Appendix B Identification of COPC in Soil

Appendix B Identification of COPC in Soil

B1 Introduction

This appendix presents a review of soil data and identification of COPC associated with Impacted Soil Zones (ISZ) as defined by HLA (2006). The COPC identified in shallow and deep soil are summarised in **Section 3.3** of the main report.

B2 Impacted Soil Zones 1a, 1b and 2 (Area I)

Impacted Soil Zones 1a, 1b and 2 (ISZ 1a, 1b and 2) have been defined by HLA (2006) based on available soil data. These source zones include part of Area I with ISZ 1a extending into the southern portion of Area H and ISZ 2 overlapping with ISZ 3 that crosses into Area F. All these areas overlie groundwater Plume 1.

Soil data from these areas has been obtained from investigations undertaken by URS in 2000, CH2MHill in 2003 and 2004 and HLA in 2005 and 2006. Further data collected by HLA in 2007 suggest the most significant COPC in these areas are DDX and chlorobenzene (primarily in ISZ 1a and 1b).

Soil is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Appendix B Identification of COPC in Soil

Table B-1 Maximum Concentrations Reported in Soils in ISZ 1a, 1b and 2 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
ISZ 1a and 1b		
1,4-dichlorobenzene	12.7 mg/kg in shallow soil 28 mg/kg in soil 1 m and deeper	13 mg/kg RSL
1,2-dichloroethane (EDC)	1.1 mg/kg in shallow soil*	2.2 mg/kg RSL
trichloroethene (TCE)	10 mg/kg in shallow soil*	14 mg/kg RSL
chlorobenzene (MCB)	535 mg/kg in shallow soil 3040 mg/kg in soil 1m and deeper	1500 mg/kg RSL
carbon tetrachloride	5.8 mg/kg in shallow soil*	1.3 mg/kg RSL
chloroform	1.2 mg/kg in shallow soil 10.6 in soil 1m and deeper	1.5 mg/kg RSL
benzene	1.5 mg/kg in soil 1m and deeper	1 mg/kg NSW Service Station Guidelines
toluene	1500 mg/kg in shallow soil*	130 mg/kg NSW Service Station Guidelines
xylenes	82.2 mg/kg in shallow soil*	25 mg/kg NSW Service Station Guidelines
α-BHC	167 mg/kg in shallow soil*	0.27 mg/kg RSL
β- and γ-BHC	61.3 mg/kg in shallow soil*	0.96 mg/kg RSL
γ-BHC	100 mg/kg in shallow soil*	0.96 mg/kg RSL
δ-BHC	84.5 mg/kg in shallow soil*	0.96 mg/kg RSL
dieldrin	542 mg/kg in shallow soil*	50 mg/kg NEPM HIL for aldrin + dieldrin
DDX	988000 mg/kg in shallow soil*	5.1 mg/kg RSL (lower from group)
hexachlorobenzene	5.76 mg/kg in shallow soil*	1.1 mg/kg RSL
PAHs: benzo(a)pyrene benzo(a)anthracene benzo(b)fluroanthene dibenz(ah)anthracene indeno(1,2,3-c,d)pyrene	Concentrations in shallow soil* 31.9 mg/kg 31.5 mg/kg 47 mg/kg 1.4 mg/kg 22.8 mg/kg	 5 mg/kg NEPM HIL 2.1 mg/kg RSL 2.1 mg/kg RSL 0.21 mg/kg RSL 2.1 mg/kg RSL
ethylbenzene	< screening level in shallow soil 110 mg/kg in soil 1 m and deeper	130 mg/kg NSW Service Station Guidelines
ISZ 2		
1,4-dichlorobenzene	7.7 mg/kg in shallow soil 15.2 mg/kg in soil 1 m and deeper	13 mg/kg RSL
1,2-dichloroethane (EDC)	51 mg/kg in shallow soil*	2.2 mg/kg RSL
trichloroethene (TCE)	154 mg/kg in shallow soil 385 mg/kg in soil 1 m and deeper	14 mg/kg RSL
chlorobenzene (MCB)	432 mg/kg in shallow soil*	1500 mg/kg RSL
vinyl chloride	13 mg/kg in shallow soil*	1.7 mg/kg RSL
chloroform	77 mg/kg in shallow soil*	1.5 mg/kg RSL
benzene	3.2 mg/kg in shallow soil*	1 mg/kg NSW Service Station Guidelines
toluene	21 mg/kg in shallow soil*	130 mg/kg NSW Service Station Guidelines
xylenes	0.8 mg/kg in shallow soil 4.9 in soil 1m and deeper	25 mg/kg NSW Service Station Guidelines
γ-BHC	0.13 mg/kg in shallow soil*	0.96 mg/kg RSL
dieldrin	0.97 mg/kg in shallow soil*	50 mg/kg NEPM HIL for aldrin + dieldrin
DDX	1480 mg/kg in shallow soil*	5.1 mg/kg RSL (lower from group)
hexachlorobenzene	0.18 mg/kg in shallow soil*	1.1 mg/kg RSL
PAHs: benzo(a)pyrene	Concentrations in shallow soil 5.6 mg/kg	 5 mg/kg NEPM HIL

Notes:
All data assessed was collected at the site to April 2006.
* COPC present at maximum concentrations within the shallow soils (ie soils to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.
COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B3 Impacted Soil Zone 3 (Area F)

Impacted Soil Zone 3 (ISZ 3) has been defined by HLA (2006) based on available soil data. ISZ 3 is located across a large portion of the site originally defined as Area F. ISZ 3 and other portions of Area F also overlie groundwater Plume 2.

Soil data from ISZ 3 and Area F has been obtained from investigations undertaken by URS in 2000, CH2MHill in 2003 and 2004 and HLA in 2005 and 2006.

Soil is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Table B-2 Maximum Concentrations Reported in Soils in ISZ 3 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
1,2-dichloroethane (EDC)	226 mg/kg in shallow soil 687 mg/kg in soil deeper than 1m	2.2 mg/kg RSL
trichloroethene (TCE)	1 mg/kg in shallow soil 2 mg/kg in soil deeper than 1m	14 mg/kg RSL
β- and γ-BHC	10 mg/kg in shallow soil*	0.96 mg/kg RSL
DDX	1220 mg/kg in shallow soil*	5.1 mg/kg RSL (lower of group)

Notes:

All data assessed was collected at the site to April 2006.

* COPC present at maximum concentrations within the shallow soils (ie soils to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.

COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B4 Impacted Soil Zone 4 (Area A)

Impacted Soil Zone 4 (ISZ 4) has been defined by HLA (2006) based on available soil data. ISZ 4 is located within the portion of the site originally defined as Area A.

Soil data from ISZ 4 has been predominantly obtained from investigations undertaken by URS in 2000 and CH2MHill in 2003 and 2004. Most of the data obtained from CH2MHill is associated with validation data collected during remediation of the area. It is noted that the area has been excavated and validated down to bedrock (CH2MHill, 2007), with concentrations of benzene, TCE and EDC above the remediation criteria remaining within the weathered shale bedrock. Excavated soil has been placed onsite within the storage shed and the area has been backfilled with validated clean fill. The assessment of risk based on the available data prior to remediation is expected to provide a conservative assessment of risk.

Impacted soil in Area A, and assumed be predominantly located within ISZ4, that was characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Table B-3 Maximum Concentrations Reported in Soils in ISZ 4 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
α-BHC	3.2 mg/kg in shallow soil*	0.27 mg/kg RSL
β- and γ-BHC	32 mg/kg in shallow soil*	0.96 mg/kg RSL
δ-BHC	1.6 mg/kg in shallow soil*	0.96 mg/kg RSL
DDX	45750 mg/kg in shallow soil*	5.1 mg/kg RSL (lower of group)
hexachlorobenzene (HCB)	33 mg/kg in shallow soil*	1.1 mg/kg RSL
mercury	116 mg/kg in shallow soil*	75 mg/kg NEPM HIL
1,2-dichloroethane (EDC)	< screening level in shallow soil 292 mg/kg from all depths** 45 mg/kg at bedrock following remediation**	2.2 mg/kg RSL
trichloroethene (TCE)	< screening level in shallow soil 79 mg/kg from all depths** 292 mg/kg at bedrock following remediation**	14 mg/kg RSL
chloroform	< screening level in shallow soil 2.7 mg/kg from all depths**	1.5 mg/kg RSL
benzene	< screening level in shallow soil 9.8 mg/kg from all depths** 9.8 mg/kg at bedrock following remediation**	1 mg/kg NSW EPA Service Station Guidelines

Notes:

All data assessed was collected from the site to April 2006.

* COPC present at maximum concentrations within the shallow soil (ie soil to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.

** COPC identified only from depths greater than 1 m. Impacts associated with the presence of these COPC are at a depth of 2.5 to 3.5 m which is considered to be associated with the presence of these chemicals in groundwater (EDC and chlorobenzene plume identified (Plume 3 (HLA, 2006) in this area) as the depth to groundwater in this area ranges from 2.8 to 4.1 m (CH2MHill, 2004) and saturated soils do not warrant further consideration.

On this basis, the COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B5 Impacted Soil Zone 5 (Area B)

Impacted Soil Zone 5 (ISZ 5) has been defined by HLA (2006) based on available soil data. ISZ 5 is located across the portion of the site originally defined as Area B with some overlap into Area G.

Soil data from ISZ 5 (including remaining soils outside of the ISZ in Area B) have been obtained from investigations undertaken by URS in 2000, CH2MHill in 2003 and 2004 and HLA in 2005 and 2006. Soil in this area is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soils have been defined in this assessment as soils from 0 to 1 m below ground surface level):

Table B-4 Maximum Concentrations Reported in Soils in ISZ 5 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
α-BHC	0.64 mg/kg in shallow soil 2 mg/kg in soil deeper than 1m	0.27 mg/kg RSL
β- and γ-BHC	3 mg/kg in shallow soil 7.8 mg/kg in soil deeper than 1m	0.96 mg/kg RSL
DDX	16183 mg/kg in shallow soil	5.1 mg/kg RSL (lower of group)
hexachlorobenzene (HCB)	1.38 mg/kg in shallow soil*	1.1 mg/kg RSL
PAHs: benzo(a)pyrene benzo(a)anthracene benzo(b)fluroanthene dibenz(ah)anthracene indeno(1,2,3-c,d)pyrene	Concentrations in shallow soil* 9.4 mg/kg 5.1 mg/kg 9 mg/kg 1.1 mg/kg 6.1 mg/kg	 5 mg/kg NEPM HIL 2.1 mg/kg RSL 2.1 mg/kg RSL 0.21 mg/kg RSL 2.1 mg/kg RSL
ethylbenzene	127 mg/kg in shallow soil	50 mg/kg NSW Service Station
xylenes	595 mg/kg in shallow soil	25 mg/kg NSW Service Station
δ-BHC	< screening level in shallow soil 3.7 mg/kg in soil deeper than 1m	0.96 mg/kg RSL
1,4-dichlorobenzene	< screening level in shallow soil 10.8 mg/kg from all depths**	13 mg/kg RSL
1,2,3-trichloropropane	< screening level in shallow soil 0.5 mg/kg from all depths**	0.41 mg/kg RSL
1,2-dichloroethane (EDC)	< screening level in shallow soil 29.4 mg/kg from all depths**	2.2 mg/kg RSL
trichloroethene (TCE)	< screening level in shallow soil 3.4 mg/kg from all depths**	14 mg/kg RSL
chlorobenzene (MCB)	28.6 mg/kg in shallow soil 546 mg/kg from all depths**	1500 mg/kg RSL
chloroform	< screening level in shallow soil 3.3 mg/kg from all depths**	1.5 mg/kg RSL
1,1,2-trichloroethane	< screening level in shallow soil 9.2 mg/kg in soil 1 m and deeper	5.5 mg/kg RSL

Notes:

All data assessed was collected at the site to April 2006.

* COPC present at maximum concentrations within the shallow soils (ie soils to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.

** COPC identified only from depths greater than 1 m. Impacts could be associated with the presence of these chemicals in groundwater (EDC plume identified (Plume 4 (HLA, 2006)) in this area) however the maximum concentrations reported are above the groundwater table. As the impacts are identified at depth, consideration with respect to potential vapour migration should be considered in areas above these impacts and impacts from groundwater.

COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B6 Impacted Soil Zone 6 (Area C)

Impacted Soil Zone 6 (ISZ 6) has been defined by HLA (2006) based on available soil data. ISZ 6 is located within the portion of the site originally defined as Area C however it does not include the Pharmaceutical Building.

Soil data from ISZ 6 (including remaining soils outside of the ISZ in Area C, not including the Pharmaceutical Building) has been predominantly obtained from investigations undertaken by URS in 2000 and CH2MHill in 2003 and 2004. Most of the data obtained from CH2MHill is associated with validation data collected during remediation of the area. It is noted that the this area has been excavated and validated down to bedrock (CH2MHill, 2007). Excavated soil has been placed onsite within the storage shed and the area has been backfilled with validated clean fill. The assessment of risk based on the available data prior to remediation is expected to provide a conservative assessment of risk.

Soil in this area is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Table B-5 Maximum Concentrations Reported in Soils in ISZ 6 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
α-BHC	10.6 mg/kg in shallow soil*	0.27 mg/kg RSL
β- and γ-BHC	7.5 mg/kg in shallow soil*	0.96 mg/kg RSL
δ-BHC	3.04 mg/kg in shallow soil*	0.96 mg/kg RSL
DDX	6079 mg/kg in shallow soil*	5.1 mg/kg RSL (lower of group)
hexachlorobenzene (HCB)	2.84 mg/kg in shallow soil*	1.1 mg/kg RSL
PAHs: benzo(a)pyrene benzo(a)anthracene benzo(b)fluroanthene dibenz(ah)anthracene indeno(1,2,3-c,d)pyrene	Concentrations in shallow soil* 43 mg/kg 37 mg/kg 48 mg/kg 7 mg/kg 27 mg/kg	 5 mg/kg NEPM HIL 2.1 mg/kg RSL 2.1 mg/kg RSL 0.21 mg/kg RSL 2.1 mg/kg RSL

Notes:

All data assessed was collected at the site to April 2006.

* COPC present at maximum concentrations within the shallow soils (i.e. soils to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.

COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B7 Impacted Soil Zone 10 (Area C)

In addition to validation samples collected from soils in Area C, soil sampling has also been undertaken beneath the Pharmaceuticals Building located in Area C. Impacts identified beneath the building are defined as Impacted Soil Zone 10 (ISZ 10). These impacts were not remediated as part of the Pharmaceuticals Site remediation (VH2MHill 2007) and remain beneath the building. The building has been retained on site, however if the building were to be removed in the future, then there is the potential for COPC identified beneath the building to require consideration with respect to remediation requirements.

Soil is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Table B-6 Maximum Concentrations in Soils beneath Pharmaceutical Building ISZ 10 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
benzene	1.1 mg/kg	1 mg/kg NSW Service Station Guidelines
xylenes	34 mg/kg	25 mg/kg NSW Service Station Guidelines
α-BHC	9 mg/kg	0.27 mg/kg RSL
β- and γ-BHC	50 mg/kg	0.96 mg/kg RSL
DDX	5083 mg/kg	5.1 mg/kg RSL (lower of group)
PAHs: benzo(a)pyrene benzo(a)anthracene benzo(b)fluroanthene dibenz(ah)anthracene indeno(1,2,3-c,d)pyrene	 14.6 mg/kg 6.3 mg/kg 14.7 mg/kg 2.4 mg/kg 13.6 mg/kg	 5 mg/kg NEPM HIL 2.1 mg/kg RSL 2.1 mg/kg RSL 0.21 mg/kg RSL 2.1 mg/kg RSL

Notes:

All data assessed was collected at the site to April 2006.

COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B8 Impacted Soil Zones 7 and 8 (Area H)

Impacted Soil Zone 7 (ISZ 7) and ISZ 8 have been defined by HLA (2006) based on available soil data. ISZ 7 is located in the north-eastern corner of the site within the area originally defined as Area H. ISZ 8 is located across the remainder Area H, to the south of ISZ 7 with some overlap with the northern tip of ISZ 1a.

ISZ 7 and ISZ 8 are not located of any groundwater plumes identified by HLA (2006) with the southern edge of ISZ 8 on the boundary of Plume 1.

Soil data from ISZ 7 and 8 has been obtained from investigations undertaken by URS in 2000, CH2MHill in 2003 and 2004 and HLA in 2005 and 2006.

Soil in these areas is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Appendix B Identification of COPC in Soil

Table B-7 Maximum Concentrations Reported in Soils in ISZ 7 and ISZ 8 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
ISZ 7		
α-BHC	598 mg/kg in shallow soil*	0.27 mg/kg RSL
β- and γ-BHC	571 mg/kg in shallow soil*	0.96 mg/kg RSL
γ-BHC	951 mg/kg in shallow soil*	0.96 mg/kg RSL
DDX	5390 mg/kg in shallow soil*	5.1 mg/kg RSL (lower of group)
hexachlorobenzene	40.2 mg/kg in shallow soil*	1.1 mg/kg RSL
ISZ 8		
1,4-dichlorobenzene	165 mg/kg in shallow soil*	13 mg/kg RSL
tetrachloroethene (PCE)	22.5 mg/kg in shallow soil 603 mg/kg in soil 1 m and deeper	2.7 mg/kg RSL
chloroform	41.9 mg/kg in shallow soil*	1.5 mg/kg RSL
benzene	21.4 mg/kg in shallow soil*	1 mg/kg NSW Service Station Gdlns
toluene	116 mg/kg in shallow soil*	130 mg/kg NSW Service Station Gdlns
α-BHC	17400 mg/kg in shallow soil*	0.27 mg/kg RSL
β- and γ-BHC	14200 mg/kg in shallow soil*	0.96 mg/kg RSL
γ-BHC	8000 mg/kg in shallow soil*	0.96 mg/kg RSL
δ-BHC	899 mg/kg in shallow soil*	0.96 mg/kg RSL
dieldrin	542 mg/kg in shallow soil*	50 mg/kg NEPM HIL for aldrin + dieldrin
DDX	12170 mg/kg in shallow soil*	5.1 mg/kg RSL (lower of group)
hexachlorobenzene	6 mg/kg in shallow soil 250 mg/kg in soil 1 m and deeper	1.1 mg/kg RSL
pentachlorophenol	10 mg/kg in surface soil	9 mg/kg RSL
PAHs: benzo(a)pyrene benzo(a)anthracene benzo(b)fluroanthene dibenz(ah)anthracene indeno(1,2,3-c,d)pyrene	 4.1 mg/kg shallow, 4.7 mg/kg deep 3 mg/kg shallow, 4.2 mg/kg deep 6 mg/kg shallow, 11 mg/kg deep 0.7 mg/kg shallow and deep 3.7 mg/kg shallow and deep	 5 mg/kg NEPM HIL 2.1 mg/kg RSL 2.1 mg/kg RSL 0.21 mg/kg RSL 2.1 mg/kg RSL
trichloroethene (TCE)	14.2 mg/kg in soil 1 m and deeper	14 mg/kg RSL
1,2-dichloroethane (EDC)	< screening level in shallow soil 1.4 mg/kg in soil 1 m and deeper	2.2 mg/kg RSL
mercury	< screening level in shallow soil 187 mg/kg in soil 1 m and deeper	75 mg/kg NEPM HIL

Notes for Table 3-9:

All data assessed was collected at the site to April 2006.

* COPC present at maximum concentrations within the shallow soils (ie soils to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.

COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B9 Impacted Soil Zone 9 (Area G)

Impacted Soil Zone 9 (ISZ 9) has been defined by HLA (2006) based on available soil data. ISZ 9 is located at the top of the site overlapping the boundary between areas originally defined as Areas G and H. ISZ 9 and other portions of Area G are not located of any groundwater plumes identified by HLA (2006).

Soil data from ISZ 9 (including soil in Area G that remains outside of the ISZ, excluding ISZ 5) has been predominantly obtained from investigations undertaken by URS in 2000, CH2MHill in 2003 with some data derived from HLA in 2005 (no additional data for this area is presented in HLA 2006).

Soil in this area is characterised by the following chemicals that are present above the adopted human health risk-based screening levels (note shallow soil has been defined in this assessment as soil from 0 to 1 m below ground surface level):

Table B-8 Maximum Concentrations Reported in Soils in ISZ 9 and Identification of COPC

COPC	Maximum Concentrations Reported	Screening Level
tetrachloroethene (PCE)	4.1 mg/kg in shallow soil*	2.7 mg/kg RSL
benzene	4.7 mg/kg in shallow soil 5.8 mg/kg in soil 1m and deeper	1 mg/kg NSW Service Station Gdlns
α-BHC	25.4 mg/kg in shallow soil*	0.27 mg/kg RSL
β- and γ-BHC	19 mg/kg in shallow soil*	0.96 mg/kg RSL
Dieldrin	562 mg/kg in shallow soil*	50 mg/kg NEPM HIL for aldrin + dieldrin
DDX	22265 mg/kg in shallow soil*	5.1 mg/kg RSL (lower of group)
endosulfan II	15900 mg/kg in shallow soil*	3700 mg/kg RSL
PAHs: benzo(a)pyrene benzo(a)anthracene benzo(b)fluroanthene dibenz(ah)anthracene indeno(1,2,3-c,d)pyrene	Concentrations in shallow soil* 8.5 mg/kg 8.1 mg/kg 3.5 mg/kg 0.5 mg/kg 2.9 mg/kg	 5 mg/kg NEPM HIL 2.1 mg/kg RSL 2.1 mg/kg RSL 0.21 mg/kg RSL 2.1 mg/kg RSL

Notes:

All data assessed was collected at the site to April 2006.

* COPC present at maximum concentrations within the shallow soils (ie soils to a depth of 1 m). Lower concentrations reported in depths greater than 1 m.

COPC identified in shallow soils that are highlighted above have been considered further in the risk assessment for long-term commercial/industrial workers. All chemicals listed have been assessed for intrusive worker exposure.

Appendix B Identification of COPC in Soil

B-10 Soil in Remaining On-Site Areas

No source zones have been identified in former Areas D and E (with the exception of the northwest corner of Area E which forms part of ISZ 5), and these areas along with the southern portion of Area C overlie groundwater Plume 2. Review of soil data from these areas indicates low concentrations of COPC (below adopted screening criteria), consistent with no source zone being identified. Hence no further assessment of soils in these areas has been presented.

Appendix C Identification of COPC in Vapour Data

Appendix C Identification of COPC in Vapour Data

C1 Introduction

This appendix presents a review of available vapour data (soil gas (including sub-slab), flux emissions and indoor air) and identification of COPC associated with areas located above Impacted Soil Zones (ISZ) and groundwater plumes on and off the site. The COPC identified are summarised in **Section 3.5** of the main report.

C2 Impacted Soil Zones 1a, 1b and 2 and Plume 1 (Area I)

Air emission data is available for these zones, from the following:

- HLA sampling of soil gas and flux emissions in 2006 from AS6, AS7 and AS8 located within ISZs 1a, 1b and 2 and overlying groundwater Plume 1. These samples were collected in the vicinity of maximum impacts of volatile chemicals identified in soils and groundwater in the area.
- URS sampling of soil gas in 2007 and 2008 from locations AS6, AS7 and AS8. An additional soil gas sample was collected from location AS18 located between AS6 and AS8 within ISZ 2, also above Plume 1.

It is noted that there are no areas identified on the site that are located above groundwater Plume 1 alone (i.e. not also associated with other ISZs or other groundwater plumes) and hence no separate assessment of on-site areas above Plume 1 has been presented.

In addition it is noted that the vapour data collected from these locations is considered to be representative of vapour impacts from maximum impacted soil in both ISZ 1a/1b and ISZ 2. No separate assessment of vapour from ISZ 1a/1b and ISZ 2 has been presented. This is expected to be conservative for the assessment of exposures in ISZ1a/1b as the highest concentrations of volatile COPC were identified in ISZ 2 and are addressed within the vapour data collected from this area.

The following presents a summary of the available data from these locations. The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table).

Appendix C Identification of COPC in Vapour Data

Table C-1 Soil Gas and Flux Emissions Data for ISZ 1a, 1b and 2 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)											Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)		
	AS6			AS7			AS8			AS18			AS6[1]	AS7[1]	AS8[1]
	2006[*1]	2007[2]	2008[2*]	2006[1]	2007[2]	2008[2]	2006[1]	2007[2]	2008[2*]	2007[2]	2008[2*]		2006	2006	2006
cis-1,2-dichloroethene	1075	90000	1400	--	--	--	--	--	--	3100	2.3	26000[U] (s, trans-)	--	--	--
trichloroethene (TCE)	12060	3100000	29000	35124	--	--	--	5.3	--	90	--	230[W]	--	--	--
tetrachloroethene (PCE)	--	49000	230	9711	--	--	3589	8.4	--	75	4.2	25000[W]	--	--	--
chlorobenzene	3920	38000	--	9380165	--	310	22931	--	--	--	--	50000[W]	--	--	--
chloroform	32.2	63000	1800	--	6100	120	--	130	42	68	240	2400[W]	--	0.154	--
1,2-dichloroethane (EDC)	17.1	21000	310	--	--	--	--	--	--	--	--	200[W]	--	--	--
vinyl chloride	--	23000	--	--	--	9.8	--	--	--	1100	--	100[W]	--	--	--
benzene	5930#	--	--	23760	--	92	6181#	4.7	1.4	16	--	958[N]	--	--	--
toluene	4221	1600000	1400	21384297	--	1100	16949	310	260	170	1	37680[N]	--	0.195	0.11
1,2,4-trimethylbenzene	--	9800	--	--	--	--	--	140	--	290	--	3100[U]	--	--	--
1,3,5-trimethylbenzene	--	31000	--	--	--	--	--	170	--	290	--	2600[U]	--	--	--
ethylbenzene	--	--	--	54752	--	--	--	50	3.6	24	--	2200000[W]	--	--	0.32
xylenes	--	27000	--	84711	--	22	--	160	14.3	127	--	86850[N]	--	0.10	3.25
carbon disulfide	--	--	--	--	--	--	--	--	6.5	--	--	310000[U]	--	--	--
p-isopropyltoluene	--	--	--	--	--	--	--	--	--	--	--	180000[U] (s, cumene)	--	0.19	--
trans-1,2-dichloroethene	22.1	--	100	--	--	--	--	--	--	350	7	26000[U]	--	--	--
1,1-dichloroethane	--	--	--	--	--	--	--	--	--	39	--	770[U]	--	--	--
1,4-dichlorobenzene	--	--	--	47521	--	--	--	--	--	--	--	100000[W]	--	--	--
1,2-dichlorobenzene	--	--	--	8678	--	--	--	--	--	--	--	88000[U]	--	--	--
1,1,1-trichloroethane	--	--	--	--	--	--	--	--	--	17	3.7	2200000[U]	--	--	--
1,3-butadiene	--	--	--	--	--	5.6	--	2.1	--	--	--	41[U]	--	--	--
2-butanone (MEK)	--	--	--	--	--	140	--	14	--	40	2.3	2200000[U]	--	--	--
2-propanol	--	--	--	--	--	37	--	--	--	--	--	NA	--	--	--
ethanol	--	--	160	--	--	2300	--	13	160	280	8.5	NA	--	--	--
4-methyl-2-pentanone (MIBK)	--	--	--	--	--	58	--	--	--	--	--	1300000[U]	--	--	--
4-ethyltoluene	--	9900	--	--	--	--	--	42	--	81	--	2200000[W] (s, toluene)	--	--	--
acetone	--	20000	--	--	--	290	--	130	--	59	11	14000000[U]	--	--	--
carbon tetrachloride	--	--	--	--	--	--	--	4.7	--	--	--	6100[W]	--	--	--
chloromethane	--	--	--	--	--	15	--	2.2	2	--	0.73	680[U]	--	--	--
cumene	--	9900	--	--	--	--	--	1100	--	--	--	180000[U]	--	--	--
cyclohexane	--	--	--	--	--	43	--	160	6.3	310	--	2600000[U]	--	--	--

Appendix C Identification of COPC in Vapour Data

Chemical Detected	Soil Gas Concentration (µg/m^3)											Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)		
	AS6			AS7			AS8			AS18			AS6[1]	AS7[1]	AS8[1]
	2006[*1]	2007[2]	2008[2*]	2006[1]	2007[2]	2008[2]	2006[1]	2007[2]	2008[2*]	2007[2]	2008[2*]		2006	2006	2006
heptane	--	--	--	--	--	33	--	11	8.9	120	--	310000[U] (s, hexane)	--	--	--
hexane	--	--	--	--	--	12	--	46	2.4	220	--	310000[U]	--	--	--
methylene chloride	--	15000	--	--	--	47	--	--	--	27	--	460000[UX]	--	--	--
naphthalene	--	--	--	--	--	--	--	310	--	--	--	1300[UX]	--	--	--
propylbenzene	--	12000	--	--	--	--	--	--	--	--	--	15000[U04]	--	--	--
styrene	--	--	--	--	--	--	--	1000	--	28	--	440000[U]	--	--	--
trichlorofluoromethane	--	--	--	--	--	1.5	--	--	--	--	1.5	310000[U]	--	--	--
bromodichloromethane	--	--	--	--	--	--	--	--	--	--	18**	11[U04]	--	--	--
dibromochloromethane	--	--	--	--	--	--	--	--	--	--	2.6	8[U04]	--	--	--
dichlorodifluoromethane	--	--	--	--	--	--	--	--	--	--	1.3	88000[U]	--	--	--

Notes:

*	Maximum of data collected onto primary and secondary sample
**	Not considered to be significant as primary sample was reported at the guideline level and the duplicate (listed in the table) was just above the adopted guideline. The guideline adopted is based on the protection of ambient air (including residential), not just industrial air and is therefore considered conservative.
#	Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.
--	not detected above the LOR or not reported
1	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
2	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment

Appendix C Identification of COPC in Vapour Data

C3 Impacted Soil Zone 3 (Area F)

Air emission data is available for ISZ 3, from the following:

- HLA sampling of soil gas and flux emissions in 2006 (refer to **Figure 4** for locations) from AS4 and AS5 located within Area F. These samples were collected above ISZ 3 (soil) and the inferred edge of groundwater Plume 1 and within groundwater Plume 2. The samples have not been collected over areas of maximum EDC impacts identified in soils, however they are considered representative of potential emissions from the area; and
- URS sampling of soil gas in 2007 and 2008 (refer to **Figure 5** for locations) from locations AS4 and AS5. An additional soil gas sample was collected from location AS17 located within ISZ 3 and above groundwater Plume 2.

The following presents a summary of the available data from these locations. The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table).

Appendix C Identification of COPC in Vapour Data

Table C-2 Soil Gas and Flux Emissions Data for ISZ 3 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)							Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)	
	AS4			AS5			AS17		AS4	AS5
	2006[1]	2007[2]	2008[2]	2006[1]	2007[2]	2008[2]	2007[2]		2006[1]	2006[1]
toluene	29732	3.1	9	60277	1.2	880	--	37680[N]	--	--
1,2-dichloroethane (EDC)	--	--	--	--	--	--	6400	200[W]	--	--
chlorobenzene	35679	--	--	97826	--	--	--	50000[W]	--	--
naphthalene	--	360	100	--	24	--	130	1300[UN]	--	--
benzene	6145#	--	--	6423#	--	--	--	958[N]	--	--
ethylbenzene	--	4.3	6.1	--	--	--	--	2200000[W]	--	--
xylenes	--	--	43	--	--	21	--	86850[N]	--	--
p-isopropyltoluene	--	na	--	--	na	--	na	180000[U] (s, cumene)	0.087	--
trimethylbenzenes	--	3.5	108	--	--	--	--	2600[U] (1,3,5-)	--	--
tetrachloroethene (PCE)	--	2.2	--	--	60	8.9	--	25000[W]	--	--
trichloroethene (TCE)	--	1.1	--	--	83	--	--	230[W]	--	--
2-butanone (MEK)	--	16	4.7	--	--	71	--	2200000[U]	--	--
2-hexanone (MBK)	--	3.9	--	--	--	--	--	1300000[U] (s, MIBK)	--	--
2-propanol	--	3.8	--	--	--	23	--	NA	--	--
4-ethyltoluene	--	0.95	51	--	--	--	--	2200000[W] (s, toluene)	--	--
propylbenzene	--	--	8.1	--	--	--	--	15000[U04]	--	--
4-methyl-2-pentanone (MIBK)	--	1.4	--	--	--	61	--	1300000[U]	--	--
acetone	--	91	16	--	25	140	79	14000000[U]	--	--
bromodichloromethane	--	21**	--	--	3.2	--	--	11[U04]	--	--
carbon disulfide	--	4.5	9.3	--	--	--	--	310000[U]	--	--
chloromethane	--	0.7	--	--	6.1	--	--	680[U]	--	--
dibromochloromethane	--	5.4	--	--	--	--	--	8[U04]	--	--
ethanol	--	7.2	9.7	--	3.8	1500	--	NA	--	--
chloroform	--	--	--	--	--	7.4	--	2400[W]	--	--
cyclohexane	--	--	--	--	--	23	--	2600000[U]	--	--
heptane	--	--	--	--	--	26	--	310000[U] (s, hexane)	--	--
hexane	--	--	--	--	--	5.2	--	310000[U]	--	--

Notes:

** Not considered to be significant as only one detection reported at a concentration just above the adopted guideline. The guideline adopted is based on the protection of ambient air, not just industrial air and is therefore considered conservative.

Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.

Appendix C Identification of COPC in Vapour Data

--	not detected above the LOR or not reported
1	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
2	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment

Appendix C Identification of COPC in Vapour Data

C4 Impacted Soil Zone 4 and Plume 3 (Area A)

Air emission data is available for ISZ 4 and areas located above groundwater Plume 3 (HLA 2006) within the portion of the site formerly defined as Area A. Data is available from the following:

- CH2MHill sampling in 2005 (refer to **Figure 4** for locations) at locations #1, #3, #5 (all above the identified groundwater plume) and #2. It is noted that the depth of soil gas sampling is not known, however for location #5 it is inferred from a hand written map that the depth for soil gas sampling was approximately 0.5 m above the groundwater table, i.e. 2-2.5 m below ground level;
- HLA sampling of soil gas and flux emissions in 2006 (refer to **Figure 4** for locations) from AS1 (refer to Figure 4 for locations); and
- URS sampling of soil gas in 2007 and 2008 (refer to **Figure 5** for locations) from locations AS1 (above Plume 3 and ISZ 4) and AS14 (above Plume 3, closer to the western boundary of the site). It is noted that lower concentrations were reported in soil gas from AS14 compared with AS1 where the contribution from soil impacts (from ISZ 4) are greater.

The data presented in this section reflects areas located above both ISZ 4 and Plume 3. There are a number of other areas within Area A that are located downgradient of ISZ 4 and therefore located over Plume 3 alone. Plume 3 also extends further downgradient beneath off-site areas. Review of data relevant to the assessment of areas overlying Plume 3 alone is presented separately.

The following table presents a summary of the available data from these locations. The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table).

Appendix C Identification of COPC in Vapour Data

Table C-3 Soil Gas and Flux Emissions Data from ISZ 4 and Plume 3 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)									Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)				
	#1	#2	#3	#5	AS1			AS14			#1	#2	#3	#5	AS1
	2003[1]	2003[1]	2003[1] M	2003[1]	2006[2*]	2007[3]	2008[3]	2007[3]	2008[3]		2003[1]	2003[1]	2003[1]	2003[1***]	2006[2]
benzene	600	1200	--	640000	6151#	830	--	0.69	--	958[N]	0.007	0.009	0.015	0.022	--
trichloroethene (TCE)	300	7800	86	4000000	12896	140000	970	--	--	230[W]	0.017	0.007	--	0.22	0.080
toluene	170	9000	230	2300000	5258	--	--	4.5	1.3	37680[N]	0.025	0.047	0.056	0.098	0.31
chlorobenzene	50	5000	130	660000	4068	1400	--	--	--	50000[W]	--	--	--	0.027	--
chloroform	180	210	--	100000	38.7	5300	96	28	47	2400[W]	--	--	--	0.046	--
1,2-dichloroethane (EDC)	1,100	16000	--	13000000	2133	200000	34	--	--	200[W]	0.049	0.022	--	0.262	--
vinyl chloride	--	--	--	--	--	2400	--	0.51	--	100[W]	--	--	--	--	--
ethylbenzene	--	400	--	46000	--	--	--	2.2	--	2200000[W]	--	0.007	0.008	0.008	0.060
xylenes	80	2500	130	260000	--	--	--	15.6	--	86850[N]	0.016	0.042	0.044	0.053	0.050
1,2,4-trimethylbenzene	180	210	--	4900	--	--	--	98	--	3100[U]	--	0.007	--	0.016	0.070
1,3,5-trimethylbenzene	50	110	--	3200	--	--	--	290	--	2600[U]	--	--	--	--	--
1,2-dichlorobenzene	--	--	--	500	--	--	--	--	--	88000[U]	--	--	--	--	--
1,3-dichlorobenzene	--	--	--	100	--	--	--	--	--	88000[U] (s, 1,2-)	--	--	--	--	--
1,4-dichlorobenzene	--	90	--	3200	--	--	--	15	--	100000[W]	--	--	--	--	--
tetrachloroethene (PCE)	--	140	--	24000	--	1100	160	14	--	25000[W]	--	--	--	--	--
trans-1,2-dichloroethene	--	--	--	--	--	--	5.1	--	--	26000[U]	--	--	--		
sec-butylbenzene	--	--	--	300	--	na	--	na	--	180000[U] (s, cumene)	--	--	--	--	--
cumene	--	50	--	2500	--	--	--	2.2	--	180000[U]	--	--	--	--	--
naphthalene	--	--	--	80	--	--	--	--	8.3	1300[UX]	--	--	--	--	--
n-proylbenzene	--	50	--	1400	--	--	--	4	--	15000[U04]	--	--	--	--	--
2-butanone (MEK)	--	--	--	--	--	--	5.8	15	19	2200000[U]	--	--	--	--	--
2-hexanone (MBK)	--	--	--	--	--	--	--	--	5.2	1300000[U] (s, MIBK)	--	--	--		
2-propanol	--	--	--	--	--	--	--	3.4	2.7	NA	--	--	--	--	--
4-ethyltoluene	--	--	--	--	--	--	--	32	--	2200000[W] (s, toluene)	--	--	--	--	--
4-methyl-2-pentanone (MIBK)	--	--	--	--	--	--	--	1.4	1.4	1300000[U]	--	--	--	--	--
acetone	--	--	--	--	--	--	13	79	48	14000000[U]	--	--	--	--	--
bromodichloromethane	--	--	--	--	--	--	--	1.4	1.6	11[U04]	--	--	--	--	--
dichlorodifluoromethane	--	--	--	--	--	--	--	--	1.3	88000[U]	--	--	--		
trichlorofluoromethane	--	--	--	--	--	--	--	--	1.5	310000[U]	--	--	--		
heptane	--	--	--	--	--	--	--	1	--	310000[U] (s, hexane)	--	--	--	--	--
hexane	--	--	--	--	--	--	--	2.4	1	310000[U]	--	--	--	--	--
ethanol	--	--	--	--	--	--	--	8.6	8.2	NA	--	--	--		

Appendix C Identification of COPC in Vapour Data

Chemical Detected	Soil Gas Concentration (µg/m^3)									Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)				
	#1	#2	#3	#5	AS1			AS14			#1	#2	#3	#5	AS1
	2003[1]	2003[1]	2003[1] M	2003[1]	2006[2*]	2007[3]	2008[3]	2007[3]	2008[3]		2003[1]	2003[1]	2003[1]	2003[1***]	2006[2]
methylene chloride	--	--	--	--	--	460	--	--	--	460000[UX]	--	--	--	--	--

Notes:

*	Maximum of data collected onto primary and secondary sample
**	Not considered to be significant as only one detection reported at a concentration just above the adopted guideline. The guideline adopted is based on the protection of ambient air, not just industrial air and is therefore considered conservative.
M	Sample reported to be affected by the presence of moisture that could have resulted in an underestimate of soil gas concentrations.
#	Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.
--	not detected above the LOR or not reported
1	Samples collected in 2003 by CH2MHill. Note no details on sampling method or QR available.
2	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
3	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment

Appendix C Identification of COPC in Vapour Data

C5 Impacted Soil Zone 5 (Area B)

Air emission data is available for ISZ 5 within the portion of the site formerly defined as Area B (with some overlap into Areas E and G). Data is available from the following:

- HLA sampling of soil gas and flux emissions in 2006 (refer to **Figure 4** for locations) from AS2. These data were collected above maximum impacts of volatile chemicals identified in soils in the area; and
- URS sampling of soil gas in 2007 and 2008 (refer to **Figure 5** for locations) from location AS2, close to (within 3m) the HLA AS2 sampling location.

No soil gas or flux emissions data have been collected from areas in Area B that are not associated with ISZ 5. Review of groundwater data from Area B (Plume 4, HLA 2006) indicates that the key chemicals identified are similar to those identified in Area A (Plume 3, HLA 2006). The concentrations of volatile chemicals in Plume 4 (Area B) are lower than those reported in Plume 3. Hence potential emissions to air and risks associated with the inhalation of these chemicals above the contaminated groundwater plume are expected to be lower than presented in areas located above Plume 3 (groundwater only).

The following table presents a summary of the available data from these locations. It is noted that due to elevated concentrations reported in the soil gas sample collected, the analytical LOR was elevated such that many volatile chemicals were not reported above the LOR. Review of the soil and groundwater data suggest that no other volatile chemicals not detected in the soil gas sampling from AS2 are expected to significantly contribute to the calculated risk.

The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table).

Appendix C Identification of COPC in Vapour Data

Table C-4 Soil Gas and Flux Emissions Data for ISZ 5 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)			Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)
	AS2				AS2
	2006[1]	2007[2]	2008[2]		2006[1]
toluene	120000	--	--	37680[N]	0.18
1,1-dichloroethene	--	100000	64000	88000[U]	--
1,2-dichloroethane (EDC)	889000	250000	20000	200[W]	--
trichloroethene (TCE)	35000	5900	3000	230[W]	--
1,2-dichloropropane	168000	23000	12000	120[U]	0.13
1,1,2-trichloroethane	4660000	640000	200000	77[U]	7.3
chlorobenzene	133000	9800	--	50000[W]	--
1,1,1,2-tetrachloroethane	16000	--	--	170[U]	--
1,2,3-trichloropropane	125000	8442**	--	0.3[U04]	--
chloroform	--	20000	1600	2400[W]	--
tetrachloroethene (PCE)	51000	--	1100	25000[W]	--
vinyl chloride	--	81000	19000	110[W]	--
cis-1,2-dichloroethene	--	--	360	26000[U] (s, trans-)	--
trans-1,2-dichloroethene	--	--	1500	26000[U]	--
benzene	5200#	--	--	958[N]	--
xylenes	--	--	--	86850[N]	0.099
1,2,4-trimethylbenzene	--	--	--	3100[U]	0.069
2-chlorotoluene	7300	--	--	73000[U04]	--
carbon disulfide	--	2600	--	310000[U]	--

Notes:

#	Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.
--	not detected above the LOR or not reported
1	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
2	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of 1x10^{-6}.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment

Appendix C Identification of COPC in Vapour Data

C6 Impacted Soil Zone 8 (Area H)

No air emission data has been collected from areas located above ISZ 8 which is located within Area H. However it is noted that data has been collected from ISZ 1a, 1b and 2 above soil that have similar volatile COPC as ISZ8. The concentrations of many volatile COPC in soil in ISZ 1a, 1b and 2 are greater than in ISZ 8 and hence air emissions data collected from ISZ 1a, 1b and 2 is considered to provide a conservative estimate of potential emissions to air in areas above impacts identified in ISZ 8 (on the basis that the depth to contamination and characteristics of soils are similar).

It is also noted that many areas within ISZ 1a, 1b and 2 are also located above groundwater Plume 1 (**Figure 3**), whereas no significant groundwater plume has been identified beneath ISZ 8. Hence the data collected from ISZ 1a, 1b and 2 (presented in **Table C-1**) will also reflect the presence of volatile COPC in groundwater, further contributing to the conservative evaluation of potential exposures in ISZ 8.

C7 Impacted Soil Zone 9 (Areas G and H)

No soil gas or flux emissions data have been collected from ISZ 9. In addition the following can be noted:

- No significant groundwater plumes have been identified beneath ISZ 9 and hence no significant contribution to the emissions to air are expected to derive from groundwater; and
- Low levels of volatile COPC have been identified in soil in ISZ 9 with benzene, PCE and 1,1,2-trichloroethane being the volatile COPC identified in soil.

While the vapour migration pathway is not expected to be significant within this area, to provide a conservative assessment of potential issues that might be associated with ISZ 9, maximum measured soil gas concentrations for benzene and PCE reported above ISZ 1a, 1b and 2 (where higher concentrations in soils have been reported) have been adopted for the purpose of this assessment. Note that no other areas on the site have identified 1,1,2-trichloroethane as a COPC in soil and hence no soil gas data is available that specifically relates to the presence of this volatile chemical in soil. No further assessment of this chemical in ISZ 9 has been presented.

The maximum soil gas concentrations of benzene and PCE presented in **Table C-1** for these chemicals have been adopted for ISZ 9.

C8 Remaining Impacted Soil Zones 6, 7 and 10

No soil gas or flux emissions data have been collected from areas above ISZ 6, 7 or 10. In addition the following can be noted:

- The detection of volatile COPC in soil in these areas is limited. The only area where volatile COPC have been identified in soil is ISZ 10 where the maximum concentrations of benzene and xylenes just exceeded the adopted soil screening criteria. These concentrations are considered to be low when compared with other areas of the site where higher levels of benzene and xylenes are reported in soil (refer to **Section 6** and risk calculations presented in **Appendix F**). Further assessment would only be warranted in the event that unacceptable risks were identified for benzene and xylenes at other areas; and
- Review of available groundwater data indicates that the extent of plumes identified beneath the site does not extend beneath these source areas.

On this basis, no significant emissions to air derived from soil or groundwater are expected within ISZ 6, 7 and 10 hence further assessment of this pathway would only be warranted in the event that other areas affected by higher concentrations of COPC indicated potentially unacceptable risks.

Appendix C Identification of COPC in Vapour Data

C9 On-Site Groundwater Plume 2 (Areas C and D)

Air emission data is available for a number of areas located on the site that are above groundwater Plume 2, including areas that overlap with Plume 1. These areas are located within areas formerly defined as Area C and Area D. The assessment of Areas C and D does not overlap with ISZ6 and ISZ10 as these zones are not located above the identified groundwater plume and review of soil data for ISZ6 and ISZ10 does not indicate the presence of volatile chemicals. Hence vapour migration from the ISZs is not considered significant with respect to the assessment of potential exposures in areas on the site above Plume 2.

Data is available from the following in relation to Groundwater Plume 2:

- CH2MHill sampling in 2005 (refer to **Figure 4** for locations) at locations #4 (to the east of the Pharmaceutical Building on the western edge of the inferred Plume 2) and #6 (above Plume 2). It is noted that the depth of soil gas sampling is not known, however it is inferred from a handwritten map that the depth for soil gas sampling was approximately 0.5 m above the groundwater table, i.e. 2-2.5 m below ground level;
- HLA sampling of soil gas and flux emissions in 2006 (refer to **Figure 4** for locations) from AS3 (which is located in a similar area to location #6 sampled by CH2MHill), AS4 and AS9 (located above both Plume 1 and Plume 2); and
- URS sampling of soil gas in 2007 and 2008 (refer to **Figure 5** for locations) from location AS3, AS4 and AS9 (above Plume 2). In addition, soil gas samples have been collected at locations AS15 and AS16 located above Plume 2.

Only samples collected and reported have been included in the table. Some locations could not be sampled on some occasions due to saturation of the soil gas well. Where possible further data has been collected following reinstallation of the soil gas well.

Review of the groundwater data in the area suggests that soil gas and flux emissions data collected from AS11 and AS13 (located off-site and downgradient of Area D above similar concentrations in groundwater) are also relevant to the estimation of potential emissions to air in Area D above the impacted groundwater.

The following table presents a summary of the available data from these locations. The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table along with chemicals detected in flux emissions sampling).

