ -C ₁₄	2440	1000 ²	
TPH C ₁₅ -C ₂₈	2870		
TPH C ₂₉ -C ₃₆	2720		
benzene	31.3	1 ²	BTEX was assessed by Woodward-Clyde, exceedances follow similar distribution noted for TPH in later investigations
toluene	4.8	130 ²	
ethylbenzene	141	50 ²	
m- & p-xylene	35	25 ²	
o-xylene	6.4		
isopropylbenzene	14.4	1977 ³	Region IX PRG
n-propylbenzene	70.2	240 ³	Region IX PRG
1,3,5-trimethylbenzene	1.5	70 ³	Region IX PRG
sec-butylbenzene	3.1	220 ³	Region IX PRG
1,2,4-trimethylbenzene	20	170 ³	Region IX PRG
Volatile Organic Compounds			
vinyl chloride	53	0.75 ³	Maximum reported at TP37, with other detections reported at TP29, SP056 and SP057
1,1-dichloroethene	2	413 ³	
trans-1,2-dichloroethene	106	234 ³	
1,1-dichloroethane	22.9	1738 ³	
cis-1,2-dichloroethene	165	146.3 ³	Maximum reported at TP29
1,2-dichloroethane (EDC)	4.5	0.603 ³	Maximum reported at TP29
trichloroethene (TCE)	95.1	0.11 ³	Maximum reported at TP105
1,1,2-trichloroethane	2	1.6 ³	Maximum and only exceedance of criteria in stockpile sample SP30
tetrachloroethene	90.8	3.4 ³	Maximum reported at TP105
1,1,2,2-tetrachloroethane	10.2	0.93 ³	Maximum reported at TP105
chloroform	3.9	0.47 ³	Maximum reported at HA01
chloromethane	0.05	160 ³	
Polynuclear Aromatic Hydrocarbons			
naphthalene	28.3	187.6 ³	
2-methylnaphthalene	51.9	187.6 (S)	No data available, assume naphthalene as surrogate
acenaphthylene	2.1	29219 (S)	Limited data available, assume acenaphthene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
acenaphthene	1.2	29219 ³	
phenanthrene	9.8	29126 (S)	Limited data available, assume pyrene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
anthracene	3.2	10000 ³	
fluoranthene	21.8	22000 ³	
pyrene	23.8	29126 ³	
benzo(a)anthracene	12	2.1 ³	
chrysene	10.4	210.9 ³	

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Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Comments
benzo(b)&(k)fluoranthene	11.2	2.1 ³	Based on lower value for group
benzo(a) pyrene	6.6	5 ¹	
indeno(1,2,3-c,d)pyrene	2.8	2.1 ³	
dibenz(a,h)anthracene	1	0.21 ³	
benzo(g,h,i)perylene	3.2	210.9 (S)	Limited data available, assume chrysene as surrogate – consistent with TEFs
PAHs (Sum of total)	106.4	100 ¹	Total PAH and individual PAHs exceed criteria at one location (TP16)
Phthalates			
di-n-butyl phthalate	1.8	62560 ³	
butyl benzyl phthalate	2	10000 ³	
bis(2-ethylhexyl) phthalate	43.1	123.1 ³	
Chlorinated Hydrocarbons			
hexachloroethane (HCE)	7.8	120 ³	
hexachlorobutadiene (HCBd)	326	22 ³	Maximum reported at HA02 with exceedance of criteria identified at HA01
pentachlorobenzene	7.3	490 ³	
hexachlorobenzene (HCB)	1170	1.1 ³	Maximum reported at HA02 with exceedance of criteria identified at HA01, HA03, SS049, HA006
1,3,5-trichlorobenzene	15	215 (S)	No data available, adopt data for 1,2,4-trichlorobenzene as surrogate
1,2,4,5-tetrachlorobenzene	35.6	180 ³	
1,3-dichlorobenzene	0.007	600 ³	
1,4-dichlorobenzene	0.017	7.9 ³	
1,2-dichlorobenzene	0.013	600 ³	
1,2,4-trichlorobenzene	0.094	215 ³	
tetrachlorobenzene	0.5	180 (S)	No data available, adopt data for 1,2,4,5-tetrachlorobenzene as surrogate
Others			
n-nitrosodiphenyl & diphenylamine	268	255503	Criteria for diphenylamine
dibenzofuran	1.5	15633	
carbazole	2.3	863	

1 – Soil criteria derived from NEPM HIL for commercial industrial land use

2 – NSW EPA Service Station Guidelines (1994)

3 – Region IX PRG for industrial soils

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

Shaded rows are chemicals where maximum concentration reported is greater than the adopted criteria

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Table 2 – Review of Soil Data and Identification of Chemicals of Potential Concern –
Southlands Block 2

Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Reference
arsenic	41	500 ¹	
cadmium	3	100 ¹	
chromium	114	60 ¹	Maximum detected in stockpile sample SP49, concentrations in stockpiles greater than soil samples
copper	288	5000 ¹	
lead	2220	1500 ¹	Maximum detected in stockpile sample SP50, concentrations in stockpiles greater than soil samples
nickel	52	3000 ¹	
zinc	2960	35000 ¹	
mercury	1233	75 ¹	Maximum reported at TP50, no other exceedances of criteria reported
Volatile Organic Compounds			
1,1-dichloroethene	0.07	413 ³	
tetrachloroethene	0.5	3.4 ³	
chloroethane	0.03	18000 ³	
Polynuclear Aromatic Hydrocarbons			
naphthalene	5.2	187.6 ³	
phenanthrene	2.5	29126 (S)	Limited data available, assume pyrene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
acenaphthene	0.9	29219 ³	
anthracene	1.1	10000 ³	
fluoranthene	5.2	22000 ³	
pyrene	6.1	29126 ³	
benzo(a)anthracene	3.6	2.1 ³	Not identified as COPC as total PAH meet NEPM HIL
chrysene	3.2	210.9 ³	
benzo(b)&(k)fluoranthene	7.7	2.1 ³	Based on lower value for group, not identified as COPC as total PAH meet NEPM HIL and screening value used is conservative.
benzo(a) pyrene	4	5 ¹	
indeno(1,2,3-c,d)pyrene	2.4	2.1 ³	Not identified as COPC as total PAH meet NEPM HIL
benzo(g,h,i)perylene	2.8	210.9 (S)	Limited data available, assume chrysene as surrogate – consistent with TEFs
PAHs (Sum of total)	39.5	100 ¹	
Phthalates			
di-n-butyl phthalate	0.5	62560 ³	
butyl benzyl phthalate	1.9	10000 ³	
bis(2-ethylhexyl) phthalate	10.4	123.1 ³	
Chlorinated Hydrocarbons			
hexachloroethane (HCE)	5.2	120 ³	
hexachlorobutadiene (HCBD)	7.7	22 ³	
pentachlorobenzene	0.12	490 ³	
hexachlorobenzene (HCB)	8.8	1.1 ³	Maximum reported from TP50 with other exceedance noted at SS037

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Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Reference
1,3,5-trichlorobenzene	1.9	215 (S)	No data available, adopt data for 1,2,4-trichlorobenzene as surrogate
1,2,4,5-tetrachlorobenzene	0.8	180 ³	
1,3-dichlorobenzene	0.028	600 ³	
1,4-dichlorobenzene	0.039	7.9 ³	
1,2-dichlorobenzene	0.016	600 ³	
1,2,4-trichlorobenzene	0.11	215 ³	
tetrachlorobenzene	0.006	180 (S)	No data available, adopt data for 1,2,4,5-tetrachlorobenzene as surrogate
Others			
n-nitrosodiphenyl & diphenylamine	8.4	25550 ³	Criteria for diphenylamine
bis(2-chloroethyl)ether	2.1	0.55 ³	Maximum and only exceedance of criteria at TP52

1 – Soil criteria derived from NEPM HIL for commercial industrial land use

2 – NSW EPA Service Station Guidelines (1994)

3 – Region IX PRG for industrial soils

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

Shaded rows are chemicals where maximum concentration reported is greater than the adopted criteria

In addition, it is noted that asbestos has been identified in soils within Block 2 and is therefore included as a COPC.

On the basis of the above the following COPC have been identified in soils at the site:

Block 1	Block 2
chromium lead; mercury TPH benzene ethylbenzene m-, p- and o-xylenes vinyl chloride cis-1,2-dichloroethene 1,2-dichloroethane (EDC) trichloroethene (TCE) 1,1,2-trichloroethane tetrachloroethene (PCE) 1,1,2,2-tetrachloroethane chloroform PAHs, namely benzo(a)pyrene, benzo(a)anthracene, benzo(b&k)fluoranthene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene hexachlorobutadiene (HCBD) hexachlorobenzene (HCB)	chromium lead mercury bis(2-chloroethyl)ether hexachlorobenzene (HCB) asbestos

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The data has not been further screened with respect to impacts at the surface (defined to be soil at depths 0 to 1m²) or at depth (defined as depths greater than 1m) within this preliminary assessment as it has been assumed that development of the site may result in the movement of soil across the site with the potential for soil at depth to be moved closer to the surface during such works.

A2.3 Ponds on Block 1

A number of ponds are present within Block 1. Four of the ponds are deep enough to be permanent features (they extend below the water table) with the remaining ponds ephemeral including some drainage lines. As the ponds were originally associated with paper pulp wastes, there is the potential for the water in the ponds to be odorous.

Development plans for Block 1 are not currently finalised and it is unclear what is proposed for the ponds present on Block 1. Hence the preliminary assessment has considered the most conservative approach where the ponds remain on the site and are accessible for the purpose of routine maintenance (weed control etc). Available data on water quality and sediment quality from the pond areas has been obtained from the following:

- Surface water samples collected by HLA in April 2005 from six locations within the permanent and ephemeral ponds. Note that the ponds were also sampled as part of the Stage 1 Survey (Woodward-Clyde, 1990) but were only analysed for general water quality parameters;
- Sediment data collected from a number of the ponds by Woodward-Clyde as part of the Stage 2 Survey (1996); and
- Sediment data collected by HLA in April 2005 from the same six locations where surface water samples were collected. Sediment samples were collected from two depths at each location.

Tables 3 and 4 present a summary of the sediment and surface water data available. Note that sediment data has been screened against the soil criteria adopted for industrial sites and surface water data has been screened against drinking water guidelines.

Table 3 – Review of Sediment Data and Identification of Chemicals of Potential Concern – Southlands Block 1

Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Reference
arsenic	58	500 ¹	
cadmium	2	100 ¹	
chromium	188	60 ¹	Maximum reported at SED06, 0.5-0.6 m, only one

² Surface soils have been defined as depth from the surface to 1m at this site. Typically surface soils are defined as 0-0.5m depth as per ANZECC (1992) however, as this site is to be redeveloped, it is considered likely that such works would involve excavation and mixing of soil within at least the top 1m of the site.