Appendix C Identification of COPC in Vapour Data

Table C-5 Soil Gas Data Relevant to On-Site Areas above Plume 2 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)																	Soil Gas Screening Level (µg/m^3)
	#6	AS3			#4[1]	AS4		AS9			AS11		AS13	AS15		AS16		
	2003[1]	2006[2]	2007[3]	2008[3]	2003[1]	2006[2]	2007[3]	2006[2]	2007[3]	2008[3]	2006[2]	2007[3]	2006[2]	2007[3]	2008[3]	2007[3]	2008[3*]	
trichloroethene (TCE)	1900	--	--	--	420	--	1.1	--	36	--	--	--	--	--	--	--	--	230[W]
toluene	2400	8036	5.1	460	570	29732	3.1	--	5.4	53	65000	--	7079	3.8	180	2.6	870	37680[N]
1,1,2-trichloroethane	--	--	--	13	--	--	--	--	--	--	--	--	229	--	--	--	--	77[U]
chlorobenzene	1600	8631	--	--	370	35679	--	--	--	--	19000	--	--	--	--	--	--	50000[W]
1,2-dichloroethane (EDC)	3300	--	--	--	890	--	--	--	160	--	--	--	10	--	--	--	--	200[W]
1,2,3-trichloropropane	--	--	na	--	--	--	na	--	na	--	9400	--***	14	--	--	--	--	0.3[U04]
naphthalene	--	--	160	--	--	--	360	--	25	--	--	--	--	--	--	--	--	1300[UX]
benzene	480	5952#	1.7	6	80	6145#	--	6163#	--	3.7	6800#	--	6780#	--	110	1.6	4.1	958[N]
ethylbenzene	140	--	3.1	--	40	--	4.3	--	1.5	1.4	--	1.1	--	--	6.8	--	--	2200000[W]
xylenes	890	--	6.9	--	250	--	10.7	--	7	5.3	--	--	--	3.45	14	3.3	5.9	86850[N]
trimethylbenzenes	70	--	38	--	--	--	3.5	--	83	--	--	--	--	--	--	17.4	--	2600[U] (1,3,5-)
1,4-dichlorobenzene	40	12.9	--	--	--	--	--	--	--	--	--	--	14	--	--	--	--	100000[W]
chloroform	130	--	7.7	--	--	--	230	--	77	4.5	--	1.4	--	--	--	8.1	4	2400[W]
tetrachloroethene (PCE)	50	--	16	--	--	--	2.2	--	15	1.8	--	--	--	--	--	4.6	--	25000[W]
1,1,1-trichloroethane	--	--	--	--	--	--	--	--	--	1.9	--	--	--	--	--	--	--	2200000[U]
carbon tetrachloride	--	--	--	--	--	--	--	--	--	11	--	--	--	--	--	--	--	6100[W]
chloroethane	--	--	--	--	--	--	--	--	--	1	--	--	--	--	--	--	--	4400000[U]
1,3-butadiene	--	--	1.2	3.5	--	--	--	--	--	2.4	--	--	--	--	26	--	2	41[U]
2-butanone (MEK)	--	--	17	89	--	--	16	--	3.7	26	--	--	--	18	56	13	61	2200000[U]
2-hexanone	--	--	4.6	--	--	--	3.9	--	--	--	--	--	--	--	--	--	--	1300000[U] (s, MIBK)
2-propanol	--	--	3.4	17	--	--	3.8	--	--	6.4	--	--	--	4.6	20	2.8	17	NA
4-ethyltoluene	--	--	10	--	--	--	0.95	--	6	1.1	--	--	--	--	--	3.1	--	2200000[W] (s, toluene)
4-methyl-2-pentanone	--	--	2	8	--	--	1.4	--	--	9.6	--	--	--	1.2	58	1.2	26	314000[U]
acetone	--	--	98	170	--	--	91	--	29	93	--	--	3.7	100	220	65	180	14000000[U]
carbon disulfide	--	--	4.1	--	--	--	4.5	--	--	8	--	--	--	--	23	3.7	--	310000[U]
chloromethane	--	--	--	6.4	--	--	--	--	--	1.6	--	--	--	1.2	5.5	--	1.5	680[U]
cumene	--	--	1.2	--	--	--	--	--	--	--	--	--	--	--	--	0.7	--	180000[U]
cyclohexane	--	--	--	120	--	--	--	--	--	2	--	--	--	--	840	0.7	2.7	2600000[U]
dibromochloromethane	--	--	--	--	--	--	5.4	--	--	--	--	--	--	--	--	--	--	8[U04]
dichlorodifluoromethane	--	--	--	--	--	--	--	--	--	1.8	--	--	--	--	--	--	--	88000[U]
trichlorofluoromethane	--	--	--	--	--	--	--	--	--	1.6	--	--	--	--	--	--	--	310000[U]

Appendix C Identification of COPC in Vapour Data

Chemical Detected	Soil Gas Concentration (µg/m^3)																	Soil Gas Screening Level (µg/m^3)
	#6	AS3			#4[1]	AS4		AS9			AS11		AS13	AS15		AS16		
	2003[1]	2006[2]	2007[3]	2008[3]	2003[1]	2006[2]	2007[3]	2006[2]	2007[3]	2008[3]	2006[2]	2007[3]	2006[2]	2007[3]	2008[3]	2007[3]	2008[3*]	
heptane	--	--	3.1	21	--	--	--	--	--	5.5	--	--	--	1.2	480	1.6	9.9	310000[U] (s, hexane)
hexane	--	--	9.3	9.8	--	--	--	--	--	4.8	--	--	--	0.75	920	7	6.4	310000[U]
methylene chloride	--	--	2.7	--	--	--	--	--	1.5	--	--	--	--	--	11	--	--	460000[UX]
propylbenzene	--	--	5.3	--	--	--	--	--	--	--	--	--	--	--	--	--	--	15000[U04]
ethanol	--	--	8.6	1200	--	--	7.2	--	4.5	240	--	--	--	16	590	8.2	460	NA

Notes:

*	Maximum of data collected onto primary and secondary sample
**	Not considered to be significant as only one detection reported at a concentration just above the adopted guideline. The guideline adopted is based on the protection of ambient air, not just industrial air and is therefore considered conservative.
M	Sample reported to be affected by the presence of moisture. Could have resulted in an underestimate of soil gas concentrations.
#	Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.
--	not detected above the LOR or not reported
1	Samples collected in 2003 by CH2MHill. Note no details on sampling method or QR available.
2	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
3	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment.

Appendix C Identification of COPC in Vapour Data

Table C-6 Flux Emissions Data Relevant to On-Site Areas above Plume 2

COPC Detected	Flux Emission Rate (µg/m^2/min)								
	#6	AS3	#4	AS4	AS9	AS11		AS13	
	2003[1]	2006[2]	2003[1]	2006[2]	2006[2]	2006[2]	2007[3]	2006[2]	2007[3]
Potentially significant Chemicals Identified in Soil Gas									
trichloroethene (TCE)	--	--	--	--	--	--	--	--	--
toluene	0.037	0.36	0.088	--	--	0.13	0.089	0.053	0.066
1,1,2-trichloroethane	--	--	--	--	--	--	--	--	--
chlorobenzene	--	--	--	--	--	--	--	--	--
1,2,3-trichloropropane	--	--	--	--	--	--	na	--	na
naphthalene	--	--	--	--	--	--	--	--	--
Additional Volatile Chlorinated Chemicals Detected in Flux Emissions									
carbon tetrachloride	--	--	0.009	--	--	--	--	--	--

Notes:

1 Samples collected in 2003 by CH2MHill. Note no details on sampling method or QA available.

2 Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes).

3 Samples collected in 2007 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).

Chemicals presented are those identified as COPC in soil gas as well as additional; chlorinate compounds detected in flux emissions.

Appendix C Identification of COPC in Vapour Data

C10 Groundwater Plumes 3, 4 and 5 (On and Off-Site)

C10.1 Plume 3 (On and Off-Site)

Data is available from the following:

- CH2MHill soil gas and flux emissions data from locations #3 and #1 located close to the western boundary of the site above this plume, away from ISZ 2, in areas expected to be above impacted groundwater only. In addition, CH2MHill has collected air data within the building located on the Villawood site and the warehouse building (Hui Hong building) located off-site downgradient to the west of the site. The plume (HLA 2006) has been defined as being underneath this building and ambient air data was collected in the warehouse above the plume and personal monitoring of workers within the Hui Hong building. The ambient air data collected included sampling and analysis (using WorkCover NSW methods and laboratory) for VOCs, vinyl chloride, organochlorines and organophosphates.
- URS soil gas data collected in 2007 and 2008 from AS14 which is located close to the western boundary of the site above this plume and away from ISZ 4 in an area expected to be above impacted groundwater only.

It is noted that soil gas and flux emissions data have also been collected by CH2MHill and HLA from other locations within Area A. These locations are, however above source zones where impacts have been identified in soil in ISZ 2 as well as groundwater and are therefore not considered to be representative of concentrations (or emissions) above impacted groundwater only and have not be considered in the assessment of areas above Plume 3 only.

The **Tables C-7 and C-8** provide a summary of the soil gas, flux emissions and ambient air data that are considered relevant for areas above this plume only (i.e. not above ISZ 4). The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table).

C10.2 Groundwater Plume 4 (On-Site)

No soil gas or flux emissions data have been collected from Area B above groundwater Plume 4. Plume 4 is located within ISZ 5 extending down-gradient beneath Area B. The assessment presented for ISZ 5 addresses potential emissions to air derived from the source zone, however the available air data does not include areas above Plume 4. Review of groundwater data from Area B (Plume 4, HLA 2006) indicates that key chemicals identified to be similar to those identified in Area A (Plume 3, HLA 2006). The concentrations of volatile chemicals in Plume 4 (Area B) are lower than those reported in Plume 3. Hence potential emissions to air and risks associated with the inhalation of these chemicals above the contaminated groundwater Plume 4 are expected to be lower than in areas located above Plume 3 (groundwater only). On this basis the assessment presented for exposures in areas above Plume 3 are considered to be conservative and representative of exposures in areas above Plume 4.

C10.3 Groundwater Plume 5 (On-Site)

No soil gas or flux emissions data have been collected from Area C above groundwater Plume 5. Plume 5 is located downgradient of ISZ 6 and extends further down-gradient within Area C. The plume does not extend off site. Review of groundwater data from Area C (Plume 5, HLA 2006) indicates that key volatile chemicals identified are similar to those identified in Area A (Plume 3, HLA 2006). The concentrations of volatile chemicals in Plume 5 (Area B) are lower than those reported in Plume 3. Hence potential emissions to air and risks associated with the inhalation of these chemicals above the contaminated groundwater Plume 5 are expected to be lower than in areas located above Plume 3 (groundwater only). On this basis the assessment presented for exposures in areas above Plume 3 are considered to be conservative and representative of exposures in areas above Plume 5.

Appendix C Identification of COPC in Vapour Data

Table C-7 Soil Gas and Flux Emissions Data for Areas above Plume 3 (On and Off the Site) and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)				Soil Gas Screening Level (µg/m^3)	Flux Emission Rate (µg/m^2/min)	
	#1	#3	AS14			#1	#3
	2003[1]	2003[1M]	2007[2]	2008[2]		2003[1]	2003[1]
trichloroethene (TCE)	300	86	--	--	230[W]	0.017	--
1,2-dichloroethane (EDC)	1100	--	--	--	200[W]	0.049	--
benzene	600	--	0.69	--	958[N]	0.007	0.015
toluene	170	230	4.5	1.3	37680[N]	0.025	0.056
ethylbenzene	--	--	2.2	--	2200000[W]	--	0.008
xylenes	80	130	15.6	--	86850[N]	0.016	0.044
tetrachloroethene (PCE)	--	--	14	--	25000[W]	--	--
chlorobenzene	50	130	--	--	50000[W]	--	--
chloroform	180	--	28	47	2400[W]	--	--
1,2,4-trimethylbenzene	180	--	98	--	3100[U]	--	--
1,3,5-trimethylbenzene	50	--	290	--	2600[U]	--	--
naphthalene	--	--	--	8.3	1300[UN]		
2-butanone (MEK)	--	--	15	19	2200000[U]	--	--
2-hexanone (MBK)	--	--	--	5.2	1300000[U] (s, MIBK)	--	--
2-propanol	--	--	3.4J	2.7	NA	--	--
4-ethyltoluene	--	--	32	--	2200000[W] (s, toluene)	--	--
4-methyl-2-pentanone (MIBK)	--	--	1.4	1.4	1300000[U]	--	--
acetone	--	--	79	48	14000000[U]	--	--
bromodichloromethane	--	--	1.4	1.6	11[U04]	--	--
dichlorodifluoromethane	--	--	--	1.3	88000[U]	--	--
trichlorofluoromethane	--	--	--	1.5	310000[U]	--	--
cumene	--	-- W	2.2	--	180000[U]	--	--
heptane	--	-- W	1.0	--	310000[U] (s, hexane)	--	--
hexane	--	-- W	2.4	1	310000[U]	--	--
propylbenzene	--	-- W	4.0	--	15000[U04]	--	--
ethanol	--	--	8.6	8.2	NA	--	--

Notes:

M	Sample reported to be affected by the presence of moisture. Could have resulted in an underestimate of soil gas concentrations.
--	not detected above the LOR or not reported
1	Samples collected in 2003 by CH2MHill. Note no details on sampling method or QR available.
2	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours.
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment

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Appendix C Identification of COPC in Vapour Data

Table C-8 Indoor Air Data from Off Site Building Above Plume 3

Chemicals Detected	Ambient Air Concentration (mg/m^3)						
	HH-V01 Tube 2	HH-V02 Tube 5	HH-V03 Tube 8	HH-V04 Tube 11	HH-V05 Tube 14	HH-V06 Tube 17	NOHSC Exposure Standard TWA
ethanol	nd	0.02	0.01	0.05	0.18	0.01	1880
n-hexane	nd	0.03	0.07	0.03	0.07	0.08	176
benzene	nd	0.00	0.01	0.01	0.01	0.01	5
cyclohexane	nd	0.01	0.10	0.05	0.17	0.11	1010
3-methyl hexane	nd	0.01	0.07	0.04	0.17	0.08	1610
n-heptane	nd	0.01	0.11	0.09	0.38	0.13	1640
toluene	nd	0.34	0.43	0.51	2.12	0.53	337
octane	nd	nd	0.01	0.01	0.05	0.01	1400
ethylbenzene	nd	0.01	0.01	0.01	0.03	0.01	434
xylene	nd	0.09	0.04	0.07	0.17	0.03	350
a-pinene	nd	0.01	0.25	0.09	0.26	0.16	NA
b-pinene	nd	0.01	0.35	0.02	0.15	0.15	NA

HH-V01 – With Orica Villawood site over Plume 3.
HH-V02 – sample collected at the rear boundary of the Hui Hong site, next to a doorway, on outside of building
HH-V03 – sample collected within the factory area of the building (Hui Hong), on top of a stormwater trench
HH-V04 – sample collected from a mobile worker within the factory area building (Hui Hong) - (Forklift driver)
HH-V05 – sample collected from a worker who is relative fixed in the location during his/her work-shift (Hui Hong) - (up-stair workshop area); and
HH-V06 – sample collected from within the factory area (Hui Hong) of the building, inside the stormwater trench.

The exact locations of the samples collected in the Hui Hong building, the location of which is shown in **Figure 3**, are not available. Sample location HHV01 (on-site) is also unknown with descriptions in the report suggesting that it is location above Plume 3.

The ambient air data collected by CH2MHill indicates concentrations below the adopted NOHSC TWA exposure standards. In addition the range of analytes reported appear to reflect urban air and localised industrial emissions rather than chemicals derived from the presence of Plume 3 beneath the area. Of note is the absence of detection of key chlorinated chemicals reported in groundwater and soil gas in the area. Hence the ambient air data collected by CH2MHill have not been directly used within the risk calculations for workers within this area.

Appendix C Identification of COPC in Vapour Data

C11 Groundwater Plume 2 (Off Site)

Data is available from the following (refer to **Figures 4 and 5** for locations):

- Soil gas and flux emissions data:
  - CH2MHill soil gas and flux emissions data collected from location #6 on the southern boundary of the site above Plume 2. This location is similar to subsequent sampling location AS3;
  - HLA soil gas and flux emission data collected in 2006 from AS3 and AS9 located on the southern boundary of the site (AS9 also above Plume 1) and AS11, AS12 and AS13 located off site above this plume (as well as Plume 1); and
  - URS soil gas data collected in 2007 and 2008 from locations AS3, AS9 and AS15 located on the site near the southern boundary of the site (AS9 also above Plume 1);
  - URS soil gas flux emissions data collected in 2007 and 2008 from locations AS11, AS12 and AS13 in off-site areas (also above Plume 1);
  - URS soil gas data collected in 2008 from l off-site location AS27 (also located above Plume 1);
  - URS sub-slab soil gas samples collected in 2007 and 2008 from six locations (AS19-AS24) beneath the off-site downgradient premises located at No 25-27 Christina Road.
- Indoor air data:
  - CH2MHill air data collected from down-gradient commercial/industrial properties located above this plume at No 25-27 Christina Road in September 2005 (**Figure 5**). The sampling was undertaken using WorkCover NSW methods and laboratory and reported concentrations of chloroform, 1,2-dichloroethane (EDC), chlorobenzene, TCE, benzene ethylbenzene and 1,4-dichlorobenzene;
  - URS indoor air data collected in 2007 and 2008 within off-site downgradient commercial premises located at No 25-27 Christina Road (collected in conjunction with the sub-slab soil gas samples), No 29 Christina Road and No 33A Christina Road.

It is noted that soil gas and flux emissions data have also been collected from other locations on the site that are above Plume 2. These locations are, however above source zones where impacts have been identified in soil as well as groundwater and are therefore not considered to be representative of concentrations (or emissions) above impacted groundwater only and have not be considered in the assessment of areas above Plume 2 only.

The following tables provide a summary of the soil gas, flux emissions, sub-slab soil gas and indoor air data that are considered relevant for off-site areas above this plume. The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the relevant tables).

Only samples collected and reported have been included in the table. Some locations could not be sampled on some occasions due to saturation of the soil gas well. Where possible further data has been collected following reinstallation of the soil gas well.

Appendix C Identification of COPC in Vapour Data

Table C-9 Soil Gas Data Relevant to Off-Site Areas above Plume 2 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)															Soil Gas Screening Level (µg/m^3)
	#6	AS3			AS9			AS15		AS11		AS12		AS13	AS27	
	2003[1]	2006[2]	2007[3]	2008[3]	2006[2]	2007[3]	2008[3]	2007[3]	2008[3]	2006[2]	2007[3]	2006[2]	2007[3]	2006[2]	2008[3]	
trichloroethene (TCE)	1900	--	--	--	--	36	--	--	--	--	--	111665	3200	--	1500	230[W]
1,1,2-trichloroethane	--	--	--	13	--	--	--	--	--	89000	--	--	--	229	--	77[U]
1,2-dichloroethane (EDC)	3300	--	--	--	--	160	--	--	--	--	--	--	25	10	16	200[W]
1,2,3-trichloropropane	--	--	--	--	--	--	--	--	--	9400	--	--***	--	--	--	0.3[U04]
naphthalene	--	--	160	--	--	25	--	--	--	--	--	--	4800	--	50	1300[UX]
toluene	2400	8035	5.1	460	--	5.4	53	3.8	180	65000	--	10967	--	7079	6.4	37680[N]
benzene	480	5952#	1.7	6	6163#	--	3.7	--	110	6800#	--	6879#	--	6780#	--	958[N]
ethylbenzene	140	--	3.1	--	--	1.5	1.4	--	6.8	--	--	--	390	--	--	2200000[W]
xylenes	890	--	6.9	--	--	6	5.3	3.45	14	--	--	--	1280	--	--	86850[N]
tetrachloroethene (PCE)	50	--	16	--	--	15	1.8	--	--	--	--	--	80	--	45	25000[W]
chlorobenzene	1600	8631	--	--	--	--	--	--	--	19000	--	2595	--	--	--	50000[W]
chloroform	130	--	7.7	--	--	77	4.5	--	--	--	1.4	--	80	--	41	2400[W]
1,1,1-trichloroethane	--	--	--	--	--	--	1.9	--	--	--	--	--	--	--	6.9	2200000[U]
carbon tetrachloride	--	--	--	--	--	--	11	--	--	--	--	--	--	--	9.6	6100[W]
chloroethane	--	--	--	--	--	--	1	--	--	--	--	--	--	--	--	4400000[U]
1,2,4-trimethylbenzene	70	--	14	--	--	47	--	--	--	--	--	--	--	--	--	3100[U]
1,3,5-trimethylbenzene	--	--	24	--	--	36	--	--	--	--	--	--	--	--	--	2600[U]
1,4-dichlorobenzene	40	12.9	--	--	--	--	--	--	--	--	--	--	--	14	--	100000[W]
1,3-butadiene	--	--	1.2	3.5	--	--	2.4	--	26	--	--	--	--	--	--	41[U]
2-butanone (MEK)	--	--	17	89	--	3.7	26	18	56	--	--	--	--	--	7.9	2200000[U]
2-propanol	--	--	3.4	17	--	--	6.4	4.6	20	--	--	--	--	--	--	NA
4-ethyltoluene	--	--	10	--	--	6	1.1	--	--	--	--	--	--	--	--	2200000[W] (s, toluene)
4-methyl-2-pentanone (MIBK)	--	--	2	8	--	--	9.6	1.2	58	--	--	--	--	--	--	1300000[U]
acetone	--	--	98	170	--	29	93	100	220	--	3.7	--	--	--	50	14000000[U]
carbon disulfide	--	--	4.1	--	--	--	8	--	23	--	--	--	64	--	--	310000[U]
chloromethane	--	--	--	6.4	--	--	1.6	1.2	5.5	--	--	--	--	--	--	680[U]
cumene	--	--	1.2	--	--	--	--	--	--	--	--	--	35	--	--	180000[U]
cyclohexane	--	--	--	120	--	--	--	--	840	--	--	--	--	--	--	2600000[U]
heptane	--	--	3.1	21	--	--	5.5	1.2	480	--	--	--	--	--	--	310000[U] (s, hexane)
hexane	--	--	9.3	9.8	--	--	4.8	0.75	920	--	--	--	--	--	3.9	310000[U]
methylene chloride	--	--	2.7	--	--	1.5	--	--	11	--	--	--	--	--	--	460000[UX]

Appendix C Identification of COPC in Vapour Data

Chemical Detected	Soil Gas Concentration (µg/m^3)															Soil Gas Screening Level (µg/m^3)
	#6	AS3			AS9			AS15		AS11		AS12		AS13	AS27	
	2003[1]	2006[2]	2007[3]	2008[3]	2006[2]	2007[3]	2008[3]	2007[3]	2008[3]	2006[2]	2007[3]	2006[2]	2007[3]	2006[2]	2008[3]	
propylbenzene	--	--	5.3	--	--	--	--	--	--	--	--	--	19	--	--	15000[U04]
dichlorodofluoromethane	--	--	--	--	--	--	1.8	--	--	--	--	--	--	--	--	88000[U]
trichlorofluoromethane	--	--	--	--	--	--	1.6	--	--	--	--	--	--	--	--	310000[U]
ethanol	--	--	8.6	1200	--	4.5	240	16	590	--	--	--	71	--	48	NA

Notes:

#	Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.
--	not detected above the LOR or not reported
1	Samples collected in 2003 by CH2MHill. Note no details on sampling method or QR available.
2	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
3	Samples collected in 2007 and 2008 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment.

Appendix C Identification of COPC in Vapour Data

Table C-10 Flux Emissions Data Relevant to Off-Site Areas above Plume 2

COPC Detected	Flux Emission Rate (µg/m^2/min)								
	#6	AS3	AS9	AS11		AS12		AS13	
	2003[1]	2006[2]	2006[2]	2006[2]	2007[3]	2006[2]	2007[3]	2006[2]	2007[3]
Potentially Significant Chemicals in Identified in Soil Gas									
trichloroethene (TCE)	--	--	--	--	--	--	0.085	--	--
1,1,2-trichloroethane	--	--	--	--	--	--	--	--	--
1,1-dichloroethane	--	--	--	--	--	--	--	--	--
1,2,3-trichloropropane	--	--	--	--	--**	--	na	--	na
naphthalene	--	--	--	--	--	--	--	--	--
toluene	0.037	0.36	--	0.13	0.089	0.066	0.058	0.053	0.066

Notes:

-- not detected above the LOR or reported in analytical suite

1 Samples collected in 2003 by CH2MHill. Note no details on sampling method or QA available.

2 Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes). Note a number of data from analysis of carbon tubes where presence of benzene in solvent extraction method is likely to have affected the results (not corrected or detailed as no QA available).

3 Samples collected in 2007 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).

** Estimated value for compound based on review requested for sample, refer to URS (2007) report for detail.

Chemicals presented are those identified as COPC in soil gas as well as additional; chlorinate compounds detected in flux emissions.

Appendix C Identification of COPC in Vapour Data

Table C-11 Sub-Slab Soil Gas Data from 25-27 Christina Road

Chemical Detected	Sub-Slab Soil Gas Concentration (µg/m^3)									Soil Gas Screening Level (µg/m^3)
	AS19	AS20	AS21		AS22	AS23		AS24		
	2007[1]	2007[1]	2007[1]	2008[2*]	2007[1]	2007[1]	2008[2]	2007[1]	2008[2]	
benzene	--	3.6	0.92	3.1	--	--	--	0.99	3.5	958[N]
trichloroethene (TCE)	1.9	--	0.94	--	--	--	--	--	--	230[W]
naphthalene	--	5	--	--	--	--	15	5.3	7.4	1300[UX]
tetrachloroethene (PCE)	2.7	1.7	1.4	--	2.6	--	--	17	18	25000[W]
chlorobenzene	0.78	23	7.7	--	--	0.99	3.8	26	8.8	50000[W]
chloroform	4	1.8	1.7	1.6	14	4	--	21	19	2400[W]
toluene	1.4	4.3	5.2	18	--	7.1	3.6	27	62	37680[N]
ethylbenzene	--	--	--	2.1	--	--	--	1.2	3.4	2200000[W]
xylenes	2.9	2.8	1.1	7	--	0.98	--	5.8	11	86850[N]
styrene	--	--	--	--	--	--	--	--	1.6	440000[U]
1,2,4-trimethylbenzene	6.1	18	4.3	--	--	--	--	23	1.8	3100[U]
1,3,5-trimethylbenzene	--	18	2.8	--	--	0.91	--	130	--	2600[U]
1,2-dichlorobenzene	--	--	--	--	--	--	--	23	11	88000[U]
1,3-dichlorobenzene	--	--	--	--	--	--	--	1.4	1	88000[U] (s, 1,2-)
1,4-dichlorobenzene	--	1.3	--	--	--	14	--	3.7	13	100000[W]
1,2-dichloropropane	0.79	--	--	--	--	--	--	--	--	10[U]
2-butanone (MEK)	--	--	62	14	--	92	2.5	6.4	13	2200000[U]
2-propanol	--	--	7.6	4.2	--	7.2	--	--	16	NA
4-ethyltoluene	1.3	2.3	0.93	--	--	--	--	6.3	1.3	2200000[W] (s, toluene)
4-methyl-2-pentanone (MIBK)	1.7	--	--	--	1.5	4.7	--	--	1.3	1300000[U]
acetone	100	27	130	58	100	130	9.5	67	42	14000000[U]
carbon disulfide	4.7	46	41	43	--	--	--	5.6	--	310000[U]
chloromethane	0.43	0.92	1.2	0.48	--	--	--	0.59	0.77	680[U]
cyclohexane	--	4.2	470	22	--	13	0.85	69	6.3	2600000[U]
heptane	0.86	1.0	9.7	0.93	--	2.4	--	--	4.3	310000[U] (s, hexane)
hexane	--	1.1	170	16	--	6.2	3.6	32	25	310000[U]
methylene chloride	--	1.4	--	--	--	--	--	--	3.8	460000[UX]
dichlorodifluoromethane	--	--	--	1.4	--	--	2.2	--	0.9	88000[U]
trichlorofluoromethane	--	--	--	1.7	--	--	1.5	--	1.6	310000[U]
ethanol	7.7	1.6	31	15	11	34	2.4	6	94	NA

Notes:

* Maximum of data collected onto primary multi-sorbent tube and secondary carbon tube

Appendix C Identification of COPC in Vapour Data

--	not detected above the LOR or not reported
1	Samples collected in 2007 by URS using summa canisters over 8 hours.
2	Samples collected in 2008 by URS using summa canisters over 8 hours.
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment. No soil gas concentrations reported in sub slab samples are considered to be of significance.

Appendix C Identification of COPC in Vapour Data

Table C-12 Indoor Air Data for Off-Site Areas above Plume 2

Chemical Detected#	Ambient Air Concentration (µg/m^3)								
	33 (A-D) Christina Road (currently a printers)			29 Christina Road (currently a panel beaters)			25-27 Christina Road (currently a mechanic's office)		
	2005[1]	(AM4) 2007[2]	(AM4) 2008[3]*	2005[1]	(AM3) 2007[2]	(AM3) 2008[3]*	2005[1]	(AM2) 2007[2]	(AM2) 2008[3]*
chloroform	2300	--	--	143	--	--	2300	--	--
1,2-dichloroethane (EDC)	197	--	--	185	--	--	197	--	--
chlorobenzene	--	--	--	13	--	--	--	--	--
trichloroethene (TCE)	303	--	--	63	--	--	303	--	--
benzene	7	--	--	33	29	7.7	7	72	--
toluene	--	13	1900	--	190	430	--	210	24000
ethylbenzene	7	--	1300	46	22	38	7	18	270
xylenes	44	14	7900	143	114	186	44	110	1230
1,4-dichlorobenzene	21	--	--	55	--	--	21	--	--
2-propanol	--	20000	66000	--	58	120	--	42	220
cyclohexane	--	1900	4800	--	23	30	--	48	170
heptane	--	1100	590	--	60	60	--	25	240
hexane	--	1600	1200	--	170	82	--	40	960
2-butanone (MEK)	--	--	--	--	--	1200	--	--	690
4-methyl-2-pentanone (MIBK)	--	--	--	--	--	27	--	--	360
acetone	--	--	1700	--	--	320	--	--	13000
trimethylbenzenes	--	--		--	--	27.6	--	--	--

Notes:

1 Maximum reported from 6 samples collected by CH2MHIll in 2005. Note no details on sampling methodology, locations, indoor air sources and QA available for the data presented.

2 Reported from data collected by URS in 2007.

3 Reported from data collected by URS in 2008.

* Maximum of primary and secondary sample.

Chemicals detected in air during sampling in 2003 with additional chemicals detected during sampling in 2007 only included where the concentrations reported differ from expected range of urban\industrial air concentrations (refer to URS 2007 for further discussion). Additional chemicals have been included based on review of data collected by URS in 2008. Additional chemicals are those where detected concentrations were significantly higher than reported by URS in 2007.

Appendix C Identification of COPC in Vapour Data

With respect to the data collected by CH2MHill within offsite buildings, the following can be noted:

- It is not known whether the work places where the CH2MHill data were collected utilised products (in particular glues and solvents) that might contain chlorinated chemicals as the business areas sampled were not fully surveyed and the quality and reliability of the data is unknown. Businesses in the area identified included car repairs, kitchen manufacturing, towing, cheese packaging warehouse, designs and warehousing. It is reasonable to expect that some of these businesses could utilise degreasers or other products that contain the chemicals detected in ambient air. Hence it is difficult to identify whether the data collected reflects emissions to air derived from the groundwater plumes, or whether there is contribution from fugitive emissions from the business areas sampled and regional urban air contaminants.
- Sub-slab soil gas data collected by URS beneath 25-27 Christina Road (where the highest levels of chlorinated chemicals were previously reported in ambient air) do not show the presence of elevated levels of chlorinated chemicals beneath the slab that would indicate the presence of a significant sub surface groundwater source. Low levels of a range of compounds were detected reflecting the porous nature of the concrete slab and the expected interaction between air inside the building and sub-slab soil gas.
- Ambient air data collected by URS within 25-27 Christina Road and other buildings sampled did not report detectable concentrations of the chlorinated chemicals in groundwater; in particular 1,2-dichloroethane (EDC) was not detected at any location. Other data collected in some locations appears consistent with fugitive emissions that might be associated with the businesses operating at the time of sampling. On the basis of the sub-slab soil gas and ambient air data significant vapour intrusion does not appear to be occurring within these buildings.

Hence it is considered likely that the ambient air data collected by CH2MHill where elevated levels of chlorinated compounds were reported might be associated with fugitive emissions at the time of sampling, rather than indicating emissions from a groundwater source.

Similarly data collected by URS in 2007 and 2008 suggest localised fugitive sources associated with business operations (or cleaning) within the buildings assessed.

The quantification of risk has been undertaken on the basis of maximum measured soil gas and flux emissions data only. The sub-slab soil gas data and ambient air data has not been used further in this assessment as it does not suggest vapour intrusion is significant. It should be noted that while this data has not been used further in the calculation of risk, the collection of the data is relevant when reviewing the potential significance of vapour intrusion into these buildings.

Appendix C Identification of COPC in Vapour Data

C12 Groundwater Plume 1 (Off-Site)

Data is available from the following (refer to Figures 4 and 5 for locations):

- Soil gas and flux emissions:
  - HLA soil gas and flux emission sampling undertaken in 2006 from AS9 and AS8 located on the southern boundary of the site and location AS10 located off site above this plume;
  - URS soil gas sampling from 2007 and 2008 from AS9 and AS8 located on the southern boundary of the site;
  - URS sampling of soil gas and flux emissions in 2007 only from location AS10 located off site above this plume; and
  - URS sampling in 2008 of soil gas from off-site location AS25.
- Indoor air data:
  - CH2MHill air data collected from down-gradient commercial/industrial properties located above this plume (No 23 Christina Road and No 19-21 Christina Road) in September 2005. The sampling was undertaken using WorkCover NSW methods and laboratory and reported concentrations of chloroform, 1,2-dichloroethane (EDC), chlorobenzene, TCE, benzene ethylbenzene and 1,4-dichlorobenzene;
  - URS indoor air data collected in 2007 and 2008 from within the building located at No 23 Christina Road.

It is noted that soil gas and flux emissions data have also been collected from other locations within Area I (southern portion of the site overlying Plume 1). These locations are, however above source zones where impacts have been identified in soil (ISZ areas) as well as groundwater and are therefore not considered to be representative of concentrations (or emissions) above impacted groundwater only and have not be considered in the assessment of areas above Plume 1 only.

The following tables provide a summary of the soil gas, flux emissions and ambient air data that are considered relevant for off-site areas above this plume. The soil gas data has been presented and compared against adopted soil gas screening levels to identify potentially significant chemicals in soil gas (highlighted in the table along with chemicals detected in flux emissions sampling).

Appendix C Identification of COPC in Vapour Data

Table C-13 Soil Gas and Flux Emissions Data Relevant to Off-Site Areas above Plume 1 and Identification of COPC

Chemical Detected	Soil Gas Concentration (µg/m^3)									Soil Gas Screening Level (µg/m^3)
	AS8			AS9			AS10		AS25	
	2006[1]	2007[2]	2008[3]*	2006[1]	2007[2]	2008[3]	2006[1]	2007[2]	2008[3]	
1,1,2-trichloroethane	--	--	--	--	--	--	42000	--	--	77[U]
1,2-dichloroethane (EDC)	--	--	--	--	160	--	--	--	--	200[W]
benzene	6181#	4.7	1.4	6163#	--	3.7	6500#	--	1.6	958[N]
toluene	16949	310	260	--	5.4	53	12000	--	40	37680[N]
ethylbenzene	--	50	3.6	--	1.5	1.4	--	--	0.91	2200000[W]
xylenes	--	160	14.3	--	7	5.3	--	--	14.5	86850[N]
carbon disulfide	--	--	6.5	--	--	8	--	--	2.9	310000[U]
trichloroethene (TCE)	--	5.3	--	--	36	--	--	--	--	230[W]
tetrachloroethene (PCE)	3589	8.4	--	--	15	1.8	--	3.5	--	25000[W]
chloroform	--	130	42	--	77	4.5	--	--	--	2400[W]
1,1,1-trichloroethane	--	--	--	--	--	1.9	--	--	1.5	2200000[U]
1,2,4-trimethylbenzene	--	140	--	--	47	--	--	--	1.3	3100[U]
1,3,5-trimethylbenzene	--	170	--	--	36	--	--	--	--	2600[U]
1,3-butadiene	--	2.1	--	--	--	2.4	--	--	1.5	41[U]
2-butanone (MEK)	--	14	--	--	3.7	26	--	--	23	2200000[U]
2-propanol	--	--	--	--	--	6.4	--	--	6.6	NA
4-methyl-2-pentonone (MIBK)	--	--	--	--	--	9.6	--	--	2.9	1300000[U]
4-ethyltoluene	--	42	--	--	6	1.1	--	--	0.91	2200000[W] (s, toluene)
acetone	--	130	--	--	29	93	--	5	--	14000000[U]
carbon tetrachloride	--	4.7	--	--	--	11	--	--	1	6100[W]
chloroethane	--	--	--	--	--	1	--	--	--	4400000[U]
chloromethane	--	2.2	2	--	--	1.6	--	--	1.5	680[U]
cumene	--	1100	--	--	--	--	--	--	--	180000[U]
cyclohexane	--	160	6.3	--	--	2	--	--	1.2	2600000[U]
heptane	--	11	8.9	--	--	5.5	--	--	1.8	310000[U] (s, hexane)
hexane	--	46	2.4	--	--	4.8	--	--	1.1	310000[U]
methylene chloride	--	--	--	--	1.5	--	--	--	--	460000[UX]
naphthalene	--	310	--	--	25	--	--	--	--	1300[UX]
styrene	--	1000	--	--	--	--	--	--	--	440000[U]
dichlorodifluoromethane	--	--	--	--	--	1.8	--	--	2.8	88000[U]
trichlorofluoromethane	--	--	--	--	--	1.6	--	--	1.7	310000[U]
ethanol	--	13	160	--	4.5	240	--	--	41	NA

Appendix C Identification of COPC in Vapour Data

Notes:

*	Maximum of data collected onto primary and secondary sample
#	Benzene data not considered further in this assessment. Data reported by HLA in 2006 at these locations utilised a carbon tube with analysis via solvent extraction. No QA assessment was conducted on the data which would have identified the need to raise the LOR for benzene as it is a component of the solvent used for extraction. The levels reported in the samples highlighted are representative of the solvent extraction methodology and are not considered positive detections for the purpose of further consideration and use in this assessment.
--	not detected above the LOR or not reported
1	Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)
2	Samples collected in 2007 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).
3	Samples collected in 2008 by URS using summa canisters over 8 hours. The location of the URS soil gas installation varies slightly from the HLA location and URS 2007 locations (within 3m).
N	Soil gas screening level based on air investigation level presented in NEPM (2004), annual average.
W	Soil gas screening level based on ambient air guideline from the WHO, annual average and where relevant a non-threshold risk of $1x10^{-6}$.
U	Soil gas screening level based on industrial air RSL from USEPA, annual average. Where a RSL is not available a value (noted as U04) adopted from USEPA Region IX PRG (2004) for ambient air.
S	No guideline available hence an appropriate surrogate compound adopted for the purpose of screening. The surrogate adopted is noted.
X	Guideline adopted based on noncarcinogenic endpoints. Review of the dose-response data for the chemical indicates a threshold approach is relevant (not genotoxic carcinogens) and the data adopted for deriving the noncarcinogenic guideline is consistent with that adopted for the purpose of addressing health effects on the basis of a threshold approach.
NA	No guideline available for ethanol and propanol. However as these compounds have a low toxicity and detections of these compounds in soil gas are not considered to be of concern with respect to human health.

Rows highlighted are chemicals considered to be present in soil gas (and flux emissions) at levels considered to be potentially significant and have been considered COPC for further assessment.

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Appendix C Identification of COPC in Vapour Data

Table C-14 Flux Emissions Data Relevant to Off-Site Areas above Plume 1 and Identification of COPC

Chemical Detected	Flux Emission Rate (µg/m^2/min)			
	AS8	AS9	AS10	
	2006[1]	2006[1]	2006[1]	2007[2]
Potentially Significant Chemicals Identified in Soil Gas				
1,1,2-trichloroethane	--	--	--	--
1,2-dichloroethane	--	--	--	--
Other Chemicals Detected in Flux Emissions				
toluene	0.11	--	--	0.025
ethylbenzene	0.32	--	--	--
xylenes	3.25	--	--	--
2-butanone (MEK)	--	--	--	0.054
4-methyl-2-pentonone (MIBK)	--	--	--	0.027
acetone	--	--	--	0.25
chloromethane	--	--	--	0.015
methylene chloride	--	--	--	0.33

Notes:

-- not detected above the LOR or not reported

1 Samples collected in 2006 by HLA using sorbent tubes (variable time of sampling ranging up to 80 minutes)

2 Samples collected in 2007 by URS using summa canisters over 8 hours. The location of the URS permanent soil gas installation varies slightly from the HLA location (within 3m).

Table C-15 Indoor Air Data Relevant to Off-Site Areas above Plume 1

Chemical Detected#	Ambient Air Concentration (µg/m^3)			
	19-21 Christina Road	23 Christina Road Currently a furniture factory		
	2005[1]	2005[1]	(AM1) 2007[2]	(AM1) 2008[3]
1,2-dichloroethane (EDC)	--	17	--	--
chlorobenzene	10	--	--	--
benzene	4	--	--	1.8
ethylbenzene	4	54	380	90
xylenes	27	77	850	227
1,4-dichlorobenzene	12	--	--	--
toluene	--	--	840	390

Notes:

1 Maximum reported from 6 samples collected by CH2MHIll in 2005. Note no details on sampling methodology, locations, indoor air sources and QA available for the data presented.

2 Reported from data collected by URS in 2007.

3 Reported from data collected by URS in 2008.

* Maximum of primary and secondary sample.

Chemicals detected in air during sampling in 2003 with additional chemicals detected during sampling in 2007 only included where the concentrations reported differ from expected range of urban\industrial air concentrations (refer to URS 2007 for further discussion). Additional chemicals have been included based on review of data collected by URS in 2008. Additional chemicals are those where detected concentrations were significantly higher than reported by URS in 2007.

With respect to the data collected within the off-site buildings, the following can be noted:

- It is not known whether the work places in areas where the CH2MHill data were collected utilised products (in particular glues and solvents) containing chlorinated chemicals as the business areas were not fully surveyed and the quality and reliability of the data is unknown. Businesses in the area identified included a furniture manufacturer. It is reasonable to expect that some of these businesses could utilise cleaners or other products that contain the chemicals detected in ambient air. Hence it is difficult to identify whether the data reflects emissions to air derived from the groundwater plumes, or whether there is contribution from fugitive emissions from the business areas and regional urban air contaminants.

Appendix C Identification of COPC in Vapour Data

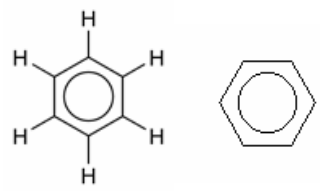
- Ambient air data collected by URS in 2007 or 2008 within 23 Christina Road and other buildings sampled did not report detectable concentrations of chlorinated chemicals in groundwater; in particular 1,2-dichloroethane (EDC) was not detected at any location. Data collected within this building appears consistent with fugitive emissions that might be associated with the business currently operating within the premises. On the basis of the ambient air data collected above Plume 1 by URS in 2007 and 2008, and consideration of the discussion presented for areas located above Plume 2, significant vapour intrusion does not appear to be occurring within buildings located above Plume 1.

Hence it is considered likely that the ambient air data collected by CH2MHill where elevated levels of chlorinated compounds were reported could be associated with fugitive emissions at the time of sampling, rather than indicating emissions from a groundwater source.

The quantification of risk has been undertaken on the basis of maximum measured soil gas and flux emissions data only. It should be noted that while the ambient air data has not been used further in the calculation of risk, the collection of the data is important in reviewing the potential significance of vapour intrusion into these buildings.

Appendix D Toxicity Summaries

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 2006 and ORNL Database 2006):

CAS No	71-43-2
Chemical Formula	C_6H_6
Molecular Weight	78.12
Vapour Pressure	95.2 mmHg at 25^{o}C
Vapour Density	2.77
Density	0.8787 g/ml at 20^{o}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^{o}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.

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

General

There is no clinical disease which is unique to benzene toxicity. However the effects on the haemotopoietic 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).

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 thrombocytopema. Continued exposure may result in marrow aplasia and pancytopenia, an often fa6eaql 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.

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 µg/kg/day. The guideline dose suggested is 1.7 µg/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 2006) 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/m^3 *STEL: none 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 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:

$SF\ (mg/kg/day)^{-1}$ = *Risk/Intake(mg/kg/day)*

= *[Risk x Body Weight]/[Concentration (in water) x Ingestion Rate)]*

= $[1x10^{-6}$ *x 70kg]/[0.001mg/L x 2L/day]*

= $0.035\ (mg/kg/day)^{-1}$

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 $4.4 – 7.5x10^{-6}\ (\mu g/m^3)^{-1}$ with a geometric mean of $6x10^{-6}\ (\mu g/m^3)^{-1}$ (i.e. for an air concentration of 1 $\mu g/m^3$, the lifetime risk is estimated to be $6x10^{-6}$). As no value within the range is recommended, the geometric mean has been used, which is equivalent to the following slope factor:

$SF\ (mg/kg/day)^{-1}$ = *Risk/Intake(mg/kg/day)*

= *[Risk x Body Weight]/[Concentration (in air) x Inhalation Rate)]*

= $[6x10^{-6}$ *x 70kg]/[0.001mg/*m^3 *x 20* m^3*/day]*

= $0.021\ (mg/kg/day)^{-1}$

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/m3), fertility effects (NOAEL of 96 mg/m3) and developmental toxicity (NOAEL of 31.9 mg/m3) has been undertaken to determine relevant Margins of Safety (MOS).

US

The USEPA (available from IRIS 2006) 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.
- Oral slope factor of 0.015 to 0.055 $(mg/kg/day)^{-1}$ 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 $2.2x10^{-6}$ to $7.8x10^{-6}$ $(\mu g/m^3)^{-1}$ 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 (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	Slope Factor = 0.035 $(mg/kg/day)^{-1}$ (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 = $6x10^{-6}$ $(\mu g/m^3)^{-1}$ (WHO, 2000), with an equivalent inhalation slope factor of 0.021 $(mg/kg/day)^{-1}$. Occupational inhalation exposure (NOHSC, current): TWA: 1ppm = 3.2 mg/m^3 STEL: NA

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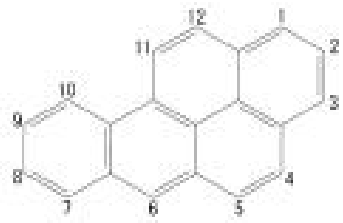
GENERAL



Benzo(a)pyrene (also known as benzo(d,e,f)chrysene, 3,4-benzopyrene, 3,4-benzpyrene, 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	5.49×10^{-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	9×10^{-6} cm^2/s
Henry's Law Coefficient	4.9×10^{-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/m^3 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/m^3. 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 and 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:

$SF\ (mg/kg/day)^{-1}$ = *Risk/Intake(mg/kg/day)*

= *[Risk x Body Weight]/[Concentration (in water) x Ingestion Rate)]*

= $[1x10^{-5} \times 70kg]/[0.0007\ mg/L \times 2\ L/day]$

= $\mathbf{0.5\ (mg/kg/day)^{-1}}$

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 $8.7x10^{-5}$ $(ng/m^3)^{-1}$ (ie for an air concentration of 1 ng/m^3, the lifetime risk is estimated to be $8.7x10^{-5}$).

The unit risk value is equivalent to the following slope factor:

$SF\ (mg/kg/day)^{-1}$ = *Risk/Intake(mg/kg/day)*

= *[Risk x Body Weight]/[Concentration (in air) x Inhalation Rate)]*

= $[8.7x10^{-5} \times 70kg]/[0.000001mg/m^3 \times 20\ m^3/day]$

= $\mathbf{304.5\ (mg/kg/day)^{-1}}$

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 epidemiologicl studies, the working group recommended the unit risk adopted by the WHO Air Qaulity Guidelined 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, despit 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 un 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 $(mg/kg/day)^{-1}$ and an inhalation unit risk for the evaluation of chronic exposure to BaP that ranges from $1.1x10^{-3}$ to $3.3x10^{-3}$ $(\mu g/m^3)^{-1}$. The unit risk value of $1.1x10^{-3}$ $(\mu g/m^3)^{-1}$ is considered the best value for the assessment of inhalation exposures as it is based on an in halation 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 $(mg/kg/day)^{-1}$ (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 $8.7x10^{-5}$ (per ng/m^3), equivalent to 304.5 $(mg/kg/day)^{-1}$ (WHO 2000 and NEPC 2002). Occupational inhalation exposure evaluated using the following (NOHSC): *No guideline available, exposure to be controlled to the lowest practicable level.*

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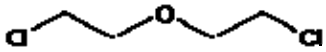

Bis(2-chloroethyl) ether (also known as dichloroethyl ether, dichloroethyl oxide, and bis(β -chloroethyl) ether and abbreviated to **BCEE**) is a β-chloroalky1 ether. BCEE is reported to be used as an intermediate reactant in the synthesis of the methyldithiocarbamic acid fungicide commonly known as 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 polymeners 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	$C_4H_8Cl_2O$
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-chloralkyl 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 $(mg/kg/day)^{-1}$ (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 $(\mu g/m^3)^{-1}$ (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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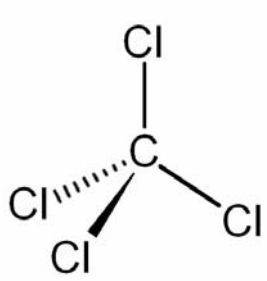

Carbon Tetrachloride (also known as carbona, carbon chloride, tetrachloromethane, carbon tet, methane tetrachloride, perchloromethane, tetrachlorocarbon) is predominantly a man-made compound, however it has been detected in volcanic emission gasses. It has also been suggested that carbon tetrachloride 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 chloroflurocarbons (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^{o}C
Vapour Density	5.32
Density	1.59 g/ml at 25^{o}C
Solubility	800 mg/L at 25^{o}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^{o}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 maybe 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 it 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/m^3 *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/m^3 based on 90-day inhalation study on rats (NOAEL 6.1 mg/m^3) and 100 uncertainty factor.
- TC = 0.0067 mg/m^3 based on 6-month inhalation study on rats (NOAEL 32 mg/m^3), 1000 uncertainty factor and 5/7 conversion.
- TC = 0.0114 mg/m^3 based on a 2-year inhalation study on rats (LOAEL 32 mg/m^3), 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/m^3*** 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/m^3)$^{-1}$*** 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-chronc 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/m^3) based on hepatic effects in animals (inhalation study).
- Chronic inhalation MRL = 0.03 ppm (0.19 mg/m^3) 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 $\boldsymbol{4.2x10^{-5}\ (\mu g/m^3)^{-1}}$.
- Chronic Inhalation reference Exposure Level ($\boldsymbol{REL) = 0.04\ mg/m^3}$ based on hepatic effects in guinea pigs.
- Acute inhalation REL = 1.9 mg/m^3 (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/m^3 (average) to 0.004 mg/m^3 (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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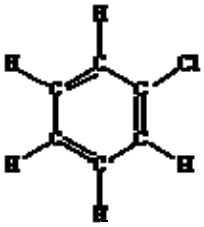
GENERAL



Chlorobenzene (also known as monochlorobenzene; benzene chloride, phenyl chloride and commonly abbreviated to **MCB**) is a monocyclic aromatic compound with one hydrogen atom on the benzene ring substituted with one chlorine. It is produced by chlorination of benzene in the liquid phase with a catalyst. MCB is most commonly used as a carrier solvent for pesticides, in the formulation of rubber polymers and as a carrier for textile dyes.

PROPERTIES

MCB is a colourless liquid at ambient temperature with an aromatic almond-like odour. MCB has a relatively high vapour pressure, moderate octanol-water partition coefficient and moderate to low water solubility. MCB is soluble in alcohol, ether and benzene. Technical grade MCB is typically 99% pure with < 0.05% benzene and < 0.1% dichlorobenzenes as contaminants. . Key properties are presented below (ATSDR 1990, USEPA 2004 and ORNL Database 2006):

CAS No	108-90-7
Chemical Formula	C_6H_5Cl
Molecular Weight	112.56
Vapour Pressure	8.8 mmHg at 20^oC
Vapour Density	3.9
Density	1.106 g/ml at 20^oC
Solubility	500 mg/L at 20^oC
Air Diffusion Coefficient	0.073 cm^2/s
Water Diffusion Coefficient	8.7 x 10^{-6} cm^2/s
Henry's Law Coefficient	0.00358 $atm.m^3/mol$
	= 0.127 at 25^oC (unitless)
Koc	268 cm^3/g
Log Kow	2.84
Odour Threshold	1-8 mg/m^3
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0407

EXPOSURE

Exposure of the general population to MCB may 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 MCB in drinking water or soils (particularly near contaminated sites. MCB has not been found in Australian drinking waters (NHMRC, 2004).

If released into the environment the following can be noted with respect to MCB (CEPA, 1992):

- Air: Based on its relatively high vapour pressure and moderate to low water solubility, it is likely that the atmosphere plays an important role in the distribution and ultimate fate of MCB. Once released to the atmosphere, either directly or by volatilization from other media, MCB is expected to

photooxidize relatively quickly in a reaction with hydroxyl radicals producing phenols and their subsequent degradation products. The hydroxyl radical consumed by this reaction may impact negatively on the atmospheric fate of a number of greenhouse gases since it represents an important sink for these substances. Photolysis, another prevalent atmospheric process, is considered an unlikely transformation process because MCB does not absorb in the range of wavelengths reaching the surface of the earth.

- Soil: The bulk of the MCB released onto soil during, for example, pesticide application, is expected to volatilize, either directly or subsequently, to the atmosphere. Based on its organic carbon sorption coefficient the soil mobility potential of MCB is medium.

- Water: On the basis of several studies in which the behaviour of MCB in the aquatic environment has been examined, it has been concluded that none of microbiological degradation, hydrolysis or photolysis were significant transformation processes. Furthermore, MCB was not found to bioconcentrate in significant quantities in aquatic biota.

HEALTH EFFECTS

General

The following information is available from RIVM (2001), CEPA (1992) and ATSDR (1990). There is no clinical disease which is unique to MCB toxicity.

MCB is readily absorbed through the lungs and gastrointestinal tract. Given its lipophilic nature, it is likely that it is also absorbed well through the skin; however, quantitative data on uptake are not available for any of these routes of exposure. Once absorbed, MCB is rapidly distributed to many tissues with concentrations being greatest in adipose tissue.

MCB is primarily metabolized by oxidative reactions involving the mixed-function oxidase mediated enzymes to ortho-, meta- or parachlorophenols, the glutathione, glucuronic acid or sulphate conjugates of which are excreted in the urine. Reactive intermediates of the metabolism of MCB, possibly arene oxides and chlorophenols, can bind to cellular proteins. Binding of these metabolites appears to be correlated with necrotic pathological damage in the kidneys and liver in studies undertaken on rodents.

Data on the health effects of MCB in humans are limited. Effects on the central nervous system, neonatal development and skin have been reported in occupationally exposed workers, but the available data is not adequate to provide conclusive data as these studies also involved exposures to other chemicals. Studies in animals have reported exposures to high concentrations of MCB affects the neurological system, liver and kidneys.

Long term studies have shown liver and kidney toxicity as well as depression of bone marrow activity in mice and myeloid depletion of the thymus, spleen or bone marrow in rats and mice.

Available data shows that MCB does not affect reproduction of cause birth defects.

Genotoxic Effects

MCB was not mutagenic in a number of bacterial test systems. It has induced chromosomal aberrations in bacteria and plants (one test only) but not in mammalian systems. On the basis of the available information MCB is not considered to be genotoxic.

Cancer

There is limited evidence for carcinogenicity in animals and insufficient evidence in humans. Review of carcinogenicity by RIVM (2001) found that there was no evidence for carcinogenicity of MCB.

TOXICITY CLASSIFICATION

MCB is classified by the USEPA as "not classifiable as to human carcinogenicity" (Category D) due to inadequate human and animal data and predominantly negative genetic toxicity data.

IARC has not evaluated MCB.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have derived a drinking water guideline of 0.3 mg/L for MCB using a ***TDI of 0.086 mg/kg/day*** based on a no effect level of 60 mg/kg/day from a 2-year oral study on rats, a safety factor of 500 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 MCB, the following have been established:

TWA: 10 ppm, equivalent to 46 mg/m^3

STEL: none established

WHO

The WHO (Drinking Water Guideline 2004) provide a guideline value for MCB of 0.3 mg/L based on a ***TDI of 0.086 mg/kg/day*** following the same approach outlined by NHMRC (as above with a body weight of 60kg rather than 70kg).

The WHO (2000) has established a tolerable concentration (TC) for MCB in air. The ***TC of 0.5 mg/m^3***, based on an annual average, is based on decreased food intake, increased organ weight, lesions and changes in blood parameters in rats.

EU

No assessment of MCB is available from the EU.

RIVM

The Dutch (RIVM, 2001) have provided a review of MCB and identified the following threshold values for the assessment of exposures to MCB:

- Oral TDI = 0.2 mg/kg/day;
- Inhalation provisional tolerable concentration in air = 0.5 mg/m^3; and
- Background intakes expected to be <0.0009 mg/kg/day.

US

The USEPA (IRIS current in 2006) has derived an oral reference dose ***(RfD) of 0.02 mg/kg/day*** based on liver effects in a 13-week dog study. No inhalation reference concentration was derived and, as MCB was considered non-carcinogenic, no slope factors were derived.

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with MCB. The levels established (valid in 2006) are:

- Intermediate oral MRL = 0.4 mg/kg/day based on liver effects

The California Air Resources Board (CARB and OEHHA) has established a reference exposure level of 1 mg/m^3 for MCB in air based in increased liver weights, hepatocellular hypertrophy, renal degeneration and inflammation and testicular degeneration 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. Limited data is available regarding environmental levels of MCB in Australia, noting that MCB has not been found in drinking water in Australia (NHMRC). MCB 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 MCB 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 MCB 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 MCB 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 = 0.086 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	TC = 0.5 mg/m3 (WHO, 2000), which is equivalent to a TDI of 0.14 mg/kg/day (based on70 kg body weight and 20m^3 air inhaled per day) Occupational inhalation exposure (NOHSC, current to 2006): TWA: 10 ppm = 46 mg/m^3 STEL: NA
Background	Low – considered to be 0%

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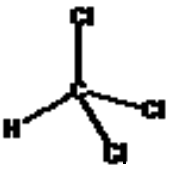
GENERAL



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^{o}C
Vapour Density	4.1
Density	1.48 g/ml at 25^{o}C
Solubility	7920 mg/L at 25^{o}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^{o}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 (CCl_2O). The reaction is P450-mediated and occurs in both the liver and the kidney. The major end product of chloroform metabolism is carbon dioxide (CO_2), 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 hepatoxicity 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 area 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/m^3 *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 (hepatoxicity) 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 hepatoxicity in dogs (derived in 1994 from oral studies) and a ***TC =0.14 mg/m^3 (WHO, 2004).*** WHO (2000b) have also published an inhalation unit risk of ***4.2x10^{-7}(μg/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 ***2.3x10^{-5} (μg/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 reviwed 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/m^3) based on hepatic effects in mice (inhalation study)
- Intermediate inhalation MRL = 0.05 ppm (0.248 mg/m^3) based on toxic hepatitis in workers (inhalation study)
- Chronic inhalation MRL = 0.02 ppm (0.099 mg/m^3) 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 $\boldsymbol{5.3x10^{-6}\ (\mu g/m^3)^{-1}}$.
- Chronic Inhalation reference Exposure Level ($\boldsymbol{REL) = 0.3\ mg/m^3}$ based on liver toxicity, kidney toxicity and developmental toxicity.
- Acute inhalation $\boldsymbol{REL = 0.15\ mg/m^3}$ (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 µg/kg/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 = 4.2x10^{-7} (μg/m^3)$^{-1}$ (WHO, 2000), with an equivalent inhalation slope factor of 0.0015 (mg/kg/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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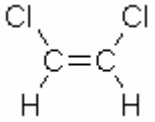
GENERAL

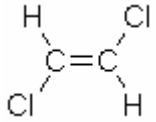
1,2-Dichloroethene (also known as **1,2-DCE**, 1,2-dichloroethylene, acetylene dichloride and dioform) exists in two isomeric forms, *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene, that are colourless, volatile liquids with a slightly acrid odour. 1,2-Dichloroethene is prepared commercially 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 alkalies to form chloracetylene 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 tetraoxide. 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	**cis-isomer**	**trans-isomer**
CAS No:	156-59-2	156-60-5
Chemical Formula	$C_2H_2Cl_2$	$C_2H_2Cl_2$
Molecular Weight	96.95	96.95
Vapour Pressure	180 mmHg at 20^{o}C	265 mmHg at 20^{o}C
Vapour Density	1.6	1.8
Density	1.28 g/ml at 20^{o}C	1.26 g/ml at 20^{o}C
Solubility	3500 mg/L at 20^{o}C	6300 mg/L at 20^{o}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^{o}C (unitless)	0.00938 atm.m^3/mol = 0.384 at 25^{o}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-DEC 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/m^3

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.

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/m^3. A TC of 0.060 mg/m^3 was established for *trans*-1,2-DCE based on a LOAEL of 185 mg/m^3 (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 μg/m^3) and a maximum reported concentration of 2.1 ppb (8.3 μg/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 μg/kg/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.

	***Cis*-1,2-DCE**	***Trans*-1,2-DCE**
	The *cis*-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 *trans*-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 *cis*-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 *trans*-isomer. This value is used for both isomers, but is only derived from studies on the *trans*-isomer)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.	
Inhalation	No inhalation studies are available for the evaluation of exposure to *cis*-1,2-DCE, hence the oral value has been used for the evaluation of inhalation exposures. **RfD = 0.01 mg/kg/day** (it is noted that the RIVM present a provisional TC for the *cis*-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). Occupational inhalation exposure (NOHSC) for 1,2-DCE (both isomers): TWA: 200 ppm, equivalent to 793 mg/m^3 STEL: NA	Limited inhalation studies are available for the evaluation of exposure to *trans*-1,2-DCE, hence the oral TDI has been used for the evaluation of inhalation exposures. **TDI = 0.017 mg/kg/day** (it is noted that the RIVM present a provisional TC for the *trans*-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). Occupational inhalation exposure (NOHSC) for 1,2-DCE (both isomers): TWA: 200 ppm, equivalent to 793 mg/m^3 STEL: NA
Background	Negligible	Negligible

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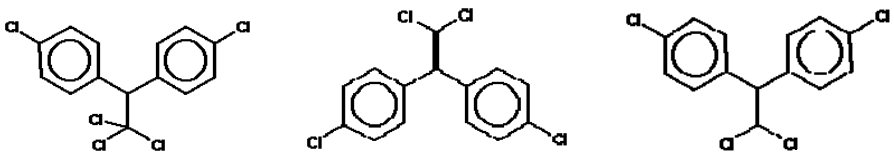
GENERAL

DDT is an organochlorine insecticide that has found a broad range of agricultural and non-agricultural applications. In 1972, DDT use was banned in many parts of the world, except for use in controlling emergency public health problems. DDT is still used in certain parts of the world to control vector-borne diseases, such as malaria. The release of DDT into the environment occurs primarily through spraying applications onto agricultural crops, forest lands, other nonagricultural land, and homes (for the control of disease-bearing vectors). Both DDD and DDE are degradation products of DDT. DDD was also manufactured and used as an insecticide, but to a much lesser extent than DDT. DDE has no commercial use, but is commonly detected along with DDT at concentrations in the environment that often exceed those measured for DDT.

PROPERTIES

The term DDT is generally understood throughout the world and refers to *p,p'* -DDT (1,1 -[2,2,2-trichloroethylidine]-bis [4-chloro-benzene]). The compound's structure permits several different isomeric forms, such as *o,p'* -DDT (1-chloro-2-[2,2,2-trichloro-1-(4-chloro-phenyl) ethyl] benzene). The term DDT is also applied to commercial products consisting predominantly of *p,p'* -DDT with smaller amounts of other compounds. All isomers of DDT are white crystalline, tasteless and almost odourless solids. Technical DDT, which is principally the *p,p'* isomer (also referred to a 4,4'-isomer), has been formulated in almost every conceivable form. Key properties relevant to the *p,p'*-isomers are presented below (ATSDR 2002 and ORNL Database 2006):

Property	DDT	DDE	DDD
CAS No:	50-29-3	72-55-9	72-54-8
Chemical Formula	$C_{14}H_9Cl_5$	$C_{14}H_8Cl_4$	$C_{14}H_{10}Cl_4$
Molecular Weight	354.49	318.03	320.05
Vapour Pressure	$1.6x10^{-7}$ mmHg at 20°C	$6.0x10^{-6}$ mmHg at 20°C	$1.35x10^{-6}$ mmHg at 20°C
Vapour Density	NA	NA	NA
Density	0.98-0.99 g/ml at 20°C	NA	1.385 g/ml at 20°C
Solubility	0.025 mg/L at 25°C	0.12 mg/L at 25°C	0.090 mg/L at 25°C
Air Diffusion Coefficient	0.0137 cm^2/s	0.0144 cm^2/s	0.0169 cm^2/s
Water Diffusion Coefficient	4.95×10^{-6} cm^2/s	5.87×10^{-6} cm^2/s	4.76×10^{-6} cm^2/s
Henry's Law Coefficient	$8.3x10^{-6}$ atm.m^3/mol = 0.00034 at 25°C (unitless)	$2.1x10^{-5}$ atm.m^3/mol = 0.0017 at 25°C (unitless)	$4.6x10^{-6}$ atm.m^3/mol = 0.00027 at 25°C (unitless)
Koc	220000 cm^3/g	153000 cm^3/g	153000 cm^3/g
Log Kow	6.91	6.51	6.02
Odour Threshold	NA	NA	NA
Permeability Constant	1.06	0.916	0.4



EXPOSURE

Exposure of the general public and workers to DDT, DDE, and DDD has been declining since the ban on the use of DDT, at rates that depend on geographic location and environmental conditions. The predominant route of exposure of the public to DDT and its metabolites is through the diet. Although DDT and its metabolites are ubiquitous in the atmosphere, they are present in such low concentrations that exposures through inhalation or dermal contact are considered to be negligible. Exposure to DDT in drinking water is considered negligible.

Upon introduction into the environment, DDT will enter soil, water, or air. The following is available from ATSDR (2002):

> DDT and its metabolites are essentially immobile in soil, becoming strongly absorbed onto the surface layer of soils. Likewise, as a consequence of their extremely low water solubilities, DDT and its metabolites become absorbed onto particulates in water and settle into sediments. Because of its chemical characteristics, DDT can undergo long-range transport through the atmosphere in a process known as "global distillation" where DDT migrates from warmer regions to colder regions through repeated cycles of volatilization from soil and water surfaces followed by deposition of DDT onto surfaces through dry and wet deposition processes.
>
> This long-range transport of DDT results in the wide dispersion of DDT and its metabolites throughout the world, even into remote areas, such as the Arctic or Antarctic regions. The rate and extent of disappearance of DDT may result from transport processes as well as from degradation and transformation. Both the slow degradation of DDT and its metabolites and the ban on DDT use in the early 1970s in most of the world have contributed to a decrease in the levels of these compounds in the environment over the past 30 years. DDT can be degraded through atmospheric photooxidation in air or photolysis on the surface of water or soil. DDT can undergo slow biodegradation through reductive dechlorination to form DDE and DDD, and then be further degraded to form other metabolites.
>
> The persistence of DDT and its metabolites, in combination with their high lipophilicity, have contributed to the bioaccumulation (increasing concentration of a chemical in an organism which exceeds that in its environment) and biomagnification (increasing concentration of a chemical in an organism as a function of trophic level) of DDT and its degradation products in the environment. DDT, DDE, and DDD accumulate in fatty tissues, with tissue concentrations typically increasing with the trophic level of the organism. In addition to DDT, DDE, and DDD, methylsulfonyl derivatives of DDE can be formed in higher organisms, such as seals, polar bears, beluga whales, and humans, and are found predominantly in the liver and fatty tissues.

HEALTH EFFECTS

General

The following information is available from WHO (2003), USDOE (1994) and ATSDR (2002). There is no clinical disease which is unique to DDT, DDE or DDD toxicity. Typically, exposure does not occur to DDT, DDE or DDD individually, rather it typically occurs to a mixture of the three compounds as DDE and DDD are degradation and metabolic products of DDT. In addition DDT, DDE and DDD can exist in three isometric forms based on the relative proportion of the chlorine substitutions on the two chlorophenyl rings. The most common isomer of DDT, DDE or DDD in the environment is the *p,p'*-isomer. Technical grade DDT contains 65-8-% of *p,p'*-DDT.

DDT, DDD and DDE are absorbed following inhalation, oral and dermal exposure. All three compounds are readily distributed in the lymph and blood to all the body tissues and ultimately stored in proportion to the lipid content of the tissues, regardless of the route of exposure. Excretion of DDT in the form of its metabolites is largely via urine. Metabolism of DDT results in primary transformation ot DDD and DDE which are further metabolised to the major urinary metabolite DDA.

Once absorbed DDT (DDE and DDD) are very slowly transformed. Once stored in fatty tissue, DDT residues are sequestrated and stabilised unless they are mobilised either through lactation or significant weight loss, which burns fat. Organochlorines appear to transfer freely across the placenta from mother to foetus.

DDT is best known for its impairment of nerve impulse conduction. In addition to being a neurotoxicant, DDT is capable of producing marked alterations of reproduction and development in animals. This is attributed to hormone-altering actions of DDT isomers and/or metabolites, with *o,p*'-DDT isomer having the strongest estrogen-like properties. The isomer p,p'-DDE, a metabolite of DDT and persistent environmental pollutant has been shown to alter the development of reproductive organs in rates. Some studies in human have suggested high DDT/DDE burdens hay be associated with alterations in end points controlled by hormone functions (such as lactation, maintenance of pregnancy and fertility).

DDT, DDD and DDE are all strongly suspected of being environmental endocrine disrupters (chemicals that affect the hormonal system). DDT can have reproductive endocrine effects (as above) and also has a major toxic effect on the adrenal glands. DDT-related deformities in birds include clubbed feet and crossed bills. There is also concern that it has the potential to disrupt the endocrine system of humans.

Cancer and Genotoxic Effects

Studies in animals have shown DDT, DDE and DDD can cause cancer, primarily in the liver. There has been no conclusive link between DDT (and associated compounds) exposure and cancer in humans.

Possible genotoxic effects in humans have been reported in a few studies but due to the simultaneous exposure to other compounds and other confounding effects, the results obtained are considered inconclusive. DDT and related compounds are not mutagenic.

There is sufficient information indicating that the adrenal gland is a target for *o,p'*-DDD toxicity in humans and animals. *o,p'*-DDD has been used in humans to treat adrenocortical carcinoma and benign Cushing's disease.

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 DDT, DDE and DDD in weight-of-evidence group B2, probable human carcinogen, based on increased incidence of lung tumours in mice (DDT), liver tumours in mice (DDT and DDE) and hamsters (DDE) and thyroid tumours in rats (DDT). DDD is structurally similar to DDT and a known metabolite of DDT is also classified as a B2 carcinogen.

IARC has assigned a weight-of-evidence classification of 2B to DDT, possibly carcinogenic to humans based on sufficient evidence in experimental animals but inadequate evidence in humans. DDT was considered a non-genotoxic carcinogen in rodents.

DDT is listed under the Stockholm Convention on Persistent Organic Pollutants.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC 2004) have derived a drinking water guideline of 0.02 mg/L for DDT and its derivatives based on an ADI of 0.02 mg/kg/day (based on the ADI previously used by WHO in derivation of old drinking water guideline, 1984 study). The current listing of ADIs provided by the TGA (2006) presents a Tolerable Daily Intake (TDI) of 0.002 mg/kg/day for DDT based on a NOEL of 0.25 mg/kg/day. No further detail is available on the basis for the TDI.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment" (available within the Hazardous Substances Information System, NIOSH 2005). For DDT, the following have been established:

TWA: 1 mg/m^3

STEL: NA

No occupational exposure levels have been established by NOHSC for DDE or DDD.

WHO

The WHO (Drinking Water Guidelines 2004) has derived a guideline of 0.001 mg/L for DDT and its metabolites based on a PTDI of 0.01 mg/kg/day based on a NOAEL of 1 mg/kg fore developmental toxicity in rats and an uncertainty factor of 100.

The WHO (2000, 2000b) have not evaluated or provided an inhalation guideline values for DDT or its derivatives.

EU

No assessment of DDT or its derivatives is available from the EU.

RIVM

Review of DDT and its metabolites DDD and DDE by RIVM (2001) has provided tolerable daily intakes for DDT, DDD and DDE of 0.0005 mg/kg/day, individually as well as for the sum of DDT, DDD and DDE. This value was established prior to the revision of the ADI established by JPMR (as noted above from WHO).

No inhalation values have been derived.

US

The USEPA (IRIS current in 2006) has derived the following for DDT, DDE and DDD:

- Non-cancer effects: oral reference dose (RfD) of 0.0005 mg/kg/day for DDT based on a 27 week study in rats.
- Oral slope factor for DDT and DDE of 0.34 $(mg/kg/day)^{-1}$.
- Oral slope factor for DDD of 0.24 $(mg/kg/day)^{-1}$.
- No data is currently available for inhalation exposure (carcinogenic or non-cancer) to DDT, however the ATSDR (2002) note that an inhalation unit risk of 9.7x10-5 $(\mu g/m^3)^{-1}$ was derived based on the oral slope factor..

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with DDT. The levels established (valid in 2005) are:

- Acute oral MRL of 0.0005 mg/kg/day based on neurotoxicity effects; and
- Intermediate oral MRL of 0.0005 mg/kg/day based on hepatic effects.

Occupational Limits:

The OSHA and ACGIH has established and 8-hour TLV (or PEL) of 1 mg/m^3 for DDT.

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 DDT, intakes from soil, water and air can be considered to be insignificant as the primary route of exposure is associated with ingestion of food. Based on data presented by Food Standards Australia, total DDT (sum of DDT, DDE and DDD) in the market basket survey comprised <1% of the adopted ADI (the maximum intake was for toddlers and was 0.001 µg/kg/day which is well below the NHMRC TDI, from below, of 0.002 mg/kg/day). On this basis background intakes of DDT and its metabolites are considered to be low.

Toxicity Values

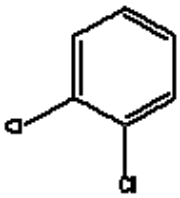
Toxicity data relevant for use in the characterisation of risk to human health have been selected for DDT, DDE and DDD following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

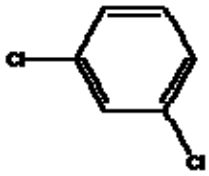
	DDT, DDE and DDD
	DDT is considered a non-genotoxic carcinogen – hence assessment on the basis of threshold approach appropriate. Limited data is available for DDE and DDD however as these are degradation products and metabolites of DDT their toxicity can be considered to be the similar to DDT. As presented within RIVM, the assessment of DDT and its metabolites should consider potential exposure to the sum of these compounds and hence the threshold value adopted for DDT has been considered relevant to the sum of DDT, DDE and DDD. Evaluation of exposure via all routes must be undertaken on the basis of a threshold approach.
Oral	**TDI = 0.002 mg/kg/day** (NRMRC, 2006)
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	No inhalation studies are available for the evaluation of exposure to DDX, hence the oral value has been used for the evaluation of inhalation exposures. Occupational inhalation exposure for DDX: TLV: 1 mg/m^3 (ACGIH and OSHA) STEL: NA
Background	Low

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	$C_6H_4Cl_2$	$C_6H_4Cl_2$	$C_6H_4Cl_2$
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 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.

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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.

- 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:

	1,2-DCB	1,3-DCB	1,4-DCB
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/m^3***, 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/m^3 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/m^3 based on a NOAEL of 60 mg/m^3 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/m^3 based on a NOAEL of 67 mg/m^3 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/m^3 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/m^3 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	Data from NOHSC: TWA = 25ppm = 150 mg/m^3 STEL = 50ppm = 301 mg/m^3	No data available, adopt data for 1,2-DCB	Data from NOHSC: 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 m^3 inhaled per day.

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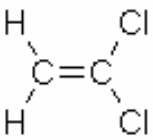
GENERAL



1,1-Dichloroethene (vinylindene 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 hydrochloroflurocarbons (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 photo chemically 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 polyvinylindene (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	$C_2H_2Cl_2$
Molecular Weight	96.95
Vapour Pressure	634 mmHg at 25^{o}C
Vapour Density	3.25
Density	1.21 g/ml at 20^{o}C
Solubility	2420 mg/L at 20^{o}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^{o}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/m^3). 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 (form 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/m^3 (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

REFERENCES

ATSDR, 1994. *Toxicological Profile for 1,1-Dichloroethene*, available on website and current to 2006.

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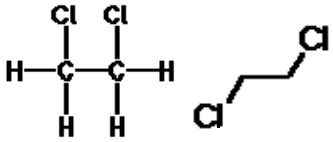
GENERAL



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 trichloethane, 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 on 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 alcohol, chloroform and ether. Key properties are presented below (ATSDR 2001, USEPA 2004 and ORNL Database 2006):

CAS No	107-06-2
Chemical Formula	$C_2H_4Cl_2$
Molecular Weight	98.96
Vapour Pressure	79.1 mmHg at 25^{o}C
Vapour Density	3.4
Density	1.23 g/ml at 20^{o}C
Solubility	8690 mg/L at 20^{o}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
	= 0.0401 at 25^{o}C (unitless)
Koc	17.4 cm^3/g
Log Kow	1.48
Odour Threshold	48.6 to 405 mg/m^3
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00534

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. Intake from food sources is expected to be negligible. Children maybe exposed via the same pathways as adults. EDC has been detected in human milk and hence infancies could be exposed via breast-feeding. Occupational exposures (particularly inhalation and dermal contact) may occur in industries which handle the product.

If released into the environment the following can be noted with respect to EDC:

- 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 in 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 is not expected to be important fate processes. The potential for bioaccumulation in aquatic or terrestrial organisms appears to be low.

HEALTH EFFECTS

General

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.

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. Un-metabolised 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.

The following summary of more specific health effects has been derived from ATSDR (2001).

Death	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.
Hepatic Effects	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.
Renal Effects	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.
Immunological	Immunological effects have not been reported in humans. In mice,

Effects	immunological effects have been reported following both acute inhalation and oral exposure. Due 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 *in vitro* and *in vivo* assays, and binds to DNA in rodents *in vivo*. Review of genotoxicity by Woodward-Clyde (1996) indicated that the available evidence in animals suggests that EDC is genotoxic.
Cancer	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). 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).

TOXICITY CLASSIFICATION

EDC was classified as a "probable" human carcinogen (Category B2) by the USEPA for all routes of exposure based upon evidence from animal studies.

IARC (1999) 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.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004 and derived from WHO, see below) have derived a drinking water guideline of 0.003 mg/L for EDC based on an lifetime excess cancer risk of 1 in 1,000,000. The slope factor used in the derivation of the drinking water guideline can be calculated as follows:

SF (mg/kg/day)$^{-1}$ *= Risk/Intake(mg/kg/day)*

= [Risk x Body Weight]/[Concentration (water) x Ingestion Rate)]

= [1 x10^{-6} x 70kg]/[0.003mg/L x 2 L/day]

= 0.012 (mg/kg/day)$^{-1}$

Worksafe Australia (NOHSC) 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/m^3

STEL: NA

It should be noted that this chemical is flagged for further review based on carcinogenic potential.

WHO

The WHO (Drinking Water Guideline 1993 and 2004) 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 0.012 (mg/kg/day)$^{-1}$*** (as used by NHMRC).

The WHO also notes that data indicate that EDC is less potent when inhaled.

WHO (2000) has undertaken a review of EDC for inhalation exposures. The review indicates that there is sufficient evidence of carcinogenicity in animals based on oral ingestion data. However, animal inhalation data do not provide sufficient evidence of carcinogenicity. Because of deficiencies in extrapolating oral data to inhalation, neither the oral slope factor nor any inhalation value has been recommended by the WHO in this assessment. A guideline value of 0.7 mg/m^3 for a 24-hour average has been derived for non-

carcinogenic endpoints by the WHO (2000) based on a lowest-observed-adverse-effect level from animal studies. It is noted that this guideline value is of short duration exposures only and is recommended for the assessment of accidental release episodes or specific indoor pollution problems.

WHO (2000b) have published a parallel review from a task force which had the aim of providing a more global application of air quality guidelines, in conjunction with the Air Quality Guidelines for Europe (WHO, 2000). WHO (2000b) provide a range of inhalation unit risk values for exposure to EDC in air based on tumour formation in rats. The range of inhalation unit risk values is (0.5 to 2.8)x10^{-6} ($\mu g/m^3$)$^{-1}$ (i.e. for an air concentration of 1 $\mu g/m^3$, the lifetime risk is estimated to be (0.5 to 2.8)x10^{-6}). This is equivalent to the following inhalation slope factor:

SF (mg/kg/day)$^{-1}$ = *Risk/Intake(mg/kg/day)*

= *[Risk x Body Weight]/[Concentration (in air) x Inhalation Rate)]*

= *[(0.5 to 2.8) x10^{-6} x 70kg]/[0.001mg/m^3 x 20 m^3/day]*

= ***0.0018 to 0.0098 (mg/kg/day)$^{-1}$***

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 0.012 (mg/kg/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.

US

The USEPA (IRIS current) 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} ($\mu g/m3$)$^{-1}$*** 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. The levels established (current) 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 (CARB 2002, OEHHA 2000) has established ***inhalation unit risk value of 2.1x10^{-5} (μg/m^3)$^{-1}$*** and a ***chronic reference exposure level for EDC of 0.4 mg/m^3*** based on hepatotoxicity (elevated liver enzyme levels in serum of 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. 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 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 EDC following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

Oral	Slope Factor = 0.012 (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 = 2.8x10^{-6} (μg/m^3)$^{-1}$ (WHO, 2000), with an equivalent inhalation slope factor of 0.0098 (mg/kg/day)$^{-1}$. Occupational inhalation exposure (NOHSC, current): TWA: 10ppm = 40 mg/m^3 STEL: NA
Background	NA

REFERENCES

ATSDR, 2001. *Toxicological Profile for 1,2-Dichloroethane,* available on website and current to 2004.

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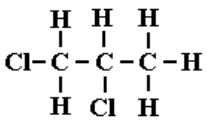
GENERAL



1,2-Dichloropropane (dichloro-1,2-propane, propylene chloride, propylene dichloride and commonly abbreviated to **1,2-DCP**) is produced by chlorination of propylene. 1,2-DCP was once used alone as an insecticide for stored grain, and as a component of several insecticidal and nematocidal soil fumigants. Use of 1,2-DCP is no longer used as a soil fumigant and its content limited in pesticides. Currently 1,2-DCP is used primarily as a chemical intermediate in the production of carbon tetrachloride and the dry-cleaning agent tetrachloroethene (PCE). It is also used as an industrial solvent for fats, oils, resins, waxes and rubber.

Releases into air occur during 1,2-DCP production and its many industrial uses as well as from volatilisation during wastewater treatment and incomplete incineration. As a component of fumigants, 1,2-DCP is released into the air as fugitive emissions. Following its application, it volatilizes and diffuses into ambient air. If released on soil (e.g., as a fumigant, or from landfill and spills), 1,2-DCP will partially volatilise with the remainder leaching into the subsurface soil and groundwater. 1,2-DCP is not rapidly degraded in air or soil and bioaccumulation is not significant.

EXPOSURE AND HEALTH EFFECTS

The general population is primarily exposed to 1,2-DCP from inhalation of contaminated ambient air and from consumption of contaminated drinking water. Workers may be exposed to 1,2-DCP via inhalation and dermal contact during use and/or production. A route of major exposure in the past was by accidentally or intentionally drinking cleaning products that contained 1,2-DCP, but these cleaning materials are no longer produced. 1,2-DCP is rapidly absorbed and quickly removed in the urine and faeces and by the lungs (exhaling).

Primary health effects associated with exposure to 1,2-DCP include effects to the central nervous system, liver and kidney. Drinking 1,2-DCP by humans (i.e., drinking cleaning solutions) has produced poisoning. At these high levels of exposure, effects include dizziness, headache, nausea, injury to the liver and kidneys, anaemia, coma and, ultimately, death. Inhaling high levels of 1,2-DCP by humans, as in deliberate breathing of vapours from cleaning solutions, produces similar effects. No reports have been made of any health effects in humans following low-level exposure to 1,2-DCP for either short or long time periods.

In animal experiments, low concentrations of 1,2-DCP inhaled over short- and long-term periods result in damage to the liver, kidney, and respiratory systems, while exposure to high concentrations resulted in death. Short-term exposure to high concentrations of vapours also causes irritation to the eyes and throat. Oral exposure to 1,2-DCP over short- or long-term periods resulted in damage to the liver and kidneys is seen at low doses, and death occurs at high doses. Irritation of the skin after contact with 1,2-DCP has been seen in both humans and rabbits.

Available studies in humans indicate that oral or inhalation exposures to 1,2-DCP has not been reported to produce cancer in humans. Long-term oral exposure studies in animals has produced evidence of liver

cancer in mice and breast cancer in female rats. The significance of the animal cancer studies to humans is not well understood. 1,2-DCP has not been shown to cause birth defects in humans or animals, but a delay in the growth of bones has been observed in foetal rats following exposure of the mother rats.

Results from in vitro assays for mutagenicity were mixed. The limited in vivo studies indicated negative results. Base on the review presented by WHO (2004), in accordance with IARC, it was concluded that the use of a threshold approach for the toxicological evaluation of 1,2-DCP was appropriate.

PROPERTIES

1,2-DCP is a colourless liquid with a chloroform-like odour that evaporates quickly at room temperature. . Key properties are presented below (ATSDR 1989, USEPA 2004 and ORNL Database 2006):

CAS No	78-87-5
Chemical Formula	$C_3H_6Cl_2$
Molecular Weight	112.99
Vapour Pressure	53.3 mmHg at 25^{o}C
Vapour Density	3.9
Density	1.16 g/ml at 20^{o}C
Solubility	2800 mg/L at 20^{o}C
Air Diffusion Coefficient	0.0782 cm^2/s
Water Diffusion Coefficient	8.73 x 10^{-6} cm^2/s
Henry's Law Coefficient	0.0028 atm.m^3/mol = 0.115 at 25^{o}C (unitless)
Koc	67.7 cm^3/g
Log Kow	1.98
Odour Threshold	0.25 ppm = 1.19 mg/m^3
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00992

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 1,2-DCP in Australia. Review of 1,2-DCP by the WHO (2004) estimated a background daily intakes based on ambient air levels in Japan of 0.074 µg/kg and drinking water levels from the US of 0.54 µg/kg. The inhalation exposures assumed contribute 6% of the adopted threshold value (below for inhalation exposures) and drinking water exposures assumed contribute 3% of the adopted threshold value (below for oral intakes). A background contribution of 10% has been conservatively adopted for 1,2-DCP.

Toxicity Values

Review of available data with respect to 1,2-DCP by the WHO (2004) indicates that the chemical is a potential carcinogen based on limited evidence in animals with limited, equivocal evidence of

genotoxicity. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to 1,2-DCP on the basis of a threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,2-DCP 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: Not evaluated.
IARC: Group 3 not classifiable as to its carcinogenicity to humans.

Toxicity Values:	
Oral	TDI = 0.014 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	Inhalation RfC = 0.004 mg/m^3 (USEPA, current). This is equivalent to a daily intake of 0.0011 mg/kg/day (based on inhalation of 20 m^3/day and 70kg bodyweight). Occupational inhalation exposure (NOHSC, current to 2006): TWA: 75 ppm = 347 mg/m^3 STEL: 110 ppm = 508 mg/m^3
Background	10%

REFERENCES

ATSDR, 1989. *Toxicological Profile for 1,2-Dichloropropane*, available on website and current to 2006.

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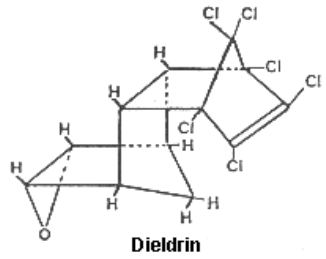

Dieldrin

Dieldrin is an organochlorine pesticide that was manufactured commercially since 1950 and used throughout the world up to the early 1970s. It was used as an insecticide in agriculture for the control of many soil pests and in the treatment of seed. Insects controlled by organochlorine pesticides include termites, grasshoppers, wood borers, beetles, and textile pests. Dieldrin has also been used in public health for the control of tsetse flies and other vectors of debilitating tropical diseases. Dieldrin acts as a contact and stomach poison for insects.

Since the early 1970s, dieldrin has been severely restricted or banned, in a number of countries, from use, especially in agriculture. Aldrin and dieldrin are still present in the environment from these past uses. Dieldrin is the epoxide of aldrin that is formed in animals by biotransormation, and in the environment by chemical oxidation. Therefore dieldrin can be found in areas where Aldrin was originally released. Dieldrin, as with aldrin and endrin are not readily biodegradable, are persistent chlorinated pesticides that accumulate within the food chain.

Dieldrin in soil or water breaks down (degrades) very slowly and persists in the environments for many years. Dieldrin is not considered mobile as it adheres to soil. Dieldrin does not dissolve in water very well and is therefore not found in water at high concentrations. Most dieldrin in the environment attaches to soil and to sediments at the bottoms of lakes, ponds, and streams. Dieldrin can travel large distances by attaching to dust particles, which can then be transported great distances by the wind. Dieldrin can evaporate slowly from surface water or soil. In the air, dieldrin changes to photodieldrin within a few days. Plants can take up dieldrin from the soil and store it in their leaves and roots. Fish or animals that eat dieldrin-contaminated materials store a large amount of the dieldrin in their fat. Animals or fish that eat other animals have levels of dieldrin in their fat many times higher than animals or fish that eat plants.

EXPOSURE AND HEALTH EFFECTS

The following has been obtained from WHO (1989) and RIVM (2001) with respect to dieldrin:

Dieldrin is highly hazardous by the oral and dermal routes. The most common form of human exposure occur through the ingestion of contaminated foodstuff or water, inhalation of vapour, or absorption through skin. The later being an important factor leading to occupational poisoning by dieldrin.

Dieldrin is absorbed from the gastrointestinal tract, the lungs and through intact skin. Dieldrin is selectively stored in body fat, and may be found in fat of people in the general population. It persists in body fat for several years after cessation of exposure. Dieldrin may also be found in blood and in various organs of people in the general population.

The storage of dieldrin is influenced by other chlorinated hydrocarbon insecticides. In periods of starvation stored chlorinated hydrocarbon insecticides may be released into the circulation, sometimes with marked effects.

Most organochlorines are metabolised slowly. In common with other lipophilic xenobiotics, chlorinated hydrocarbon insecticides can be metabolised by the microsomal cytochrome P-450 system in the liver to mainly unknown hydrophilic metabolites. At least one metabolite of dieldrin excreted in the faeces has been identified (9-hydroxydieldrin). Hepatic enzyme induction can result from exposure to dieldrin.

Chlorinated hydrocarbon insecticides act by altering the electrophysiological and associated enzymatic properties of nerve cell membranes, causing a change in the kinetics of Na^+ and K^+ ion flow through the membrane.

Chronic exposure to dieldrin (humans and animals) results in stimulation of the central nervous system. In addition, immunosuppression and adverse reproductive effects have been reported. No data are available on the teratogenity risks.

Dieldrin has been reviewed by the IARC. Orally given to the mouse dieldrin can produce liver tumours. The significance of the marginal increase in incidence of hepatic tumours in mice, following lifetime exposure to dieldrin, is doubtful. In the rat, dog and monkey dieldrin is respectively negative and inconclusive for the oral hepatocarcinogenicity. The IARC has not stated that dieldrin pose no carcinogenic risk to humans.

There is a lack of evidence for genotoxicity of dieldrin and as such a threshold approach is relevant.

PROPERTIES

Technical dieldrin consists of buff to light tan flakes with a mild odour. Pure dieldrin is a white crystalline solid. Key properties are presented below (ATSDR 2002, USEPA 2004 and ORNL Database 2006):

CAS No	60-57-1
Chemical Formula	$C_{12}H_8Cl_6O$
Molecular Weight	380.91
Vapour Pressure	$3x10^{-6}$ mmHg at 25°C
Vapour Density	NA
Density	1.75 g/ml at 20°C
Solubility	0.25 mg/L at 20°C
Air Diffusion Coefficient	0.0125 cm^2/s
Water Diffusion Coefficient	4.74×10^{-6} cm^2/s
Henry's Law Coefficient	$5.2x10^{-6}$ $atm.m^3/mol$
	= 0.000409 at 25°C (unitless)
Koc	10600 cm^3/g
Log Kow	5.2
Odour Threshold	0.041 ppm = 0.64 mg/m^3
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.0445

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 dieldrin in Australia. NHMRC (2004) note that both Aldrin and dieldrin have occasionally been detected in large water storages with concentrations <0.001 mg/L. The frequency of detection has dropped markedly since 1980. Dieldrin was not reported (above the LOR) in the Australian Total Diet Survey (Food Standards Australia, 2003), hence background intakes are expected to be low. This is consistent with review of background exposures presented by RIVM (2001).

Toxicity Values

Review of available data with respect to dieldrin indicates that the chemical is not considered to be genotoxic. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to dieldrin on the basis of a threshold approach.

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 – probable human carcinogen
IARC: Group 3 not classifiable as to carcinogenicity to humans.

Toxicity Values:	
Oral	ADI/PTDI = 0.0001 mg/kg/day (NHMRC 2004 and WHO 2004 for combined total of aldrin and dieldrin).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	No threshold value available from NHMRC, WHO, ATSDR or USEPA and hence adopt oral ADI/PTDI. It is noted that a provisional tolerable concnetyartion in air has been derived by RIVM (2001) of 0.00035 mg/m^3, which is equivalent to the above oral ADI which converted to an tolerable daily intake as it is based on the above oral value. Occupational inhalation exposure (NOHSC, current to 2006): TWA: 0.25 mg/m^3 STEL: NA
Background	Low

REFERENCES

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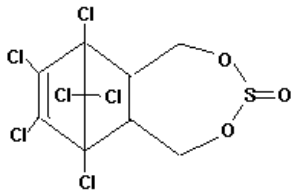
GENERAL



Endosulfan

Endosulfan is a chlorinated cylodiene pesticide predominantly used in Australia within the cotton industry. It is sold as a mixture of two different forms of the same chemical (referred to as α-and β-endosulfan or endosulfan I and endosulfan II). Technical endosulfan (6,7,8,9,10, 10-hexachloro-1,5,5a,6,9,9a, hexahydro 6,9-methano-2,4,3-benzodioxathiepin, 3-oxide) is a brown crystalline substance consisting of alpha- and beta-isomers in the ratio of approximately 70:30. Endosulfan is a manufactured pesticide. It is used to control a number of insects on food crops such as grains, tea, fruits, and vegetables and on non-food crops such as tobacco and cotton. It is also used as a wood preservative.

Both endosulfan isomers are fairly resistant to photo-degradation, but the metabolites endosulfan sulfate and endosulfan diol are susceptible to photolysis. Its half-life in water is estimated to be 4 days, but anaerobic conditions and/or a low pH will lengthen the half-life. In water, it is mainly degraded to endosulfan diol. Fish are extremely sensitive to endosulfan and fish kills have been reported as a result of the discharge of endosulfan into rivers. Agricultural run-off has not caused such a problem.

In soil, endosulfan isomers and its metabolites are not very mobile. The alpha-isomer disappears more rapidly than the beta-isomer. Endosulfan sulfate is the major degradation product in soil. These compounds are not prone to leaching. Biodegradation in soil and water is dependent on climatic conditions and on the type of micro-organisms present.

EXPOSURE AND HEALTH EFFECTS

The following has been obtained from WHO (1998) and EPHC (2003) with respect to dieldrin:

The main source of exposure of the general population is food, but residues have generally been found to be well below maximum residue limits. Because of its use in tobacco farming, smoking may be an additional source of endosulfan exposure. Endosulfan can be absorbed following ingestion, inhalation, and skin contact. Following oral or parenteral dosing, it is rapidly excreted via faeces and urine. Following acute over-exposure, high endosulfan concentrations can temporarily be found in the liver; the concentration in plasma decreases rapidly. The major metabolites are endosulfan sulfate and endosulfan diol. Endosulfan has been found in breast milk of women environmentally exposed and in fatty tissue of children in agricultural areas.

Endosulfan is a chlorinated cyclodiene pesticide with a predominant toxicological effect following acute exposure in humans and animals being over stimulation of the central nervous system. The α-isomer is reported to be more toxic than the β-isomer . Signs of neurotoxicity reported in humans and animals include hyperactivity, involuntary muscle movements, pronounced sensitivity to noise and light, seizures and convulsions.

Review by the WHO and JMPR concluded that endosulfan is not genotoxic, and no carcinogenic effects were noted in long-term studies using mice and rats. The kidney is the target organ for toxicity associated with repeated exposure.

Several studies have shown that endosulfan, alone or in combination with other pesticides, may bind to oestrogen receptors and perturb the endocrine system. Limited information is available on the toxicity of endosulfan sulfate.

PROPERTIES

The following presents key properties are presented below (ATSDR 2000, USEPA 2004 and ORNL Database 2006). It is noted that endosulfan sulfate is a reaction product found in technical endosulfan; it is also found in the environment due to photolysis and in organisms as a result of oxidation by biotransformation. In addition β-endosulfan is slowly converted to α-endosulfan.