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Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Reference
			other exceedance noted at locations SD023 (0-0.15m) of 159 mg/kg. All other concentrations less than adopted criteria
copper	369	5000 ¹	
lead	285	1500 ¹	
nickel	49	3000 ¹	
zinc	2040	35000 ¹	
mercury	45.1	75 ¹	
TPH and MAH			
TPH C ₆ -C ₉	113	65 ²	Maximum concentrations reported by Woodward Clyde (TPH not reported by HLA) at locations SD018 and SD019 which is in the same ephemeral drain as SED06
TPH C ₁₀ -C ₁₄	498	1000 ²	
TPH C ₁₅ -C ₂₈	3460		
TPH C ₂₉ -C ₃₆	0		
benzene	0.6	1 ²	
toluene		130 ²	
ethylbenzene	1.7	50 ²	
m- & p-xylene	1.5	25 ²	
o-xylene	1.0		
1,2,4-trimethylbenzene	0.8	170 ³	
Volatile Organic Compounds			
vinyl chloride	384	0.75 ³	Maximum reported at location SED06 at depth 0.5-0.6m. Shallow sample (0.2-0.3m) reported other exceedance of 285 mg/kg
1,1-dichloroethene	0.5	413 ³	
trans-1,2-dichloroethene	13.4	234 ³	
1,1-dichloroethane	1.4	1738 ³	
cis-1,2-dichloroethene	302	146.3 ³	Maximum reported at SED06, 0.2-0.3 m
1,2-dichloroethane	29.8	0.603 ³	Maximum reported at SED06, 0.2-0.3 m
trichloroethene (TCE)	1050	0.11 ³	Maximum reported at SED06, 0.5-0.6 m
1,1,2-trichloroethane	115	1.6 ³	Maximum reported at SED06, 0.2-0.3 m
tetrachloroethene	7130	3.4 ³	Maximum reported at SED06, 0.5-0.6 m
1,1,2,2-tetrachloroethane	42.7	0.93 ³	Maximum reported at SED06, 0.2-0.3 m
hexachlorobutadiene	112	22 ³	Maximum reported at SED06, 0.2-0.3 m
chloroform	213	0.47 ³	Maximum reported at SED06, 0.2-0.3 m
chloromethane	0.05	160 ³	
carbon tetrachloride	0.07	0.55 ³	
Semivolatile Organic Compounds			
naphthalene	0.7	187.6 ³	
2-methylnaphthalene	1.2	187.6 (S)	No data available, assume naphthalene as surrogate
acenaphthene	0.9	29219 ³	
phenanthrene	8.3	29126 (S)	Limited data available, assume pyrene as surrogate – consistent with TEFs and non-carcinogenic classification of chemicals
anthracene	1.3	10000 ³	
fluoranthene	0.9	22000 ³	
pyrene	2.9	29126 ³	
benzo(a)anthracene	0.8	2.1 ³	
chrysene	0.8	210.9 ³	
di-n-butyl phthalate	2.3	62560 ³	
bis(2-ethylhexyl) phthalate	22.4	123.1 ³	
hexachloroethane (HCE)	1.5	120 ³	
pentachlorobenzene	0.01	490 ³	
hexachlorobenzene (HCB)	0.21	1.1 ³	
1,3,5-trichlorobenzene	0.06	215 (S)	No data available, adopt data for 1,2,4-trichlorobenzene as surrogate
1,2,4-trichlorobenzene	0.81	215 ³	
1,2,4,5-tetrachlorobenzene	0.6	180 ³	
1,3-dichlorobenzene	0.36	600 ³	
1,4-dichlorobenzene	1.1	7.9 ³	
1,2-dichlorobenzene	0.62	600 ³	
tetrachlorobenzene	0.55	180 (S)	No data available, adopt data for 1,2,4,5-tetrachlorobenzene as surrogate

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Chemicals Detected in Soils	Maximum Concentration Reported (mg/kg)	Screening Criteria (mg/kg)	Reference
Phenolic Compounds			
3- &4-methylphenol	0.5	3078 ³	Minimum guideline available for 3- or 4-methylphenol
2,4-dichlorophenol	7.1	1847 ³	
2,4,6-trichlorophenol	0.6	62 ³	

1 – Soil criteria derived from NEPM HIL for commercial industrial land use

2 – NSW EPA Service Station Guidelines (1994)

3 – Region IX PRG for industrial soils

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

Shaded rows are chemicals where maximum concentration reported is greater than the adopted criteria

Table 4 – Review of Surface Water Data from Ponds and Identification of Chemicals of Potential Concern – Southlands Block 1

Chemicals Detected in Soils	Maximum Concentration Reported in Ephemeral Pond (mg/L)	Maximum Concentration Reported in Permanent Ponds (mg/L)	Screening Criteria (mg/L)	Reference
arsenic	0.193	0.003	0.007 ¹	Maximum concentration reported at SW06 (ephemeral drainage area) with all other concentrations reported lower than adopted criteria
cadmium	0.0117	Not detected	0.002 ¹	
chromium	0.665	Not detected	0.05 ¹	
copper	1.14	0.003	2 ¹	Maximum concentration reported at SW06 (ephemeral drainage area) with all other concentrations reported lower than adopted criteria
lead	1.48	0.004	0.01 ¹	
nickel	0.424	0.002	0.02 ¹	
zinc	11.9	0.026	11 ³	
mercury	0.0003	0.0005	0.001 ¹	
Volatile Organic Compounds				
vinyl chloride	14.2	0.3	0.0003 ¹	Maximum reported at SW06 (ephemeral drainage area) with elevated concentrations also reported at SW05 (ephemeral drainage area) and SW02 and SW04 which are permanent ponds
1,1-dichloroethene	0.135	Not detected	0.03 ¹	Maximum and only detection reported at SW06 (ephemeral drainage area)
trans-1,2-dichloroethene	0.332	0.013	0.06 ¹	Maximum reported at SW06 (ephemeral drainage area) with elevated concentrations also reported at SW05 (ephemeral drainage area) and SW02 and SW04 which are permanent ponds
1,1-dichloroethane	0.610	0.006	70 ³	
cis-1,2-dichloroethene	13.9	0.166	0.06 ¹	Maximum reported at SW06 with exceedances reported in all other locations
1,1,1-trichloroethane	0.028	Not detected	2 ²	Maximum and only detection reported at SW06 (ephemeral drainage area)
carbon tetrachloride	Not detected	0.013	0.003 ¹	Maximum and only detection reported at SW04
1,2-dichloroethane	3.11	0.019	0.003 ¹	Maximum reported at SW06 (ephemeral drainage area) with elevated concentrations also reported at SW05 (ephemeral drainage area) and SW02 and SW04 which are permanent ponds
trichloroethene (TCE)	18.0	0.115	0.02 ²	Maximum reported at SW06 with exceedances reported in most other locations
1,1,2-trichloroethane	6.1	0.019	0.0002 ³	Maximum reported at SW06 (ephemeral