	Endosulfan	**α-Endosulfan (Endosulfan I)**
CAS No	115-29-7	959-98-8
Chemical Formula	$C_9H_6Cl_6O_3S$	$C_9H_6Cl_6O_3S$
Molecular Weight	406.92	406.93
Physical state	Crystalline, waxy solid	Crystalline solid
Vapour Density	14	NA
Vapour Pressure	$1x10^{-5}$ mmHg at 25°C	$1x10^{-5}$ mmHg at 25°C
Density	1.735 g/ml at 20°C	NA
Solubility	0.45 mg/L at 25°C	0.53 mg/L at 20°C
Air Diffusion Coefficient	0.0115	NA
Water Diffusion Coefficient	4.55×10^{-6} cm^2/s	NA
Henry's Law Coefficient	$1x10^{-5}$ $atm.m^3/mol$ = 0.00266 at 25°C (unitless)	$1x10^{-5}$ $atm.m^3/mol$ = 0.00266 at 25°C (unitless)
Koc	22000 cm^3/g	NA
Log Kow	3.55	3.83
Odour Threshold	NA	NA
Dermal Absorption	0.01 (unitless)	0.01 (unitless)
Permeability Constant	0.00329	

	β-Endosulfan (Endosulfan II)	**Endosulfan sulfate**
CAS No	33213-65-9	1031-07-8
Chemical Formula	$C_9H_6Cl_6O_3S$	$C_9H_6Cl_6O_4S$
Molecular Weight	406.93	422.9
Physical state	Cream/tan crystalline solid	Brown crystalline solid
Vapour Pressure	$1x10^{-5}$ mmHg at 25°C	$1x10^{-5}$ mmHg at 25°C
Vapour Density	NA	NA
Density	NA	NA
Solubility	0.28 mg/L at 25°C	0.22 mg/L at 20°C
Air Diffusion Coefficient	NA	NA
Water Diffusion Coefficient	NA	NA
Henry's Law Coefficient	$1.91x10^{-5}$ $atm.m^3/mol$	$2.61x10^{-5}$ $atm.m^3/mol$
Koc	NA	NA
Log Kow	3.52	3.66
Odour Threshold	NA	NA
Dermal Absorption	0.01 (unitless)	0.01 (unitless)
Permeability Constant	NA	NA

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. A review of background intakes of endosulfan in Australia has been undertaken (EPHC, 2003) which indicated for a 2-year old, the estimated background exposure from endosulfan residues in air, food and water contributed approximately 7% of the ADI. For the purpose of risk assessment a 10% background intake has been assumed.

Toxicity Values

Review of available data with respect to endosulfan indicates that the chemical (and isomers) is not considered to be carcinogenic or genotoxic. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to dieldrin on the basis of a threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for endosulfan (all isomers and endosulfan sulfate) 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: Not available
IARC: Not available.

Toxicity Values:	
Oral	ADI = 0.006 mg/kg/day (WHO 2004, NHMRC 2004 and TGA).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	No threshold value available from NHMRC, WHO, ATSDR or USEPA and hence it is recommended to adopt the oral ADI. Occupational inhalation exposure (NOHSC, current to 2006): TWA: 0.1 mg/m^3 STEL: NA
Background	10%

REFERENCES

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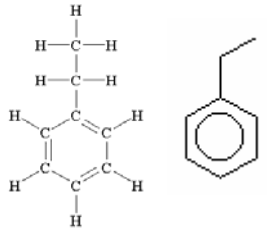
GENERAL



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, HSBD 2006 and ORNL Database 2006):

CAS No	100-41-4
Chemical Formula	C_8H_{10}
Molecular Weight	106.17
Vapour Pressure	9.53 mmHg at 25^{o}C
Vapour Density	3.66
Density	0.867 g/ml at 20^{o}C
Solubility	169 mg/L at 20^{o}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^{o}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

If released into the environment the following can be noted with respect to ethlybenzene (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 removeal 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 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. 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.
Genotoxic Effects	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).

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, applying a safety factor of 1000 and a study duration conversion factor of 5/7.

Worksafe Australia (NOHSC, available in 2006) 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/m^3

STEL: 125 ppm, equivalent to 543 mg/m^3

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/m^3**. 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.

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/m^3 established based on developmental toxicity from a rat and rabbit inhalation studies.

The ATSDR (1997) 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/m^3 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 (2002) and NEPM (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 STEL: 125 ppm, equivalent to 543 mg/m^3
Background	Low

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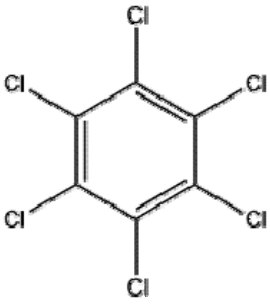
GENERAL



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^{o}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 chlorinates 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_6Cl_6
Molecular Weight	284.79
Vapour Pressure	0.000011 mmHg at 20^{o}C
Vapour Density	9.8
Density	1.57 g/ml at 23^{o}C
Solubility	0.006 mg/L at 20^{o}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^{o}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, tyroid 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, tyroid 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 $Dose_5$, or TD_5 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 TD_5 is calculated directly from the experimental data rather than using the upper or lower confidence limits. Uncertainty factors are then applied to the TD_5 to obtain a guidance value. The TD_5 value was calculated from the results of a two-generation study in rats using a multistage model where the TD_5 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 TD_5 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 TD_5 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 TD_5 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 TD_5 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 $4.6x10^{-4}$ $(\mu g/m^3)^{-1}$*** 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 $5.1x10^{-4}$ $(\mu g/m^3)^{-1}$*** for HCB.

The ACGIH (American Conference of Governmental Industrial Hygienists, 1998) has established a Threshold Limit Value ***(TLV) of 0.002 mg/m^3*** 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 fro 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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GENERAL

Hexachlorocyclohexane (**HCH**) (formerly known as benzene hexachloride (**BHC**)) is a synthetic chemicals that exists in eight chemicals forms or isomers. The different isomers are named according to the position of the hydrogen atoms in the structure of the chemical.

Technical-grade HCH, a mixture of several chemical forms of HCH, was also once used as an insecticide and typically contained about 10–15% of γ-HCH as well as the alpha (α), beta (β), delta (δ), and epsilon (ε) forms of HCH. Virtually all of the insecticidal properties reside in the gamma isomer.

The key isomers of HCH (of commercial significance) are:

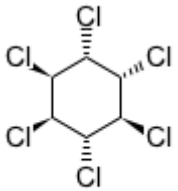
- Alpha-HCH (a-HCH, α-HCH) is a major by-product (65-70%) in the manufacture of lindane (γ -HCH). Its solubility in water is low, but it is very soluble in organic solvents such as acetone, chloroform, and xylene. It is a solid with a low vapour pressure.
- Beta-HCH (b-HCH, β-HCH) is also a by-product (7-10%) in the manufacture of lindane (γ -HCH). Its solubility in water is low, but it is very soluble in organic solvents such as acetone, cyclohexane, and xylene. It is a solid with a low vapour pressure.
- Gamma-HCH (g-HCH, or γ -HCH, commonly called **lindane**), is produced and used as an insecticide on fruit, vegetables, and forest crops, and animals and animal premises. It is a white solid whose vapour may evaporate into the air. The vapour is colourless and has a slight musty. ɣ -HCH is also used in prescription medicine (lotion, cream, or shampoo) to treat and/or control scabies (mites) and head lice in humans.
- Delta-HCH (d-HCH or δ-HCH) is also by-product (7%) in the manufacture of lindane (γ -HCH).

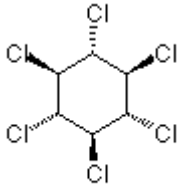
PROPERTIES

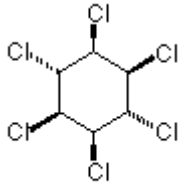
Key properties of HCHs are presented below (ATSDR 2005, USEPA 2004 and ORNL Database 2006):

Physical Properties of Hexachlorocyclohexanes

	α-HCH	β-HCH	γ-HCH	δ-HCH
CAS No	319-84-6	319-85-7	58-89-9	319-86-8
Chemical Formula	$C_6H_6Cl_6$	$C_6H_6Cl_6$	$C_6H_6Cl_6$	$C_6H_6Cl_6$
Molecular Weight	290.83	290.83	290.83	290.83
Vapour Pressure	$4.5x10^{-5}$ mmHg at 25°C	$3.6x10^{-7}$ mmHg at 20°C	$4.2x10^{-5}$ mmHg at 20°C	$3.5x10^{-5}$ mmHg at 25°C
Vapour Density	1.85 (mixed isomer)	1.85 (mixed isomer)	1.85 (mixed isomer)	1.85 (mixed isomer)
Density	1.87 g/ml at 20°C	1.89 g/ml at 19°C	1.89 g/ml at 20°C	NA
Solubility	8 mg/L at 20°C	8 mg/L at 20°C	8 mg/L at 20°C	8 mg/L at 20°C
Air Diffusion Coefficient	0.0142 cm^2/s	0.0142 cm^2/s	0.0142 cm^2/s	NA
Water Diffusion Coefficient	7.34×10^{-6} cm^2/s	7.34×10^{-6} cm^2/s	7.34×10^{-6} cm^2/s	NA
Henry's Law Coefficient	$6.6x10^{-6}$ atm.m^3/mol = 0.000499 at 25°C (unitless)	$4.5x10^{-7}$ atm.m^3/mol = 0.000018 at 25°C (unitless)	$3.2x10^{-6}$ atm.m^3/mol = 0.00021 at 25°C (unitless)	0.0000175 at 25°C (unitless)
Koc	3380 cm^3/g	3380 cm^3/g	3380 cm^3/g	3380 cm^3/g
Log Kow	3.82	3.78	3.72	4.14
Odour Threshold	NA	NA	12 ppm = 59.5 mg/m^3	NA
Dermal Absorption	0.01 (unitless)	0.01 (unitless)	0.01 (unitless)	0.01 (unitless)
Permeability Constant	0.0279 cm/hr	0.0279 cm/hr	0.0279 cm/hr	0.0279 cm/hr







EXPOSURE

Exposure of the general population to HCH is primarily associated with γ-HCH which is present within prescription medication to treat/control scabies and head lice. Exposure to all isomers of HCH may occur from foods, drinking water, inhalation or soils. Exposures to α, β and δ-HCH is less frequent than γ-HCH because these other isomers are less common. Exposure is most likely to occur in occupational environments which handle or produce the chemical. γ-HCH has been reported on a number of occasions in major Australian water storage sources, usually with low concentrations (less than 0.002 mg/L) (NHMRC, 2004).

The following is noted with respect to the environmental fate of HCH isomers (WHO 1991):

- α-HCH: Biodegradation and abiotic degradation (dechlorination) by ultraviolet irradiation occur in the environment and produce, respectively, delta-3,4,5,6-tetrachloro-hexene and pentachlorocyclohexene. This breakdown process is slower than in the case of lindane. The persistence of α-HCH in soil is determined by environmental factors such as the action of micro-organisms, organic matter content, and co-distillation and evaporation from soils. No isomerization occurs from lindane to alpha-HCH. Rapid bioconcentration takes place in microorganisms, invertebrates and fish. However, biotransformation and elimination is also fairly rapid in these organisms.

- β-HCH: Biodegradation and abiotic degradation (dechlorination) by ultraviolet irradiation occur in the environment and produce pentachlorocyclohexane, but at a much slower rate than in the case of lindane (gamma-HCH). β-HCH is the most persistent HCH isomer. Its persistence in soil is determined by environmental factors such as the action of micro-organisms, content of organic matter and water, and co-distillation and evaporation from soil. Owing to the persistence of β -HCH, rapid bioconcentration takes place in invertebrates, fish, birds and man. The bioconcentration is higher and the elimination is slower for β -HCH than for the other HCH isomers.

- γ-HCH: Lindane is strongly adsorbed on soils that contain a large amount of organic matter; furthermore, it can move downward through the soil with water from rainfall or artificial irrigation. Volatilization appears to be an important route of its dissipation under the high-temperature conditions of tropical regions. Lindane undergoes rapid degradation (dechlorination) in the presence of ultra-violet irradiation, to form pentachlorocyclohexenes (PCCHs) and tetrachlorocyclohexenes (TCCHs). When lindane undergoes environmental degradation under humid or submerged conditions and in field conditions, its half-time varies from a few days to three years, depending on type of soil, climate, depth of application and other factors. In agricultural soils common in Europe, its half-time is 40-70 days. The biodegradation of lindane is much faster in unsterilised than in sterilized soils. Anaerobic conditions are the most favourable for its microbial metabolisation. Lindane present in water is degraded mostly by microorganisms in sediments to form the same degradation products.

  Limited amounts of lindane and gamma-PCCHs are taken up by and translocated into plants, especially in soils with a high content of organic matter. Residues are found mainly in the roots of plants, and little, if any, is translocated into stems, leaves or fruits. Rapid bioconcentration takes

place in microorganisms, invertebrates, fish, birds and humans, but biotransformation and elimination are relatively rapid when exposure is discontinued. In aquatic organisms, uptake from water is more important than uptake from food.

HCHs have been identified as persistent organic pollutants (POPs) listed within the Protocol on POPs to the UNECE Convention on Long Range Transboundary Air Pollution.

HEALTH EFFECTS

General

The following information is available from ATSDR (2005) and RIVM (2001). The database for the HCH-isomers is limited, with more data available for especially for technical and γ-HCH than other isomers. There is no clinical disease which is unique to HCH toxicity.

HCHs are rapidly and almost completely absorbed from the gastrointestinal tract. There are indications that HCHs are also well absorbed via inhalation and dermal exposure. Biotransformation involves dechlorination, mainly producing a number of chlorophenols as well as chlorinated hydroxy-benzenes and –cyclohexanes.

Except for skin rashes observed in humans following topical application of γ -HCH, there is no evidence that the toxicity of HCH is route-dependent. In humans and animals, the main target of acute exposure to high amounts of HCH is the nervous system, and the effects consist of hyperexcitability, seizures, and convulsions that eventually may lead to death. Although the available reports on humans describe a wide array of effects associated with exposure to HCH, it is difficult to define a clear target organ or system for HCH toxicity, largely because of limitations of the studies, such as lack of exposure data and simultaneous exposure to other chemicals in occupational settings, or exposure to lethal or near lethal amounts, which caused generalized non-specific toxicities.

The following can be noted with respect to the toxicity of HCHs:

- α-HCH: Oral exposure results in liver enzyme induction, increased liver weights, histological changes in the liver, kidney dysfunction and immunosuppression. There are no indications of α-HCH being mutagenic. Carcinogenic studies revealed equivocal results. Further studies indicate that the α-HCH induced tumorogenicity has a non-genotoxic mechanism.

- β –HCH: This isomer accumulates more strongly than other isomers. Its toxicity profile is comparable with that of α and γHCH, but is expressed at lower doses. In addition some estrogenic effects have been identified, the uterus being the target organ. There are no indications for mutagenic potential. Of the studies undertaken on carcinogenicity, one showed hyperplastic changes in the liver and an increase in benign and malignant liver tumours, the other study was negative.

- γ-HCH: The toxicity of γ-HCH is partly comparable with α-HCH. The toxicity is primarily expressed in the liver and kidney, but also neuro- and immunotoxicity. There are no indications of γ -

HCH being mutagenic or teratogenic. Use of γ -HCH pesticides by farmers was associated with a 50% increased risk of non- Hodgkin's lymphoma. However, a causal relationship could not be determined due to confounding effects such as use of other pesticides. Benign lung adenomas were also increased in mice following chronic exposure to γ -HCH. A metabolite of γ -HCH, 2,4,6-trichlorophenol, accounts for 10–20% of γ -HCH-derived excretion products; this metabolite is carcinogenic in animals and might account for some or all of the carcinogenic activity observed in animals. A stable halogenated epoxide of another γ -HCH metabolite, pentachlorocyclohexene, could also contribute to the hepatocarcinogenicity of γ -HCH.

- δ-HCH: Data is limited to one study.

TOXICITY CLASSIFICATION

The International Agency for Research on Cancer (IARC, 1987) evaluated the HCH and concluded that for the technical grade and the α isomer there is sufficient evidence for carcinogenicity to animals, whereas this evidence is limited for the β and γ isomers. There is inadequate evidence for their carcinogenicity to human beings. The HCHs were classified in group 2B – possibly carcinogenic to humans.

The USEPA has provided the following assessments for HCH:

- α-HCH: Classified as Group B2: probable human carcinogen based on increased incidence of liver tumours in mice and rats.
- β –HCH: Classified as Group C: possible human carcinogen based on increases in benign liver tumours in mice.
- γ-HCH: No assessment available
- δ-HCH: Classified as Group D: not classifiable as to human carcinogenicity.
- Technical HCH: Classified as Group B2: probable human carcinogen based four positive studies in mice.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 2004) have derived a drinking water guideline of 0.02 mg/L for lindane (**γ-HCH**) using a ***ADI of 0.008 mg/kg/day*** based on a short-term study on rats and includes a safety factor of 100. The ADI has been derived from a review of lindane by the Joint Meeting on Pesticide Residues (Toxicological evaluation of pesticides produced by the WHO/FAO, JMPR, 1989). It is noted that the ADI established by JMPR has been reviewed since 1989 with the ADI revised,

however the revision has not been incorporated in the Australian Drinking Water Guidelines. Further discussion on revisions by JMPR are discussed below (refer to WHO data).

The Agricultural and Veterinary Chemicals list of ADIs (OCS, 2006), that includes recommendations from the former Pesticides and Agricultural Chemicals Standing Committee (PACSC) of the NHMRC and by the Office of Chemical Safety (OCS) in the Therapeutics Goods Administration (TGA), provides an ADI for lindane of ***0.003 mg/kg/day*** which is derived from a NOEL of 0.31 mg/kg/day (date referenced as 5/11/1986).

No guidelines have been established for other HCH isomers.

Worksafe Australia (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For HCHs, the following have been established:

	α-HCH	β –HCH	γ-HCH	δ-HCH
TWA:	NA	NA	0.008 ppm = 0.1 mg/m^3	NA
STEL:	NA	NA	NA	NA

WHO

The JMPR have provided a number of review of lindane. The review provided in 1989 presented an ADI of 0 to 0.008 mg/kg/day, with 0.008 mg/kg/day adopted in the Australian Drinking Water Guidelines (1996 and 2004) and WHO Drinking Water Guidelines (1996). Further review of lindane in 1997 provided a temporary ADI of 0-0.001 mg/kg/day with the recommendation for further review. Further review on 2002 concluded that lindane was not genotoxic, was not likely to pose a carcinogenic risk to humans and that the existing database was adequate to characterise the potential hazard of lindane to foetuses, infants and children. On this basis an ADI of ***0-0.005 mg/kg/day*** was established on the basis of a NOAEL of 10 ppm (0.47 mg/kg/day) from a long term study of toxicity and carcinogenicity in rats and a safety factor of 100. In addition, an acute reference dose (RfD) of 0.06 mg/kg/day was established on the basis of a NOAEL of 6 mg/kg/day from a study of acute neurotixicty in rats and a safety factor of 100.

The WHO (Drinking Water Guideline 2004) have derived a drinking water guideline of 0.002 mg/L for lindane (**γ-HCH**) using a ***ADI of 0.005 mg/kg/day*** based on a NOAEL of 0.47 mg/kg/day in a 2-year study in rats and includes a safety factor of 100 (ADI adopted from the JMPR review, 2002). No guidelines have been established for other HCH isomers.

The WHO have not established any air guidelines for HCH.

EU

The EU has not provided an assessment of HCH.

RIVM

The Dutch (RIVM, 2001) have provided a review of HCHs and identified the following:

	α-HCH	β –HCH	γ-HCH
Oral TDI (mg/kg/day)	0.001	0.00002	0.00004
Inhalation TC (mg/m^3)	0.00025	NA	0.00014
Background Intake (µg/kg/day)	<0.03	<0.01	<0.03

The TDI for α-HCH was based on a LOAEL for leucocytopenia and liver changes of 10 mg/kg feed (0.5 mg/kg/day) and a NOAEL of 2 mg/kg feed (0.1 mg/kg/day) in a 90 day study with rats (Slooff and Matthijssen, 1988 as referenced by RIVM, 2001) and an uncertainty factor of 100.

The TDI for β –HCH was derived from two semi-chronic oral studies on reproduction in rats, both of which resulted in a NOAEL of 0.4 mg/kg feed (0.02 mg/kg/day) for infertility (Slooff and Matthijssen, 1988 as referenced by RIVM 2001) and an uncertainty factor of 1000 was used (including a factor of 10 fold to account for a poor database).

No new data was available at the time of review (RIVM, 2001) and hence these values were not revised.

The earlier TDI derived by RIVM in 1991 for γ-HCH was the same as derived for α-HCH based on a LOAEL for liver and kidney damage of 10 mg/kg feed (0.5 mg/kg/day) and a NOAEL of 2 mg/kg feed (0.1 mg/kg/day) in a 90 day study with rats (different study to that of α-HCH, Slooff and Matthijssen, 1988) and an uncertainty factor of 100. Review in 2001 suggested immunotoxic effects at lower levels than reported in 1991. Hence a lower value was derived for γ-HCH on the basis of a LOAEL of 0.012 mg/kg/day obtained from Meera (1992 as referenced by RIVM 2001) with an uncertainty factor of 300. The JMPR (2002) has criticised the study from Meera due to the purity of the preparation used, however RIVM has not accepted this criticism on the basis that there are no indications for impurities that would result in higher toxicities than reported in the study.

US

The USEPA (IRIS current in 2006) has derived the following for HCHs:

	α-HCH	β –HCH	γ-HCH	δ-HCH
Oral RfD (mg/kg/day)	NA	NA	0.0003	NA
Inhalation RfC (mg/m^3)	NA	NA	NA	NA
Oral Slope Factor (mg/kg/day)$^{-1}$	6.3	1.8	NA	NA
Inhalation Unit Risk (µg/m^3)$^{-1}$	0.0018	0.00053	NA	NA

The ATSDR has established Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with HCH. The levels established (valid in 2006) are:

	α-HCH	β –HCH	γ-HCH	δ-HCH
Acute Oral MRL (mg/kg/day)	NA	0.05	0.003	NA
Intermediate Oral MRL (mg/kg/day)	NA	0.0006	0.00001	NA
Chronic Oral MRL (mg/kg/day)	0.008	NA	NA	NA

The chronic MRL established for α-HCH was derived on the basis of a NOAEL of 0.8 mg/kg/day and a LOAEL of 3.5 mg/kg/day for lever effects in rats and an uncertainty factor of 100. While the study data used was from 1950, review in 2005 suggested this data remained valid for the purpose of establishing a chronic MRL.

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 HCHs in Australia, noting that low concentrations of lindane has been reported in drinking water supplies in Australia (NHMRC). RIVM (2001) provide a more comprehensive review of potential background intakes of HCHs where the contribution from background is estimated to be less than 1% of the ADI. On this basis background intakes are considered low and the threshold values adopted for the assessment of HCHs 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 HCHs following review of the available information in general accordance with guidelines from enHealth (2002) and NEPM (1999), accounting for background intake where relevant.

As the four isomers of HCH addressed in this toxicity summary are not considered genotoxic it is considered relevant (and in line with enHealth guidance) to consider health effects on the basis of a threshold approach.

Summary of Suggested Toxicity Values for Assessment of HCHs

	α-HCH	β –HCH	γ-HCH	δ-HCH
Oral	TDI = 0.008 mg/kg/day (ATSDR, 2005)	TDI = 0.00002mg/kg/day (RIVM, 2001)	ADI = 0.003 mg/kg/day (NHMRC and OCS 2006)	No data available, adopt assessment presented for β – HCH (due to assumed similarity in effects)
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 = 0.00025 mg/m^3 (RIVM, 2001), equivalent to 0.000071 mg/kg/day tolerable intake*.	No data available, adopt assessment presented for α–HCH	The only inhalation specific value derived is the TC = 0.00014 mg/m^3 (RIVM, 2001), equivalent to 0.00004 mg/kg/day tolerable intake*.	No data available, adopt assessment presented for β – HCH (due to assumed similarity in effects)
Occupational Inhalation Data	NA – adopt value for lindane where required	NA – adopt value for lindane where required	Data from NOHSC: TWA = 0.008 ppm = 0.1 mg/m^3	NA – adopt value for lindane where required
Background	Low	Low	Low	Low

* Tolerable intake in air calculated based on 70 kg body weight and 20 m^3 inhaled per day.

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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 methylmercury.

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.

- Soil: The majority of mercury in surface soil is in the form of oxidized mercury complexes/compounds; however, a small fraction is methylmercury 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 methylmercury. The methylmercury-containing bacteria may be consumed by the next level in the food chain, or they may excrete the methylmercury 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 membranes into the

blood. Liquid metallic mercury is poorly absorbed via the oral route with studies indicating less than 0.01% absorption. Dermal absorption of mercury vapour contributes 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.

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. No dermal absorption data is available.

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 glomerulonepkritis, 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 methylmercury compounds, however studies on rats indicates rapid and almost complete absorption of inhaled methylmercury vapour. Ingested methylmercury is almost completely absorbed. No dermal absorption data are available.

Methylmercury is distributed via the blood to all tissues. It can cross into the brain and foetus. The major site of systemic deposition of methylmercury 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. Methylmercury is converted to mercuric mercury in animals and humans, though less readily than for elemental mercury.

The key target of methylmercury in humans is the CNS, particularly the brain. Evidence from animal and human studies indicates that the embryo and foetus are more sensitive to methylmercury than adults.

Other effects associated with methylmercury 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 methylmercury 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 methylmercury in humans is inadequate. The USEPA (2001) have concluded that methylmercury is not a potent genotoxic agent. Methylmercury induced tumours in mice were considered likely to have a non-genotoxic origin.

EXPOSURE LIMITS AND TOXICITY EVALUATIONS

Australia

The Australian Drinking Water Guidelines (NHMRC, 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 methylmercury 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 (NOHSC) have established "Exposure Standards for Atmospheric Contaminants in the Occupational Environment". For mercury the following have been established:

Elemental mercury: *TWA = 0.025 mg/m^3*, STEL = NA

Monovalent mercury: *TWA = 0.1 mg/m^3*, STEL = NA

Divalent mercury: *TWA = 0.025 mg/m^3*, STEL = NA

Alkyl mercury compounds: *TWA = 0.01 mg/m^3, STEL = 0.03 mg/m^3*

Aryl mercury compounds: *TWA = 0.1 mg/m^3, STEL* = NA

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 methylmercury recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA). The value for methylmercury 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 methylmercury 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".

Draft revision to the WHO Drinking Water Guideline as part of rolling revisions to the guidelines for mercury released in July 2004 (not endorsed) 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. As

this guideline is in draft form which has been released for comment and has not been endorsed or adopted by WHO, it has not been considered at this stage for quantification of effects associated with mercury exposure.

WHO (2000) have derived a ***guideline value of 0.001 mg/m^3*** 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 methylmercury due to the potential for adverse health impacts associated with post-depositional methylmercury formation and bioaccumulation.

EU

No assessment of mercury is available from the EU.

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/m^3*** 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).

Methylmercury: 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 Minimum Risk levels (MRLs) associated with non-carcinogenic effects associated with mercury. The levels established (valid in 2006) are:

- Chronic inhalation MRL for mercury (metallic, vapour) = 0.0002mg/m^3 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;
- Chronic oral MRL for methylmercury = 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/m^3*** 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/m^3*** 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/m^3 for methylmercury

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. Review of methyl mercury intake by Food Standards Australia (2004) has indicated that background intake of methylmercury 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 potential exposure to mercury has assumed 50% background intake of inorganic (total) mercury. 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 methylmercury) has been assumed. Hence the suggested toxicity 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 mercury 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 = 0.00071 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 methylmercury (based on revised PTWI of 0.0016 mg/kg provided by JECFA 2003 for the protection of the developing foetus) Intake adjusted for background = 0.00036 mg/kg/day for total mercury (inorganic and elemental) and 0.00019 mg/kg/day for methylmercury (accounting for 50% and 15% background intake respectively)

Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	GV = 0.001 mg/m^3 as an annual average (WHO 2000 for elemental mercury, inorganic mercury and methylmercury) Intake adjusted for background = 0.0005 mg/m^3 (accounting for 50% background intake) Occupational inhalation exposure (NOHSC, current to 2006): Elemental mercury: *TWA = 0.025 mg/m^3*, STEL = NA Monovalent mercury: *TWA = 0.1 mg/m^3*, STEL = NA Divalent mercury: *TWA = 0.025 mg/m^3*, STEL = NA Alkyl mercury compounds: *TWA = 0.01 mg/m^3, STEL = 0.03 mg/m^3* Aryl mercury compounds: *TWA = 0.1 mg/m^3, STEL* = NA

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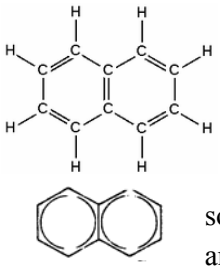
GENERAL



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 anhyride. 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^{o}C
Vapour Density	4.4
Density	1.15 g/ml at 20^{o}C
Solubility	31.7 mg/L at 25^{o}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^{o}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 maybe 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 absorb to sols 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/m^3

STEL: 15 ppm, equivalent to 79 mg/m^3

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/m^3 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/m^3 based on a LOAEL (9.3 mg/m^3 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/m^3) 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 $3.4x10^{-5}$ $(\mu g/m^3)^{-1}$ 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/m^3. Data from Australian cities (DEH, 1999) indicate the highest annual average concentration of naphthalene of 0.058 µg/m^3. Similarly data from Brisbane (Berko, 1999) indicates average urban air concentrations of naphthalene of 0.075 µg/m^3. 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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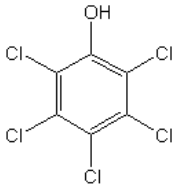
GENERAL



Pentachlorophenol (chlorophen, penchlorol, penta and commonly abbreviated to **PCP**) in its pure form exists as colourless crystals. Typically, commercial grade pentachlorophenol is 86% pure. Contaminants generally consist of other polychlorinated phenols, polychlorinated dibenzop-dioxins (CDDs), and polychlorinated dibenzofurans (CDFs), which are formed during the manufacturing process and can impart a darker colour to the crystals. PCP was, in the past, one of the most heavily used pesticides, but is now regulated in many countries. In Australia PCP is deregistered for use with requirements to apply for permits for the use of the chemical. The sodium salt of PCP (sodium pentachlorophenate) was once the active concentrate used in fungicide, herbicide, molluscicide, slimicide and fermentation disinfectant preparations. It is only now available for use in industrial or manufacturing with some other controlled uses including the treatment of utility poles, railroad ties, and wharf pilings.

PCP is found in all environmental media as a result of its past widespread use; current releases to the environment are more limited as a result of changing use patterns. In addition, a number of other environmental contaminants, including hexachlorobenzene, pentachlorobenzene, pentachloronitrobenzene, and hexachlorocyclohexane isomers, are known to be metabolized to PCP.

The relatively high volatility of PCP and the water solubility of its ionized form have led to widespread contamination of the environment with this compound. Depending on the solvent, temperature, pH, and type of wood, up to 80% of PCP may evaporate from treated wood within 12 months.

The adsorption and leaching behaviour of PCP varies from soil to soil. Adsorption of PCP decreases with rising pH and so PCP is most mobile in mineral soils, and least mobile in acidic clay and sandy soils. Solid or water-dissolved PCP can be photolysed by sunlight within a few days. Traces of PCDDs, mainly OCDD are formed photochemically on irradiation of Na-PCP in aqueous solution. High organic matter and moisture content, median temperatures, and high pH enhance microbial breakdown in soil. Low oxygen conditions are generally unfavourable for the biodegradation of PCP, allowing it to persist in soil, water, and sediments.

Numerous metabolites have been identified resulting from the methylation, acetylation, dechlorination, or hydroxylation of PCP. Of the possible metabolites, at least tetrachlorocatechol seems to be relatively persistent. However, there is a lack of data concerning the fate of the intermediate products of both the abiotic and biotic degradation of PCP. PCP is rather persistent, quite mobile, and found in all environmental systems.

EXPOSURE AND HEALTH EFFECTS

Humans may be exposed to PCP in occupational settings through inhalation of contaminated workplace air and dermal contact with the compound or with wood products treated with the compound. The general population exposure may occur through contact with contaminated environmental media, particularly in the vicinity of wood treatment facilities and hazardous waste sites. Important routes of exposure appear to be inhalation of contaminated air, inhalation exposure to PCP that has volatilized from treated wood

surfaces, ingestion of contaminated groundwater used as a source of drinking water, ingestion of contaminated food, and dermal contact with contaminated soils or wood products treated with the compound. The primary route of human exposure to pentachlorophenol at hazardous waste sites is ingestion of contaminated media, and to a lesser extent, inhalation and dermal contact with contaminated media.

PCP is efficiently absorbed through the skin, the lungs, and the gastrointestinal tract. Usually, the highest PCP levels can be found in the urine immediately after exposure. Consequently, the PCP concentrations in the tissues account for only a small fraction of the PCP dose. Studies do not show a uniform distribution pattern of PCP, but indicate that very high levels can be found in the liver and kidneys. After chronic exposure, most PCP is absorbed by the central nervous system.

PCP is rapidly eliminated by most animals. The PCP concentration in human urine has been widely used as an indicator of the PCP body burden, based on the fact that, in man, renal excretion of PCP is the major elimination route.

Adverse health effects have been observed in humans and experimental animals following short- and long-term exposure to PCP by the inhalation, oral, and dermal exposure routes. The liver, thyroid, immune system, reproductive system, and the developing organism are the primary targets of PCP toxicity. Case reports of individuals acutely exposed to PCP via inhalation and dermal contact and longer-term occupational exposure via inhalation and/or dermal contact identify a number of adverse health effects. The observed effects include symptoms associated with uncoupling of oxidative phosphorylation (tachycardia, increased respiratory rate, laboured breathing, profuse sweating, fever, metabolic acidosis), liver effects, and impaired immune function.

Since humans are generally exposed to technical-grade PCP, which usually contains such toxic impurities as polychlorinated dibenzo-p-dioxins and dibenzofurans, some of the effects observed in humans (or the severity and dose-response characteristics of the effects) may be related, at least in part, to the presence of the impurities.

In addition to the liver, thyroid, immune, reproductive, and developmental effects, exposure to pentachlorophenol is also associated with carcinogenic, renal, and neurological effects. The results of several epidemiology studies suggest that pentachlorophenol may be a human carcinogen. This assessment is supported by the findings in chronic oral rodent studies of increased incidences of liver tumours (hemangiosarcomas, adenomas, and carcinomas) and adrenal gland pheochromocytomas in mice and mesotheliomas and nasal squamous cell carcinomas in rats.

It is generally accepted that uncoupling of the mitochondrial oxidative phosohorylation is the basis for the toxic effects of PCP.

PCP has not demonstrated genotoxicity in *in vitro* and *in vivo* test systems.

PROPERTIES

Pure PCP exists as colourless crystals. It has a very sharp characteristic phenolic smell when hot but very little odour at room temperature. Impure PCP (the form usually found at hazardous waste sites) is dark grey to brown and exists as dust, beads, or flakes. PCP can be found in two forms: PCP itself or as the sodium salt of PCP. The sodium salt dissolves easily in water, but PCP does not. These two forms have some different physical properties, but are expected to have similar toxic effects. . Key properties of PCP are presented below (ATSDR 2001, USEPA 2004 and ORNL Database 2006):

CAS No	87-86-5
Chemical Formula	C_6HCL_5O
Molecular Weight	266.34
Vapour Pressure	0.00011 mmHg at 25^oC
Vapour Density	9.2
Density	1.987 g/ml at 20^oC
Solubility	14 mg/L at 20^oC
Air Diffusion Coefficient	0.056 cm^2/s
Water Diffusion Coefficient	6.1 x 10^{-6} cm^2/s
Henry's Law Coefficient	3.4x10^{-6} atm.m^3/mol
	= 1x10^{-6} at 25^oC (unitless)
Koc	3380 cm^3/g
Log Kow	4.64
Odour Threshold	NA
Dermal Absorption	0.25 (unitless)
Permeability Constant	0.195

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 PCP in Australia. Review of PCP by RIVM (2001) estimated a background daily intake of 0.001 µg/kg which is only a small fraction of the adopted ADI (below) and therefore background intakes have been considered to be low and does not consideration within risk assessments.

Toxicity Values

Review of available data with respect to PCP by the RIVM (2001) indicates the chemical is not considered to be genotoxic. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to PCP on the basis of a non-threshold approach as is adopted by NHMRC (2004).

Toxicity data relevant for use in the characterisation of risk to human health have been selected for PCP 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 2B possibly carcinogenic to humans.

Toxicity Values:	
Oral	ADI = 0.003 mg/kg/day (NHMRC 2004 and RIVM 2001).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	No inhalation data available, hence recommend adopting oral data as relevant for inhalation exposures. Occupational inhalation exposure (NOHSC, current to 2006): TWA: 0.5 mg/m^3 STEL: NA Also noted: Absorption through the skin may be a significant source of exposure
Background	Low – consider to be 0%

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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 studies) 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 photdecomposition.

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 threhsold 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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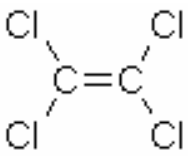

Tetrachloroethene (also known as tetrachloroethylene, perchloroethylene, ethylene tetrachloride, per, perc, perchlor, 1,1,2,2-tetrachloroethylene and commonly abbreviated to **PCE**) is a synthetic chemical that is widely used for dry cleaning of fabrics and for metal-degreasing operations. It is also 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_2Cl_4
Molecular Weight	165.83
Vapour Pressure	18.5 mmHg at 25^{o}C
Vapour Density	5.8
Density	1.62 g/ml at 20^{o}C
Solubility	200 mg/L at 20^{o}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^{o}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).

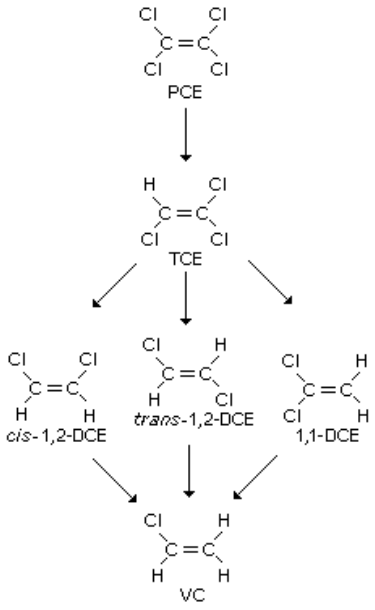

Figure 1. Pathway for anaerobic microbial degradation of chlorinated ethenes to form vinyl chloride (from: WHO, 1999)

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 encephalophy (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	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. 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.

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/m^3

STEL: 150 ppm, equivalent to 1020 mg/m^3

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/m^3. 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/m^3 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/m^3)$^{-1}$ (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 μg/m^3 (reviewed 1999) based on CNS effects and a chronic inhalation value of 35 μg/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 mg/kg/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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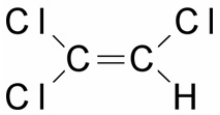

Trichloroethene (also known as 1,1,2-trichloroethylene, ethylene trichloride, and commonly abbreviated to **TCE**) is a synthetic product that was first prepared in 1864 by the reduction of hexachloroethane with hydrogen. It is mainly used as a liquid or vapour degreasing solvent, particularly in the 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_2HCl_3
Molecular Weight	131.4
Vapour Pressure	74 mmHg at 25^{o}C
Vapour Density	4.53
Density	1.465 g/ml at 20^{o}C
Solubility	1100 mg/L at 20^{o}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^{o}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 pf 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).

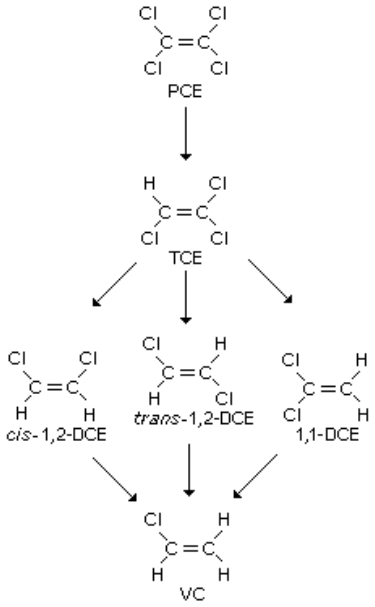

Figure 1. Pathway for anaerobic microbial degradation of chlorinated ethenes to form vinyl chloride (from: WHO, 1999)

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 inducted 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

	properties of the metabolite trichloroethanol (TCOH).
Reproductive Effects	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 – "*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*."
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/m^3

STEL: 40 ppm, equivalent to 216 mg/m^3

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/m^3) for the assessment of exposures to TCE in air has been established. (i.e. for an air concentration of 1 µg/m^3, 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:

$SF\ (mg/kg/day)^{-1}$ = *Risk/Intake(mg/kg/day)*

= *[Risk x Body Weight]/[Concentration (in air) x Inhalation Rate)]*

= $[4.3x10^{-7}\ x\ 70kg]/[0.001mg/m^3\ x\ 20\ m^3/day]$

= $\mathbf{0.0015\ (mg/kg/day)^{-1}}$

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/m^3*** 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 $2x10^{-6}$ to $3x10^{-6}$ $(\mu g/m^3)^{-1}$.

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 mg/m^3 (representing an intake of approximately 0.00018 mg/kg/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 $4.3x10^{-7}$ (per $\mu g/m^3$), equivalent to 0.0015 $(mg/kg/day)^{-1}$ (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 mg/m^3 STEL: 40 ppm, equivalent to 216 mg/m^3
Background	Low

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GENERAL

Tetrachloroethane (TCA) is a chemical compound occurring in two isomers: 1,1,12- and 1,1,2,2-.

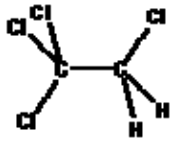
1,1,1,2-Tetrachloroethane (also known as asym-tetrachloroethane and abbreviated to **1,1,1,2-TCA**) is a synthetic chemical that does not occur naturally in the environment. 1,1,1,2-TCA is not produced on an industrial scale, however it is a common by-product of many chlorination reactions of C2 hydrocarbons 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).

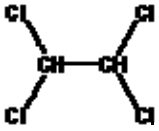
1,1,2,2-Tetrachloroethane (also known as acetylene tetrachloride; sym-tetrachloroethane; s-tetrachloroethane; tetrachloroethane, 1,1-dichloro-2,2-dichloroethane and commonly abbreviated to **1,1,2,2-TCA**) is a synthetic chemical not known to occur naturally. 1,1,2,2-TCA is widely used as an industrial 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):

	1,1,1,2-Tetrachloroethane	**1,1,2,2-Tetrachloroethane**
CAS No.	603-20-6	79-34-5
Chemical Formula	$C_2H_2Cl_4$	$C_2H_2Cl_4$
Molecular Weight	167.85	167.85
Vapour Pressure	12 mmHg at 21^{o}C	13.3 mmHg at 21^{o}C
Vapour Density	5.32	5.32
Density	1.54 g/ml at 20^{o}C	1.59 g/ml at 20^{o}C
Solubility	1070 mg/L at 20^{o}C	2870 mg/L at 20^{o}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^{o}C (unitless)	0.000345 atm.m^3/mol = 0.0141 at 25^{o}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 arts 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 rates 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/m^3 *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 µg/m^3 (applying 5000 margin) or 0.34 to 1.6 µg/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 µg/kg/day (applying 5000 margin) or 0.12 to 0.56 µg/kg/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 to 3)x10^{-6} (µg/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 (mg/kg/day)$^{-1}$*** based on a linear multistage model based on hepatocellular carcinomas; and an ***inhalation unit risk of 5.8x10^{-5} (µg/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 mg/kg/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 mg/kg/day*** based on a LOAEL of 89.3 mg/kg/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/m^3)$^{-1}$*** 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/m^3) 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 $3x10^{-6}$ $(\mu g/m^3)^{-1}$ 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

REFERENCES

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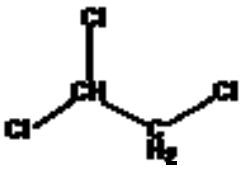
GENERAL



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	$C_2H_3Cl_3$
Molecular Weight	133.41
Vapour Pressure	22.49 mmHg at 25^{o}C
Vapour Density	4.63
Density	1.4 g/ml at 20^{o}C
Solubility	1100 mg/L at 20^{o}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^{o}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/m^3 *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

REFERENCES

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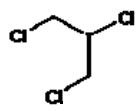
GENERAL



1,2,3-Trichloropropane (allyl trichloride, trchlorohyrdin and glycerol trichlorohydrin) is a chlorinated alkane. Not only is it manufactured itself, but it is produced in significant quantities as a by-product of the production of other chlorinated compounds, including epichlorohydrin. It is used as an intermediate in the synthesis of other chemicals (e.g., pesticides) and as a crosslinking agent in the production of polymers, such as polysulfides and hexafluoropropylene.

1,2,3-trichloropropane predominantly found in the environment in air (about 85%), followed by water (about 11%). 1,2,3-Trichloropropane released into the environment is only poorly converted by abiotic processes (e.g., transformation by photochemically produced hydroxyl radicals) and might therefore remain present for extended periods. However, it can be removed from aquatic systems by evaporation and might be leached from soil into groundwater, due to the low soil sorption coefficients reported for this compound. It is not readily biodegraded and is only slowly transformed by bacteria under aerobic and anaerobic conditions. The data available on the bioconcentration of this chlorinated alkane indicate that it is unlikely to bioaccumulate.

EXPOSURE AND HEALTH EFFECTS

The main routes of exposure to 1,2,3-trichloropropane are via inhalation of contaminated air and ingestion of contaminated drinking-water. The chemical can be absorbed dermally to a more limited extent. In animal studies, 1,2,3-trichloropropane is rapidly absorbed from the gastrointestinal tract, metabolized, and excreted.

1,2,3-Trichloropropane is of moderate acute toxicity, with prominent toxic effect is irritation of the mucosa of eyes and nose and liver and kidney damage.

Limited data is available from human studies, however studies in animals indicated the primary effects associated with repeated exposures include toxic effects in the forestomach, pancreas, liver, kidney, fertility and reproduction (oral studies) and upper respiratory tract, lung, and liver (inhalation studies).

From a variety of *in vitro* genotoxicity tests, a genotoxic potential of 1,2,3-trichloropropane in the presence of metabolic activation systems is evident. Single data on a direct genotoxic action of 1,2,3-trichloropropane seem questionable. *In vivo*, DNA single strand breaks were detectable by alkaline elution, whereas genotoxic action in a dominant lethal test was absent. The major DNA adduct, *S*-[l-(hydroxymethyl)-2-(N^7-guanyl)ethyl]glutathione, and other DNA adducts were identified in preneoplastic and neoplastic lesions of target organs.

1,2,3-Trichloropropane is carcinogenic in rats and mice, inducing a wide range of tumours in both sexes. Data on mode of action, including studies on metabolism, genotoxicity, and determination of DNA adduct formation, suggest that the mechanism of induction of tumours involves direct interaction of an active metabolite with genetic material. Hence, exposure to 1,2,3-trichloropropane should be avoided.

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PROPERTIES

At room temperature, 1,2,3-trichloropropane is a clear, colourless, moderately flammable liquid with a distinct odour. . Key properties are presented below (ATSDR 1992, USEPA 2004 and ORNL Database 2006):

CAS No	96-18-4
Chemical Formula	$C_3H_5Cl_3$
Molecular Weight	147.43
Vapour Pressure	3.1 mmHg at 25°C
Vapour Density	5.1
Density	1.39 g/ml at 20°C
Solubility	2700 mg/L at 20°C
Air Diffusion Coefficient	0.071 cm^2/s
Water Diffusion Coefficient	7.9×10^{-6} cm^2/s
Henry's Law Coefficient	0.00034 $atm.m^3/mol$
	= 0.014 at 25°C (unitless)
Koc	130.8 cm^3/g
Log Kow	2.27
Odour Threshold	NA
Dermal Absorption	0.01 (unitless)
Permeability Constant	0.00983

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 1,2,3-trichloropropane in Australia. Review of 1,2,3-trichloropropane by the WHO (2003) estimated a background daily intake of 0.15 µg/kg with higher levels reported from maximum concentrations reported in Kuwait which is considered to be negligible compared with the adopted threshold levels presented below.