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Summary of Data and Identification of COPC

Chemicals Detected in Soils	Maximum Concentration Reported in Ephemeral Pond (mg/L)	Maximum Concentration Reported in Permanent Ponds (mg/L)	Screening Criteria (mg/L)	Reference
				drainage area) with elevated concentrations also reported at SW05 (ephemeral drainage area) and SW02 and SW04 which are permanent ponds
tetrachloroethene	40.6	0.28	0.05 ¹	Maximum reported at SW06 with exceedances reported in most other locations
1,1,2,2-tetrachloroethane	0.645	0.034	0.00006 ³	Maximum reported at SW06 (ephemeral drainage area) with elevated concentrations also reported at SW05 (ephemeral drainage area) and SW02 and SW04 which are permanent ponds
hexachlorobutadiene	0.136	0.002	0.0007 ¹	Maximum and only detection reported at SW06 (ephemeral drainage area)
chloroform	11.8	0.051	0.25 ¹	Maximum reported at SW06 with exceedances reported at two other locations
Semivolatile Organic Compounds				
di-n-butyl phthalate	0.006	Not detected	1.5 (S)	Value not available, hence data for di-n-octyl phthalate used as surrogate
pentachlorobenzene	0.005	Not detected	0.03 ³	
Chlorinated Hydrocarbons				
1,4-dichlorobenzene	0.007	Not detected	0.04 ¹	
1,2-dichlorobenzene	0.004	Not detected	1.5 ¹	
chlorobenzene	0.064	Not detected	0.3 ¹	
Phenolic Compounds				
phenol	0.006	Not detected	11 ³	
3- & 4-methylphenol	0.044	0.013	1.8 ³	
2,4-dichlorophenol	0.077	0.006	0.2 ¹	
2,4,6-trichlorophenol	0.010	Not detected	0.02 ¹	

1 – Australian Drinking Water Guideline (2004)

2 – WHO Drinking Water Guideline (2004) and rolling revisions

3 – Region IX PRG for tap water

(S) - criteria based on surrogate compound (no criteria available for chemical detected)

Shaded rows are chemicals where maximum concentration reported is greater than the adopted criteria

On the basis of the above the following COPC have been identified in surface water and sediments on Block 1:

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Summary of Data and Identification of COPC

Block 1 – Surface Water in Ponds	Block 1 – Sediments in Ponds
arsenic cadmium chromium lead nickel zinc vinyl chloride 1,1-dichloroethene trans-1,2-dichloroethene cis-1,2-dichloroethene carbon tetrachloride 1,2-dichloroethane (EDC) trichloroethene (TCE) 1,1,2-trichloroethane tetrachloroethene (PCE) 1,1,2,2-tetrachloroethane chloroform hexachlorobutadiene (HCBD).	chromium TPH vinyl chloride cis-1,2-dichloroethene 1,2-dichloroethane (EDC) trichloroethene (TCE) 1,1,2-trichloroethane tetrachloroethene (PCE) 1,1,2,2-tetrachloroethane chloroform hexachlorobenzene (HCB).

A2.4 Groundwater

Impacted groundwater is present beneath Block 1 and Block 2 of the Southlands site. The main contaminants present in groundwater are derived from the central plumes dominated by EDC, PCE, TCE and VC and the southern plumes dominated by CTC, PCE, TCE and VC, however a range of other chemicals have also been reported in groundwater.

As discussed in the HHRA, the only exposure pathway associated with the presence of impacts in groundwater that is considered complete is the inhalation of volatile chemicals that may be present at the top of the groundwater table, the vapours of which may migrate through overlying soils and into buildings, outdoor air and excavations. Hence the identification of COPC considered relevant to the presence of impacts in groundwater and the inhalation pathway have been identified using soil gas and flux emissions data rather than groundwater.

A2.5 Soil Gas and Flux Emission Rates

Flux emissions and some soil gas data have been collected from the southlands property by URS as part of a number of sampling programs since 1995. These include the following (refer to Figure XXX for locations):

- 1995 air emissions sampling undertaken as part of the Stage 2 Health Risk Assessment (and reported in Appendix D of the Stage 2 Health Risk Assessment Report). Both flux emissions and soil gas data were collected. Samples collected that are relevant to the assessment of the Southlands property

Appendix A

Summary of Data and Identification of COPC

include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05) and AS6 (AS06) within Block 2;