Toxicity Values

Review of available data with respect to 1,2,3-trichloropropane by the EU (2002) indicates that the chemical is a carcinogen and exhibits a genotoxic effect both in vitro and in vivo. On this basis it is considered relevant (in accordance with guidance from enHealth (2002)) to consider potential exposures to 1,2,3-trichloropropane on the basis of a non-threshold approach.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for 1,2,3-trichloropropane 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 2A probable human carcinogen based on sufficient evidence in animals.

Toxicity Values:	
Oral	Slope Factor = 7 $(mg/kg/day)^{-1}$ (USEPA, available from HEAST and listed on RAIS website (ORNL, 2006).
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	No inhalation data available, hence recommend adopting oral data as relevant for inhalation exposures. Occupational inhalation exposure (NOHSC, current to 2006): TWA: 10 ppm = 60 mg/m^3 STEL: NA
Background	Negligible

REFERENCES

ATSDR, 1992. *Toxicological Profile for 1,2,3-Trichloropropane*, available on website and current to 2006.

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WHO, 2004 Guidelines for Drinking-Water Quality, 3rd Edition, Geneva, 2004.

GENERAL

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.

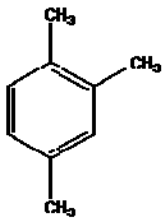
1,2,4-Trimethylbenzene depresses the central nervous system. Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Exposure to 1,3,5-trimethylbenzene may cause an asthma like allergy with future exposure potentially resulting in further asthma attacks, shortness of breath, wheezing, cough and/or chest tightness. 1,3,5-Trimethylbenzene may damage the live and cause anaemia.

No information was found on the carcinogenicity or genotoxicity of 1,2,4- or 1,3,5-trimethylbenzene.

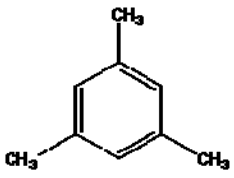
PROPERTIES

Trimethylbenzene is a colourless liquid with a distinct odour. . Key properties are presented below (USEPA 1994, HSDB online database 2007, USEPA 2004 and ORNL Database 2007):

	1,2,4-trimethylbenzene	1,3,5-trimethylbenzene
Cas No	95-63-6	108-67-8
Chemical Formula	C_9H_{12}	C_9H_{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-trimethylbvenzene in air have been reported by the NSW EPA 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-trimethylbenzene was less frequently detected in air samples we the average concentration reported in the Sydney CBD of 0.2 pppv. 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/m^3 (at 25^{o}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 (2002)) 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 both 1,2,4- and 1,3,5-trimethylbenzene 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: Not classified
IARC: Not classified

Toxicity Values:	
Oral	Reference Dose = 0.05 mg/kg/day (USEPA NCEA 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 (2002)).
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 NCEA 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 (2002)). 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 (NIOSH, current to 2007): TWA: 25 ppm = 123 mg/m^3 STEL: NA
Background	50%

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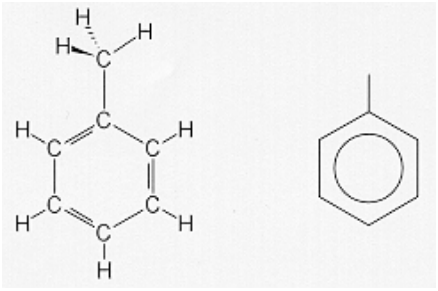
GENERAL



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 2007 and ORNL Database 2007):

CAS No	108-88-3
Chemical Formula	$C_6H_5CH_3$
Molecular Weight	92.12
Vapour Pressure	28.4 mmHg at 25^{o}C
Vapour Density	3.2
Density	0.867 g/ml at 20^{o}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^{o}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

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, 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 Koc 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.

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 Effects	Inhalation of toluene has been associated with irritation of the upper airways, throat and degeneration of the nasal epithelium.

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. Dermal contact with toluene may cause skin damage because it removes skin lipids. Toluene is slightly to moderately irritating to the eyes.
Immunological Effects	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.

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, applying a safety factor of 1000 and a study duration conversion factor of 5/7.

Worksafe Australia (NOHSC, 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/m^3

STEL: 150 ppm, equivalent to 574 mg/m^3

WHO

The WHO Drinking Water Guidelines (2004) have established a guideline value of 0.7 mg/L based on a 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)[1] (based on 1 week average) of 0.26 mg/m^3.

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 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.

[1] 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.

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/m^3 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/m^3) based on neurological effects in humans;
- Chronic inhalation MRL of 0.08 ppm (0.3 mg/m^3) 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/m^3 and 0.019 mg/m^3. 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/m^3. 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 (2002) and NEPM (1999), accounting for background intake where relevant.

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[2] (based on 1 week average) of 0.26 mg/m^3.

The USEPA also provides in inhalation reference concentration (associated with potential chronic exposures) based on the same study, associated with CNS effects, of 0.4 mg/m^3.

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 NEPM) 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

[2] 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.

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/m^3**.

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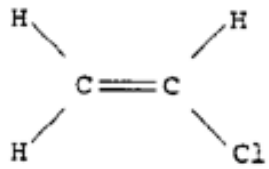
GENERAL



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^{o}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_2H_3Cl
Molecular Weight	62.5
Vapour Pressure	2600 mmHg at 25^{o}C
Vapour Density	2.16
Density	0.91 g/ml at 20^{o}C
Solubility	2760 mg/L at 20^{o}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^{o}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).

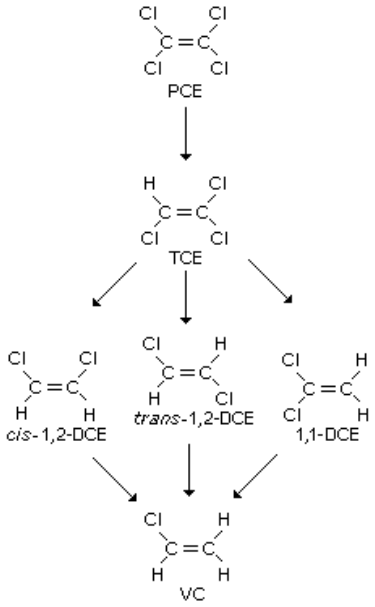


Figure 1. Pathway for anaerobic microbial degradation of chlorinated ethenes to form vinyl chloride (from: WHO, 1999)

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 studied 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 *in vitro* 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/m^3
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:

SF (mg/kg/day)$^{-1}$ *= Risk/Intake(mg/kg/day)*

= [Risk x Body Weight]/[Concentration (water) x Ingestion Rate)]

= [2 x10^{-5} x 70kg]/[0.0003mg/L x 2 L/day]

= 2.3 (mg/kg/day)$^{-1}$

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/m^3)$^{-1}$*** (i.e. for an air concentration of 1 μg/m^3, 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 $= 0.72\ (mg/kg/day)^{-1}$ exposures in adulthood

$=1.44\ (mg/kg/day)^{-1}$ 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 $= 4.4x10^{-6}\ (\mu g/m^3)^{-1} = 0.016\ (mg/kg/day)^{-1}$ exposures during adulthood

$= 8.8x10^{-6}\ (\mu g/m^3)^{-1} = 0.03\ (mg/kg/day)^{-1}$ 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 (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 $(mg/kg/day)^{-1}$ (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 $(mg/kg/day)^{-1}$ can be adopted.
Dermal	No dermal guidelines are available, hence it has been assumed that dermal toxicity is equivalent to oral toxicity.
Inhalation	Inhalation slope factor = 0.016 $(mg/kg/day)^{-1}$ exposures during adulthood (USEPA 2000)* = 0.03 $(mg/kg/day)^{-1}$ exposures over a lifetime (USEPA 2000)* Occupational inhalation exposure (NOHSC): *TWA: 5 ppm, equivalent to 13 mg/m^3* *STEL: NA*
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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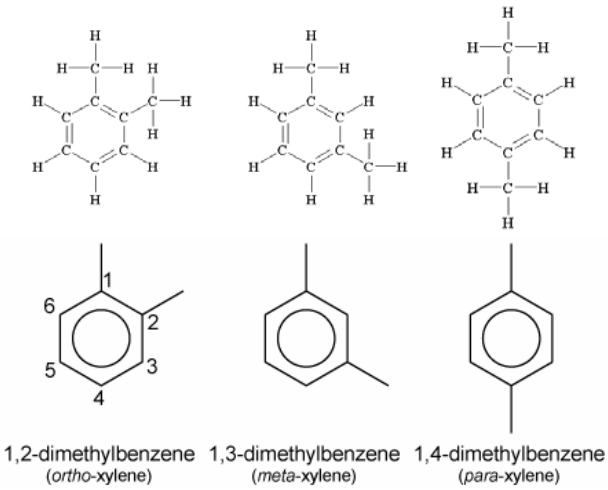
GENERAL



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). T hese 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, 1995).

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 1995, USEPA 2004, HSDB 2006 and ORNL Database 2006):

CAS No	1330-20-7 (for mixed xylene)
Chemical Formula	C_8H_{10} (single isomer)
Molecular Weight	106.16
Vapour Pressure	6 to 16 mmHg at 20^{o}C
Vapour Density	3.7
Density	0.864 g/ml at 20^{o}C
Solubility	130 mg/L at 25^{o}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^{o}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)

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 photoxidation 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.

- 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 bioacumulate to low to modest levels, however food-chain biomagnification has not been observed

HEALTH EFFECTS

General

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 1995 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.
	Acute oral ingestion of xylenes can result in respiratory depression.
Cardiovascular Effects	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 Effects	Workers exposed to xylene vapours have reported nausea, vomiting and gastric 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 of 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 rates 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 concentration 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 Effects	Both mixed and individual isomers produce foetotoxic effects in laboratory animals, These include skeletal variations in foetuses, delayed ossification, 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.

Worksafe Australia (NOHSC, available in 2006) 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/m^3

STEL: 150 ppm, equivalent to 655 mg/m^3

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.

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/m^3**.

EU

No review of xylene (mixed or individual isomers) are available from the EU or OECD.

US

The USEPA (available from IRIS 2006) 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/m^3 established based on impaired coordination in a subchronic inhalation rat study.

The ATSDR (1997) 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 1, 0.7 and 0.1 ppm respectively;
- Acute oral MRL for p-xylene of 1 mg/kg/day;
- Intermediate oral MRLs for mixed xylene and m-xylene of 0.2 and 0.6 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/m^3 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/m^3. 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.

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 (2002) and NEPM (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). Occupational inhalation exposure (NOHSC, current): TWA: 80 ppm, equivalent to 350 mg/m^3

	STEL: 150 ppm, equivalent to 655 mg/m^3
Background	Low – 2%

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Appendix E Vapour Modelling Approach

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 1990[1] and Johnson and Ettinger 1991[2]) 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:

$$C_{source} = C_{water} \bullet HL \qquad (g/cm^3) \qquad \textbf{...Equation VS1}$$

Where:

C_{water} = concentration in water (at top of groundwater, g/cm^3)
HL = Henry's Law constant (unitless)

[1] Johnson, P.C., Hertx 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:

$$SVPC = \frac{VP \bullet MW}{T \bullet 62361} \quad (g/cm^3) \quad \textbf{...Equation VS2}$$

Where:

VP = vapour pressure of the contaminant (mmHg)
MW = molecular weight (g/mol)
T = soil temperature (K)
62361 = conversion (mmHg/K* cm^3/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:

$$C_{source} = \frac{C_{soil} \bullet H' \bullet \rho_S}{\theta_{ws} + k_d \bullet \rho_S + H' \bullet \theta_{as}} \quad (g/cm^3) \quad \textbf{...Equation VS3}$$

where:

Csoil = Concentration in soil source zone (g/g)
H' = Henry's Law constant (unitless)
ρ_S = Soil bulk density (g soil/cm^3 soil)
θ_{ws} = Volumetric water content in soil source zone (cm^3 water/cm^3 soil)
θ_{as} = Volumetric air content in soil source zone (cm^3 air/cm^3 soil)
K_d = Soil-water partition coefficient (cm^3 air/g soil) = K_{oc} x f_{oc}
Koc = Soil organic carbon partition coefficient, chemical specific (cm^3/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):

$$C_{sat} = \frac{S}{\rho_s} \bullet [H' \bullet \theta_{as} + \theta_{ws} + K_d \bullet \rho_s] \quad (mg/kg) \quad \textbf{...Equation VS4}$$

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:

$$C_{NAPL\ source} = \frac{x_i \bullet P_i(T_S) \bullet MW}{1000 \bullet R \bullet T_S} \qquad \textbf{...Equation VS5}$$

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^{-1}K^{-1};
T_S = Average soil temperature (^{o}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.

$$D_T^{eff} = \frac{L_T}{\sum_{i=1}^{n} L_i / D_i^{eff}} \qquad \textbf{...Equation D1}$$

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 (cm^2/s) – refer to Equation D2

$$D_i^{eff} = D_a \bullet \left[\frac{\theta_{ai}^{3.33}}{n_i^2}\right] + \left[\frac{D_w}{H'}\right] \bullet \left[\frac{\theta_{wi}^{3.33}}{n_i^2}\right] \qquad \textbf{...Equation D2}$$

D_a = diffusivity in air, chemical specific (cm^2/s)
θ_{ai} = soil air-filled volume of layer i (cm^3/cm^3)
n_i = soil total porosity of layer i (cm^3/cm^3)
= 1-ρ_b/ρ_s
ρ_b = soil dry bulk density, (g/cm^3)
ρ_s = soil particle density, (g/cm^3) - typically 2.65
D_w = diffusivity in water, chemical specific (cm^2/s)
θ_{wi} = soil water-filled volume of layer i, (cm^3/cm^3)

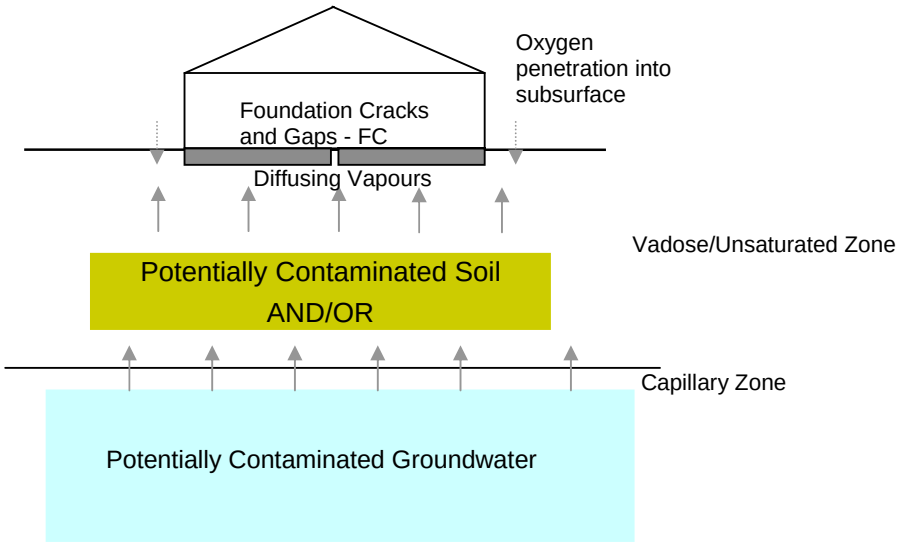
Farmer Vapour Intrusion Model

Introduction

The potential concentration of volatile chemicals inside a range of building types can be estimated using a box model as detailed by Farmer (1980[1]) as modified by SD DEH (2000[2]). 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

$$C_{indoors} = \frac{E_{surface} \bullet FS \bullet FC}{ER \bullet V} \quad \mu g/m^3 \qquad \textbf{...Equation F1}$$

$E_{surface}$	= Emission rate measured from the surface of the ground beneath the building (µg/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)

$$ER = \frac{C_{source} \bullet D_{eff} \bullet A_{area}}{D} \quad (g/s) \qquad \textbf{.....Equation F2}$$

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 (1985[3]), which is summarised by Markey and Anderson (1996[4]) 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.

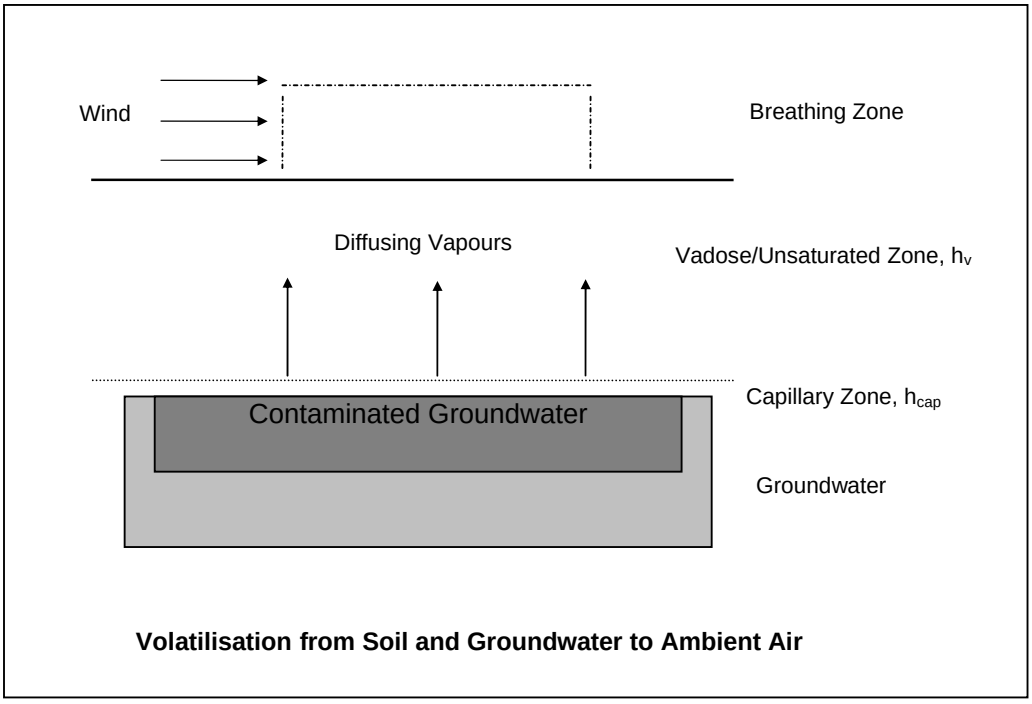
Outdoor Air and Excavations

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, 1996[1]) and the Risk Based Corrective Action at Petroleum Release Sites (ASTM, 2002[2]).

The model is used to assess vapour intrusion indoors only and assumed that the source is non-depleting.

Conceptual Model



Volatilisation from Soil and Groundwater to Ambient Air

[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.

$$C_o = \frac{J_S}{Q/C \bullet 10^{-9}} \qquad \textbf{...Equation O1}$$

Where:
C_o = Outdoor air concentration ($\mu g/m^3$)
J_S = Contaminant flux from the surface of the ground (measured) ($g/s/m^2$)
Q/C = Dispersion term calculated for area ($g/s/m^2$ per kg/m^3)
10^{-9} = Units conversion to from kg/m^3 to $\mu g/m^3$

$$Q/C = 11.91 \bullet \exp\left(\frac{(\ln(Acres) - 18.4385)^2}{209.7845}\right) \qquad \textbf{...Equation O2}$$

Where:
Q/C = Dispersion term calculated for area ($g/s/m^2$ 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.

$$C_{outdoor} = C_S \bullet VF \qquad (mg/m^3) \qquad \textbf{...Equation O3}$$

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:

$$VF = \frac{D_s^{eff} \bullet W}{U_{air} \bullet \delta \bullet L_{GW}}$$ **....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.

ASTM (2002) also provides equations for estimating emissions to outdoor air from sources that are close to or at the surface of the ground.

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 1992[3] 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 1999[4]). The EPA method examines the mass of a chemical that could be transferred from water to air and assumes:

$$VF_w\left(\frac{l}{m^3}\right) = \frac{\left(k_{lg}\right)}{\left(k\right)\left(\frac{\mu}{L}\right)\left(H\right)} \bullet \left(\frac{1000l}{m^3}\right)$$

(mg/m^3 air)/(mg/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 x 10^{-6} m/s (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 in excavation of 1 mph (0.45 m/sec) over a year's time (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 VFw is applied directly to the relevant groundwater concentrations assumed to seep into an excavation to estimate an air concentration in the flooded trench. If this VF were considered in relation to phase partition equations for dissolved phase and LNAPL sources, the air concentration in an excavation would be approximately 2000 times lower (based on dispersion and dilution in excavation) than the vapour phase concentration estimated at an LNAPL source, assuming the product floods into an excavation.

[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

Appendix F Risk Calculations On and Off-Site

Risk Calculations ISZ 1a and 1b

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 - ISZ 1a and 1b (and ISZ 2)

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.75	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**1.75**	Depth to soil gas sample
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m^2]	emarea	**625**	Assumed size of building 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	**50**	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	**50**	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	**2500**	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
cis-1,2-dichloroethene	3.92E-4			3.92E-4
trimethylbenzenes	3.39E-4			3.39E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	21000	2.1E-08	6.7E-10		**4.0E-05**		**1.0E-05**
vinyl chloride	23000	2.3E-08	7.3E-10		**4.4E-05**		**1.1E-05**
chloroform	63000	6.3E-08	2.0E-09	2.6E-09	**1.5E-04**		**3.9E-05**
trichloroethene (TCE)	3100000	3.1E-06	7.4E-08		**4.4E-03**		**1.1E-03**
tetrachloroethene (PCE)	49000	4.9E-08	1.1E-09		**6.4E-05**		**1.6E-05**
chlorobenzene	9380165	9.4E-06	2.1E-07		**1.2E-02**		**3.2E-03**
benzene	23760	2.4E-08	6.3E-10		**3.8E-05**		**9.6E-06**
toluene	21384297	2.1E-05	5.6E-07	3.3E-09	**2.0E-04**		**5.0E-05**
cis-1,2-dichloroethene	90000	9.0E-08	2.0E-09		**1.2E-04**		**3.1E-05**
trimethylbenzenes	40800	4.1E-08	7.9E-10		**4.7E-05**		**1.2E-05**

↑ Maximum soil gas concentration from source zone

↑ Maximum emissions rate from source sone

↑ Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Impacted Soil Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**4.5E-7**	**1.8E-2**			**TOTAL**	**4.3E-8**	**1.8E-3**
1,2-dichloroethane (EDC)	9.8E-03		4.0E-05	1.9E-06	4.4E-06	1.9E-8	--	1.0E-05	1.8E-07	4.3E-07	1.8E-9	--
vinyl chloride	3.1E-02		4.4E-05	2.1E-06	4.8E-06	6.4E-8	--	1.1E-05	2.0E-07	4.6E-07	6.1E-9	--
chloroform	1.5E-03	2.0E-02	1.5E-04	7.3E-06	1.7E-05	1.1E-8	8.5E-4	3.9E-05	6.9E-07	1.6E-06	1.0E-9	8.1E-5
trichloroethene (TCE)	1.5E-03		4.4E-03	2.1E-04	4.9E-04	3.1E-7	--	1.1E-03	2.0E-05	4.7E-05	3.0E-8	--
tetrachloroethene (PCE)		4.7E-02	6.4E-05	3.0E-06	7.0E-06	--	1.5E-4	1.6E-05	2.9E-07	6.7E-07	--	1.4E-5
chlorobenzene		1.4E-01	1.2E-02	5.9E-04	1.4E-03	--	9.6E-3	3.2E-03	5.6E-05	1.3E-04	--	9.2E-4
benzene	2.1E-02		3.8E-05	1.8E-06	4.2E-06	3.8E-8	--	9.6E-06	1.7E-07	4.0E-07	3.6E-9	--
toluene		6.7E-02	2.0E-04	9.2E-06	2.1E-05	--	3.2E-4	5.0E-05	8.8E-07	2.0E-06	--	3.1E-5
cis-1,2-dichloroethene		1.0E-02	1.2E-04	5.7E-06	1.3E-05	--	1.3E-3	3.1E-05	5.4E-07	1.3E-06	--	1.3E-4
trimethylbenzenes		8.6E-04	4.7E-05	2.2E-06	5.2E-06	--	6.1E-3	1.2E-05	2.1E-07	5.0E-07	--	5.8E-4

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Impacted Soil Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**4.5E-7**	**3.0E-4**			**TOTAL**	**4.3E-8**	**2.8E-5**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	4.0E-05	1.9E-06	4.4E-06	1.9E-8	7.8E-7	1.0E-05	1.8E-07	4.3E-07	1.8E-9	7.4E-8
vinyl chloride	3.1E-02	1.9E+00	4.4E-05	2.1E-06	4.8E-06	6.4E-8	2.6E-6	1.1E-05	2.0E-07	4.6E-07	6.1E-9	2.5E-7
chloroform	1.5E-03	1.4E+00	1.5E-04	7.3E-06	1.7E-05	1.1E-8	1.2E-5	3.9E-05	6.9E-07	1.6E-06	1.0E-9	1.1E-6
trichloroethene (TCE)	1.5E-03	7.7E+00	4.4E-03	2.1E-04	4.9E-04	3.1E-7	6.3E-5	1.1E-03	2.0E-05	4.7E-05	3.0E-8	6.0E-6
tetrachloroethene (PCE)		4.8E+01	6.4E-05	3.0E-06	7.0E-06	--	1.5E-7	1.6E-05	2.9E-07	6.7E-07	--	1.4E-8
chlorobenzene		6.6E+00	1.2E-02	5.9E-04	1.4E-03	--	2.1E-4	3.2E-03	5.6E-05	1.3E-04	--	2.0E-5
benzene	2.1E-02	4.6E-01	3.8E-05	1.8E-06	4.2E-06	3.8E-8	9.1E-6	9.6E-06	1.7E-07	4.0E-07	3.6E-9	8.7E-7
toluene		2.7E+01	2.0E-04	9.2E-06	2.1E-05	--	7.9E-7	5.0E-05	8.8E-07	2.0E-06	--	7.5E-8
cis-1,2-dichloroethene		1.1E+02	1.2E-04	5.7E-06	1.3E-05	--	1.2E-7	3.1E-05	5.4E-07	1.3E-06	--	1.1E-8
trimethylbenzenes		1.8E+01	4.7E-05	2.2E-06	5.2E-06	--	2.9E-7	1.2E-05	2.1E-07	5.0E-07	--	2.8E-8

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

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 - ISZ 1a and 1b (and ISZ 2) - Excavations

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
cis-1,2-dichloroethene	3.92E-4			3.92E-4
trimethylbenzenes	3.39E-4			3.39E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	21000	2.1E-08	1.2E-07			**1.6E-03**	
vinyl chloride	23000	2.3E-08	1.3E-07			**1.7E-03**	
chloroform	63000	6.3E-08	3.5E-07			**4.6E-03**	
trichloroethene (TCE)	3100000	3.1E-06	1.3E-05			**1.7E-01**	
tetrachloroethene (PCE)	49000	4.9E-08	1.9E-07			**2.5E-03**	
chlorobenzene	9380165	9.4E-06	3.6E-05			**4.9E-01**	
benzene	23760	2.4E-08	1.1E-07			**1.5E-03**	
toluene	21384297	2.1E-05	9.8E-05			**1.3E+00**	
cis-1,2-dichloroethene	90000	9.0E-08	3.5E-07			**4.7E-03**	
trimethylbenzenes	40800	4.1E-08	1.4E-07			**1.8E-03**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Impacted Soil Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04** **5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.7E-7**	**7.9E-3**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.6E-03	1.2E-06	8.1E-05	1.1E-8	1.4E-5
vinyl chloride	3.1E-02	1.9E+00	1.7E-03	1.3E-06	8.8E-05	3.9E-8	4.8E-5
chloroform	1.5E-03	1.4E+00	4.6E-03	3.4E-06	2.4E-04	5.0E-9	1.7E-4
trichloroethene (TCE)	1.5E-03	7.7E+00	1.7E-01	1.3E-04	8.9E-03	1.9E-7	1.2E-3
tetrachloroethene (PCE)		4.8E+01	2.5E-03	1.8E-06	1.3E-04	--	2.7E-6
chlorobenzene		6.6E+00	4.9E-01	3.6E-04	2.5E-02	--	3.8E-3
benzene	2.1E-02	4.6E-01	1.5E-03	1.1E-06	7.6E-05	2.3E-8	1.7E-4
toluene		2.7E+01	1.3E+00	9.7E-04	6.8E-02	--	2.5E-3
cis-1,2-dichloroethene		1.1E+02	4.7E-03	3.5E-06	2.4E-04	--	2.1E-6
trimethylbenzenes		1.8E+01	1.8E-03	1.4E-06	9.5E-05	--	5.3E-6

↑ Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Impacted Soil Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.3E-6**	**1.2E+2**			**TOTAL**	**1.9E-8**	**2.9E+1**
hexachlorobenzene (HCB)		1.6E-04	5.76	5.8E-07	1.4E-06	--	8.5E-3	5.76	4.8E-09	3.4E-07	--	2.1E-3
chloroform		6.5E-03	1.2	1.2E-07	2.8E-07	--	4.3E-5	10.6	8.9E-09	6.2E-07	--	9.6E-5
carbon tetrachloride		5.0E-04	5.8	5.8E-07	1.4E-06	--	2.7E-3	5.8	4.9E-09	3.4E-07	--	6.9E-4
chlorobenzene		8.6E-02	535	5.4E-05	1.3E-04	--	1.5E-3	3040	2.5E-06	1.8E-04	--	2.1E-3
benzene	3.5E-02		1.5	1.5E-07	3.5E-07	5.3E-9	--	1.5	1.3E-09	8.8E-08	4.4E-11	--
toluene		2.0E-01	1500	1.5E-04	3.5E-04	--	1.8E-3	1500	1.3E-06	8.8E-05	--	4.4E-4
ethylbenzene		9.7E-02				--	--	110	9.2E-08	6.5E-06	--	6.7E-5
xylenes		1.8E-01	82.2	8.3E-06	1.9E-05	--	1.1E-4	82.2	6.9E-08	4.8E-06	--	2.8E-5
PAHs (BaP equivalent)	5.0E-01		45.63	4.6E-06	1.1E-05	2.3E-6	--	45.63	3.8E-08	2.7E-06	1.9E-8	--
1,4-dichlorobenzene		1.1E-02	19.6	2.0E-06	4.6E-06	--	4.3E-4	35.5	3.0E-08	2.1E-06	--	1.9E-4
a-HCH (a-BHC)		8.0E-03	167	1.7E-05	3.9E-05	--	4.9E-3	167	1.4E-07	9.8E-06	--	1.2E-3
b-HCH (b-BHC)		2.0E-05	61.3	6.2E-06	1.4E-05	--	7.2E-1	61.3	5.1E-08	3.6E-06	--	1.8E-1
d-HCH (d-BHC)		2.0E-05	84.5	8.5E-06	2.0E-05	--	9.9E-1	84.5	7.1E-08	5.0E-06	--	2.5E-1
g-HCH (g-BHC or lindane)		3.0E-03	100	1.0E-05	2.3E-05	--	7.8E-3	100	8.4E-08	5.9E-06	--	2.0E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	988000	9.9E-02	2.3E-01	--	1.2E+2	988000	8.3E-04	5.8E-02	--	2.9E+1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soil from all depths

Dermal Exposure to Chemicals via Contact with Soil

Impacted Soil Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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: Non-Threshold Slope Factor	Toxicity Data: Threshold ADI, TDI or RfD	Concentration in Soil	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Hazard Quotient	Concentration in Soil	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**9.7E-7**	**5.0E+1**			**TOTAL**	**1.7E-8**	**2.6E+1**
hexachlorobenzene (HCB)		1.6E-04	5.76	2.4E-07	5.7E-07	--	3.6E-3	5.76	4.3E-09	3.0E-07	--	1.9E-3
chloroform		6.5E-03	1.2	5.1E-08	1.2E-07	--	1.8E-5	10.6	7.9E-09	5.6E-07	--	8.5E-5
carbon tetrachloride		5.0E-04	5.8	2.5E-07	5.8E-07	--	1.2E-3	5.8	4.3E-09	3.0E-07	--	6.1E-4
chlorobenzene		8.6E-02	535	2.3E-05	5.3E-05	--	6.2E-4	3040	2.3E-06	1.6E-04	--	1.9E-3
benzene	3.5E-02		1.5	6.4E-08	1.5E-07	2.2E-9	--	1.5	1.1E-09	7.9E-08	3.9E-11	--
toluene		2.0E-01	1500	6.4E-05	1.5E-04	--	7.5E-4	1500	1.1E-06	7.9E-05	--	4.0E-4
xylenes		1.8E-01	82.2	3.5E-06	8.2E-06	--	4.6E-5	82.2	6.2E-08	4.3E-06	--	2.5E-5
PAHs (BaP equivalent)	5.0E-01		45.63	1.9E-06	4.5E-06	9.7E-7	--	45.63	3.4E-08	2.4E-06	1.7E-8	--
1,4-dichlorobenzene		1.1E-02	19.6	8.3E-07	1.9E-06	--	1.8E-4	35.5	2.7E-08	1.9E-06	--	1.7E-4
a-HCH (a-BHC)		8.0E-03	167	7.1E-06	1.7E-05	--	2.1E-3	167	1.2E-07	8.7E-06	--	1.1E-3
b-HCH (b-BHC)		2.0E-05	61.3	2.6E-06	6.1E-06	--	3.0E-1	61.3	4.6E-08	3.2E-06	--	1.6E-1
d-HCH (d-BHC)		2.0E-05	84.5	3.6E-06	8.4E-06	--	4.2E-1	84.5	6.3E-08	4.4E-06	--	2.2E-1
g-HCH (g-BHC or lindane)		3.0E-03	100	4.2E-06	9.9E-06	--	3.3E-3	100	7.5E-08	5.2E-06	--	1.7E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	988000	4.2E-02	9.8E-02	--	4.9E+1	988000	7.4E-04	5.2E-02	--	2.6E+1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soil from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment ISZ 1a, 1b a

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**7.0E-6**	**8.2E+1**			**TOTAL**	**1.2E-7**	**4.3E+1**
hexachlorobenzene (HCB)		1.6E-04	0.10	5.76	1.4E-06	3.2E-06	--	2.0E-2	5.76	2.4E-08	1.7E-06	--	1.0E-2
chloroform		6.5E-03	0.01	1.2	2.8E-08	6.6E-08	--	1.0E-5	10.6	4.4E-09	3.1E-07	--	4.7E-5
carbon tetrachloride		5.0E-04	0.01	5.8	1.4E-07	3.2E-07	--	6.4E-4	5.8	2.4E-09	1.7E-07	--	3.4E-4
chlorobenzene		8.6E-02	0.01	535	1.3E-05	2.9E-05	--	3.4E-4	3040	1.3E-06	8.8E-05	--	1.0E-3
benzene	3.5E-02		0.01	1.5	3.5E-08	8.3E-08	1.2E-9	--	1.5	6.2E-10	4.4E-08	2.2E-11	--
toluene		2.0E-01	0.01	1500	3.5E-05	8.3E-05	--	4.2E-4	1500	6.2E-07	4.4E-05	--	2.2E-4
xylenes		1.8E-01	0.01	82.2	1.9E-06	4.5E-06	--	2.6E-5	82.2	3.4E-08	2.4E-06	--	1.4E-5
PAHs (BaP equivalent)	5.0E-01		0.13	45.63	1.4E-05	3.3E-05	7.0E-6	--	45.63	2.5E-07	1.7E-05	1.2E-7	--
1,4-dichlorobenzene		1.1E-02	0.01	19.6	4.6E-07	1.1E-06	--	1.0E-4	35.5	1.5E-08	1.0E-06	--	9.7E-5
a-HCH (a-BHC)		8.0E-03	0.01	167	3.9E-06	9.2E-06	--	1.2E-3	167	6.9E-08	4.9E-06	--	6.1E-4
b-HCH (b-BHC)		2.0E-05	0.01	61.3	1.4E-06	3.4E-06	--	1.7E-1	61.3	2.5E-08	1.8E-06	--	8.9E-2
d-HCH (d-BHC)		2.0E-05	0.01	84.5	2.0E-06	4.7E-06	--	2.3E-1	84.5	3.5E-08	2.5E-06	--	1.2E-1
g-HCH (g-BHC or lindane)		3.0E-03	0.01	100	2.4E-06	5.5E-06	--	1.8E-3	100	4.2E-08	2.9E-06	--	9.7E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	988000	7.0E-02	1.6E-01	--	8.2E+1	988000	1.2E-03	8.6E-02	--	4.3E+1
				↑ Maximum concentration in top 1m soils					↑ Maximum concentration in soil from all depths				

Summary of Risk - ISZ 1a and 1b

Exposure to COPC Derived from Soil and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
ISZ 1a, 1b and 2 - Commercial/Industrial Worker			
Ingestion of COPC Indetified in Shallow Soils	**2.3E-06**	**118**	**118**
Dermal Contact with COPC Identified in Shallow Soils	**9.7E-07**	**50**	**50**
Inhalation of volatile COPC derived from soils and groundwater	**4.5E-07**	**0.018**	**0.00030**
Total Risk	**4E-06**	**167**	**167**

ISZ 1a, 1b and 2 - Intrusive Worker		
Ingestion of COPC Indetified in Shallow Soils	**1.9E-08**	**29**
Dermal Contact with COPC Identified in Shallow Soils	**1.7E-08**	**26**
Inhalation of volatile COPC derived from soils and groundwater	**2.7E-07**	**0.0079**
Total Risk	**3E-07**	**56**

Risk Calculations ISZ 2

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 - ISZ 2 (and ISZ 1a and 1b)

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.75	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**1.75**	Depth to soil gas sample
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m^2]	emarea	**625**	Assumed size of building 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	**50**	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	**50**	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	**2500**	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
cis-1,2-dichloroethene	3.92E-4			3.92E-4
trimethylbenzenes	3.39E-4			3.39E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	21000	2.1E-08	6.7E-10		**4.0E-05**		**1.0E-05**
vinyl chloride	23000	2.3E-08	7.3E-10		**4.4E-05**		**1.1E-05**
chloroform	63000	6.3E-08	2.0E-09	2.6E-09	**1.5E-04**		**3.9E-05**
trichloroethene (TCE)	3100000	3.1E-06	7.4E-08		**4.4E-03**		**1.1E-03**
tetrachloroethene (PCE)	49000	4.9E-08	1.1E-09		**6.4E-05**		**1.6E-05**
chlorobenzene	9380165	9.4E-06	2.1E-07		**1.2E-02**		**3.2E-03**
benzene	23760	2.4E-08	6.3E-10		**3.8E-05**		**9.6E-06**
toluene	21384297	2.1E-05	5.6E-07	3.3E-09	**2.0E-04**		**5.0E-05**
cis-1,2-dichloroethene	90000	9.0E-08	2.0E-09		**1.2E-04**		**3.1E-05**
trimethylbenzenes	40800	4.1E-08	7.9E-10		**4.7E-05**		**1.2E-05**

↑ Maximum soil gas concentration from source zone

↑ Maximum emissions rate from source sone

↑ Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Impacted Soil Zone 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**4.5E-7**	**1.8E-2**			**TOTAL**	**4.3E-8**	**1.8E-3**
1,2-dichloroethane (EDC)	9.8E-03		4.0E-05	1.9E-06	4.4E-06	1.9E-8	--	1.0E-05	1.8E-07	4.3E-07	1.8E-9	--
vinyl chloride	3.1E-02		4.4E-05	2.1E-06	4.8E-06	6.4E-8	--	1.1E-05	2.0E-07	4.6E-07	6.1E-9	--
chloroform	1.5E-03	2.0E-02	1.5E-04	7.3E-06	1.7E-05	1.1E-8	8.5E-4	3.9E-05	6.9E-07	1.6E-06	1.0E-9	8.1E-5
trichloroethene (TCE)	1.5E-03		4.4E-03	2.1E-04	4.9E-04	3.1E-7	--	1.1E-03	2.0E-05	4.7E-05	3.0E-8	--
tetrachloroethene (PCE)		4.7E-02	6.4E-05	3.0E-06	7.0E-06	--	1.5E-4	1.6E-05	2.9E-07	6.7E-07	--	1.4E-5
chlorobenzene		1.4E-01	1.2E-02	5.9E-04	1.4E-03	--	9.6E-3	3.2E-03	5.6E-05	1.3E-04	--	9.2E-4
benzene	2.1E-02		3.8E-05	1.8E-06	4.2E-06	3.8E-8	--	9.6E-06	1.7E-07	4.0E-07	3.6E-9	--
toluene		6.7E-02	2.0E-04	9.2E-06	2.1E-05	--	3.2E-4	5.0E-05	8.8E-07	2.0E-06	--	3.1E-5
cis-1,2-dichloroethene		1.0E-02	1.2E-04	5.7E-06	1.3E-05	--	1.3E-3	3.1E-05	5.4E-07	1.3E-06	--	1.3E-4
trimethylbenzenes		8.6E-04	4.7E-05	2.2E-06	5.2E-06	--	6.1E-3	1.2E-05	2.1E-07	5.0E-07	--	5.8E-4

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Impacted Soil Zone 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**4.5E-7**	**3.0E-4**			**TOTAL**	**4.3E-8**	**2.8E-5**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	4.0E-05	1.9E-06	4.4E-06	1.9E-8	7.8E-7	1.0E-05	1.8E-07	4.3E-07	1.8E-9	7.4E-8
vinyl chloride	3.1E-02	1.9E+00	4.4E-05	2.1E-06	4.8E-06	6.4E-8	2.6E-6	1.1E-05	2.0E-07	4.6E-07	6.1E-9	2.5E-7
chloroform	1.5E-03	1.4E+00	1.5E-04	7.3E-06	1.7E-05	1.1E-8	1.2E-5	3.9E-05	6.9E-07	1.6E-06	1.0E-9	1.1E-6
trichloroethene (TCE)	1.5E-03	7.7E+00	4.4E-03	2.1E-04	4.9E-04	3.1E-7	6.3E-5	1.1E-03	2.0E-05	4.7E-05	3.0E-8	6.0E-6
tetrachloroethene (PCE)		4.8E+01	6.4E-05	3.0E-06	7.0E-06	--	1.5E-7	1.6E-05	2.9E-07	6.7E-07	--	1.4E-8
chlorobenzene		6.6E+00	1.2E-02	5.9E-04	1.4E-03	--	2.1E-4	3.2E-03	5.6E-05	1.3E-04	--	2.0E-5
benzene	2.1E-02	4.6E-01	3.8E-05	1.8E-06	4.2E-06	3.8E-8	9.1E-6	9.6E-06	1.7E-07	4.0E-07	3.6E-9	8.7E-7
toluene		2.7E+01	2.0E-04	9.2E-06	2.1E-05	--	7.9E-7	5.0E-05	8.8E-07	2.0E-06	--	7.5E-8
cis-1,2-dichloroethene		1.1E+02	1.2E-04	5.7E-06	1.3E-05	--	1.2E-7	3.1E-05	5.4E-07	1.3E-06	--	1.1E-8
trimethylbenzenes		1.8E+01	4.7E-05	2.2E-06	5.2E-06	--	2.9E-7	1.2E-05	2.1E-07	5.0E-07	--	2.8E-8

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

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 - ISZ 2 (and ISZ 1a and 1b) - Excavations

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
cis-1,2-dichloroethene	3.92E-4			3.92E-4
trimethylbenzenes	3.39E-4			3.39E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	21000	2.1E-08	1.2E-07			**1.6E-03**	
vinyl chloride	23000	2.3E-08	1.3E-07			**1.7E-03**	
chloroform	63000	6.3E-08	3.5E-07			**4.6E-03**	
trichloroethene (TCE)	3100000	3.1E-06	1.3E-05			**1.7E-01**	
tetrachloroethene (PCE)	49000	4.9E-08	1.9E-07			**2.5E-03**	
chlorobenzene	9380165	9.4E-06	3.6E-05			**4.9E-01**	
benzene	23760	2.4E-08	1.1E-07			**1.5E-03**	
toluene	21384297	2.1E-05	9.8E-05			**1.3E+00**	
cis-1,2-dichloroethene	90000	9.0E-08	3.5E-07			**4.7E-03**	
trimethylbenzenes	40800	4.1E-08	1.4E-07			**1.8E-03**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Impacted Soil Zone 2

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04** **5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.7E-7**	**7.9E-3**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.6E-03	1.2E-06	8.1E-05	1.1E-8	1.4E-5
vinyl chloride	3.1E-02	1.9E+00	1.7E-03	1.3E-06	8.8E-05	3.9E-8	4.8E-5
chloroform	1.5E-03	1.4E+00	4.6E-03	3.4E-06	2.4E-04	5.0E-9	1.7E-4
trichloroethene (TCE)	1.5E-03	7.7E+00	1.7E-01	1.3E-04	8.9E-03	1.9E-7	1.2E-3
tetrachloroethene (PCE)		4.8E+01	2.5E-03	1.8E-06	1.3E-04	--	2.7E-6
chlorobenzene		6.6E+00	4.9E-01	3.6E-04	2.5E-02	--	3.8E-3
benzene	2.1E-02	4.6E-01	1.5E-03	1.1E-06	7.6E-05	2.3E-8	1.7E-4
toluene		2.7E+01	1.3E+00	9.7E-04	6.8E-02	--	2.5E-3
cis-1,2-dichloroethene		1.1E+02	4.7E-03	3.5E-06	2.4E-04	--	2.1E-6
trimethylbenzenes		1.8E+01	1.8E-03	1.4E-06	9.5E-05	--	5.3E-6

↑ Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Impacted Soil Zone 2

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.4E-6**	**1.8E-1**			**TOTAL**	**2.8E-8**	**4.5E-2**
1,2-dichloroethane (EDC)	1.2E-02		51	5.1E-06	1.2E-05	6.2E-8	--	51	4.3E-08	3.0E-06	5.1E-10	--
vinyl chloride	2.3E+00		13	1.3E-06	3.1E-06	3.0E-6	--	13	1.1E-08	7.6E-07	2.5E-8	--
chloroform		6.5E-03	77	7.7E-06	1.8E-05	--	2.8E-3	77	6.5E-08	4.5E-06	--	7.0E-4
trichloroethene (TCE)		2.4E-02	154	1.5E-05	3.6E-05	--	1.5E-3	385	3.2E-07	2.3E-05	--	9.5E-4
benzene	3.5E-02		3.2	3.2E-07	7.5E-07	1.1E-8	--	3.2	2.7E-09	1.9E-07	9.4E-11	--
PAHs (BaP equivalent)	5.0E-01		5.6	5.6E-07	1.3E-06	2.8E-7	--	5.6	4.7E-09	3.3E-07	2.3E-9	--
1,4-dichlorobenzene		1.1E-02	7.7	7.7E-07	1.8E-06	--	1.7E-4	15.2	1.3E-08	8.9E-07	--	8.3E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	1480	1.5E-04	3.5E-04	--	1.7E-1	1480	1.2E-06	8.7E-05	--	4.3E-2
			↑					↑				
			Maximum concentration in top 1m soils					Maximum concentration in soil from all depths				

Dermal Exposure to Chemicals via Contact with Soil

Impacted Soil Zone 2

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.4E-6**	**7.5E-2**			**TOTAL**	**2.5E-8**	**4.0E-2**
1,2-dichloroethane (EDC)	1.2E-02		51	2.2E-06	5.1E-06	2.6E-8	--	51	3.8E-08	2.7E-06	4.6E-10	--
vinyl chloride	2.3E+00		13	5.5E-07	1.3E-06	1.3E-6	--	13	9.7E-09	6.8E-07	2.2E-8	--
chloroform		6.5E-03	77	3.3E-06	7.6E-06	--	1.2E-3	77	5.8E-08	4.0E-06	--	6.2E-4
trichloroethene (TCE)		2.4E-02	154	6.5E-06	1.5E-05	--	6.4E-4	385	2.9E-07	2.0E-05	--	8.5E-4
benzene	3.5E-02		3.2	1.4E-07	3.2E-07	4.8E-9	--	3.2	2.4E-09	1.7E-07	8.4E-11	--
PAHs (BaP equivalent)	5.0E-01		5.6	2.4E-07	5.6E-07	1.2E-7	--	5.6	4.2E-09	2.9E-07	2.1E-9	--
1,4-dichlorobenzene		1.1E-02	7.7	3.3E-07	7.6E-07	--	7.1E-5	15.2	1.1E-08	8.0E-07	--	7.4E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	1480	6.3E-05	1.5E-04	--	7.3E-2	1480	1.1E-06	7.8E-05	--	3.9E-2

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soil from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment ISZ 2