- 1996 air emissions sampling program carried out prior to the commencement of the Stage 3 monitoring rounds. The data from this monitoring is presented in the Air Emissions Report, Woodward-Clyde 1997. Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05) and AS6 (AS06) within Block 2;
- 1998 air emissions sampling round completed as part of the Stage 3 monitoring program (reported in the progress report WCIE-233, March 1999). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor), AS03 (to the north of Block 1 in the Nant St tank farm) and AS5 (AS05) and AS6 (AS06) within Block 2
- 1999 air emissions sampling round completed as part of the Stage 3 monitoring program (reported in the progress report WCIE-298, December 1999). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor), AS3 (AS03) (to the north of Block 1 in the Nant St tank farm) and AS5 (AS05), AS6 (AS06) and AS13 (AS13) within Block 2;
- 2001 and 2002 air emissions sampling round completed as part of the Stage 3 monitoring program (reported in the Air Emissions Sampling Program July 2001 Report, URS 2001 and Air Emissions Sampling Program November 2002 Report, URS 2002). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05), AS6 (AS06) and AS13 (AS13) within Block 2;
- 2004 and 2005 air emissions sampling round completed as part of the Stage 3 monitoring program (reported in Air Emissions Sampling Program March 2004 Report, URS 2004 and Air Emissions Sampling Program July 2005 Report, URS 2006). Samples collected that are relevant to the assessment of the Southlands property include AS9 (AS09) (adjacent to Block 1 in the rail corridor) and AS5 (AS05), AS6 (AS06), AS13 (AS13) and AS15 (AS15) within Block 2.
- 2005 and 2006 sampling of soil gas from one location on the southern portion of Southlands (Block 2) at location AS86 (SG01) (reported in Soil Gas Sampling - Southlands Report, URS 2006).
- 2006 sampling of flux emissions and soil gas (limited locations due to the presence of shallow groundwater) from a number of locations on Southlands. Flux emissions data were collected from locations AS65 (FH02), AS66 (FH03), AS67 (FH04), AS68 (FH05) (located on Block 1) and AS69 (FH07) and AS70 (FH08) (located on Block 2). Soil gas data were collected from locations AS71 (SG09) and AS72 (SG10) (located on Block 2).

Tables 5 and 6 present a summary of the chemicals reported in flux emissions data relevant to the assessment of Southlands. Table 7 presents a summary of the soil gas data relevant to the assessment of Southlands.

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Summary of Data and Identification of COPC

As there are no screening criteria that are relevant to measured flux emission rates, COPC have been identified as those chemicals detected in at least one sample.

With respect to soil gas concentrations, the reported concentration has been screened against the adopted ambient air criteria. This provides a conservative methodology for identifying COPC in soil gas as the concentrations present within the soil profile would not be inhaled directly. There would be further diffusion, degradation and dilution prior to inhalation.

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Summary of Data and Identification of COPC

Table 5- Flux Emission Rates – Relevant to the Assessment of Southlands Block 1

Location	Sample Date	Chemical Detected – Flux Emission Rate (µg/min/m ²)																					
		vinyl chloride	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	1,1,1,2-tetrachloroethane	trichlorofluoromethane	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	1,1-dichloroethane	benzene	toluene	ethylbenzene	xylene	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	p-isopropyltoluene	naphthalene
AS9	May '95	nd	10.5	2.5	13	nd	0.5	nd	0.1	nd													
	Oct '96	2.38	0.73	0.089	nd	1.37	0.64	0.041	0.14	nd													
	Dec '98	nd	nd	nd	nd	nd	0.4	nd	0.079	nd													
	Aug '99	nd	nd	0.011	nd	0.34	0.34	0.026	0.068	0.38													
	Jul '01	nd	0.018*	nd	nd	nd	0.01	nd	0.014	0.024													
	Nov '02	nd	0.1*	nd	nd	nd	5	0.19	39	nd													
	Mar '04	nd	nd	nd	nd	nd	0.11	nd	0.25	nd													
	Jul '05	nd	0.296	nd	nd	0.12	nd	nd	nd	nd													
AS3	Dec '98	0.021	nd	nd	0.016	0.14	0.014	nd	0.018	nd													
	Aug '99	nd	nd	nd	nd	0.2	0.99	nd	0.012	nd													
AS65	Aug '06	nd	nd	nd	nd	0.113	0.25	nd	0.094	0.035	nd	0.18	0.63	nd	nd	0.047	0.44	0.025	0.13	nd	0.069	nd	0.047
AS66	Aug '06	nd	nd	nd	nd	1.38 ^A	0.055	nd	0.046	nd	nd	nd	nd	nd	nd	0.097	0.13	nd	0.12	nd	0.088	nd	3.2 ^A
AS67	Aug '06	nd	nd	0.066	0.022	0.082	0.44	nd	0.16	nd	nd	nd	nd	nd	nd	0.047	0.38	0.016	0.085	nd	0.047	nd	0.06
AS68	Aug '06	nd	nd	nd	nd	2.8 ^A	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.037	0.11	nd	0.069	nd	nd	nd	1.7
Max		2.38	10.5	2.5	13	2.8	5	0.19	39	0.38		0.18	0.63	nd	nd	0.97	0.44	0.025	0.13	nd	0.088	nd	3.2