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**1.6E-6**	**1.2E-1**			**TOTAL**	**2.8E-8**	**6.5E-2**
1,2-dichloroethane (EDC)	1.2E-02		0.01	51	1.2E-06	2.8E-06	1.4E-8	--	51	2.1E-08	1.5E-06	2.5E-10	--
vinyl chloride	2.3E+00		0.01	13	3.1E-07	7.2E-07	7.1E-7	--	13	5.4E-09	3.8E-07	1.2E-8	--
chloroform		6.5E-03	0.01	77	1.8E-06	4.2E-06	--	6.5E-4	77	3.2E-08	2.2E-06	--	3.4E-4
trichloroethene (TCE)		2.4E-02	0.01	154	3.6E-06	8.5E-06	--	3.6E-4	385	1.6E-07	1.1E-05	--	4.7E-4
benzene	3.5E-02		0.01	3.2	7.6E-08	1.8E-07	2.6E-9	--	3.2	1.3E-09	9.3E-08	4.7E-11	--
PAHs (BaP equivalent)	5.0E-01		0.13	5.6	1.7E-06	4.0E-06	8.6E-7	--	5.6	3.0E-08	2.1E-06	1.5E-8	--
1,4-dichlorobenzene		1.1E-02	0.01	7.7	1.8E-07	4.2E-07	--	4.0E-5	15.2	6.3E-09	4.4E-07	--	4.1E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	1480	1.0E-04	2.4E-04	--	1.2E-1	1480	1.8E-06	1.3E-04	--	6.5E-2
				↑					↑				
				Maximum concentration in top 1m soils					Maximum concentration in soil from all depths				

Summary of Risk - ISZ 2

Exposure to COPC Derived from Soil and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
ISZ 1a, 1b and 2 - Commercial/Industrial Worker			
Ingestion of COPC Indetified in Shallow Soils	**3.4E-06**	**0.18**	**0.18**
Dermal Contact with COPC Identified in Shallow Soils	**1.4E-06**	**0.075**	**0.075**
Inhalation of volatile COPC derived from soils and groundwater	**4.5E-07**	**0.018**	**0.00030**
Total Risk	**5E-06**	**0.3**	**0.3**

ISZ 1a, 1b and 2 - Intrusive Worker		
Ingestion of COPC Indetified in Shallow Soils	**2.8E-08**	**0.045**
Dermal Contact with COPC Identified in Shallow Soils	**2.5E-08**	**0.040**
Inhalation of volatile COPC derived from soils and groundwater	**2.7E-07**	**0.0079**
Total Risk	**3E-07**	**0.09**

Risk Calculations ISZ 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 - ISZ 3 and Plume 2

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.1	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**1.1**	Average depth to soil gas data
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m^2]	emarea	**625**	Assumed size of building 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	**50**	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	**50**	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	**2500**	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
chlorobenzene	3.89E-4			3.89E-4
toluene	4.60E-4			4.60E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	6400	6.4E-09	3.3E-10		**2.0E-05**		**5.0E-06**
chlorobenzene	97826	9.8E-08	3.5E-09		**2.1E-04**		**5.3E-05**
toluene	60277	6.0E-08	2.5E-09		**1.5E-04**		**3.8E-05**

↑ Maximum soil gas concentration from source zone

↑ Calculated based on maximum soil gas concentration

Inhalation of Volatile Chemicals

Identified Soil Zone 3 and Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**9.1E-9**	**4.1E-4**			**TOTAL**	**8.7E-10**	**3.9E-5**
1,2-dichloroethane (EDC)	9.8E-03		1.96E-05	9.2E-07	2.2E-06	9.1E-9	--	5.0E-06	8.8E-08	2.1E-07	8.7E-10	--
chlorobenzene		1.4E-01	2.07E-04	9.8E-06	2.3E-05	--	1.6E-4	5.3E-05	9.3E-07	2.2E-06	--	1.5E-5
toluene		6.7E-02	1.51E-04	7.1E-06	1.7E-05	--	2.5E-4	3.8E-05	6.8E-07	1.6E-06	--	2.4E-5

Calculated from maximum measured emission rates and soil gas concentrations

Calculated from maximum measured emission rates and soil gas concentrations

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Soil Zone 3 and Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**9.1E-9**	**4.5E-6**			**TOTAL**	**8.7E-10**	**4.3E-7**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.0E-05	9.2E-07	2.2E-06	9.1E-9	3.8E-7	5.0E-06	8.8E-08	2.1E-07	8.7E-10	3.6E-8
chlorobenzene		6.6E+00	2.1E-04	9.8E-06	2.3E-05	--	3.5E-6	5.3E-05	9.3E-07	2.2E-06	--	3.3E-7
toluene		2.7E+01	1.5E-04	7.1E-06	1.7E-05	--	6.1E-7	3.8E-05	6.8E-07	1.6E-06	--	5.8E-8

Calculated from maximum measured emission rates and soil gas concentrations

Calculated from maximum measured emission rates and soil gas concentrations

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 - Excavations in ISZ 3 and Plume 2

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavation
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
chlorobenzene	3.89E-4			3.89E-4
toluene	4.60E-4			4.60E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	6400	6.4E-09	3.6E-08			**4.8E-04**	
chlorobenzene	97826	9.8E-08	3.8E-07			**5.1E-03**	
toluene	60277	6.0E-08	2.8E-07			**3.7E-03**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Soil Zone 3 and Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04** **5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	3.5E-9	5.1E-5
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	4.8E-04	3.5E-07	2.5E-05	3.5E-9	4.3E-6
chlorobenzene		6.6E+00	5.1E-03	3.7E-06	2.6E-04	--	4.0E-5
toluene		2.7E+01	3.7E-03	2.7E-06	1.9E-04	--	7.0E-6

Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Identified Soil Zone 3

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.7E-7**	**2.6E-1**			**TOTAL**	**6.9E-9**	**6.5E-2**
1,2-dichloroethane (EDC)	1.2E-02		226	2.3E-05	5.3E-05	2.7E-7	--	687	5.8E-07	4.0E-05	6.9E-9	--
trichloroethene (TCE)		2.4E-02	1	1.0E-07	2.3E-07	--	9.9E-6	2	1.7E-09	1.2E-07	--	4.9E-6
b-HCH (b-BHC)		2.0E-05	10	1.0E-06	2.3E-06	--	1.2E-1	10	8.4E-09	5.9E-07	--	2.9E-2
g-HCH (g-BHC or lindane)		3.0E-03	10	1.0E-06	2.3E-06	--	7.8E-4	10	8.4E-09	5.9E-07	--	2.0E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1220	1.2E-04	2.9E-04	--	1.4E-1	1220	1.0E-06	7.2E-05	--	3.6E-2

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Soil Zone 3

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.2E-7**	**1.1E-1**			**TOTAL**	**6.2E-9**	**5.8E-2**
1,2-dichloroethane (EDC)	1.2E-02		226	9.6E-06	2.2E-05	1.2E-7	--	687	5.1E-07	3.6E-05	6.2E-9	--
trichloroethene (TCE)		2.4E-02	1	4.2E-08	9.9E-08	--	4.2E-6	2	1.5E-09	1.0E-07	--	4.4E-6
b-HCH (b-BHC)		2.0E-05	10	4.2E-07	9.9E-07	--	5.0E-2	10	7.5E-09	5.2E-07	--	2.6E-2
g-HCH (g-BHC or lindane)		3.0E-03	10	4.2E-07	9.9E-07	--	3.3E-4	10	7.5E-09	5.2E-07	--	1.7E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1220	5.2E-05	1.2E-04	--	6.0E-2	1220	9.1E-07	6.4E-05	--	3.2E-2

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 3**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*FE*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**6.4E-8**	**1.3E-1**			**TOTAL**	**3.4E-9**	**6.8E-2**
1,2-dichloroethane (EDC)	1.2E-02		0.01	226	5.3E-06	1.2E-05	6.4E-8	--	687	2.9E-07	2.0E-05	3.4E-9	--
trichloroethene (TCE)		2.4E-02	0.01	1	2.4E-08	5.5E-08	--	2.3E-6	2	8.3E-10	5.8E-08	--	2.4E-6
b-HCH (b-BHC)		2.0E-05	0.01	10	2.4E-07	5.5E-07	--	2.8E-2	10	4.2E-09	2.9E-07	--	1.5E-2
g-HCH (g-BHC or lindane)		3.0E-03	0.01	10	2.4E-07	5.5E-07	--	1.8E-4	10	4.2E-09	2.9E-07	--	9.7E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	1220	8.6E-05	2.0E-04	--	1.0E-1	1220	1.5E-06	1.1E-04	--	5.3E-2

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Summary of Risk - ISZ 3

Exposure to COPC Derived from Soils and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
ISZ 3 - Commercial/Industrial Workers			
Ingestion of COPC Indetified in Shallow Soils	**2.7E-07**	**0.26**	**0.26**
Dermal Contact with COPC Identified in Shallow Soils	**1.2E-07**	**0.11**	**0.11**
Inhalation of volatile COPC derived from soils and groundwater	**9.1E-09**	**0.00041**	**0.0000045**
Total Risk	**4E-07**	**0.4**	**0.4**

ISZ 3 - Intrusive Workers		
Ingestion of COPC Indetified in Shallow Soils	**6.9E-09**	**0.065**
Dermal Contact with COPC Identified in Shallow Soils	**6.2E-09**	**0.058**
Inhalation of volatile COPC derived from soils and groundwater	**3.5E-09**	**0.000051**
Total Risk	**2E-08**	**0.1**

Risk Calculations ISZ 4

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 - ISZ 4 and GW Impacts from Plume 3

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.5	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1.5	Assumed depth to soil gas samples
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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	50	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	50	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	2500	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
ethylbenzene	3.96E-4			3.96E-4
xylenes	3.70E-4			3.70E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
chlorobenzene	3.89E-4			3.89E-4
chloroform	5.52E-4			5.52E-4
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
1,2,4-trimethylbenzene	3.39E-4			3.39E-4
1,3,5-trimethylbenzene	3.39E-4			3.39E-4
vinyl chloride	5.58E-4			5.58E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
benzene	64000	6.4E-08	2.0E-09	3.7E-10	**2.2E-05**		**5.6E-06**
toluene	2300000	2.3E-06	7.1E-08	5.2E-09	**3.1E-04**		**7.9E-05**
ethylbenzene	46000	4.6E-08	1.2E-09	1.0E-09	**6.0E-05**		**1.5E-05**
xylenes	260000	2.6E-07	6.4E-09	8.5E-10	**5.1E-05**		**1.3E-05**
trichloroethene (TCE)	4000000	4.0E-06	1.1E-07	3.7E-09	**2.2E-04**		**5.6E-05**
chlorobenzene	660000	6.6E-07	1.7E-08	4.5E-10	**2.7E-05**		**6.9E-06**
chloroform	100000	1.0E-07	3.7E-09	7.7E-10	**4.6E-05**		**1.2E-05**
1,2-dichloroethane (EDC)	13000000	1.3E-05	4.9E-07	4.4E-09	**2.6E-04**		**6.7E-05**
1,2,4-trimethylbenzene	4900	4.9E-09	1.1E-10	1.2E-09	**7.0E-05**		**1.8E-05**
1,3,5-trimethylbenzene	3200	3.2E-09	7.2E-11		**4.3E-06**		**1.1E-06**
vinyl chloride	2400	2.4E-09	8.9E-11		**5.4E-06**		**1.4E-06**

Maximum soil gas concentration from source zone

Maximum emissions rate from source sone

Calculated based on maximum emission rate, then maximum soil gas data (note maximum SG data from 2007 provides concentrations equal to those estimated on the basis of maximum flux emissions from old data)

Inhalation of Volatile Chemicals

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02** **1.1E-01**	**NonThreshold** **Threshold**	**1.8E-02** **4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**1.7E-7**	**1.0E-2**			**TOTAL**	**1.6E-8**	**9.9E-4**
1,2-dichloroethane (EDC)	9.8E-03		2.62E-04	1.2E-05	2.9E-05	1.2E-7	--	6.7E-05	1.2E-06	2.8E-06	1.2E-8	--
vinyl chloride	3.1E-02		5.36E-06	2.5E-07	5.9E-07	7.8E-9	--	1.4E-06	2.4E-08	5.6E-08	7.4E-10	--
chloroform	1.5E-03	2.0E-02	4.60E-05	2.2E-06	5.1E-06	3.2E-9	2.5E-4	1.2E-05	2.1E-07	4.8E-07	3.0E-10	2.4E-5
trichloroethene (TCE)	1.5E-03		2.20E-04	1.0E-05	2.4E-05	1.6E-8	--	5.6E-05	9.9E-07	2.3E-06	1.5E-9	--
chlorobenzene		1.4E-01	2.70E-05	1.3E-06	3.0E-06	--	2.1E-5	6.9E-06	1.2E-07	2.8E-07	--	2.0E-6
benzene	2.1E-02		2.20E-05	1.0E-06	2.4E-06	2.2E-8	--	5.6E-06	9.9E-08	2.3E-07	2.1E-9	--
toluene		6.7E-02	3.10E-04	1.5E-05	3.4E-05	--	5.1E-4	7.9E-05	1.4E-06	3.3E-06	--	4.9E-5
ethylbenzene		6.3E+00	6.00E-05	2.8E-06	6.6E-06	--	1.0E-6	1.5E-05	2.7E-07	6.3E-07	--	1.0E-7
xylenes		2.4E-01	5.10E-05	2.4E-06	5.6E-06	--	2.3E-5	1.3E-05	2.3E-07	5.4E-07	--	2.2E-6
trimethylbenzenes		8.6E-04	7.43E-05	3.5E-06	8.2E-06	--	9.5E-3	1.9E-05	3.3E-07	7.8E-07	--	9.1E-4

Calculated from maximum measured emission rates and soil gas concentrations

Calculated from maximum measured emission rates and soil gas concentrations

Inhalation of Volatile Chemicals in Areas Above Main Plumes (Evaluated using Occupational TWA Values)

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**1.7E-7**	**2.0E-5**			**TOTAL**	**1.6E-8**	**1.9E-6**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.6E-04	1.2E-05	2.9E-05	1.2E-7	5.0E-6	6.7E-05	1.2E-06	2.8E-06	1.2E-8	4.8E-7
vinyl chloride	3.1E-02	1.9E+00	5.4E-06	2.5E-07	5.9E-07	7.8E-9	3.2E-7	1.4E-06	2.4E-08	5.6E-08	7.4E-10	3.0E-8
chloroform	1.5E-03	1.4E+00	4.6E-05	2.2E-06	5.1E-06	3.2E-9	3.5E-6	1.2E-05	2.1E-07	4.8E-07	3.0E-10	3.4E-7
trichloroethene (TCE)	1.5E-03	7.7E+00	2.2E-04	1.0E-05	2.4E-05	1.6E-8	3.1E-6	5.6E-05	9.9E-07	2.3E-06	1.5E-9	3.0E-7
chlorobenzene		6.6E+00	2.7E-05	1.3E-06	3.0E-06	--	4.5E-7	6.9E-06	1.2E-07	2.8E-07	--	4.3E-8
benzene	2.1E-02	4.6E-01	2.2E-05	1.0E-06	2.4E-06	2.2E-8	5.3E-6	5.6E-06	9.9E-08	2.3E-07	2.1E-9	5.1E-7
toluene		2.7E+01	3.1E-04	1.5E-05	3.4E-05	--	1.2E-6	7.9E-05	1.4E-06	3.3E-06	--	1.2E-7
ethylbenzene		6.2E+01	6.0E-05	2.8E-06	6.6E-06	--	1.1E-7	1.5E-05	2.7E-07	6.3E-07	--	1.0E-8
xylenes		5.0E+01	5.1E-05	2.4E-06	5.6E-06	--	1.1E-7	1.3E-05	2.3E-07	5.4E-07	--	1.1E-8
trimethylbenzenes		1.8E+01	7.4E-05	3.5E-06	8.2E-06	--	4.6E-7	1.9E-05	3.3E-07	7.8E-07	--	4.4E-8

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

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 - Excavations in Area A

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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	4.66E-4			4.66E-4
Toluene	4.60E-4			4.60E-4
Ethylbenzene	3.96E-4			3.96E-4
Xylenes	3.70E-4			3.70E-4
Trichloroethene (TCE)	4.17E-4			4.17E-4
Chlorobenzene	3.89E-4			3.89E-4
Chloroform	5.52E-4			5.52E-4
1,2-Dichloroethane (EDC)	5.62E-4			5.62E-4
1,2,4-Trimethylbenzene	3.39E-4			3.39E-4
1,3,5-Trimethylbenzene	3.39E-4			3.39E-4
vinyl chloride	5.58E-4			5.58E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
Benzene	64000	6.4E-08	3.0E-07			**4.0E-03**	
Toluene	2300000	2.3E-06	1.1E-05			**1.4E-01**	
Ethylbenzene	46000	4.6E-08	1.8E-07			**2.4E-03**	
Xylenes	260000	2.6E-07	9.6E-07			**1.3E-02**	
Trichloroethene (TCE)	4000000	4.0E-06	1.7E-05			**2.2E-01**	
Chlorobenzene	660000	6.6E-07	2.6E-06			**3.4E-02**	
Chloroform	100000	1.0E-07	5.5E-07			**7.4E-03**	
1,2-Dichloroethane (EDC)	13000000	1.3E-05	7.3E-05			**9.7E-01**	
1,2,4-Trimethylbenzene	4900	4.9E-09	1.7E-08			**2.2E-04**	
1,3,5-Trimethylbenzene	3200	3.2E-09	1.1E-08			**1.4E-04**	
vinyl chloride	2400	2.4E-09	1.3E-08			**1.8E-04**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04** **5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	7.4E-6	1.2E-2
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	9.7E-01	7.2E-04	5.0E-02	7.0E-6	8.8E-3
vinyl chloride	3.1E-02	1.9E+00	1.8E-04	1.3E-07	9.2E-06	4.1E-9	5.0E-6
chloroform	1.5E-03	1.4E+00	7.4E-03	5.4E-06	3.8E-04	8.0E-9	2.7E-4
trichloroethene (TCE)	1.5E-03	7.7E+00	2.2E-01	1.6E-04	1.2E-02	2.5E-7	1.5E-3
chlorobenzene		6.6E+00	3.4E-02	2.5E-05	1.8E-03	--	2.7E-4
benzene	2.1E-02	4.6E-01	4.0E-03	2.9E-06	2.1E-04	6.2E-8	4.5E-4
toluene		2.7E+01	1.4E-01	1.0E-04	7.3E-03	--	2.7E-4
ethylbenzene		6.2E+01	2.4E-03	1.8E-06	1.3E-04	--	2.0E-6
xylenes		5.0E+01	1.3E-02	9.5E-06	6.6E-04	--	1.3E-5
trimethylbenzenes		1.8E+01	3.7E-04	2.7E-07	1.9E-05	--	1.1E-6

↑ Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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		**5.9E+0**			**TOTAL**	**3.2E-9**	**1.5E+0**
hexachlorobenzene (HCB)		1.6E-04	33	3.3E-06	7.7E-06	--	4.8E-2	33	2.8E-08	1.9E-06	--	1.2E-2
1,2-dichloroethane (EDC)	1.2E-02					--	--	292	2.4E-07	1.7E-05	2.9E-9	--
chloroform		6.5E-03				--	--	2.7	2.3E-09	1.6E-07	--	2.4E-5
trichloroethene (TCE)		2.4E-02				--	--	79	6.6E-08	4.6E-06	--	1.9E-4
benzene	3.5E-02					--	--	9.8	8.2E-09	5.8E-07	2.9E-10	--
a-HCH (a-BHC)		8.0E-03	3.2	3.2E-07	7.5E-07	--	9.4E-5	3.2	2.7E-09	1.9E-07	--	2.3E-5
b-HCH (b-BHC)		2.0E-05	32	3.2E-06	7.5E-06	--	3.8E-1	32	2.7E-08	1.9E-06	--	9.4E-2
d-HCH (d-BHC)		2.0E-05	1.6	1.6E-07	3.8E-07	--	1.9E-2	1.6	1.3E-09	9.4E-08	--	4.7E-3
g-HCH (g-BHC or lindane)		3.0E-03	32	3.2E-06	7.5E-06	--	2.5E-3	32	2.7E-08	1.9E-06	--	6.3E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	45750	4.6E-03	1.1E-02	--	5.4E+0	45750	3.8E-05	2.7E-03	--	1.3E+0
mercury (as total, inroganic)		3.6E-04	116	1.2E-05	2.7E-05	--	7.7E-2	116	9.7E-08	6.8E-06	--	1.9E-2

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL		**2.5E+0**			**TOTAL**	**2.9E-9**	**1.3E+0**
hexachlorobenzene (HCB)		1.6E-04	33	1.4E-06	3.3E-06	--	2.0E-2	33	2.5E-08	1.7E-06	--	1.1E-2
1,2-dichloroethane (EDC)	1.2E-02					--	--	292	2.2E-07	1.5E-05	2.6E-9	--
chloroform		6.5E-03				--	--	2.7	2.0E-09	1.4E-07	--	2.2E-5
trichloroethene (TCE)		2.4E-02				--	--	79	5.9E-08	4.1E-06	--	1.7E-4
benzene	3.5E-02					--	--	9.8	7.3E-09	5.1E-07	2.6E-10	--
a-HCH (a-BHC)		8.0E-03	3.2	1.4E-07	3.2E-07	--	4.0E-5	3.2	2.4E-09	1.7E-07	--	2.1E-5
b-HCH (b-BHC)		2.0E-05	32	1.4E-06	3.2E-06	--	1.6E-1	32	2.4E-08	1.7E-06	--	8.4E-2
d-HCH (d-BHC)		2.0E-05	1.6	6.8E-08	1.6E-07	--	7.9E-3	1.6	1.2E-09	8.4E-08	--	4.2E-3
g-HCH (g-BHC or lindane)		3.0E-03	32	1.4E-06	3.2E-06	--	1.1E-3	32	2.4E-08	1.7E-06	--	5.6E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	45750	1.9E-03	4.5E-03	--	2.3E+0	45750	3.4E-05	2.4E-03	--	1.2E+0
mercury (as total, inroganic)		3.6E-04	116	4.9E-06	1.2E-05	--	3.2E-2	116	8.7E-08	6.1E-06	--	1.7E-2
			↑ Maximum concentration in top 1m soils					↑ Maximum concentration in soils from all depths				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 4**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL		**4.0E+0**			**TOTAL**	**1.6E-9**	**2.1E+0**
hexachlorobenzene (HCB)		1.6E-04	0.10	33	7.8E-06	1.8E-05	--	1.1E-1	33	1.4E-07	9.6E-06	--	6.0E-2
1,2-dichloroethane (EDC)	1.2E-02		0.01				--	--	292	1.2E-07	8.5E-06	1.5E-9	--
chloroform		6.5E-03	0.01				--	--	2.7	1.1E-09	7.9E-08	--	1.2E-5
trichloroethene (TCE)		2.4E-02	0.01				--	--	79	3.3E-08	2.3E-06	--	9.7E-5
benzene	3.5E-02		0.01				--	--	9.8	4.1E-09	2.9E-07	1.4E-10	--
a-HCH (a-BHC)		8.0E-03	0.01	3.2	7.6E-08	1.8E-07	--	2.2E-5	3.2	1.3E-09	9.3E-08	--	1.2E-5
b-HCH (b-BHC)		2.0E-05	0.01	32	7.6E-07	1.8E-06	--	8.8E-2	32	1.3E-08	9.3E-07	--	4.7E-2
d-HCH (d-BHC)		2.0E-05	0.01	1.6	3.8E-08	8.8E-08	--	4.4E-3	1.6	6.7E-10	4.7E-08	--	2.3E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	32	7.6E-07	1.8E-06	--	5.9E-4	32	1.3E-08	9.3E-07	--	3.1E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	45750	3.2E-03	7.6E-03	--	3.8E+0	45750	5.7E-05	4.0E-03	--	2.0E+0
mercury (as total, inroganic)		3.6E-04	0.01	116	2.7E-06	6.4E-06	--	1.8E-2	116	4.8E-08	3.4E-06	--	9.5E-3

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Summary of Risk - Identified Source Zone 4

Exposure to COPC Derived from Soils and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
Commercial/Industrial Workers - GW Impacts and Source Zone 4			
Ingestion of COPC Indetified in Shallow Soils	**0.0E+00**	**5.9**	**5.9**
Dermal Contact with COPC Identified in Shallow Soils	**0.0E+00**	**2.5**	**2.5**
Inhalation of volatile COPC derived from soils and groundwater	**1.9E-07**	**0.011**	**0.000022**
Total Risk	**2.E-07**	**8**	**8**

Intrusive Workers - GW Impacts and Source Zone 4		
Ingestion of COPC Indetified in Shallow Soils	**3.2E-09**	**1.5**
Dermal Contact with COPC Identified in Shallow Soils	**2.9E-09**	**1.3**
Inhalation of volatile COPC derived from soils and groundwater (TWAs)	**7.4E-06**	**0.012**
Total Risk	**7E-06**	**3**

Risk Calculations ISZ 5

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 - ISZ 5

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.75	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1.75	Depth to soil gas sample
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m^2]	emarea	625	Assumed size of building 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	50	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	50	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	2500	calculated
Q/C Dispersion term	[g/m^2/s per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
1,1,1,2-tetrachloroethane	2.28E-4			2.28E-4
1,2-dichloropropane	4.16E-4			4.16E-4
1,1-dichloroethene	4.80E-4			4.80E-4

Calculated Air Concentrations	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)	Measured Emission Rate from Surface of Ground (g/s/m^2)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	889000	8.9E-07	2.9E-08		**1.7E-03**		**4.4E-04**
vinyl chloride	81000	8.1E-08	2.6E-09		**1.6E-04**		**3.9E-05**
chloroform	20000	2.0E-08	6.3E-10		**3.8E-05**		**9.6E-06**
trichloroethene (TCE)	35000	3.5E-08	8.3E-10		**5.0E-05**		**1.3E-05**
tetrachloroethene (PCE)	51000	5.1E-08	1.1E-09		**6.6E-05**		**1.7E-05**
1,1,2-trichloroethane	4660000	4.7E-06	1.1E-07	1.2E-07	**7.3E-03**		**1.9E-03**
chlorobenzene	133000	1.3E-07	3.0E-09		**1.8E-04**		**4.5E-05**
toluene	120000	1.2E-07	3.2E-09	3.0E-09	**1.8E-04**		**4.6E-05**
1,2,3-trichloropropane	125000	1.3E-07	2.9E-09		**1.7E-04**		**4.4E-05**
1,1,1,2-tetrachloroethane	16000	1.6E-08	2.1E-10		**1.3E-05**		**3.2E-06**
1,2-dichloropropane	168000	1.7E-07	4.0E-09	2.2E-09	**1.3E-04**		**3.3E-05**
1,1-dichloroethene	100000	1.0E-07	2.7E-09		**1.6E-04**		**4.2E-05**

Maximum soil gas concentration from source zone

Maximum emissions rate from source sone

Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**6.0E-5**	**2.2E-1**			**TOTAL**	**5.6E-6**	**2.1E-2**
1,2-dichloroethane (EDC)	9.8E-03		1.7E-03	8.1E-05	1.9E-04	7.9E-7	--	4.4E-04	7.7E-06	1.8E-05	7.6E-8	--
vinyl chloride	3.1E-02		1.6E-04	7.3E-06	1.7E-05	2.2E-7	--	3.9E-05	7.0E-07	1.6E-06	2.1E-8	--
chloroform	1.5E-03	2.0E-02	3.8E-05	1.8E-06	4.2E-06	2.6E-9	2.1E-4	9.6E-06	1.7E-07	4.0E-07	2.5E-10	2.0E-5
trichloroethene (TCE)	1.5E-03		5.0E-05	2.4E-06	5.5E-06	3.6E-9	--	1.3E-05	2.3E-07	5.3E-07	3.4E-10	--
tetrachloroethene (PCE)		4.7E-02	6.6E-05	3.1E-06	7.3E-06	--	1.5E-4	1.7E-05	3.0E-07	7.0E-07	--	1.5E-5
1,1,2-trichloroethane		4.0E-03	7.3E-03	3.4E-04	8.0E-04	--	2.0E-1	1.9E-03	3.3E-05	7.7E-05	--	1.9E-2
chlorobenzene		1.4E-01	1.8E-04	8.3E-06	1.9E-05	--	1.4E-4	4.5E-05	8.0E-07	1.9E-06	--	1.3E-5
toluene		7.4E-02	1.8E-04	8.5E-06	2.0E-05	--	2.7E-4	4.6E-05	8.1E-07	1.9E-06	--	2.5E-5
1,2,3-trichloropropane	7.0E+00		1.7E-04	8.2E-06	1.9E-05	5.8E-5	--	4.4E-05	7.9E-07	1.8E-06	5.5E-6	--
1,1,1,2-tetrachloroethane		3.0E-02	1.3E-05	5.9E-07	1.4E-06	--	4.6E-5	3.2E-06	5.6E-08	1.3E-07	--	4.4E-6
1,2-dichloropropane		1.0E-03	1.3E-04	6.1E-06	1.4E-05	--	1.4E-2	3.3E-05	5.8E-07	1.4E-06	--	1.3E-3
1,1-dichloroethene		5.7E-02	1.6E-04	7.7E-06	1.8E-05	--	3.2E-4	4.2E-05	7.4E-07	1.7E-06	--	3.0E-5

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**5.9E-5**	**1.6E-4**			**TOTAL**	**5.6E-6**	**1.5E-5**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.7E-03	8.1E-05	1.9E-04	7.9E-7	3.3E-5	4.4E-04	7.7E-06	1.8E-05	7.6E-8	3.1E-6
vinyl chloride	3.1E-02	1.9E+00	1.6E-04	7.3E-06	1.7E-05	2.2E-7	9.2E-6	3.9E-05	7.0E-07	1.6E-06	2.1E-8	8.8E-7
chloroform	1.5E-03	1.4E+00	3.8E-05	1.8E-06	4.2E-06	2.6E-9	2.9E-6	9.6E-06	1.7E-07	4.0E-07	2.5E-10	2.8E-7
trichloroethene (TCE)	1.5E-03	7.7E+00	5.0E-05	2.4E-06	5.5E-06	3.6E-9	7.1E-7	1.3E-05	2.3E-07	5.3E-07	3.4E-10	6.8E-8
tetrachloroethene (PCE)		4.8E+01	6.6E-05	3.1E-06	7.3E-06	--	1.5E-7	1.7E-05	3.0E-07	7.0E-07	--	1.5E-8
1,1,2-trichloroethane		7.9E+00	7.3E-03	3.4E-04	8.0E-04	--	1.0E-4	1.9E-03	3.3E-05	7.7E-05	--	9.8E-6
chlorobenzene		6.6E+00	1.8E-04	8.3E-06	1.9E-05	--	3.0E-6	4.5E-05	8.0E-07	1.9E-06	--	2.8E-7
toluene		2.7E+01	1.8E-04	8.5E-06	2.0E-05	--	7.3E-7	4.6E-05	8.1E-07	1.9E-06	--	6.9E-8
1,2,3-trichloropropane	7.0E+00	8.6E+00	1.7E-04	8.2E-06	1.9E-05	5.8E-5	2.2E-6	4.4E-05	7.9E-07	1.8E-06	5.5E-6	2.1E-7
1,1,1,2-tetrachloroethane			1.3E-05	5.9E-07	1.4E-06	--	--	3.2E-06	5.6E-08	1.3E-07	--	--
1,2-dichloropropane		5.0E+01	1.3E-04	6.1E-06	1.4E-05	--	2.9E-7	3.3E-05	5.8E-07	1.4E-06	--	2.8E-8
1,1-dichloroethene		2.9E+00	1.6E-04	7.7E-06	1.8E-05	--	6.3E-6	4.2E-05	7.4E-07	1.7E-06	--	6.0E-7

Calculated from maximum measured emission rates and soil gas concentrations

Calculated from maximum measured emission rates and soil gas concentrations

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 - Excavations in ISZ 5

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

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)
1,2-dichloroethane (EDC)	8690	98.96	17.4	0.104	9.9E-06	79.1	0.0401
vinyl chloride	2760	62.5	18.6	0.106	1.2E-06	2600	1.107
chloroform	7920	119.38	39.8	0.104	1.0E-05	160	0.15
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
1,1,2-trichloroethane	1100	133.41	67.7	0.078	8.8E-06	22.49	0.0374
chlorobenzene	500	112.56	268	0.073	8.7E-06	8.8	0.127
toluene	526	92	182	0.087	8.6E-06	30	0.27
1,2,3-trichloropropane	2700	147.43	130.8	0.071	7.9E-06	3.1	0.014
1,1,1,2-tetrachloroethane	1070	167.85	96.63	0.0423	9.1E-06	12	0.0989
1,2-dichloropropane	2800	112.99	67.7	0.0782	8.7E-06	53.3	0.115
1,1-dichloroethene	2420	96.95	35.04	0.09	1.0E-04	634	1.07

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
1,1,1,2-tetrachloroethane	2.28E-4			2.28E-4
1,2-dichloropropane	4.16E-4			4.16E-4
1,1-dichloroethene	4.80E-4			4.80E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	889000	8.9E-07	5.0E-06			**6.7E-02**	
vinyl chloride	81000	8.1E-08	4.5E-07			**6.0E-03**	
chloroform	20000	2.0E-08	1.1E-07			**1.5E-03**	
trichloroethene (TCE)	35000	3.5E-08	1.5E-07			**1.9E-03**	
tetrachloroethene (PCE)	51000	5.1E-08	1.9E-07			**2.6E-03**	
1,1,2-trichloroethane	4660000	4.7E-06	2.0E-05			**2.6E-01**	
chlorobenzene	133000	1.3E-07	5.2E-07			**6.9E-03**	
toluene	120000	1.2E-07	5.5E-07			**7.4E-03**	
1,2,3-trichloropropane	125000	1.3E-07	5.1E-07			**6.8E-03**	
1,1,1,2-tetrachloroethane	16000	1.6E-08	3.7E-08			**4.9E-04**	
1,2-dichloropropane	168000	1.7E-07	7.0E-07			**9.3E-03**	
1,1-dichloroethene	100000	1.0E-07	4.8E-07			**6.4E-03**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04**	**NonThreshold**
		5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.6E-5**	**2.8E-3**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	6.7E-02	4.9E-05	3.4E-03	4.8E-7	6.0E-4
vinyl chloride	3.1E-02	1.9E+00	6.0E-03	4.5E-06	3.1E-04	1.4E-7	1.7E-4
chloroform	1.5E-03	1.4E+00	1.5E-03	1.1E-06	7.6E-05	1.6E-9	5.3E-5
trichloroethene (TCE)	1.5E-03	7.7E+00	1.9E-03	1.4E-06	1.0E-04	2.2E-9	1.3E-5
tetrachloroethene (PCE)		4.8E+01	2.6E-03	1.9E-06	1.3E-04	--	2.8E-6
1,1,2-trichloroethane		7.9E+00	2.6E-01	1.9E-04	1.4E-02	--	1.7E-3
chlorobenzene		6.6E+00	6.9E-03	5.1E-06	3.6E-04	--	5.4E-5
toluene		2.7E+01	7.4E-03	5.4E-06	3.8E-04	--	1.4E-5
1,2,3-trichloropropane	7.0E+00	8.6E+00	6.8E-03	5.0E-06	3.5E-04	3.5E-5	4.1E-5
1,1,1,2-tetrachloroethane			4.9E-04	3.6E-07	2.5E-05	--	--
1,2-dichloropropane		5.0E+01	9.3E-03	6.9E-06	4.8E-04	--	9.7E-6
1,1-dichloroethene		2.9E+00	6.4E-03	4.7E-06	3.3E-04	--	1.2E-4

Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**7.5E-7**	**1.9E+0**			**TOTAL**	**9.5E-9**	**5.1E-1**
hexachlorobenzene (HCB)		1.6E-04	1.38	1.4E-07	3.2E-07	--	2.0E-3	1.38	1.2E-09	8.1E-08	--	5.1E-4
1,2-dichloroethane (EDC)	1.2E-02					--	--	29.4	2.5E-08	1.7E-06	3.0E-10	--
chloroform		6.5E-03				--	--	3.3	2.8E-09	1.9E-07	--	3.0E-5
trichloroethene (TCE)		2.4E-02				--	--	3.4	2.9E-09	2.0E-07	--	8.4E-6
chlorobenzene		8.6E-02				--	--	546	4.6E-07	3.2E-05	--	3.7E-4
ethylbenzene		9.7E-02	127	1.3E-05	3.0E-05	--	3.1E-4	127	1.1E-07	7.5E-06	--	7.7E-5
xylenes		1.8E-01	595	6.0E-05	1.4E-04	--	8.0E-4	595	5.0E-07	3.5E-05	--	2.0E-4
PAHs (BaP equivalent)	5.0E-01		14.92	1.5E-06	3.5E-06	7.5E-7	--	14.9	1.2E-08	8.7E-07	6.2E-9	--
1,4-dichlorobenzene		1.1E-02				--	--	10.8	9.1E-09	6.3E-07	--	5.9E-5
1,2,3-trichloropropane	7.0E+00					--	--	0.5	4.2E-10	2.9E-08	2.9E-9	--
a-HCH (a-BHC)		8.0E-03	0.64	6.4E-08	1.5E-07	--	1.9E-5	2	1.7E-09	1.2E-07	--	1.5E-5
b-HCH (b-BHC)		2.0E-05	3	3.0E-07	7.0E-07	--	3.5E-2	7.8	6.5E-09	4.6E-07	--	2.3E-2
d-HCH (d-BHC)		2.0E-05				--	--	3.7	3.1E-09	2.2E-07	--	1.1E-2
g-HCH (g-BHC or lindane)		3.0E-03	3	3.0E-07	7.0E-07	--	2.3E-4	7.8	6.5E-09	4.6E-07	--	1.5E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	16183	1.6E-03	3.8E-03	--	1.9E+0	16183	1.4E-05	9.5E-04	--	4.8E-1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.2E-7**	**8.2E-1**			**TOTAL**	**8.5E-9**	**4.6E-1**
hexachlorobenzene (HCB)		1.6E-04	1.38	5.9E-08	1.4E-07	--	8.6E-4	1.38	1.0E-09	7.2E-08	--	4.5E-4
1,2-dichloroethane (EDC)	1.2E-02					--	--	29.4	2.2E-08	1.5E-06	2.6E-10	--
chloroform		6.5E-03				--	--	3.3	2.5E-09	1.7E-07	--	2.7E-5
trichloroethene (TCE)		2.4E-02				--	--	3.4	2.5E-09	1.8E-07	--	7.5E-6
chlorobenzene		8.6E-02				--	--	546	4.1E-07	2.9E-05	--	3.3E-4
ethylbenzene		9.7E-02	127	5.4E-06	1.3E-05	--	1.3E-4	127	9.5E-08	6.7E-06	--	6.9E-5
xylenes		1.8E-01	595	2.5E-05	5.9E-05	--	3.4E-4	595	4.5E-07	3.1E-05	--	1.8E-4
PAHs (BaP equivalent)	5.0E-01		14.92	6.3E-07	1.5E-06	3.2E-7	--	14.9	1.1E-08	7.8E-07	5.6E-9	--
1,4-dichlorobenzene		1.1E-02				--	--	10.8	8.1E-09	5.7E-07	--	5.3E-5
1,2,3-trichloropropane	7.0E+00					--	--	0.5	3.7E-10	2.6E-08	2.6E-9	--
a-HCH (a-BHC)		8.0E-03	0.64	2.7E-08	6.3E-08	--	7.9E-6	2	1.5E-09	1.0E-07	--	1.3E-5
b-HCH (b-BHC)		2.0E-05	3	1.3E-07	3.0E-07	--	1.5E-2	7.8	5.8E-09	4.1E-07	--	2.0E-2
d-HCH (d-BHC)		2.0E-05				--	--	3.7	2.8E-09	1.9E-07	--	9.7E-3
g-HCH (g-BHC or lindane)		3.0E-03	3	1.3E-07	3.0E-07	--	9.9E-5	7.8	5.8E-09	4.1E-07	--	1.4E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	16183	6.9E-04	1.6E-03	--	8.0E-1	16183	1.2E-05	8.5E-04	--	4.2E-1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 5**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**2.3E-6**	**1.4E+0**			**TOTAL**	**4.2E-8**	**7.3E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	1.38	3.3E-07	7.6E-07	--	4.8E-3	1.38	5.7E-09	4.0E-07	--	2.5E-3
1,2-dichloroethane (EDC)	1.2E-02		0.01				--	--	29.4	1.2E-08	8.6E-07	1.5E-10	--
chloroform		6.5E-03	0.01				--	--	3.3	1.4E-09	9.6E-08	--	1.5E-5
trichloroethene (TCE)		2.4E-02	0.01				--	--	3.4	1.4E-09	9.9E-08	--	4.2E-6
chlorobenzene		8.6E-02	0.01				--	--	546	2.3E-07	1.6E-05	--	1.8E-4
ethylbenzene		9.7E-02	0.01	127	3.0E-06	7.0E-06	--	7.2E-5	127	5.3E-08	3.7E-06	--	3.8E-5
xylenes		1.8E-01	0.01	595	1.4E-05	3.3E-05	--	1.9E-4	595	2.5E-07	1.7E-05	--	9.9E-5
PAHs (BaP equivalent)	5.0E-01		0.13	14.92	4.6E-06	1.1E-05	2.3E-6	--	14.9	8.1E-08	5.6E-06	4.0E-8	--
1,4-dichlorobenzene		1.1E-02	0.01				--	--	10.8	4.5E-09	3.1E-07	--	2.9E-5
1,2,3-trichloropropane	7.0E+00		0.01				--	--	0.5	2.1E-10	1.5E-08	1.5E-9	--
a-HCH (a-BHC)		8.0E-03	0.01	0.64	1.5E-08	3.5E-08	--	4.4E-6	2	8.3E-10	5.8E-08	--	7.3E-6
b-HCH (b-BHC)		2.0E-05	0.01	3	7.1E-08	1.7E-07	--	8.3E-3	7.8	3.2E-09	2.3E-07	--	1.1E-2
d-HCH (d-BHC)		2.0E-05	0.01				--	--	3.7	1.5E-09	1.1E-07	--	5.4E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	3	7.1E-08	1.7E-07	--	5.5E-5	7.8	3.2E-09	2.3E-07	--	7.6E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	16183	1.1E-03	2.7E-03	--	1.3E+0	16183	2.0E-05	1.4E-03	--	7.1E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Summary of Risk- Identified Source Zone 5

Exposure to COPC Derived from Soils and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
ISZ 5 - Commercial/Industrial Workers			
Ingestion of COPC Indetified in Shallow Soils	**7.5E-07**	**1.9**	**1.9**
Dermal Contact with COPC Identified in Shallow Soils	**3.2E-07**	**0.82**	**0.82**
Inhalation of volatile COPC derived from soils and groundwater	**6.5E-05**	**0.24**	**0.00018**
Total Risk	**7E-05**	**3**	**3**

ISZ 5 - Intrusive Workers		
Ingestion of COPC Indetified in Shallow Soils	**9.5E-09**	**0.51**
Dermal Contact with COPC Identified in Shallow Soils	**8.5E-09**	**0.46**
Inhalation of volatile COPC derived from soils and groundwater (TWAs)	**3.6E-05**	**0.0028**
Total Risk	**4E-05**	**1**

Risk Calculations ISZ 6

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 6

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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	**4.1E-6**	**8.4E-1**			**TOTAL**	**3.4E-8**	**2.1E-1**
hexachlorobenzene (HCB)		1.6E-04	2.84	2.9E-07	6.7E-07	--	4.2E-3	2.84	2.4E-09	1.7E-07	--	1.0E-3
PAHs (BaP equivalent)	5.0E-01		82.2	8.3E-06	1.9E-05	4.1E-6	--	82.2	6.9E-08	4.8E-06	3.4E-8	--
a-HCH (a-BHC)		8.0E-03	10.6	1.1E-06	2.5E-06	--	3.1E-4	10.6	8.9E-09	6.2E-07	--	7.8E-5
b-HCH (b-BHC)		2.0E-05	7.5	7.5E-07	1.8E-06	--	8.8E-2	7.5	6.3E-09	4.4E-07	--	2.2E-2
d-HCH (d-BHC)		2.0E-05	3.04	3.1E-07	7.1E-07	--	3.6E-2	3.04	2.5E-09	1.8E-07	--	8.9E-3
g-HCH (g-BHC or lindane)		3.0E-03	7.5	7.5E-07	1.8E-06	--	5.9E-4	7.5	6.3E-09	4.4E-07	--	1.5E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	6079	6.1E-04	1.4E-03	--	7.1E-1	6079	5.1E-06	3.6E-04	--	1.8E-1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 6

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.7E-6**	**3.6E-1**			**TOTAL**	**3.1E-8**	**1.9E-1**
hexachlorobenzene (HCB)		1.6E-04	2.84	1.2E-07	2.8E-07	--	1.8E-3	2.84	2.1E-09	1.5E-07	--	9.3E-4
PAHs (BaP equivalent)	5.0E-01		82.2	3.5E-06	8.2E-06	1.7E-6	--	82.2	6.2E-08	4.3E-06	3.1E-8	--
a-HCH (a-BHC)		8.0E-03	10.6	4.5E-07	1.1E-06	--	1.3E-4	10.6	7.9E-09	5.6E-07	--	6.9E-5
b-HCH (b-BHC)		2.0E-05	7.5	3.2E-07	7.4E-07	--	3.7E-2	7.5	5.6E-09	3.9E-07	--	2.0E-2
d-HCH (d-BHC)		2.0E-05	3.04	1.3E-07	3.0E-07	--	1.5E-2	3.04	2.3E-09	1.6E-07	--	8.0E-3
g-HCH (g-BHC or lindane)		3.0E-03	7.5	3.2E-07	7.4E-07	--	2.5E-4	7.5	5.6E-09	3.9E-07	--	1.3E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	6079	2.6E-04	6.0E-04	--	3.0E-1	6079	4.5E-06	3.2E-04	--	1.6E-1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 6**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*FE*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**1.3E-5**	**5.4E-1**			**TOTAL**	**2.2E-7**	**2.9E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	2.84	6.7E-07	1.6E-06	--	9.8E-3	2.84	1.2E-08	8.3E-07	--	5.2E-3
PAHs (BaP equivalent)	5.0E-01		0.13	82.2	2.5E-05	5.9E-05	1.3E-5	--	82.2	4.4E-07	3.1E-05	2.2E-7	--
a-HCH (a-BHC)		8.0E-03	0.01	10.6	2.5E-07	5.8E-07	--	7.3E-5	10.6	4.4E-09	3.1E-07	--	3.9E-5
b-HCH (b-BHC)		2.0E-05	0.01	7.5	1.8E-07	4.1E-07	--	2.1E-2	7.5	3.1E-09	2.2E-07	--	1.1E-2
d-HCH (d-BHC)		2.0E-05	0.01	3.04	7.2E-08	1.7E-07	--	8.4E-3	3.04	1.3E-09	8.8E-08	--	4.4E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	7.5	1.8E-07	4.1E-07	--	1.4E-4	7.5	3.1E-09	2.2E-07	--	7.3E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	6079	4.3E-04	1.0E-03	--	5.0E-1	6079	7.6E-06	5.3E-04	--	2.7E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Summary of Risk - ISZ 6

Exposure to COPC Derived from Soils

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI
ISZ 6 - Commercial/Industrial Workers		
Ingestion of COPC Indetified in Shallow Soils	**4.1E-06**	**0.84**
Dermal Contact with COPC Identified in Shallow Soils	**1.7E-06**	**0.36**
Total Risk	**6E-06**	**1**

ISZ 6 - Intrusive Workers		
Ingestion of COPC Indetified in Shallow Soils	**3.4E-08**	**0.30**
Dermal Contact with COPC Identified in Shallow Soils	**3.1E-08**	**0.27**
Total Risk	**7E-08**	**0.6**

Risk Calculations ISZ 7

Exposure to Chemicals via Ingestion of Soils

Identified Soil Zone 7

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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		**7.5E+0**			**TOTAL**		**1.9E+0**
hexachlorobenzene (HCB)		1.6E-04	40.2	4.0E-06	9.4E-06	--	5.9E-2	40.2	3.4E-08	2.4E-06	--	1.5E-2
a-HCH (a-BHC)		8.0E-03	598	6.0E-05	1.4E-04	--	1.8E-2	598	5.0E-07	3.5E-05	--	4.4E-3
b-HCH (b-BHC)		2.0E-05	571	5.7E-05	1.3E-04	--	6.7E+0	571	4.8E-07	3.4E-05	--	1.7E+0
g-HCH (g-BHC or lindane)		3.0E-03	951	9.6E-05	2.2E-04	--	7.4E-2	951	8.0E-07	5.6E-05	--	1.9E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	5390	5.4E-04	1.3E-03	--	6.3E-1	5390	4.5E-06	3.2E-04	--	1.6E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Soil Zone 7