Appendix A

Summary of Data and Identification of COPC

Table 6- Flux Emission Rates – Relevant to the Assessment of Southlands Block 2

		Chemical Detected – Flux Emission Rate (µg/min/m ²)																					
		vinyl chloride	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	1,1,2,2-tetrachloroethane	trichlorofluoromethane	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	1,1-dichloroethane	benzene	toluene	ethylbenzene	xylenes	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	p-isopropyltoluene	naphthalene
AS5	May '95	nd	nd	1.9	nd	nd	nd	nd	nd	nd													
	Oct '96	nd	0.22	nd	nd	nd	nd	nd	nd	nd													
	Dec '98	nd	0.039	nd	0.013	0.038	0.012	nd	0.017	nd													
	Aug '99	nd	nd	0.018	0.015	0.44	0.063	0.013	0.054	nd													
	Jul '01	nd	nd	nd	nd	nd	0.048	nd	nd	nd													
	Mar '04	nd	nd	nd	nd	nd	nd	nd	0.047	nd													
	Jul '05	nd	0.26	nd	nd	nd	nd	nd	nd	nd													
AS6	May '95	nd	nd	0.6	nd	nd	nd	nd	nd	nd													
	Oct '96	0.057	0.26	nd	nd	nd	0.022	nd	0.019	nd													
	Dec '98	nd	nd	nd	0.055	0.057	0.01	nd	nd	nd													
	Aug '99	nd	0.02	nd	nd	0.063	nd	nd	0.017	nd													
	Jul '01	nd	nd	nd	0.016	nd	0.016	nd	nd	nd													
	Nov '02	nd	0.044*	nd	nd	nd	nd	nd	nd	nd													
	Mar '04	nd	nd	nd	nd	1.44	nd	0.027	0.622	nd													
	Jul '05	nd	1.6	nd	nd	nd	nd	nd	nd	nd													
AS13	Aug '99	nd	nd	nd	nd	0.22	0.013	nd	nd	nd													
	Jul '00	nd	0.072	nd	0.024	0.1	0.048	nd	0.016	nd													
	Nov '02	nd	0.022*	nd	nd	nd	nd	nd	nd	nd													
	Mar '04	nd	nd	nd	nd	nd	nd	nd	nd	nd													
	Jul '05	nd	0.48	nd	nd	nd	nd	nd	nd	nd													
AS15	Mar '04	nd	nd	nd	nd	nd	nd	nd	0.047	nd													
	Jul '05	nd	0.26	nd	nd	nd	nd	nd	nd	nd													
AS69	Aug '06	nd	nd	nd	nd	0.044	0.038	nd	nd	nd	nd	nd	nd	nd	0.085	0.95	0.091	0.44	0.038	0.19	0.016	0.057	
AS70	Aug '06	nd	nd	nd	nd	0.69	0.069	nd	nd	nd	0.55	nd	nd	0.12	0.27	0.15	0.22	0.032	0.18	nd	0.092	nd	0.92

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Summary of Data and Identification of COPC

		Chemical Detected – Flux Emission Rate ($\mu\text{g}/\text{min}/\text{m}^2$)																					
		vinyl chloride	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	1,1,2-trichloroethane	tetrachloroethene (PCE)	1,1,1,2-tetrachloroethane	trichlorofluoromethane	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	1,1-dichloroethane	benzene	toluene	ethylbenzene	xylenes	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	p-isopropyltoluene	naphthalene
Max		0.057	1.6	1.9	0.055	1.4	0.069	0.027	0.62	nd	0.55	nd	nd	0.12	0.27	0.15	0.95	0.091	0.44	0.038	0.19	0.016	0.92

* Emission rate of methylene chloride (dichloromethane) which is reported is at a level, which is below the emission rate of methylene chloride detected in the field blank.

< 0.2 Values marked with "<" are those reported to be less than the analytical limit of reporting.

0.06 Values in bold were detected during analysis.

Appendix A

Summary of Data and Identification of COPC

Table 7- Soil Gas Concentrations - Relevant to the Assessment of Southlands

Location	Sample Depth (m)	Sample Date	Chemical Detected – Soil Gas Concentration (µg/m ³)									
			vinyl chloride	dichloromethane	chloroform	1,2-dichloroethane (EDC)	trichloroethene (TCE)	tetrachloroethene (PCE)	trans-1,2-dichloroethene	cis-1,2-dichloroethene	1,1-dichloroethene	
Block 1												
AS9	0.6	May '95	200	5.1	160	nd	91	4.9				
Block 2												
AS5	0.6	May '95	nd	nd	9.2	nd	nd	nd				
AS6	0.6	May '95	nd	nd	20	nd	nd	nd				
AS86	1.1	Dec '05	205	32	5.9	343	46	195	282	602	852	
AS86	1.1	Apr '06	nd	nd	nd	26	nd	nd	nd	nd	nd	nd
AS72	0.47	Aug '06	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Max Block 2			205	32	20	343	46	195	282	602	852	
Screening Level			1 ²	4.1 ¹	2.4 ²	0.36 ²	2.3 ²	200 ²	73 ³	37 ³	200 ²	

1 Air Investigation Level as per NEPM 2004, based on annual average

2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.

3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.

4 Chronic Reference Exposure Level established by OEHHA

A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group

B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group

Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs

Appendix A

Summary of Data and Identification of COPC

Table 7 (continued) – Soil Gas Concentrations – Relevant to the Assessment of Southlands

Location	Sample Depth (m)	Sample Date	Chemical Detected – Soil Gas Concentration ($\mu\text{g}/\text{m}^3$)											
			benzene	toluene	ethylbenzene	xylenes	1,3,5-trimethylbenzene	1,2,4-trimethylbenzene	acetone	hexane	cyclohexane	4-ethyltoluene	ethanol	heptane
Block 1														
AS9	0.6	May '95												
Block 2														
AS5	0.6	May '95												
AS6	0.6	May '95												
AS86	1.1	Dec '05												
AS86	1.1	Apr '06	15	300	24	290	49	150	780	54	1500	120	61	310
AS72	0.47	Aug '06	4.3	5.4	1.3	9.0	1.3	nd	nd	nd	nd	nd	nd	nd
Max Block 2			15	300	24	290	49	150	780	54	1500	120	61	310
Screening Level			9.6¹	377¹	22000²	870¹	6.2³	6.2³	3300³	209³	2605³	377^B	100000⁴	209^A

1 Air Investigation Level as per NEPM 2004, based on annual average

2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1×10^{-6} is adopted.

3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1×10^{-6} is adopted.

4 Chronic Reference Exposure Level established by OEHHA

A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group

B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group

Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs

Appendix A

Summary of Data and Identification of COPC

On the basis of the flux emissions and soil gas data collected, the following COPC have been identified have been identified on Block 1 and Block 2.

Block 1	Block 2
vinyl chloride	vinyl chloride
dichloromethane	dichloromethane
chloroform	chloroform
carbon tetrachloride	carbon tetrachloride
1,2-dichloroethane (EDC)	1,2-dichloroethane (EDC)
trichloroethene (TCE)	trichloroethene (TCE)
1,1,2-trichloroethane	1,1,2-trichloroethane
tetrachloroethane (PCE)	tetrachloroethane (PCE)
1,1,2,2-tetrachloroethane	1,1-dichloroethene
<i>cis</i> - and <i>trans</i> -1,2-dichloroethene	1,1-dichloroethane
benzene	<i>cis</i> - and <i>trans</i> -1,2-dichloroethene
toluene	trichlorofluoromethane (freon 11)
ethylbenzene	benzene
xylenes	toluene
1,2,4-trimethylbenzene	ethylbenzene
naphthalene	xylenes
	1,2,4- and 1,3,5-trimethylbenzene
	TPH C6-C9 aromatics (including isopropyltoluene)
	TPH C6-C9 aliphatics (including heptane)
	naphthalene

A2.6 Ambient Air Adjacent to Springvale Drain

Air samples have been collected at locations adjacent to Springvale Drain and Floodvale Drain to provide a measure of potential ambient air concentrations that may be associated with emissions to air from the surface water within Springvale Drain. The air samples have been collected using Summa canisters over an 8 hour period on three occasions:

- 1st August 2006 where 2 samples were collected adjacent to Springvale Drain (AS73 and AS74) (SPAM1 and SPAM2) and 2 samples were collected adjacent to Floodvale Drain (AS75 and AS76) (FDAM1 and FD2);
- 19th September 2006 where 1 sample (AS87) was collected adjacent to Springvale Drain near the location where the highest concentration was reported in August 2006;
- 29th September 2006 from location AS87; and

Appendix A

Summary of Data and Identification of COPC

- 5th October 2006 from location AS87.

The following presents a summary of the chemicals detected in ambient air samples collected on the Southlands property. The data presented in the following table presents only those chemicals detected (refer to laboratory reports for all chemicals and limits of reporting) and the maximum reported from the primary or duplicate samples (where collected).

The measured ambient air concentrations have been screened against the adopted ambient air guidelines listed in Section XXX to identify COPC.

Appendix A

Summary of Data and Identification of COPC

Table 8 – Air Concentrations Adjacent to Springvale and Floodvale Drains

Location	Sample Date	Chemical Detected – Concentration (µg/m ³)												
		freon 12	vinyl chloride	chlormethane	bromomethane	freon 11	dichloromethane	chloroform	carbon tetrachloride	1,2-dichloroethane (EDC)	trichloroethene (TCE)	tetrachloroethene (PCE)	cis-1,2-dichloroethene	1,1-dichloroethane
Floodvale Drain														
AS75	Aug '06	2.8	0.44	1.2	0.71	1.4				7				
AS76	Aug '06	2.4		1.5		1.4	2.4			3.3				
Max		2.8	0.44	1.5	0.71	1.4	2.4			7				
Springvale Drain														
AS74	Aug '06		99					59	53	1800	100	160	51	
AS73	Aug '06	2.6	4.4	1.4	1.4	1.7		2.4	3.8	96	4.1	1.8	3.0	96
AS87	19 Sept '06		89					94	77	2400	180	160	80	6.2
AS87	29 Sept '06	3.2	20	1.9				22	14	480	42	22	26	
AS87	Oct '06	3.1	1.4			1.7	1.2							
Max		3.1	99	1.9	1.4	1.7	1.2	94	77	2400	180	160	80	96
Screening Level		210³	1²	95³	5.2³	730³	4.1¹	2.4²	6.1²	0.36²	2.3²	200²	37³	521³

1 Air Investigation Level as per NEPM 2004, based on annual average

2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.

3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.

4 Chronic Reference Exposure Level established by OEHHA

A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group

B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group

Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs

Appendix A

Summary of Data and Identification of COPC

Table 8 (continued) – Air Concentrations Adjacent to Springvale and Floodvale Drains

Location	Sample Date	Chemical Detected – Concentration (µg/m ³)														
		tetrahydrofuran	benzene	toluene	ethylbenzene	xylenes	4-ethyltoluene	1,2,4-trimethylbenzene	acetone	2-butanone (MEK)	2-propanol	hexane	cyclohexane	heptane	4-methyl-2-pentanone	ethanol
Floodvale Drain																
AS75	Aug '06			3.7		2.1		0.99	6.4	5						5.5
AS76	Aug '06		1.1	4.6	0.77	3.4		0.98	12							6.5
Max			1.1	4.6	0.77	3.4		0.99	12							6.5
Springvale Drain																
AS74	Aug '06	16														
AS73	Aug '06		1.2	4.2		3.0		0.75	12	2.6						5.5
AS87	19 Sept '06		7.5	9.6					39	8.2						
AS87	29 Sept '06		1.7	3.4					8.2	3.2						5.3
AS87	Oct '06		1.9	8.0	2.5	10.2	0.94	1.1	58	3.4	6.9	1.0	0.89	0.66	2.4	66
Max		16	7.5	9.6	2.5	10.2	0.94	1.1	58	8.2	6.9	1.0	0.89	0.66	2.4	66
Screening Level		1³	9.6¹	377¹	22000²	870¹	377^B	6.2³	3300³	5110³	7000⁴	209³	2605³	209^A	3100³	100000⁴

1 Air Investigation Level as per NEPM 2004, based on annual average

2 Air quality criteria or guideline value available from the WHO, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.

3 Ambient air PRG available from USEPA Region IX, based on annual average. Where criteria is derived on the basis of a non-threshold approach a target risk of 1x10⁻⁶ is adopted.