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL		**3.2E+0**			**TOTAL**		**1.7E+0**
hexachlorobenzene (HCB)		1.6E-04	40.2	1.7E-06	4.0E-06	--	2.5E-2	40.2	3.0E-08	2.1E-06	--	1.3E-2
a-HCH (a-BHC)		8.0E-03	598	2.5E-05	5.9E-05	--	7.4E-3	598	4.5E-07	3.1E-05	--	3.9E-3
b-HCH (b-BHC)		2.0E-05	571	2.4E-05	5.7E-05	--	2.8E+0	571	4.3E-07	3.0E-05	--	1.5E+0
g-HCH (g-BHC or lindane)		3.0E-03	951	4.0E-05	9.4E-05	--	3.1E-2	951	7.1E-07	5.0E-05	--	1.7E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	5390	2.3E-04	5.3E-04	--	2.7E-1	5390	4.0E-06	2.8E-04	--	1.4E-1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 7**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*FE*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL		**2.2E+0**			**TOTAL**		**1.2E+0**
hexachlorobenzene (HCB)		1.6E-04	0.10	40.2	9.5E-06	2.2E-05	--	1.4E-1	40.2	1.7E-07	1.2E-05	--	7.3E-2
a-HCH (a-BHC)		8.0E-03	0.01	598	1.4E-05	3.3E-05	--	4.1E-3	598	2.5E-07	1.7E-05	--	2.2E-3
b-HCH (b-BHC)		2.0E-05	0.01	571	1.3E-05	3.1E-05	--	1.6E+0	571	2.4E-07	1.7E-05	--	8.3E-1
g-HCH (g-BHC or lindane)		3.0E-03	0.01	951	2.2E-05	5.2E-05	--	1.7E-2	951	4.0E-07	2.8E-05	--	9.2E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	5390	3.8E-04	8.9E-04	--	4.5E-1	5390	6.7E-06	4.7E-04	--	2.4E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Summary of Risk - ISZ 7

Exposure to COPC Derived from Soils

Adults

Exposure Pathway	Calculated HI
ISZ 7 - Commercial/Industrial Workers	
Ingestion of COPC Indetified in Shallow Soils	**7.5**
Dermal Contact with COPC Identified in Shallow Soils	**3.2**
Total Risk	**11**

ISZ 7 - Intrusive Workers	
Ingestion of COPC Indetified in Shallow Soils	**1.9**
Dermal Contact with COPC Identified in Shallow Soils	**1.7**
Total Risk	**4**

Risk Calculations ISZ 8

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 - ISZ 8 based on data from ISZ 1a, 1b and 2

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.75	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1.75	Depth to soil gas sample
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Building Characteristics				
Area of Emission - Building Area	[m^2]	emarea	625	Assumed size of building 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	50	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	50	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	2500	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
cis-1,2-dichloroethene	3.92E-4			3.92E-4
trimethylbenzenes	3.39E-4			3.39E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	21000	2.1E-08	6.7E-10		**4.0E-05**		**1.0E-05**
vinyl chloride	23000	2.3E-08	7.3E-10		**4.4E-05**		**1.1E-05**
chloroform	63000	6.3E-08	2.0E-09	2.6E-09	**1.5E-04**		**3.9E-05**
trichloroethene (TCE)	3100000	3.1E-06	7.4E-08		**4.4E-03**		**1.1E-03**
tetrachloroethene (PCE)	49000	4.9E-08	1.1E-09		**6.4E-05**		**1.6E-05**
chlorobenzene	9380165	9.4E-06	2.1E-07		**1.2E-02**		**3.2E-03**
benzene	23760	2.4E-08	6.3E-10		**3.8E-05**		**9.6E-06**
toluene	21384297	2.1E-05	5.6E-07	3.3E-09	**2.0E-04**		**5.0E-05**
cis-1,2-dichloroethene	90000	9.0E-08	2.0E-09		**1.2E-04**		**3.1E-05**
trimethylbenzenes	40800	4.1E-08	7.9E-10		**4.7E-05**		**1.2E-05**

Maximum soil gas concentration from source zone

Maximum emissions rate from source sone

Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**3.8E-7**	**1.8E-2**			**TOTAL**	**3.6E-8**	**1.8E-3**
1,2-dichloroethane (EDC)	9.8E-03		4.0E-05	1.9E-06	4.4E-06	1.9E-8	--	1.0E-05	1.8E-07	4.3E-07	1.8E-9	--
chloroform	1.5E-03	2.0E-02	1.5E-04	7.3E-06	1.7E-05	1.1E-8	8.5E-4	3.9E-05	6.9E-07	1.6E-06	1.0E-9	8.1E-5
trichloroethene (TCE)	1.5E-03		4.4E-03	2.1E-04	4.9E-04	3.1E-7	--	1.1E-03	2.0E-05	4.7E-05	3.0E-8	--
tetrachloroethene (PCE)		4.7E-02	6.4E-05	3.0E-06	7.0E-06	--	1.5E-4	1.6E-05	2.9E-07	6.7E-07	--	1.4E-5
chlorobenzene		1.4E-01	1.2E-02	5.9E-04	1.4E-03	--	9.6E-3	3.2E-03	5.6E-05	1.3E-04	--	9.2E-4
benzene	2.1E-02		3.8E-05	1.8E-06	4.2E-06	3.8E-8	--	9.6E-06	1.7E-07	4.0E-07	3.6E-9	--
toluene		6.7E-02	2.0E-04	9.2E-06	2.1E-05	--	3.2E-4	5.0E-05	8.8E-07	2.0E-06	--	3.1E-5
cis-1,2-dichloroethene		1.0E-02	1.2E-04	5.7E-06	1.3E-05	--	1.3E-3	3.1E-05	5.4E-07	1.3E-06	--	1.3E-4
trimethylbenzenes		8.6E-04	4.7E-05	2.2E-06	5.2E-06	--	6.1E-3	1.2E-05	2.1E-07	5.0E-07	--	5.8E-4

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**3.8E-7**	**3.0E-4**			**TOTAL**	**3.6E-8**	**2.8E-5**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	4.0E-05	1.9E-06	4.4E-06	1.9E-8	7.8E-7	1.0E-05	1.8E-07	4.3E-07	1.8E-9	7.4E-8
chloroform	1.5E-03	1.4E+00	1.5E-04	7.3E-06	1.7E-05	1.1E-8	1.2E-5	3.9E-05	6.9E-07	1.6E-06	1.0E-9	1.1E-6
trichloroethene (TCE)	1.5E-03	7.7E+00	4.4E-03	2.1E-04	4.9E-04	3.1E-7	6.3E-5	1.1E-03	2.0E-05	4.7E-05	3.0E-8	6.0E-6
tetrachloroethene (PCE)		4.8E+01	6.4E-05	3.0E-06	7.0E-06	--	1.5E-7	1.6E-05	2.9E-07	6.7E-07	--	1.4E-8
chlorobenzene		6.6E+00	1.2E-02	5.9E-04	1.4E-03	--	2.1E-4	3.2E-03	5.6E-05	1.3E-04	--	2.0E-5
benzene	2.1E-02	4.6E-01	3.8E-05	1.8E-06	4.2E-06	3.8E-8	9.1E-6	9.6E-06	1.7E-07	4.0E-07	3.6E-9	8.7E-7
toluene		2.7E+01	2.0E-04	9.2E-06	2.1E-05	--	7.9E-7	5.0E-05	8.8E-07	2.0E-06	--	7.5E-8
cis-1,2-dichloroethene		1.1E+02	1.2E-04	5.7E-06	1.3E-05	--	1.2E-7	3.1E-05	5.4E-07	1.3E-06	--	1.1E-8
trimethylbenzenes		1.8E+01	4.7E-05	2.2E-06	5.2E-06	--	2.9E-7	1.2E-05	2.1E-07	5.0E-07	--	2.8E-8

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

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 - ISZ 1a, 1b and 2 - Excavations in ISZ 8

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	0.01	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	1	Assumed size of excavation
Width of Contaminated Area	[m]	width	10	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	0.5	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4
cis-1,2-dichloroethene	3.92E-4			3.92E-4
trimethylbenzenes	3.39E-4			3.39E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	21000	2.1E-08	1.2E-07			**1.6E-03**	
vinyl chloride	23000	2.3E-08	1.3E-07			**1.7E-03**	
chloroform	63000	6.3E-08	3.5E-07			**4.6E-03**	
trichloroethene (TCE)	3100000	3.1E-06	1.3E-05			**1.7E-01**	
tetrachloroethene (PCE)	49000	4.9E-08	1.9E-07			**2.5E-03**	
chlorobenzene	9380165	9.4E-06	3.6E-05			**4.9E-01**	
benzene	23760	2.4E-08	1.1E-07			**1.5E-03**	
toluene	21384297	2.1E-05	9.8E-05			**1.3E+00**	
cis-1,2-dichloroethene	90000	9.0E-08	3.5E-07			**4.7E-03**	
trimethylbenzenes	40800	4.1E-08	1.4E-07			**1.8E-03**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04** **5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	2.3E-7	7.8E-3
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.6E-03	1.2E-06	8.1E-05	1.1E-8	1.4E-5
chloroform	1.5E-03	1.4E+00	4.6E-03	3.4E-06	2.4E-04	5.0E-9	1.7E-4
trichloroethene (TCE)	1.5E-03	7.7E+00	1.7E-01	1.3E-04	8.9E-03	1.9E-7	1.2E-3
tetrachloroethene (PCE)		4.8E+01	2.5E-03	1.8E-06	1.3E-04	--	2.7E-6
chlorobenzene		6.6E+00	4.9E-01	3.6E-04	2.5E-02	--	3.8E-3
benzene	2.1E-02	4.6E-01	1.5E-03	1.1E-06	7.6E-05	2.3E-8	1.7E-4
toluene		2.7E+01	1.3E+00	9.7E-04	6.8E-02	--	2.5E-3
cis-1,2-dichloroethene		1.1E+02	4.7E-03	3.5E-06	2.4E-04	--	2.1E-6
trimethylbenzenes		1.8E+01	1.8E-03	1.4E-06	9.5E-05	--	5.3E-6

Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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	**4.9E-7**	**1.8E+2**			**TOTAL**	**1.2E-8**	**4.5E+1**
hexachlorobenzene (HCB)		1.6E-04	6	6.0E-07	1.4E-06	--	8.8E-3	250	2.1E-07	1.5E-05	--	9.2E-2
1,2-dichloroethane (EDC)	1.2E-02					--	--	1.4	1.2E-09	8.2E-08	1.4E-11	--
chloroform		6.5E-03	41.9	4.2E-06	9.8E-06	--	1.5E-3	41.9	3.5E-08	2.5E-06	--	3.8E-4
trichloroethene (TCE)		2.4E-02				--	--	14.2	1.2E-08	8.3E-07	--	3.5E-5
tetrachloroethene (PCE)		9.2E-03	22.5	2.3E-06	5.3E-06	--	5.7E-4	603	5.1E-07	3.5E-05	--	3.8E-3
benzene	3.5E-02		21.4	2.2E-06	5.0E-06	7.5E-8	--	21.4	1.8E-08	1.3E-06	6.3E-10	--
toluene		2.0E-01	116	1.2E-05	2.7E-05	--	1.4E-4	116	9.7E-08	6.8E-06	--	3.4E-5
PAHs (BaP equivalent)	5.0E-01		8.17	8.2E-07	1.9E-06	4.1E-7	--	26.55	2.2E-08	1.6E-06	1.1E-8	--
1,4-dichlorobenzene		1.1E-02	165	1.7E-05	3.9E-05	--	3.6E-3	165	1.4E-07	9.7E-06	--	9.1E-4
a-HCH (a-BHC)		8.0E-03	17400	1.8E-03	4.1E-03	--	5.1E-1	17400	1.5E-05	1.0E-03	--	1.3E-1
b-HCH (b-BHC)		2.0E-05	14200	1.4E-03	3.3E-03	--	1.7E+2	14200	1.2E-05	8.3E-04	--	4.2E+1
d-HCH (d-BHC)		2.0E-05	899	9.0E-05	2.1E-04	--	1.1E+1	899	7.5E-07	5.3E-05	--	2.6E+0
g-HCH (g-BHC or lindane)		3.0E-03	8000	8.1E-04	1.9E-03	--	6.3E-1	8000	6.7E-06	4.7E-04	--	1.6E-1
dieldrin		1.0E-04	542	5.5E-05	1.3E-04	--	1.3E+0	542	4.5E-07	3.2E-05	--	3.2E-1
DDX (sum of DDT, DDE and DDD)		2.0E-03	12170	1.2E-03	2.9E-03	--	1.4E+0	12170	1.0E-05	7.1E-04	--	3.6E-1
pentachlorophenol		3.0E-03	10	1.0E-06	2.3E-06	--	7.8E-4	10	8.4E-09	5.9E-07	--	2.0E-4
mercury (as total, inroganic)		2.5E-02				--	--	187	1.6E-07	1.1E-05	--	4.4E-4

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.1E-7**	**7.6E+1**			**TOTAL**	**1.1E-8**	**4.1E+1**
hexachlorobenzene (HCB)		1.6E-04	6	2.5E-07	5.9E-07	--	3.7E-3	250	1.9E-07	1.3E-05	--	8.2E-2
1,2-dichloroethane (EDC)	1.2E-02					--	--	1.4	1.0E-09	7.3E-08	1.3E-11	--
chloroform		6.5E-03	41.9	1.8E-06	4.2E-06	--	6.4E-4	41.9	3.1E-08	2.2E-06	--	3.4E-4
trichloroethene (TCE)		2.4E-02				--	--	14.2	1.1E-08	7.4E-07	--	3.1E-5
tetrachloroethene (PCE)		9.2E-03	22.5	9.6E-07	2.2E-06	--	2.4E-4	603	4.5E-07	3.2E-05	--	3.4E-3
benzene	3.5E-02		21.4	9.1E-07	2.1E-06	3.2E-8	--	21.4	1.6E-08	1.1E-06	5.6E-10	--
toluene		2.0E-01	116	4.9E-06	1.2E-05	--	5.8E-5	116	8.7E-08	6.1E-06	--	3.1E-5
PAHs (BaP equivalent)	5.0E-01		8.17	3.5E-07	8.1E-07	1.7E-7	--	26.55	2.0E-08	1.4E-06	9.9E-9	--
1,4-dichlorobenzene		1.1E-02	165	7.0E-06	1.6E-05	--	1.5E-3	165	1.2E-07	8.6E-06	--	8.1E-4
a-HCH (a-BHC)		8.0E-03	17400	7.4E-04	1.7E-03	--	2.2E-1	17400	1.3E-05	9.1E-04	--	1.1E-1
b-HCH (b-BHC)		2.0E-05	14200	6.0E-04	1.4E-03	--	7.0E+1	14200	1.1E-05	7.4E-04	--	3.7E+1
d-HCH (d-BHC)		2.0E-05	899	3.8E-05	8.9E-05	--	4.5E+0	899	6.7E-07	4.7E-05	--	2.4E+0
g-HCH (g-BHC or lindane)		3.0E-03	8000	3.4E-04	7.9E-04	--	2.6E-1	8000	6.0E-06	4.2E-04	--	1.4E-1
dieldrin		1.0E-04	542	2.3E-05	5.4E-05	--	5.4E-1	542	4.1E-07	2.8E-05	--	2.8E-1
DDX (sum of DDT, DDE and DDD)		2.0E-03	12170	5.2E-04	1.2E-03	--	6.0E-1	12170	9.1E-06	6.4E-04	--	3.2E-1
pentachlorophenol		3.0E-03	10	4.2E-07	9.9E-07	--	3.3E-4	10	7.5E-09	5.2E-07	--	1.7E-4
mercury (as total, inroganic)		3.6E-04				--	--	187	1.4E-07	9.8E-06	--	2.8E-2

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 8**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**1.3E-6**	**4.3E+1**			**TOTAL**	**7.2E-8**	**2.3E+1**
hexachlorobenzene (HCB)		1.6E-04	0.10	6	1.4E-06	3.3E-06	--	2.1E-2	250	1.0E-06	7.3E-05	--	4.5E-1
1,2-dichloroethane (EDC)	1.2E-02		0.01				--	--	1.4	5.8E-10	4.1E-08	7.0E-12	--
chloroform		6.5E-03	0.01	41.9	9.9E-07	2.3E-06	--	3.6E-4	41.9	1.7E-08	1.2E-06	--	1.9E-4
trichloroethene (TCE)		2.4E-02	0.01				--	--	14.2	5.9E-09	4.1E-07	--	1.7E-5
tetrachloroethene (PCE)		9.2E-03	0.01	22.5	5.3E-07	1.2E-06	--	1.3E-4	603	2.5E-07	1.8E-05	--	1.9E-3
benzene	3.5E-02		0.01	21.4	5.1E-07	1.2E-06	1.8E-8	--	21.4	8.9E-09	6.2E-07	3.1E-10	--
toluene		2.0E-01	0.01	116	2.7E-06	6.4E-06	--	3.2E-5	116	4.8E-08	3.4E-06	--	1.7E-5
PAHs (BaP equivalent)	5.0E-01		0.13	8.17	2.5E-06	5.9E-06	1.3E-6	--	26.55	1.4E-07	1.0E-05	7.2E-8	--
1,4-dichlorobenzene		1.1E-02	0.01	165	3.9E-06	9.1E-06	--	8.5E-4	165	6.9E-08	4.8E-06	--	4.5E-4
a-HCH (a-BHC)		8.0E-03	0.01	17400	4.1E-04	9.6E-04	--	1.2E-1	17400	7.2E-06	5.1E-04	--	6.3E-2
b-HCH (b-BHC)		2.0E-05	0.01	14200	3.4E-04	7.8E-04	--	3.9E+1	14200	5.9E-06	4.1E-04	--	2.1E+1
d-HCH (d-BHC)		2.0E-05	0.01	899	2.1E-05	5.0E-05	--	2.5E+0	899	3.7E-07	2.6E-05	--	1.3E+0
g-HCH (g-BHC or lindane)		3.0E-03	0.01	8000	1.9E-04	4.4E-04	--	1.5E-1	8000	3.3E-06	2.3E-04	--	7.8E-2
dieldrin		1.0E-04	0.01	542	1.3E-05	3.0E-05	--	3.0E-1	542	2.3E-07	1.6E-05	--	1.6E-1
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	12170	8.6E-04	2.0E-03	--	1.0E+0	12170	1.5E-05	1.1E-03	--	5.3E-1
pentachlorophenol		3.0E-03	0.25	10	5.9E-06	1.4E-05	--	4.6E-3	10	1.0E-07	7.3E-06	--	2.4E-3
mercury (as total, inroganic)		3.6E-04	0.01				--	--	187	7.8E-08	5.4E-06	--	1.5E-2

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Summary of Risk - ISZ 8

Exposure to COPC Derived from Soils and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
ISZ 8 - Commercial/Industrial Workers			
Ingestion of COPC Indetified in Shallow Soils	**4.9E-07**	**181**	**181**
Dermal Contact with COPC Identified in Shallow Soils	**2.1E-07**	**76**	**76**
Inhalation of volatile COPC derived from soils and groundwater	**3.8E-07**	**0.018**	**0.00030**
Total Risk	**1E-06**	**258**	**258**

ISZ 8 - Intrusive Workers		
Ingestion of COPC Indetified in Shallow Soils	**1.2E-08**	**45**
Dermal Contact with COPC Identified in Shallow Soils	**1.1E-08**	**41**
Inhalation of volatile COPC derived from soils and groundwater	**2.3E-07**	**0.0078**
Total Risk	**3E-07**	**86**

Risk Calculations ISZ 9

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 - ISZ 9

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.75	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**1.75**	Depth to soil gas sample
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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	**50**	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	**50**	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	**2500**	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
trichloroethene (TCE)	4.17E-4			4.17E-4
benzene	4.66E-4			4.66E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
tetrachloroethene (PCE)	49000	4.9E-08	1.1E-09		**6.4E-05**		**1.6E-05**
benzene	23760	2.4E-08	6.3E-10		**3.8E-05**		**9.6E-06**

Inhalation of Volatile Chemicals

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**3.8E-8**	**1.5E-4**			**TOTAL**	**3.6E-9**	**1.4E-5**
tetrachloroethene (PCE)		4.7E-02	6.4E-05	3.0E-06	7.0E-06	--	1.5E-4	1.6E-05	2.9E-07	6.7E-07	--	1.4E-5
benzene	2.1E-02		3.8E-05	1.8E-06	4.2E-06	3.8E-8	--	9.6E-06	1.7E-07	4.0E-07	3.6E-9	--

↑ Calculated from maximum measured soil gas concentration

↑ Calculated from maximum measured soil gas concentration

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**3.8E-8**	**9.3E-6**			**TOTAL**	**3.6E-9**	**8.9E-7**
tetrachloroethene (PCE)		4.8E+01	6.4E-05	3.0E-06	7.0E-06	--	1.5E-7	1.6E-05	2.9E-07	6.7E-07	--	1.4E-8
benzene	2.1E-02	4.6E-01	3.8E-05	1.8E-06	4.2E-06	3.8E-8	9.1E-6	9.6E-06	1.7E-07	4.0E-07	3.6E-9	8.7E-7

↑ Calculated from maximum measured soil gas concentration

↑ Calculated from maximum measured soil gas concentration

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 - Excavations in ISZ 9

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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)
trichloroethene (TCE)	4.17E-4			4.17E-4
benzene	4.66E-4			4.66E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
tetrachloroethene (PCE)	49000	4.9E-08	1.9E-07			**2.5E-03**	
benzene	23760	2.4E-08	1.1E-07			**1.5E-03**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04**	**NonThreshold**
		5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	2.3E-8	1.7E-4
tetrachloroethene (PCE)		4.8E+01	2.5E-03	1.8E-06	1.3E-04	--	2.7E-6
benzene	2.1E-02	4.6E-01	1.5E-03	1.1E-06	7.6E-05	2.3E-8	1.7E-4

Calculated from maximum measured soil gas concentrations

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07** **2.3E-07**	**NonThreshold** **Threshold**	**8.4E-10** **5.9E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**7.6E-7**	**4.9E+0**			**TOTAL**	**6.4E-9**	**1.2E+0**
tetrachloroethene (PCE)		9.2E-03	4.1	4.1E-07	9.6E-07	--	1.0E-4	4.1	3.4E-09	2.4E-07	--	2.6E-5
1,1,2-trichloroethane		4.0E-03				--	--	3.6	3.0E-09	2.1E-07	--	5.3E-5
benzene	3.5E-02		4.7	4.7E-07	1.1E-06	1.7E-8	--	5.8	4.9E-09	3.4E-07	1.7E-10	--
PAHs (BaP equivalent)	5.0E-01		14.8	1.5E-06	3.5E-06	7.4E-7	--	14.8	1.2E-08	8.7E-07	6.2E-9	--
a-HCH (a-BHC)		8.0E-03	25.4	2.6E-06	6.0E-06	--	7.5E-4	25.4	2.1E-08	1.5E-06	--	1.9E-4
b-HCH (b-BHC)		2.0E-05	19	1.9E-06	4.5E-06	--	2.2E-1	19	1.6E-08	1.1E-06	--	5.6E-2
g-HCH (g-BHC or lindane)		3.0E-03	19	1.9E-06	4.5E-06	--	1.5E-3	19	1.6E-08	1.1E-06	--	3.7E-4
dieldrin		1.0E-04	562	5.7E-05	1.3E-04	--	1.3E+0	562	4.7E-07	3.3E-05	--	3.3E-1
DDX (sum of DDT, DDE and DDD)		2.0E-03	22265	2.2E-03	5.2E-03	--	2.6E+0	22265	1.9E-05	1.3E-03	--	6.5E-1
endosulfan II		5.4E-03	15900	1.6E-03	3.7E-03	--	6.9E-1	15900	1.3E-05	9.3E-04	--	1.7E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.2E-7**	**2.0E+0**			**TOTAL**	**5.7E-9**	**1.1E+0**
tetrachloroethene (PCE)		9.2E-03	4.1	1.7E-07	4.1E-07	--	4.4E-5	4.1	3.1E-09	2.1E-07	--	2.3E-5
1,1,2-trichloroethane		4.0E-03				--	--	3.6	2.7E-09	1.9E-07	--	4.7E-5
benzene	3.5E-02		4.7	2.0E-07	4.7E-07	7.0E-9	--	5.8	4.3E-09	3.0E-07	1.5E-10	--
PAHs (BaP equivalent)	5.0E-01		14.8	6.3E-07	1.5E-06	3.1E-7	--	14.8	1.1E-08	7.8E-07	5.5E-9	--
a-HCH (a-BHC)		8.0E-03	25.4	1.1E-06	2.5E-06	--	3.1E-4	25.4	1.9E-08	1.3E-06	--	1.7E-4
b-HCH (b-BHC)		2.0E-05	19	8.1E-07	1.9E-06	--	9.4E-2	19	1.4E-08	1.0E-06	--	5.0E-2
g-HCH (g-BHC or lindane)		3.0E-03	19	8.1E-07	1.9E-06	--	6.3E-4	19	1.4E-08	1.0E-06	--	3.3E-4
dieldrin		1.0E-04	562	2.4E-05	5.6E-05	--	5.6E-1	562	4.2E-07	2.9E-05	--	2.9E-1
DDX (sum of DDT, DDE and DDD)		2.0E-03	22265	9.5E-04	2.2E-03	--	1.1E+0	22265	1.7E-05	1.2E-03	--	5.8E-1
endosulfan II		5.4E-03	15900	6.8E-04	1.6E-03	--	2.9E-1	15900	1.2E-05	8.3E-04	--	1.5E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 9**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**2.3E-6**	**2.4E+0**			**TOTAL**	**4.0E-8**	**1.2E+0**
tetrachloroethene (PCE)		9.2E-03	0.01	4.1	9.7E-08	2.3E-07	--	2.4E-5	4.1	1.7E-09	1.2E-07	--	1.3E-5
1,1,2-trichloroethane		4.0E-03	0.01				--	--	3.6	1.5E-09	1.0E-07	--	2.6E-5
benzene	3.5E-02		0.01	4.7	1.1E-07	2.6E-07	3.9E-9	--	5.8	2.4E-09	1.7E-07	8.4E-11	--
PAHs (BaP equivalent)	5.0E-01		0.13	14.8	4.5E-06	1.1E-05	2.3E-6	--	14.8	8.0E-08	5.6E-06	4.0E-8	--
a-HCH (a-BHC)		8.0E-03	0.01	25.4	6.0E-07	1.4E-06	--	1.7E-4	25.4	1.1E-08	7.4E-07	--	9.2E-5
b-HCH (b-BHC)		2.0E-05	0.01	19	4.5E-07	1.0E-06	--	5.2E-2	19	7.9E-09	5.5E-07	--	2.8E-2
g-HCH (g-BHC or lindane)		3.0E-03	0.01	19	4.5E-07	1.0E-06	--	3.5E-4	19	7.9E-09	5.5E-07	--	1.8E-4
dieldrin		1.0E-04	0.01	562	1.3E-05	3.1E-05	--	3.1E-1	562	2.3E-07	1.6E-05	--	1.6E-1
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	22265	1.6E-03	3.7E-03	--	1.8E+0	22265	2.8E-05	1.9E-03	--	9.7E-1
endosulfan II		5.4E-03	0.01	15900	3.8E-04	8.8E-04	--	1.6E-1	15900	6.6E-06	4.6E-04	--	8.6E-2

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Summary of Risk - ISZ 9

Exposure to COPC Derived from Soils and Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
ISZ 9 - Commercial/Industrial Workers			
Ingestion of COPC Indetified in Shallow Soils	**7.6E-07**	**4.9**	**4.9**
Dermal Contact with COPC Identified in Shallow Soils	**3.2E-07**	**2.0**	**2.0**
Inhalation of volatile COPC derived from soils and groundwater	**3.8E-08**	**0.00015**	**0.0000093**
Total Risk	**1E-06**	**7**	**7**

ISZ 9 - Intrusive Workers		
Ingestion of COPC Indetified in Shallow Soils	**6.4E-09**	**1.2**
Dermal Contact with COPC Identified in Shallow Soils	**5.7E-09**	**1.1**
Inhalation of volatile COPC derived from soils and groundwater	**2.3E-08**	**0.00017**
Total Risk	**3E-08**	**2**

Risk Calculations ISZ 10

Exposure to Chemicals via Ingestion of Soils

Pharmaceutical Building - **ISZ 10**

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.4E-6**	**1.2E+0**			**TOTAL**	**1.2E-8**	**3.0E-1**
benzene	3.5E-02		1.1	1.1E-07	2.6E-07	3.9E-9	--	1.1	9.2E-10	6.5E-08	3.2E-11	--
xylenes		1.8E-01	34	3.4E-06	8.0E-06	--	4.6E-5	34	2.9E-08	2.0E-06	--	1.1E-5
PAHs (BaP equivalent)	5.0E-01		27.7	2.8E-06	6.5E-06	1.4E-6	--	27.7	2.3E-08	1.6E-06	1.2E-8	--
a-HCH (a-BHC)		8.0E-03	9	9.1E-07	2.1E-06	--	2.6E-4	9	7.5E-09	5.3E-07	--	6.6E-5
b-HCH (b-BHC)		2.0E-05	50	5.0E-06	1.2E-05	--	5.9E-1	50	4.2E-08	2.9E-06	--	1.5E-1
g-HCH (g-BHC or lindane)		3.0E-03	50	5.0E-06	1.2E-05	--	3.9E-3	50	4.2E-08	2.9E-06	--	9.8E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	5083	5.1E-04	1.2E-03	--	6.0E-1	5083	4.3E-06	3.0E-04	--	1.5E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

Pharmaceutical Building - **ISZ 10**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-08**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal Absorption 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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**5.9E-7**	**5.0E-1**			**TOTAL**	**1.0E-8**	**2.7E-1**
benzene	3.5E-02		1.1	4.7E-08	1.1E-07	1.6E-9	--	1.1	8.2E-10	5.8E-08	2.9E-11	--
xylenes		1.8E-01	34	1.4E-06	3.4E-06	--	1.9E-5	34	2.5E-08	1.8E-06	--	1.0E-5
PAHs (BaP equivalent)	5.0E-01		27.7	1.2E-06	2.7E-06	5.9E-7	--	27.7	2.1E-08	1.5E-06	1.0E-8	--
a-HCH (a-BHC)		8.0E-03	9	3.8E-07	8.9E-07	--	1.1E-4	9	6.7E-09	4.7E-07	--	5.9E-5
b-HCH (b-BHC)		2.0E-05	50	2.1E-06	5.0E-06	--	2.5E-1	50	3.7E-08	2.6E-06	--	1.3E-1
g-HCH (g-BHC or lindane)		3.0E-03	50	2.1E-06	5.0E-06	--	1.7E-3	50	3.7E-08	2.6E-06	--	8.7E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	5083	2.2E-04	5.0E-04	--	2.5E-1	5083	3.8E-06	2.7E-04	--	1.3E-1

↑ Maximum concentration in top 1m soils

↑ Maximum concentration in soils from all depths

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment PB Bldg - **ISZ 10**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability 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 and Dermal Data			Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**4.3E-6**	**5.6E-1**			**TOTAL**	**7.5E-8**	**3.0E-1**
benzene	3.5E-02		0.01	1.1	2.6E-08	6.1E-08	9.1E-10	--	1.1	4.6E-10	3.2E-08	1.6E-11	--
xylenes		1.8E-01	0.01	34	8.0E-07	1.9E-06	--	1.1E-5	34	1.4E-08	9.9E-07	--	5.6E-6
PAHs (BaP equivalent)	5.0E-01		0.13	27.7	8.5E-06	2.0E-05	4.3E-6	--	27.7	1.5E-07	1.0E-05	7.5E-8	--
a-HCH (a-BHC)		8.0E-03	0.01	9	2.1E-07	5.0E-07	--	6.2E-5	9	3.7E-09	2.6E-07	--	3.3E-5
b-HCH (b-BHC)		2.0E-05	0.01	50	1.2E-06	2.8E-06	--	1.4E-1	50	2.1E-08	1.5E-06	--	7.3E-2
g-HCH (g-BHC or lindane)		3.0E-03	0.01	50	1.2E-06	2.8E-06	--	9.2E-4	50	2.1E-08	1.5E-06	--	4.9E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	5083	3.6E-04	8.4E-04	--	4.2E-1	5083	6.3E-06	4.4E-04	--	2.2E-1

Maximum concentration in top 1m soils

Maximum concentration in soils from all depths

Summary of Risk - ISZ 10

Exposure to COPC Derived from Soils

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI
ISZ 10 - Pharmaceutical Building Area		
Ingestion of COPC Indetified in Shallow Soils	**1.4E-06**	**1.2**
Dermal Contact with COPC Identified in Shallow Soils	**5.9E-07**	**0.50**
Total Risk	**2E-06**	**2**
ISZ 10 - Intrusive Workers		
Ingestion of COPC Indetified in Shallow Soils	**1.2E-08**	**0.30**
Dermal Contact with COPC Identified in Shallow Soils	**7.5E-08**	**0.30**
Total Risk	**9E-08**	**0.6**

Risk Calculations On-Site Plume 2

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 - Plume 2 (and Plume 1)

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1	Average depth to soil gas data
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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	50	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	50	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	2500	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
naphthalene	3.33E-4			3.33E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	3300	3.3E-09	1.9E-10		**1.1E-05**		**2.8E-06**
carbon tetrachloride				1.5E-10	**9.0E-06**		**2.3E-06**
trichloroethene (TCE)	1900	1.9E-09	7.9E-11		**4.8E-06**		**1.2E-06**
1,1,2-trichloroethane	229	2.3E-10	9.7E-12		**5.8E-07**		**1.5E-07**
chlorobenzene	35679	3.6E-08	1.4E-09		**8.3E-05**		**2.1E-05**
toluene	65000	6.5E-08	3.0E-09	2.2E-09	**1.3E-04**		**3.3E-05**
1,2,3-trichloropropane	9400	9.4E-09	3.8E-10		**2.3E-05**		**5.8E-06**
naphthalene	360	3.6E-10	1.2E-11		**7.2E-07**		**1.8E-07**

↑ Maximum soil gas concentration from source zone

↑ Maximum emissions rate from source sone

↑ Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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 ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(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	**7.6E-6**	**2.0E-3**			**TOTAL**	**7.2E-7**	**1.9E-4**
1,2-dichloroethane (EDC)	9.8E-03		1.1E-05	5.2E-07	1.2E-06	5.1E-9	--	2.8E-06	5.0E-08	1.2E-07	4.9E-10	--
carbon tetrachloride		6.1E-04	9.0E-06	4.2E-07	9.9E-07	--	1.6E-3	2.3E-06	4.0E-08	9.4E-08	--	1.5E-4
trichloroethene (TCE)	1.5E-03		4.8E-06	2.2E-07	5.2E-07	3.4E-10	--	1.2E-06	2.1E-08	5.0E-08	3.2E-11	--
1,1,2-trichloroethane		4.0E-03	5.8E-07	2.7E-08	6.4E-08	--	1.6E-5	1.5E-07	2.6E-09	6.1E-09	--	1.5E-6
chlorobenzene		1.4E-01	8.3E-05	3.9E-06	9.1E-06	--	6.4E-5	2.1E-05	3.7E-07	8.7E-07	--	6.1E-6
toluene		6.7E-02	1.3E-04	6.1E-06	1.4E-05	--	2.1E-4	3.3E-05	5.8E-07	1.4E-06	--	2.0E-5
1,2,3-trichloropropane	7.0E+00		2.3E-05	1.1E-06	2.5E-06	7.6E-6	--	5.8E-06	1.0E-07	2.4E-07	7.2E-7	--
naphthalene		8.1E-04	7.2E-07	3.4E-08	7.9E-08	--	9.7E-5	1.8E-07	3.2E-09	7.6E-09	--	9.3E-6
			↑					↑				
			Calculated from maximum measured emission rates and soil gas concentrations					Calculated from maximum measured emission rates and soil gas concentrations				

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors		Exposure Calculations (RME) Outdoors	
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	10	Assume 10 hours spent indoors	2	Assume 2 hours 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 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**	**1.8E-02**	**NonThreshold**
		1.1E-01	**Threshold**	**4.1E-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
	$(mg/kg\text{-}day)^{-1}$	(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	**7.6E-6**	**1.4E-5**			**TOTAL**	**7.2E-7**	**1.3E-6**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.1E-05	5.2E-07	1.2E-06	5.1E-9	2.1E-7	2.8E-06	5.0E-08	1.2E-07	4.9E-10	2.0E-8
carbon tetrachloride		9.0E-02	9.0E-06	4.2E-07	9.9E-07	--	1.1E-5	2.3E-06	4.0E-08	9.4E-08	--	1.0E-6
trichloroethene (TCE)	1.5E-03	7.7E+00	4.8E-06	2.2E-07	5.2E-07	3.4E-10	6.8E-8	1.2E-06	2.1E-08	5.0E-08	3.2E-11	6.5E-9
1,1,2-trichloroethane		7.9E+00	5.8E-07	2.7E-08	6.4E-08	--	8.2E-9	1.5E-07	2.6E-09	6.1E-09	--	7.8E-10
chlorobenzene		6.6E+00	8.3E-05	3.9E-06	9.1E-06	--	1.4E-6	2.1E-05	3.7E-07	8.7E-07	--	1.3E-7
toluene		2.7E+01	1.3E-04	6.1E-06	1.4E-05	--	5.2E-7	3.3E-05	5.8E-07	1.4E-06	--	5.0E-8
1,2,3-trichloropropane	7.0E+00	8.6E+00	2.3E-05	1.1E-06	2.5E-06	7.6E-6	2.9E-7	5.8E-06	1.0E-07	2.4E-07	7.2E-7	2.8E-8
naphthalene		7.4E+00	7.2E-07	3.4E-08	7.9E-08	--	1.1E-8	1.8E-07	3.2E-09	7.6E-09	--	1.0E-9

↑ Calculated from maximum measured emission rates and soil gas concentrations

↑ Calculated from maximum measured emission rates and soil gas concentrations

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 - Excavations in Area of Plume 2 (and Plume 1)

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume soil gas at depth of excavations
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing height assumed depth of excavation

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
naphthalene	3.33E-4			3.33E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	3300	3.3E-09	1.9E-08			**2.5E-04**	
trichloroethene (TCE)	1900	1.9E-09	7.9E-09			**1.1E-04**	
1,1,2-trichloroethane	229	2.3E-10	9.7E-10			**1.3E-05**	
chlorobenzene	35679	3.6E-08	1.4E-07			**1.8E-03**	
toluene	65000	6.5E-08	3.0E-07			**4.0E-03**	
1,2,3-trichloropropane	9400	9.4E-09	3.8E-08			**5.1E-04**	
naphthalene	360	3.6E-10	1.2E-09			**1.6E-05**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04** **5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	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	2.6E-6	2.8E-5
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.5E-04	1.8E-07	1.3E-05	1.8E-9	2.2E-6
trichloroethene (TCE)	1.5E-03	7.7E+00	1.1E-04	7.8E-08	5.5E-06	1.2E-10	7.1E-7
1,1,2-trichloroethane		7.9E+00	1.3E-05	9.6E-09	6.7E-07	--	8.5E-8
chlorobenzene		6.6E+00	1.8E-03	1.4E-06	9.5E-05	--	1.5E-5
toluene		2.7E+01	4.0E-03	2.9E-06	2.1E-04	--	7.6E-6
1,2,3-trichloropropane	7.0E+00	8.6E+00	5.1E-04	3.8E-07	2.6E-05	2.6E-6	3.1E-6
naphthalene		7.4E+00	1.6E-05	1.2E-08	8.3E-07	--	1.1E-7

Calculated from maximum measured soil gas concentrations

Summary of Risk - On Site Areas above Plume 2

Exposure to COPC Derived from Groundwater

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
Plume 2 - Commercial/Industrial Workers			
Inhalation of volatile COPC derived from soils and groundwater	**8.3E-06**	**0.0022**	**0.000015**
Total Risk	**8E-06**	**0.002**	**0.00001**

Plume 2 - Intrusive Workers		
Inhalation of volatile COPC derived from soils and groundwater	**2.6E-06**	**0.000028**
Total Risk	**3E-06**	**0.00003**

Risk Calculations Off-Site Plumes 1 and 2

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 - Plumes 1 and 2 (no GW intersected)

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume excavation to depth of soil gas data
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing zone for excavation

Vapour Transport Calculations for COPC identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
naphthalene	3.33E-4			3.33E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	3300	3.3E-09	1.9E-08			**2.5E-04**	
trichloroethene (TCE)	111665	1.1E-07	4.7E-07			**6.2E-03**	
1,1,2-trichloroethane	89000	8.9E-08	3.8E-07			**5.0E-03**	
toluene	65000	6.5E-08	3.0E-07			**4.0E-03**	
1,2,3-trichloropropane	9400	9.4E-09	3.8E-08			**5.1E-04**	
naphthalene	4800	4.8E-09	1.6E-08			**2.1E-04**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Intrusive Workers - Plumes 1 and 2 (Soil Gas Data)

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04**	**NonThreshold**
		5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.6E-6**	**8.9E-5**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.47E-04	1.8E-07	1.3E-05	1.8E-9	2.2E-6
trichloroethene (TCE)	1.5E-03	7.7E+00	6.21E-03	4.6E-06	3.2E-04	6.9E-9	4.2E-5
1,1,2-trichloroethane		7.9E+00	5.04E-03	3.7E-06	2.6E-04	--	3.3E-5
toluene		2.7E+01	3.99E-03	2.9E-06	2.1E-04	--	7.6E-6
1,2,3-trichloropropane	7.0E+00	8.6E+00	5.10E-04	3.8E-07	2.6E-05	2.6E-6	3.1E-6
naphthalene		7.4E+00	2.13E-04	1.6E-07	1.1E-05	--	1.5E-6

↑ Calculated based on max measured soil gas concentrations

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Intrusive Workers - Groundwater Ingress Plumes 1 and 2

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 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
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	2	Assume groundwater in excavation with groundwater seppage for 2 hours each day
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-05**	**NonThreshold**
		5.2E-03	**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	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**8.4E-6**	**1.4E-2**
hexachlorobenzene (HCB)		2.9E-04				--	--
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.10E+01	8.1E-04	5.7E-02	7.9E-6	9.9E-3
vinyl chloride		1.9E+00	3.99E-03	2.9E-07	2.1E-05	--	1.1E-5
chloroform		1.4E+00	1.36E-01	1.0E-05	7.0E-04	--	4.9E-4
trichloroethene (TCE)	1.5E-03	7.7E+00	4.99E-01	3.7E-05	2.6E-03	5.5E-8	3.3E-4
tetrachloroethene (PCE)		4.8E+01	1.20E-03	8.8E-08	6.2E-06	--	1.3E-7
1,1,2-trichloroethane		7.9E+00	4.66E-03	3.4E-07	2.4E-05	--	3.1E-6
chlorobenzene		6.6E+00				--	--
benzene	2.1E-02	4.6E-01	2.65E-01	2.0E-05	1.4E-03	4.1E-7	3.0E-3
toluene		2.7E+01	6.65E-04	4.9E-08	3.4E-06	--	1.3E-7
a-HCH (a-BHC)		1.4E-02				--	--
b-HCH (b-BHC)		1.4E-02				--	--
d-HCH (d-BHC)		1.4E-02				--	--
g-HCH (g-BHC or lindane)		1.4E-02				--	--
DDX (sum of DDT, DDE and DDD)		1.4E-01				--	--
cis-1,2-dichloroethene		1.1E+02	1.20E-03	8.8E-08	6.2E-06	--	5.5E-8

Calculated assuming groundwater ingress into excavation

Exposure to Chemicals via Incidental Ingestion of Water

Off Site Plumes 1 and 2

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Water by Intrusive/Excavation Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 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 in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**8.4E-08**	**NonThreshold**
		5.9E-06	**Threshold**

Daily Intake from Water = Concentration in Water 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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Water	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**9.5E-8**	**2.8E-3**
hexachlorobenzene (HCB)		1.6E-04	0.001	8.4E-11	5.9E-09	--	3.7E-5
1,2-dichloroethane (EDC)	1.2E-02		82.5	6.9E-06	4.8E-04	8.3E-8	--
vinyl chloride	2.3E+00		0.03	2.5E-09	1.8E-07	5.8E-9	--
chloroform		6.5E-03	1.02	8.6E-08	6.0E-06	--	9.2E-4
trichloroethene (TCE)		2.4E-02	3.75	3.1E-07	2.2E-05	--	9.3E-4
tetrachloroethene (PCE)		9.2E-03	0.009	7.5E-10	5.3E-08	--	5.7E-6
1,1,2-trichloroethane		4.0E-03	0.035	2.9E-09	2.1E-07	--	5.1E-5
chlorobenzene		8.6E-02	11.6	9.7E-07	6.8E-05	--	7.9E-4
benzene	3.5E-02		1.99	1.7E-07	1.2E-05	5.8E-9	--
toluene		2.0E-01	0.005	4.2E-10	2.9E-08	--	1.5E-7
a-HCH (a-BHC)		8.0E-03	0.000087	7.3E-12	5.1E-10	--	6.4E-8
b-HCH (b-BHC)		2.0E-05	0.000078	6.5E-12	4.6E-10	--	2.3E-5
d-HCH (d-BHC)		2.0E-05	0.000011	9.2E-13	6.5E-11	--	3.2E-6
g-HCH (g-BHC or lindane)		3.0E-03	0.00008	6.7E-12	4.7E-10	--	1.6E-7
dieldrin		1.0E-04	0.000058	4.9E-12	3.4E-10	--	3.4E-6
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.025	2.1E-09	1.5E-07	--	7.3E-5
cis-1,2-dichloroethene		1.0E-02	0.009	7.5E-10	5.3E-08	--	5.3E-6

↑ Maximum concnetration reported in shallow groundwater - off site Plumes 1 and 2

Dermal Exposure to Chemicals via Contact with Water

Off Site Plumes 1 and 2

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with water by Intrusive/Excavation Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 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 durface 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 = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**5.5E-05** **3.9E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability 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: Non-Threshold Slope Factor	Toxicity Data: Threshold ADI, TDI or RfD	Toxicity Data: Dermal Permeability	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**4.1E-7**	**9.6E-2**
hexachlorobenzene (HCB)		1.6E-04	4.1E-1	1.00E-03	2.3E-08	1.6E-06	--	9.9E-3
1,2-dichloroethane (EDC)	1.2E-02		5.3E-3	8.25E+01	2.4E-05	1.7E-03	2.9E-7	--
vinyl chloride	2.3E+00		1.1E-2	3.00E-02	1.9E-08	1.3E-06	4.3E-8	--
chloroform		6.5E-03	8.9E-3	1.02E+00	5.0E-07	3.5E-05	--	5.4E-3
trichloroethene (TCE)		2.4E-02	1.6E-2	3.75E+00	3.3E-06	2.3E-04	--	9.8E-3
tetrachloroethene (PCE)		9.2E-03	4.8E-2	9.00E-03	2.4E-08	1.7E-06	--	1.8E-4
1,1,2-trichloroethane		4.0E-03	6.4E-3	3.50E-02	1.2E-08	8.7E-07	--	2.2E-4
chlorobenzene		8.6E-02	4.1E-2	1.16E+01	2.6E-05	1.8E-03	--	2.1E-2
benzene	3.5E-02		2.1E-2	1.99E+00	2.3E-06	1.6E-04	8.0E-8	--
toluene		2.0E-01	4.5E-2	5.00E-03	1.3E-08	8.8E-07	--	4.4E-6
a-HCH (a-BHC)		8.0E-03	2.8E-2	8.70E-05	1.3E-10	9.4E-09	--	1.2E-6
b-HCH (b-BHC)		2.0E-05	2.8E-2	7.80E-05	1.2E-10	8.4E-09	--	4.2E-4
d-HCH (d-BHC)		2.0E-05	2.8E-2	1.10E-05	1.7E-11	1.2E-09	--	5.9E-5
g-HCH (g-BHC or lindane)		3.0E-03	2.8E-2	8.00E-05	1.2E-10	8.6E-09	--	2.9E-6
dieldrin		1.0E-04	4.5E-2	5.80E-05	1.4E-10	1.0E-08	--	1.0E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1.0E+0	2.49E-02	1.4E-06	9.6E-05	--	4.8E-2
cis-1,2-dichloroethene		1.0E-02	1.5E-2	9.00E-03	7.4E-09	5.2E-07	--	5.2E-5

Maximum concnetration reported in shallow groundwater - off site Plumes 1 and 2

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 - Plume 1

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.2	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1.2	Average depth to soil gas sample
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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

Vapour Transport Calculations for COPC Identified	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)
1,1,2-trichloroethane	4.25E-4			4.25E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,1,2-trichloroethane	42000	4.2E-08	1.5E-09		**8.9E-05**		

Maximum soil gas concentration from source zone

Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Offsite Areas Above Plume 1

General Data/ Equations	Units	Exposure Calculations (RME) Indoors (based on soil gas and flux data)	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02** **1.1E-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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL		**2.5E-3**
1,1,2-trichloroethane		4.0E-03	8.9E-05	4.2E-06	9.8E-06	--	2.5E-3