4 Chronic Reference Exposure Level established by OEHHA

A No guideline available. As chemical is an EC7 aliphatic hydrocarbon similar to hexane, hexane has been adopted as surrogate for hydrocarbon group

B No guideline available. As chemical is an EC9 aromatic hydrocarbon similar to toluene, toluene has been adopted as surrogate for hydrocarbon group

Shaded cells indicate measured air concentrations that exceed the adopted screening level – identified as COPCs

Appendix A

Summary of Data and Identification of COPC

On the basis of the above the following COPC have been identified for the assessment of exposures related to the emissions of volatiles from Floodvale and Springvale Drains:

Springvale Drain (relevant to the assessment of Exposures in Block 1 and Block 2)	Floodvale Drain (relevant to the assessment of exposures in Block 2)
1,2-dichloroethane (EDC) trichloroethene (TCE) vinyl chloride carbon tetrachloride chloroform <i>cis</i> -1,2-dichloroethene tetrahydrofuran	1,2-dichloroethane (EDC)

A2.7 Impacts from Other Sources

A number of other sources of emissions to air are present on the BIP such as the GTP. In addition there are other projects that have been approved or are in the process of seeking approval. These include the HCB repackaging plant and the CPWE remediation project. The most current and recent assessment of risks associated with emissions to air associated with these sources, and other sources within the BIP (and background) is presented in the Human Health Impact Assessment prepared for the CPWE Remediation Project (URS, 2006). While this project has not been approved at the time of this assessment, the air dispersion modelling and assessment of risk presented is the most current and reflects all other sources on the BIP (and background) with the inclusion of the CPWE remediation project, which provides a conservative estimate of emissions should the remediation project not be approved.

The assessment of risks to human health associated with emissions to air from all sources indicated that the risks were low and acceptable for all off-site areas, including workers and food processing facilities located in the vicinity of Southlands. On this basis, no additional COPC have been identified with respect to these exposures, however the predicted concentrations in air in the vicinity of Southlands should be taken into account in the assessment of overall exposure to the COPC identified in air.

Key emissions to air derived from other sources on the BIP that have also been identified as COPC are:

- tetrachloroethene (PCE);
- trichloroethene (TCE);
- 1,2-dichloroethene (*cis*- and *trans* -isomers);
- 1,1-dichloroethene;
- vinyl chloride;
- 1,1,2-trichloroethane;
- 1,1-dichloroethane;

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- 1,2-dichloroethane;
- chloroform;
- methylene chloride (dichloromethane);
- mercury;
- hexachlorobenzene (HCB); and
- hexachlorobutadiene (HCBD).

On the basis of air modelling undertaken by Pacific Air and Environment (PAE, 2006) as part of the CPWE Remediation Assessment, the following ground level concentrations have been estimated for the above COPC at the Kelloggs site with is the closest discrete receptor to Southlands that was modelled and is relevant to the assessment of potential exposures in Southlands. The concentrations presented are relevant to emissions from all sources and based on an annual average.

Table 9 – Concentrations of COPC Derived from Other Sources on BIP

COPC	Predicted Ambient Air Concentration Derived from All Sources (Emissions from BIP) – relevant to Southlands ($\mu\text{g}/\text{m}^3$)
tetrachloroethene (PCE)	0.41
trichloroethene (TCE)	0.0025
<i>cis</i> -1,2-dichloroethene	0.0000034
<i>trans</i> -1,2-dichloroethene	0.0000074
1,1-dichloroethene	0.00011
vinyl chloride	0.0011
1,1,2-trichloroethane	0.0000006
1,1-dichloroethane	0.0000055
1,2-dichloroethane (EDC)	0.00084
chloroform	0.00054
dichloromethane	0.000046
mercury	0.00022
hexachlorobenzene (HCB)	0.0000062
hexachlorobutadiene (HCBD)	0.0019

The above concentrations have been included in the assessment of exposures for all users of the area.

Appendix A

Summary of Data and Identification of COPC

A2.8 Summary of COPC Identified

On the basis of the above the following COPC have been identified for further assessment of exposures on the Southlands site:

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Summary of Data and Identification of COPC

Table 10 – Summary of COPC Identified on Block 1 and Block 2

Chemical	COPC - Block 1					COPC - Block 2		
	Soil	Sediment	Surface Water	Emissions to Air	Emissions from Drains	Soils	Emissions to Air	Emissions from Drains
asbestos						•		
arsenic			•					
cadmium			•					
chromium	•	•	•			•		
lead	•		•			•		
nickel			•					
zinc			•					
mercury	•					•		
TPH	•	•					•	
benzene	•			•			•	
toluene				•			•	
ethylbenzene	•			•			•	
xylene	•			•			•	
1,2,4-trimethylbenzene				•			•	
1,3,5-trimethylbenzene							•	
vinyl chloride	•	•	•	•	•		•	•
1,1-dichloroethene			•				•	
trans-1,2-dichloroethene			•	•			•	
cis-1,2-dichloroethene	•	•	•	•	•		•	•
trichloroethene (TCE)	•	•	•	•	•		•	•
tetrachloroethene (PCE)	•	•	•	•			•	
1,1-dichloroethane							•	
1,2-dichloroethane (EDC)	•	•	•	•	•		•	•
1,1,2-trichloroethane	•	•	•	•			•	
1,1,1,2-tetrachloroethane	•	•	•	•				
chloroform	•	•	•	•	•		•	•
carbon tetrachloride			•	•	•		•	•
dichloromethane				•			•	
naphthalene				•			•	
PAHs (non-threshold)	•							
hexachlorobutadiene (HCBD)	•	•	•					
hexachlorobenzene (HCB)	•					•		
bis(2-chloroethyl)ether						•		
tetrahydrofuran					•			•
trichlorofluoromethane							•	