Calculated from maximum measured soil gas or emission rates from area

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Offsite Areas Above Plume 1

General Data/ Equations	Units	Exposure Calculations (RME) Indoors (based on soil gas and flux data)	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**
		1.1E-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	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL		**1.2E-6**
1,1,2-trichloroethane		7.9E+00	8.9E-05	4.2E-06	9.8E-06	--	1.2E-6

Calculated from maximum measured soil gas or emission rates from area

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 - Plume 2

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1	Calculated from layers
Soil Temperature	[C]	T	16	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1	Average depth to soil gas sample
Moisture Content	[g/g]	mocon	0.12	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
naphthalene	3.33E-4			3.33E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	3300	3.3E-09	1.9E-10		**1.1E-05**		
trichloroethene (TCE)	111665	1.1E-07	4.7E-09	1.4E-09	**8.5E-05**		
1,1,2-trichloroethane	89000	8.9E-08	3.8E-09		**2.3E-04**		
toluene	65000	6.5E-08	3.0E-09	6.0E-09	**3.6E-04**		
1,2,3-trichloropropane	9400	9.4E-09	3.8E-10		**2.3E-05**		
naphthalene	4800	4.8E-09	1.6E-10		**9.6E-06**		

↑ Maximum soil gas concentration from source zone

↑ Maximum emissions rate from source sone

↑ Calculated based on maximum emission rate, then maximum soil gas data

Inhalation of Volatile Chemicals

Offsite Areas Above Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors (based on soil gas and flux data)	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**
		1.1E-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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**7.6E-6**	**8.1E-3**
1,2-dichloroethane (EDC)	9.8E-03		1.1E-05	5.2E-07	1.2E-06	5.1E-9	--
trichloroethene (TCE)	1.5E-03		8.5E-05	4.0E-06	9.3E-06	6.0E-9	--
1,1,2-trichloroethane		4.0E-03	2.3E-04	1.1E-05	2.5E-05	--	6.2E-3
toluene		6.7E-02	3.6E-04	1.7E-05	4.0E-05	--	5.9E-4
1,2,3-trichloropropane	7.0E+00		2.3E-05	1.1E-06	2.5E-06	7.6E-6	--
naphthalene		8.1E-04	9.6E-06	4.5E-07	1.1E-06	--	1.3E-3

Calculated from maximum measured soil gas or emission rates from area

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Offsite Areas Above Plume 2

General Data/ Equations	Units	Exposure Calculations (RME) Indoors (based on soil gas and flux data)	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**
		1.1E-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	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	7.6E-6	6.5E-6
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	1.1E-05	5.2E-07	1.2E-06	5.1E-9	2.1E-7
trichloroethene (TCE)	1.5E-03	7.7E+00	8.5E-05	4.0E-06	9.3E-06	6.0E-9	1.2E-6
1,1,2-trichloroethane		7.9E+00	2.3E-04	1.1E-05	2.5E-05	--	3.2E-6
toluene		2.7E+01	3.6E-04	1.7E-05	4.0E-05	--	1.5E-6
1,2,3-trichloropropane	7.0E+00	8.6E+00	2.3E-05	1.1E-06	2.5E-06	7.6E-6	2.9E-7
naphthalene		7.4E+00	9.6E-06	4.5E-07	1.1E-06	--	1.4E-7

Calculated from maximum measured soil gas or emission rates from area

Summary of Risk

Exposure to COPC Derived from Groundwater - Offsite Areas

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
Above Plume 2			
Inhalation of volatile COPC derived from groundwater (soil gas)	**7.6E-06**	**8.1E-03**	**6.5E-06**
Above Plume 1			
Inhalation of volatile COPC derived from groundwater (soil gas)	**0.0E+00**	**2.5E-03**	**1.2E-06**
Intrusive Workers			
Inhalation of volatile COPC derived from shallow groundwater (TWA)	**8.4E-06**	**1.4E-02**	
Incidental ingestion of shallow groundwater	**9.5E-08**	**2.8E-03**	
Dermal contact with shallow groundwater	**4.1E-07**	**9.6E-02**	

Risk Calculations Off-Site Plume 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 - Plume 3 - No GW Intersected

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**0.01**	Assume excavation to depth of soil gas data
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	**1**	Assumed size of excavation
Width of Contaminated Area	[m]	width	**10**	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	**0.5**	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing zone for excavation

Vapour Transport Calculations for COPC identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	1100	1.1E-09	6.2E-09			**8.2E-05**	
trichloroethene (TCE)	300	3.0E-10	1.3E-09			**1.7E-05**	

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Intrusive Workers - Plume 3 (Soil Gas Data)

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	60	Assume intrusive works for 60 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
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 at worst, 10 hours spent in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-04**	**NonThreshold**
		5.2E-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	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**6.2E-10**	**8.6E-7**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	8.25E-05	6.1E-08	4.3E-06	6.0E-10	7.5E-7
trichloroethene (TCE)	1.5E-03	7.7E+00	1.67E-05	1.2E-08	8.6E-07	1.9E-11	1.1E-7

Calculated assuming groundwater ingress into excavation

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Intrusive Workers in OS Areas - Groundwater Ingress

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 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
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	2	Assume groundwater in excavation with groundwater seppage for 2 hours each day
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**7.4E-05**	**NonThreshold**
		5.2E-03	**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	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.8E-7**	**1.2E-3**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.00E-02	1.5E-06	1.0E-04	1.4E-8	1.8E-5
chloroform		1.4E+00	7.32E-03	5.4E-07	3.8E-05	--	2.6E-5
trichloroethene (TCE)	1.5E-03	7.7E+00	1.25E+00	9.3E-05	6.5E-03	1.4E-7	8.4E-4
tetrachloroethene (PCE)		4.8E+01	6.65E-04	4.9E-08	3.4E-06	--	7.2E-8
1,1,2-trichloroethane		7.9E+00	8.11E-03	6.0E-07	4.2E-05	--	5.3E-6
benzene	2.1E-02	4.6E-01	1.86E-02	1.4E-06	9.6E-05	2.9E-8	2.1E-4
toluene		2.7E+01	3.68E-01	2.7E-05	1.9E-03	--	7.0E-5
ethylbenzene		6.2E+01	1.16E-02	8.5E-07	6.0E-05	--	9.6E-7
xylenes		5.0E+01	3.17E-02	2.3E-06	1.6E-04	--	3.3E-6
1,2- and 1,3-dichlorobenzene		2.1E+01	6.92E-03	5.1E-07	3.6E-05	--	1.7E-6
1,4-dichlorobenzene		2.1E+01	3.06E-02	2.3E-06	1.6E-04	--	7.4E-6
cis-1,2-dichloroethene		1.1E+02	9.31E-04	6.9E-08	4.8E-06	--	4.2E-8
1,1-dichloroethene		2.9E+00	3.59E-02		1.9E-04	--	6.5E-5
1,1-dichloroethane		5.9E+01	1.05E-02		5.4E-05	--	9.2E-7
1,1,1-trichloroethane		7.9E+01	9.71E-03		5.0E-05	--	6.3E-7

↑ Calculated assuming groundwater ingress into excavation

Exposure to Chemicals via Incidental Ingestion of Water

Off Site Plume 3

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Water by Intrusive/Excavation Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 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 in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**8.4E-08** **5.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water 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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Water	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**5.6E-10**	**3.5E-3**
hexachlorobenzene (HCB)		1.6E-04	0.00055	4.6E-11	3.2E-09	--	2.0E-5
1,2-dichloroethane (EDC)	1.2E-02		0.15	1.3E-08	8.8E-07	1.5E-10	--
chloroform		6.5E-03	0.055	4.6E-09	3.2E-07	--	5.0E-5
trichloroethene (TCE)		2.4E-02	9.43	7.9E-07	5.5E-05	--	2.3E-3
tetrachloroethene (PCE)		9.2E-03	0.005	4.2E-10	2.9E-08	--	3.2E-6
1,1,2-trichloroethane		4.0E-03	0.061	5.1E-09	3.6E-07	--	9.0E-5
chlorobenzene		8.6E-02	9.68	8.1E-07	5.7E-05	--	6.6E-4
benzene	3.5E-02		0.14	1.2E-08	8.2E-07	4.1E-10	--
toluene		2.0E-01	2.77	2.3E-07	1.6E-05	--	8.2E-5
ethylbenzene		9.7E-02	0.087	7.3E-09	5.1E-07	--	5.3E-6
xylenes		1.8E-01	0.238	2.0E-08	1.4E-06	--	8.0E-6
1,2- and 1,3-dichlorobenzene		4.3E-01	0.052	4.4E-09	3.1E-07	--	7.1E-7
1,4-dichlorobenzene		1.1E-02	0.23	1.9E-08	1.4E-06	--	1.3E-4
a-HCH (a-BHC)		8.0E-03	0.0062	5.2E-10	3.6E-08	--	4.5E-6
g-HCH (g-BHC or lindane)		3.0E-03	0.0358	3.0E-09	2.1E-07	--	7.0E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.0077	6.5E-10	4.5E-08	--	2.3E-5
cis-1,2-dichloroethene		1.0E-02	0.007	5.9E-10	4.1E-08	--	4.1E-6
1,1-dichloroethene		4.6E-02	0.27	2.3E-08	1.6E-06	--	3.4E-5
1,1-dichloroethane		1.0E-01	0.079	6.6E-09	4.6E-07	--	4.6E-6
1,1,1-trichloroethane		6.0E-01	0.073	6.1E-09	4.3E-07	--	7.1E-7

↑ Maximum concnetration reported in shallow groundwater - off site Plume 3

Dermal Exposure to Chemicals via Contact with Water

Off Site Plume 3

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with water by Intrusive/Excavation Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 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 durface 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 = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**5.5E-05** **3.9E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability 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: Non-Threshold Slope Factor	Toxicity Data: Threshold ADI, TDI or RfD	Toxicity Data: Dermal Permeability	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**6.1E-9**	**7.4E-2**
hexachlorobenzene (HCB)		1.6E-04	4.1E-1	5.50E-04	1.2E-08	8.7E-07	--	5.4E-3
1,2-dichloroethane (EDC)	1.2E-02		5.3E-3	1.50E-01	4.4E-08	3.1E-06	5.3E-10	--
chloroform		6.5E-03	8.9E-3	5.50E-02	2.7E-08	1.9E-06	--	2.9E-4
trichloroethene (TCE)		2.4E-02	1.6E-2	9.43E+00	8.4E-06	5.8E-04	--	2.5E-2
tetrachloroethene (PCE)		9.2E-03	4.8E-2	5.00E-03	1.3E-08	9.3E-07	--	1.0E-4
1,1,2-trichloroethane		4.0E-03	6.4E-3	6.10E-02	2.2E-08	1.5E-06	--	3.8E-4
chlorobenzene		8.6E-02	4.1E-2	9.68E+00	2.2E-05	1.5E-03	--	1.8E-2
benzene	3.5E-02		2.1E-2	1.40E-01	1.6E-07	1.1E-05	5.6E-9	--
toluene		2.0E-01	4.5E-2	2.77E+00	6.9E-06	4.9E-04	--	2.5E-3
ethylbenzene		9.7E-02	7.4E-2	8.70E-02	3.6E-07	2.5E-05	--	2.6E-4
xylenes		1.8E-01	7.0E-2	2.38E-01	9.3E-07	6.5E-05	--	3.7E-4
1,2- and 1,3-dichlorobenzene		4.3E-01	6.6E-2	5.20E-02	1.9E-07	1.3E-05	--	3.1E-5
1,4-dichlorobenzene		1.1E-02	6.7E-2	2.30E-01	8.5E-07	6.0E-05	--	5.6E-3
a-HCH (a-BHC)		8.0E-03	2.8E-2	6.20E-03	9.6E-09	6.7E-07	--	8.4E-5
g-HCH (g-BHC or lindane)		3.0E-03	2.8E-2	3.58E-02	5.5E-08	3.9E-06	--	1.3E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	1.0E+0	7.70E-03	4.3E-07	3.0E-05	--	1.5E-2
cis-1,2-dichloroethene		1.0E-02	1.5E-2	7.00E-03	5.8E-09	4.0E-07	--	4.0E-5
1,1-dichloroethene		4.6E-02	1.6E-2	2.70E-01	2.4E-07	1.7E-05	--	3.6E-4
1,1-dichloroethane		1.0E-01	8.9E-3	7.90E-02	3.9E-08	2.7E-06	--	2.7E-5
1,1,1-trichloroethane		6.0E-01	1.7E-2	7.30E-02	6.9E-08	4.8E-06	--	8.1E-6

Maximum concnetration reported in shallow groundwater - off site Plume 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 - Plume 3

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1.4	Calculated from layers
Soil Temperature	[C]	T	**16**	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	**1.4**	Depth to soil gas sample
Moisture Content	[g/g]	mocon	**0.12**	Conservative value for silty clays
Organic Carbon Fraction	-	foc	**0.01**	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	**1.8**	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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	**50**	Assumed size of outdoor area
Width of Contaminated Area	[m]	width	**50**	Assumed size of outdoor area
Outdoor Area	[m^2]	oarea	**2500**	calculated
Q/C Dispersion term	[$g/m^2/s$ per kg/m^3]	qc	6.56E+01	Calculated for climate similar to East coast of Australia

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4

Calculated Air Concentrations	Vapour Phase Concentration at Source (ug/m^3)	Vapour Phase Concentration at Source (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Outdoor Air Concentration Box (mg/m^3)	Outdoor Air Concentration Q/C (mg/m^3)
1,2-dichloroethane (EDC)	1100	1.1E-09	4.4E-11		**2.7E-06**		
trichloroethene (TCE)	300	3.0E-10	8.9E-12		**5.4E-07**		

↑ Maximum soil gas concentration from source zone

↑ Calculated based on maximum soil gas concentration

Inhalation of Volatile Chemicals

Areas above Plume 3 On and Off Site

General Data/ Equations	Units	Exposure Calculations (RME) Indoors	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**4.7E-02**	**NonThreshold**
		1.1E-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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.3E-9**	
1,2-dichloroethane (EDC)	9.8E-03		2.7E-06	1.2E-07	2.9E-07	1.2E-9	--
trichloroethene (TCE)	1.5E-03		5.4E-07	2.5E-08	5.9E-08	3.8E-11	--

↑ Calculated from maximum measured soil gas or emission rates from area

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Areas above Plume 3 On and Off Site

General Data/ Equations	Units	Exposure Calculations (RME) Indoors	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = $\frac{InhR*ET*B*FI*EF*ED}{BW*AT}$	m^3/kg/day	**4.7E-02** **1.1E-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	
	Non-Threshold Slope Factor	Threshold TWA	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	**1.3E-9**	**5.9E-8**
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.7E-06	1.2E-07	2.9E-07	1.2E-9	5.1E-8
trichloroethene (TCE)	1.5E-03	7.7E+00	5.4E-07	2.5E-08	5.9E-08	3.8E-11	7.6E-9

Calculated from maximum measured soil gas or emission rates from area

Summary of Risk

Exposure to COPC Derived from Groundwater - Offsite Areas

Adults

Exposure Pathway	Calculated Non-Threshold Risk	Calculated HI	Calculated HI TWA Exposure Standards
Above Plume 3			
Inhalation of volatile COPC derived from groundwater (soil gas)	**1.3E-09**		**5.9E-08**
Intrusive Workers			
Inhalation of volatile COPC derived from shallow groundwater (TWA)	**1.8E-07**	**1.2E-03**	
Incidental ingestion of shallow groundwater	**5.6E-10**	**3.5E-03**	
Dermal contact with shallow groundwater	**6.1E-09**	**7.4E-02**	

Appendix G Derivation of Risk-Based Soil Concentrations

Site-Wide (RBSWC)

Risk Based Soil Concentrations
Whole Site

COPC	RBSC (mg/kg)	Calculated Risk: Ingestion non-threshold	Ingestion threshold	Dermal contact non-threshold	Dermal contact threshold	Total Risk non-threshold	Total Risk threshold	
hexachlorobenzene (HCB)	5		0.007		0.017		0.025	Revised based on site data - value from ISZ 5
1,2-dichloroethane (EDC)	700	8.5E-07		3.6E-07		1.2E-06		Revised based on site data - value from ISZ 4
vinyl chloride	10	2.3E-06		9.8E-07		3.3E-06		Criteria from ISZ 1a, 1b and 2
chloroform	80		0.0029		0.0012		0.0041	Criteria from ISZ 1a, 1b and 2
carbon tetrachloride	10		0.0047		0.0020		0.0067	Criteria from ISZ 1a, 1b and 2
trichloroethene (TCE)	400		0.0039		0.0017		0.0056	Criteria from ISZ 1a, 1b and 2
tetrachloroethene (PCE)	5		0.000		0.0001		0.000	Revised based on site data - value from ISZ 9
1,1,2-trichloroethane	10		0.00059		0.00025		0.00083	Criteria from ISZ 5
chlorobenzene	2500		0.0068		0.0029		0.0097	Criteria from ISZ 1a, 1b and 2
benzene	5	1.8E-08		7.4E-09		2.5E-08		Criteria from ISZ 1a, 1b and 2
toluene	200		0.00024		0.00010		0.00034	Criteria from ISZ 1a, 1b and 2
ethylbenzene	200		0.00048		0.00020		0.00069	Criteria from ISZ 1a, 1b and 2
xylenes	800		0.00107		0.00045		0.00152	Revised based on site data - value from ISZ 5
PAHs (BaP equivalent)	10	5.0E-07		1.5E-06		2.0E-06		Revised value
1,4-dichlorobenzene	15		0.00033		0.00014		0.0005	Revised based on site data - value from ISZ 5
1,2,3-trichloropropane	0.5	3.5E-07		1.5E-07		5.0E-07		Criteria from ISZ 5
a-HCH (a-BHC)	30		0.000881		0.000372		0.001252	Revised value
b-HCH (b-BHC)	15		0.176		0.074		0.250	Revised value
d-HCH (d-BHC)	1.2		0.014		0.0059		0.020	Criteria from ISZ 1a, 1b and 2
g-HCH (g-BHC or lindane)	15		0.00117		0.00050		0.0017	Revised value
dieldrin	15		0.04		0.015		0.05	Revised value
DDX (sum of DDT, DDE and DDD)	2000		0.23		0.17		0.40	Criteria from ISZ 10
endosulfan II	100		0.004		0.0018		0.006	Revised value
pentachlorophenol	10		0.00078		0.0046		0.0054	Revised value
mercury (as total, inroganic)	75		0.025		0.012		0.036	Criteria from ISZ 8 (also HIL)

7.1E-06	**0.8**	**Total Risk from Soil Ingestion and Dermal Contact**
2.0E-06	**0.2**	**Allowable Risk via Inhalation**
9.1E-06	**1.0**	**Total Risk**

Exposure to Chemicals via Ingestion of Soils

Site-wide

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**4.0E-6**	**5.2E-1**			**TOTAL**	**3.4E-8**	**1.3E-1**
hexachlorobenzene (HCB)		1.6E-04	5	5.0E-07	1.2E-06	--	7.3E-3	5	4.2E-09	2.9E-07	--	1.8E-3
1,2-dichloroethane (EDC)	1.2E-02		700	7.0E-05	1.6E-04	8.5E-7	--	700	5.9E-07	4.1E-05	7.0E-9	--
vinyl chloride	2.3E+00		10	1.0E-06	2.3E-06	2.3E-6	--	10	8.4E-09	5.9E-07	1.9E-8	--
chloroform		6.5E-03	80	8.1E-06	1.9E-05	--	2.9E-3	80	6.7E-08	4.7E-06	--	7.2E-4
carbon tetrachloride		5.0E-04	10	1.0E-06	2.3E-06	--	4.7E-3	10	8.4E-09	5.9E-07	--	1.2E-3
trichloroethene (TCE)		2.4E-02	400	4.0E-05	9.4E-05	--	3.9E-3	400	3.4E-07	2.3E-05	--	9.9E-4
tetrachloroethene (PCE)		9.2E-03	5	5.0E-07	1.2E-06	--	1.3E-4	5	4.2E-09	2.9E-07	--	3.2E-5
1,1,2-trichloroethane		4.0E-03	10	1.0E-06	2.3E-06	--	5.9E-4	10	8.4E-09	5.9E-07	--	1.5E-4
chlorobenzene		8.6E-02	2500	2.5E-04	5.9E-04	--	6.8E-3	2500	2.1E-06	1.5E-04	--	1.7E-3
benzene	3.5E-02		5	5.0E-07	1.2E-06	1.8E-8	--	5	4.2E-09	2.9E-07	1.5E-10	--
toluene		2.0E-01	200	2.0E-05	4.7E-05	--	2.4E-4	200	1.7E-07	1.2E-05	--	5.9E-5
ethylbenzene		9.7E-02	200	2.0E-05	4.7E-05	--	4.8E-4	200	1.7E-07	1.2E-05	--	1.2E-4
xylenes		1.8E-01	800	8.1E-05	1.9E-04	--	1.1E-3	800	6.7E-07	4.7E-05	--	2.7E-4
PAHs (BaP equivalent)	5.0E-01		10	1.0E-06	2.3E-06	5.0E-7	--	10	8.4E-09	5.9E-07	4.2E-9	--
1,4-dichlorobenzene		1.1E-02	15	1.5E-06	3.5E-06	--	3.3E-4	15	1.3E-08	8.8E-07	--	8.2E-5
1,2,3-trichloropropane	7.0E+00		0.5	5.0E-08	1.2E-07	3.5E-7	--	0.5	4.2E-10	2.9E-08	2.9E-9	--
a-HCH (a-BHC)		8.0E-03	30	3.0E-06	7.0E-06	--	8.8E-4	30	2.5E-08	1.8E-06	--	2.2E-4
b-HCH (b-BHC)		2.0E-05	15	1.5E-06	3.5E-06	--	1.8E-1	15	1.3E-08	8.8E-07	--	4.4E-2
d-HCH (d-BHC)		2.0E-05	1.2	1.2E-07	2.8E-07	--	1.4E-2	1.2	1.0E-09	7.0E-08	--	3.5E-3
g-HCH (g-BHC or lindane)		3.0E-03	15	1.5E-06	3.5E-06	--	1.2E-3	15	1.3E-08	8.8E-07	--	2.9E-4
dieldrin		1.0E-04	15	1.5E-06	3.5E-06	--	3.5E-2	15	1.3E-08	8.8E-07	--	8.8E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	2000	2.0E-04	4.7E-04	--	2.3E-1	2000	1.7E-06	1.2E-04	--	5.9E-2
endosulfan II		5.4E-03	100	1.0E-05	2.3E-05	--	4.3E-3	100	8.4E-08	5.9E-06	--	1.1E-3
pentachlorophenol		3.0E-03	10	1.0E-06	2.3E-06	--	7.8E-4	10	8.4E-09	5.9E-07	--	2.0E-4
mercury (as total, inroganic)		7.1E-04	75	7.5E-06	1.8E-05	--	2.5E-2	75	6.3E-08	4.4E-06	--	6.2E-3
			↑ RBSWC derived					↑ RBSWC derived				

Dermal Exposure to Chemicals via Contact with Soil

Site-wide

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.7E-6**	**2.2E-1**			**TOTAL**	**3.0E-8**	**1.2E-1**
hexachlorobenzene (HCB)		1.6E-04	5	2.1E-07	5.0E-07	--	3.1E-3	5	3.7E-09	2.6E-07	--	1.6E-3
1,2-dichloroethane (EDC)	1.2E-02		700	3.0E-05	6.9E-05	3.6E-7	--	700	5.2E-07	3.7E-05	6.3E-9	--
vinyl chloride	2.3E+00		10	4.2E-07	9.9E-07	9.8E-7	--	10	7.5E-09	5.2E-07	1.7E-8	--
chloroform		6.5E-03	80	3.4E-06	7.9E-06	--	1.2E-3	80	6.0E-08	4.2E-06	--	6.4E-4
carbon tetrachloride		5.0E-04	10	4.2E-07	9.9E-07	--	2.0E-3	10	7.5E-09	5.2E-07	--	1.1E-3
trichloroethene (TCE)		2.4E-02	400	1.7E-05	4.0E-05	--	1.7E-3	400	3.0E-07	2.1E-05	--	8.8E-4
tetrachloroethene (PCE)		9.2E-03	5	2.1E-07	5.0E-07	--	5.4E-5	5	3.7E-09	2.6E-07	--	2.8E-5
1,1,2-trichloroethane		4.0E-03	10	4.2E-07	9.9E-07	--	2.5E-4	10	7.5E-09	5.2E-07	--	1.3E-4
chlorobenzene		8.6E-02	2500	1.1E-04	2.5E-04	--	2.9E-3	2500	1.9E-06	1.3E-04	--	1.5E-3
benzene	3.5E-02		5	2.1E-07	5.0E-07	7.4E-9	--	5	3.7E-09	2.6E-07	1.3E-10	--
toluene		2.0E-01	200	8.5E-06	2.0E-05	--	1.0E-4	200	1.5E-07	1.0E-05	--	5.3E-5
ethylbenzene		9.7E-02	200	8.5E-06	2.0E-05	--	2.0E-4	200	1.5E-07	1.0E-05	--	1.1E-4
xylenes		1.8E-01	800	3.4E-05	7.9E-05	--	4.5E-4	800	6.0E-07	4.2E-05	--	2.4E-4
PAHs (BaP equivalent)	5.0E-01		10	4.2E-07	9.9E-07	2.1E-7	--	10	7.5E-09	5.2E-07	3.7E-9	--
1,4-dichlorobenzene		1.1E-02	15	6.4E-07	1.5E-06	--	1.4E-4	15	1.1E-08	7.9E-07	--	7.3E-5
1,2,3-trichloropropane	7.0E+00		0.5	2.1E-08	5.0E-08	1.5E-7	--	0.5	3.7E-10	2.6E-08	2.6E-9	--
a-HCH (a-BHC)		8.0E-03	30	1.3E-06	3.0E-06	--	3.7E-4	30	2.2E-08	1.6E-06	--	2.0E-4
b-HCH (b-BHC)		2.0E-05	15	6.4E-07	1.5E-06	--	7.4E-2	15	1.1E-08	7.9E-07	--	3.9E-2
d-HCH (d-BHC)		2.0E-05	1.2	5.1E-08	1.2E-07	--	5.9E-3	1.2	9.0E-10	6.3E-08	--	3.1E-3
g-HCH (g-BHC or lindane)		3.0E-03	15	6.4E-07	1.5E-06	--	5.0E-4	15	1.1E-08	7.9E-07	--	2.6E-4
dieldrin		1.0E-04	15	6.4E-07	1.5E-06	--	1.5E-2	15	1.1E-08	7.9E-07	--	7.9E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	2000	8.5E-05	2.0E-04	--	9.9E-2	2000	1.5E-06	1.0E-04	--	5.2E-2
endosulfan II		5.4E-03	100	4.2E-06	9.9E-06	--	1.8E-3	100	7.5E-08	5.2E-06	--	9.7E-4
pentachlorophenol		3.0E-03	10	4.2E-07	9.9E-07	--	3.3E-4	10	7.5E-09	5.2E-07	--	1.7E-4
mercury (as total, inroganic)		7.1E-04	75	3.2E-06	7.4E-06	--	1.0E-2	75	5.6E-08	3.9E-06	--	5.5E-3
			RBSWC derived					RBSWC derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment Site-wide

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**2.4E-6**	**2.6E-1**			**TOTAL**	**4.2E-8**	**1.4E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	5	1.2E-06	2.8E-06	--	1.7E-2	5	2.1E-08	1.5E-06	--	9.1E-3
1,2-dichloroethane (EDC)	1.2E-02		0.01	700	1.7E-05	3.9E-05	2.0E-7	--	700	2.9E-07	2.0E-05	3.5E-9	--
vinyl chloride	2.3E+00		0.01	10	2.4E-07	5.5E-07	5.4E-7	--	10	4.2E-09	2.9E-07	9.6E-9	--
chloroform		6.5E-03	0.01	80	1.9E-06	4.4E-06	--	6.8E-4	80	3.3E-08	2.3E-06	--	3.6E-4
carbon tetrachloride		5.0E-04	0.01	10	2.4E-07	5.5E-07	--	1.1E-3	10	4.2E-09	2.9E-07	--	5.9E-4
trichloroethene (TCE)		2.4E-02	0.01	400	9.4E-06	2.2E-05	--	9.3E-4	400	1.7E-07	1.2E-05	--	4.9E-4
tetrachloroethene (PCE)		9.2E-03	0.01	5	1.2E-07	2.8E-07	--	3.0E-5	5	2.1E-09	1.5E-07	--	1.6E-5
1,1,2-trichloroethane		4.0E-03	0.01	10	2.4E-07	5.5E-07	--	1.4E-4	10	4.2E-09	2.9E-07	--	7.3E-5
chlorobenzene		8.6E-02	0.01	2500	5.9E-05	1.4E-04	--	1.6E-3	2500	1.0E-06	7.3E-05	--	8.5E-4
benzene	3.5E-02		0.01	5	1.2E-07	2.8E-07	4.1E-9	--	5	2.1E-09	1.5E-07	7.3E-11	--
toluene		2.0E-01	0.01	200	4.7E-06	1.1E-05	--	5.6E-5	200	8.3E-08	5.8E-06	--	2.9E-5
ethylbenzene		9.7E-02	0.01	200	4.7E-06	1.1E-05	--	1.1E-4	200	8.3E-08	5.8E-06	--	6.0E-5
xylenes		1.8E-01	0.01	800	1.9E-05	4.4E-05	--	2.5E-4	800	3.3E-07	2.3E-05	--	1.3E-4
PAHs (BaP equivalent)	5.0E-01		0.13	10	3.1E-06	7.2E-06	1.5E-6	--	10	5.4E-08	3.8E-06	2.7E-8	--
1,4-dichlorobenzene		1.1E-02	0.01	15	3.5E-07	8.3E-07	--	7.7E-5	15	6.2E-09	4.4E-07	--	4.1E-5
1,2,3-trichloropropane	7.0E+00		0.01	0.5	1.2E-08	2.8E-08	8.3E-8	--	0.5	2.1E-10	1.5E-08	1.5E-9	--
a-HCH (a-BHC)		8.0E-03	0.01	30	7.1E-07	1.7E-06	--	2.1E-4	30	1.2E-08	8.7E-07	--	1.1E-4
b-HCH (b-BHC)		2.0E-05	0.01	15	3.5E-07	8.3E-07	--	4.1E-2	15	6.2E-09	4.4E-07	--	2.2E-2
d-HCH (d-BHC)		2.0E-05	0.01	1.2	2.8E-08	6.6E-08	--	3.3E-3	1.2	5.0E-10	3.5E-08	--	1.7E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	15	3.5E-07	8.3E-07	--	2.8E-4	15	6.2E-09	4.4E-07	--	1.5E-4
dieldrin		1.0E-04	0.01	15	3.5E-07	8.3E-07	--	8.3E-3	15	6.2E-09	4.4E-07	--	4.4E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	2000	1.4E-04	3.3E-04	--	1.7E-1	2000	2.5E-06	1.7E-04	--	8.7E-2
endosulfan II		5.4E-03	0.01	100	2.4E-06	5.5E-06	--	1.0E-3	100	4.2E-08	2.9E-06	--	5.4E-4
pentachlorophenol		3.0E-03	0.25	10	5.9E-06	1.4E-05	--	4.6E-3	10	1.0E-07	7.3E-06	--	2.4E-3
mercury (as total, inroganic)		3.6E-04	0.01	75	1.8E-06	4.1E-06	--	1.2E-2	75	3.1E-08	2.2E-06	--	6.1E-3
				↑ RBSWC derived					↑ RBSWC derived				

ISZ 1a and 1b

Risk Based Soil Concentrations
ISZ 1a and 1b

COPC	RBSC (mg/kg)	Calculated Risk: Ingestion non-threshold	Ingestion threshold	dermal contact non-threshold	dermal contact threshold	Total Risk non-threshold	Total Risk threshold
hexachlorobenzene (HCB)	10		0.015		0.034		0.049
1,2-dichloroethane (EDC)	55	6.6E-08		2.8E-08		9.4E-08	
vinyl chloride	10	2.3E-06		9.8E-07		3.3E-06	
chloroform	80		0.0029		0.0012		0.0041
carbon tetrachloride	10		0.0047		0.0020		0.0067
trichloroethene (TCE)	400		0.0039		0.0017		0.0056
chlorobenzene	2500		0.0068		0.0029		0.0097
benzene	5	1.8E-08		7.4E-09		2.5E-08	
toluene	200		0.00024		0.00010		0.00034
ethylbenzene	200		0.00048		0.00020		0.00069
xylenes	200		0.00027		0.00011		0.00038
PAHs (BaP equivalent)	24	1.2E-06		3.7E-06		4.9E-06	
1,4-dichlorobenzene	40		0.00088		0.00037		0.0012
a-HCH (a-BHC)	1.5		0.000044		0.000019		0.000063
b-HCH (b-BHC)	2.7		0.032		0.013		0.045
d-HCH (d-BHC)	1.2		0.014		0.0059		0.020
g-HCH (g-BHC or lindane)	12		0.00094		0.00040		0.0013
DDX (sum of DDT, DDE and DDD)	2300		0.27		0.19		0.46

8.3E-06	**0.60**	**Total Risk from Soil Ingestion and Dermal Contact**
4.5E-07	**0.22**	**Total Risk from Inhalation Exposures**
2.0E-06	**0.40**	**Allowable Risk via Inhalation**
1.0E-05	**1.00**	**Total Risk**

Exposure to Chemicals via Ingestion of Soils

Identified Source Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.6E-6**	**3.5E-1**			**TOTAL**	**3.0E-8**	**8.8E-2**
hexachlorobenzene (HCB)		1.6E-04	10	1.0E-06	2.3E-06	--	1.5E-2	10	8.4E-09	5.9E-07	--	3.7E-3
1,2-dichloroethane (EDC)	1.2E-02		55	5.5E-06	1.3E-05	6.6E-8	--	55	4.6E-08	3.2E-06	5.5E-10	--
vinyl chloride	2.3E+00		10	1.0E-06	2.3E-06	2.3E-6	--	10	8.4E-09	5.9E-07	1.9E-8	--
chloroform		6.5E-03	80	8.1E-06	1.9E-05	--	2.9E-3	80	6.7E-08	4.7E-06	--	7.2E-4
carbon tetrachloride		5.0E-04	10	1.0E-06	2.3E-06	--	4.7E-3	10	8.4E-09	5.9E-07	--	1.2E-3
trichloroethene (TCE)		2.4E-02	400	4.0E-05	9.4E-05	--	3.9E-3	400	3.4E-07	2.3E-05	--	9.9E-4
chlorobenzene		8.6E-02	2500	2.5E-04	5.9E-04	--	6.8E-3	2500	2.1E-06	1.5E-04	--	1.7E-3
benzene	3.5E-02		5	5.0E-07	1.2E-06	1.8E-8	--	5	4.2E-09	2.9E-07	1.5E-10	--
toluene		2.0E-01	200	2.0E-05	4.7E-05	--	2.4E-4	200	1.7E-07	1.2E-05	--	5.9E-5
ethylbenzene		9.7E-02	200	2.0E-05	4.7E-05	--	4.8E-4	200	1.7E-07	1.2E-05	--	1.2E-4
xylenes		1.8E-01	200	2.0E-05	4.7E-05	--	2.7E-4	200	1.7E-07	1.2E-05	--	6.7E-5
PAHs (BaP equivalent)	5.0E-01		24	2.4E-06	5.6E-06	1.2E-6	--	24	2.0E-08	1.4E-06	1.0E-8	--
1,4-dichlorobenzene		1.1E-02	40	4.0E-06	9.4E-06	--	8.8E-4	40	3.4E-08	2.3E-06	--	2.2E-4
a-HCH (a-BHC)		8.0E-03	1.5	1.5E-07	3.5E-07	--	4.4E-5	1.5	1.3E-09	8.8E-08	--	1.1E-5
b-HCH (b-BHC)		2.0E-05	2.7	2.7E-07	6.3E-07	--	3.2E-2	2.7	2.3E-09	1.6E-07	--	7.9E-3
d-HCH (d-BHC)		2.0E-05	1.2	1.2E-07	2.8E-07	--	1.4E-2	1.2	1.0E-09	7.0E-08	--	3.5E-3
g-HCH (g-BHC or lindane)		3.0E-03	12	1.2E-06	2.8E-06	--	9.4E-4	12	1.0E-08	7.0E-07	--	2.3E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	2300	2.3E-04	5.4E-04	--	2.7E-1	2300	1.9E-06	1.4E-04	--	6.8E-2
			RBSC derived					RBSC derived				

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zones 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.5E-6**	**1.5E-1**			**TOTAL**	**2.7E-8**	**7.8E-2**
hexachlorobenzene (HCB)		1.6E-04	10	4.2E-07	9.9E-07	--	6.2E-3	10	7.5E-09	5.2E-07	--	3.3E-3
1,2-dichloroethane (EDC)	1.2E-02		55	2.3E-06	5.5E-06	2.8E-8	--	55	4.1E-08	2.9E-06	4.9E-10	--
vinyl chloride	2.3E+00		10	4.2E-07	9.9E-07	9.8E-7	--	10	7.5E-09	5.2E-07	1.7E-8	--
chloroform		6.5E-03	80	3.4E-06	7.9E-06	--	1.2E-3	80	6.0E-08	4.2E-06	--	6.4E-4
carbon tetrachloride		5.0E-04	10	4.2E-07	9.9E-07	--	2.0E-3	10	7.5E-09	5.2E-07	--	1.1E-3
trichloroethene (TCE)		2.4E-02	400	1.7E-05	4.0E-05	--	1.7E-3	400	3.0E-07	2.1E-05	--	8.8E-4
chlorobenzene		8.6E-02	2500	1.1E-04	2.5E-04	--	2.9E-3	2500	1.9E-06	1.3E-04	--	1.5E-3
benzene	3.5E-02		5	2.1E-07	5.0E-07	7.4E-9	--	5	3.7E-09	2.6E-07	1.3E-10	--
toluene		2.0E-01	200	8.5E-06	2.0E-05	--	1.0E-4	200	1.5E-07	1.0E-05	--	5.3E-5
xylenes		1.8E-01	200	8.5E-06	2.0E-05	--	1.1E-4	200	1.5E-07	1.0E-05	--	6.0E-5
PAHs (BaP equivalent)	5.0E-01		24	1.0E-06	2.4E-06	5.1E-7	--	24	1.8E-08	1.3E-06	9.0E-9	--
1,4-dichlorobenzene		1.1E-02	40	1.7E-06	4.0E-06	--	3.7E-4	40	3.0E-08	2.1E-06	--	2.0E-4
a-HCH (a-BHC)		8.0E-03	1.5	6.4E-08	1.5E-07	--	1.9E-5	1.5	1.1E-09	7.9E-08	--	9.8E-6
b-HCH (b-BHC)		2.0E-05	2.7	1.1E-07	2.7E-07	--	1.3E-2	2.7	2.0E-09	1.4E-07	--	7.1E-3
d-HCH (d-BHC)		2.0E-05	1.2	5.1E-08	1.2E-07	--	5.9E-3	1.2	9.0E-10	6.3E-08	--	3.1E-3
g-HCH (g-BHC or lindane)		3.0E-03	12	5.1E-07	1.2E-06	--	4.0E-4	12	9.0E-09	6.3E-07	--	2.1E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	2300	9.8E-05	2.3E-04	--	1.1E-1	2300	1.7E-06	1.2E-04	--	6.0E-2
			↑ RBSC derived					↑ RBSC derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment ISZ 1a and 1b

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**4.2E-6**	**2.4E-1**			**TOTAL**	**7.5E-8**	**1.3E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	10	2.4E-06	5.5E-06	--	3.4E-2	10	4.2E-08	2.9E-06	--	1.8E-2
1,2-dichloroethane (EDC)	1.2E-02		0.01	55	1.3E-06	3.0E-06	1.6E-8	--	55	2.3E-08	1.6E-06	2.7E-10	--
vinyl chloride	2.3E+00		0.01	10	2.4E-07	5.5E-07	5.4E-7	--	10	4.2E-09	2.9E-07	9.6E-9	--
chloroform		6.5E-03	0.01	80	1.9E-06	4.4E-06	--	6.8E-4	80	3.3E-08	2.3E-06	--	3.6E-4
carbon tetrachloride		5.0E-04	0.01	10	2.4E-07	5.5E-07	--	1.1E-3	10	4.2E-09	2.9E-07	--	5.9E-4
trichloroethene (TCE)		2.4E-02	0.01	400	9.4E-06	2.2E-05	--	9.3E-4	400	1.7E-07	1.2E-05	--	4.9E-4
chlorobenzene		8.6E-02	0.01	2500	5.9E-05	1.4E-04	--	1.6E-3	2500	1.0E-06	7.3E-05	--	8.5E-4
benzene	3.5E-02		0.01	5	1.2E-07	2.8E-07	4.1E-9	--	5	2.1E-09	1.5E-07	7.3E-11	--
toluene		2.0E-01	0.01	200	4.7E-06	1.1E-05	--	5.6E-5	200	8.3E-08	5.8E-06	--	2.9E-5
xylenes		1.8E-01	0.01	200	4.7E-06	1.1E-05	--	6.3E-5	200	8.3E-08	5.8E-06	--	3.3E-5
PAHs (BaP equivalent)	5.0E-01		0.13	24	7.4E-06	1.7E-05	3.7E-6	--	24	1.3E-07	9.1E-06	6.5E-8	--
1,4-dichlorobenzene		1.1E-02	0.01	40	9.4E-07	2.2E-06	--	2.1E-4	40	1.7E-08	1.2E-06	--	1.1E-4
a-HCH (a-BHC)		8.0E-03	0.01	1.5	3.5E-08	8.3E-08	--	1.0E-5	1.5	6.2E-10	4.4E-08	--	5.5E-6
b-HCH (b-BHC)		2.0E-05	0.01	2.7	6.4E-08	1.5E-07	--	7.4E-3	2.7	1.1E-09	7.9E-08	--	3.9E-3
d-HCH (d-BHC)		2.0E-05	0.01	1.2	2.8E-08	6.6E-08	--	3.3E-3	1.2	5.0E-10	3.5E-08	--	1.7E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	12	2.8E-07	6.6E-07	--	2.2E-4	12	5.0E-09	3.5E-07	--	1.2E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	2300	1.6E-04	3.8E-04	--	1.9E-1	2300	2.9E-06	2.0E-04	--	1.0E-1
				RBSC derived					RBSC derived				

ISZ 4

Risk Based Soil Concentrations
ISZ 4

COPC	RBSC (mg/kg)	Calculated Risk					
		Ingestion		dermal contact		Total Risk	
		non-threshold	threshold	non-threshold	threshold	non-threshold	threshold
hexachlorobenzene (HCB)	15		0.022		0.052		0.074
1,2-dichloroethane (EDC)	700	8.5E-07		3.6E-07		1.2E-06	
chloroform	5		0.00018		0.000076		0.00026
trichloroethene (TCE)	100		0.00099		0.00042		0.0014
benzene	10	3.5E-08		1.5E-08		5.0E-08	
a-HCH (a-BHC)	5		0.00015		0.000062		0.00021
b-HCH (b-BHC)	15		0.18		0.074		0.25
d-HCH (d-BHC)	2		0.023		0.010		0.033
g-HCH (g-BHC or lindane)	50		0.0039		0.0017		0.0056
DDX (sum of DDT, DDE and DDD)	1800		0.21		0.15		0.36
Mercury (as total, inroganic)	120		0.079		0.034		0.11

Total Risk from Soil Ingestion and Dermal Contact	**1.3E-06**	**0.84**
Total Risk from Inhalation Exposures	**1.9E-07**	**0.011**
Allowable Risk via Inhalation	**2.0E-06**	**0.20**
Total Risk	**3.3E-06**	**1.0**

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07** **2.3E-07**	**NonThreshold** **Threshold**	**8.4E-10** **5.9E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**8.8E-7**	**5.2E-1**			**TOTAL**	**7.3E-9**	**1.3E-1**
hexachlorobenzene (HCB)		1.6E-04	15	1.5E-06	3.5E-06	--	2.2E-2	15	1.3E-08	8.8E-07	--	5.5E-3
1,2-dichloroethane (EDC)	1.2E-02		700	7.0E-05	1.6E-04	8.5E-7	--	700	5.9E-07	4.1E-05	7.0E-9	--
chloroform		6.5E-03	5	5.0E-07	1.2E-06	--	1.8E-4	5	4.2E-09	2.9E-07	--	4.5E-5
trichloroethene (TCE)		2.4E-02	100	1.0E-05	2.3E-05	--	9.9E-4	100	8.4E-08	5.9E-06	--	2.5E-4
benzene	3.5E-02		10	1.0E-06	2.3E-06	3.5E-8	--	10	8.4E-09	5.9E-07	2.9E-10	--
a-HCH (a-BHC)		8.0E-03	5	5.0E-07	1.2E-06	--	1.5E-4	5	4.2E-09	2.9E-07	--	3.7E-5
b-HCH (b-BHC)		2.0E-05	15	1.5E-06	3.5E-06	--	1.8E-1	15	1.3E-08	8.8E-07	--	4.4E-2
d-HCH (d-BHC)		2.0E-05	2	2.0E-07	4.7E-07	--	2.3E-2	2	1.7E-09	1.2E-07	--	5.9E-3
g-HCH (g-BHC or lindane)		3.0E-03	50	5.0E-06	1.2E-05	--	3.9E-3	50	4.2E-08	2.9E-06	--	9.8E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1800	1.8E-04	4.2E-04	--	2.1E-1	1800	1.5E-06	1.1E-04	--	5.3E-2
Mercury (as total, inroganic)		3.6E-04	120	1.2E-05	2.8E-05	--	7.9E-2	120	1.0E-07	7.0E-06	--	2.0E-2
			↑ RBSC Derived					↑ RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 4

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.7E-7**	**2.2E-1**			**TOTAL**	**6.5E-9**	**1.2E-1**
hexachlorobenzene (HCB)		1.6E-04	15	6.4E-07	1.5E-06	--	9.3E-3	15	1.1E-08	7.9E-07	--	4.9E-3
1,2-dichloroethane (EDC)	1.2E-02		700	3.0E-05	6.9E-05	3.6E-7	--	700	5.2E-07	3.7E-05	6.3E-9	--
chloroform		6.5E-03	5	2.1E-07	5.0E-07	--	7.6E-5	5	3.7E-09	2.6E-07	--	4.0E-5
trichloroethene (TCE)		2.4E-02	100	4.2E-06	9.9E-06	--	4.2E-4	100	7.5E-08	5.2E-06	--	2.2E-4
benzene	3.5E-02		10	4.2E-07	9.9E-07	1.5E-8	--	10	7.5E-09	5.2E-07	2.6E-10	--
a-HCH (a-BHC)		8.0E-03	5	2.1E-07	5.0E-07	--	6.2E-5	5	3.7E-09	2.6E-07	--	3.3E-5
b-HCH (b-BHC)		2.0E-05	15	6.4E-07	1.5E-06	--	7.4E-2	15	1.1E-08	7.9E-07	--	3.9E-2
d-HCH (d-BHC)		2.0E-05	2	8.5E-08	2.0E-07	--	9.9E-3	2	1.5E-09	1.0E-07	--	5.2E-3
g-HCH (g-BHC or lindane)		3.0E-03	50	2.1E-06	5.0E-06	--	1.7E-3	50	3.7E-08	2.6E-06	--	8.7E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1800	7.6E-05	1.8E-04	--	8.9E-2	1800	1.3E-06	9.4E-05	--	4.7E-2
Mercury (as total, inroganic)		3.6E-04	120	5.1E-06	1.2E-05	--	3.4E-2	120	9.0E-08	6.3E-06	--	1.8E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 4**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*FE*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**2.1E-7**	**2.7E-1**			**TOTAL**	**3.6E-9**	**1.4E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	15	3.5E-06	8.3E-06	--	5.2E-2	15	6.2E-08	4.4E-06	--	2.7E-2
1,2-dichloroethane (EDC)	1.2E-02		0.01	700	1.7E-05	3.9E-05	2.0E-7	--	700	2.9E-07	2.0E-05	3.5E-9	--
chloroform		6.5E-03	0.01	5	1.2E-07	2.8E-07	--	4.2E-5	5	2.1E-09	1.5E-07	--	2.2E-5
trichloroethene (TCE)		2.4E-02	0.01	100	2.4E-06	5.5E-06	--	2.3E-4	100	4.2E-08	2.9E-06	--	1.2E-4
benzene	3.5E-02		0.01	10	2.4E-07	5.5E-07	8.3E-9	--	10	4.2E-09	2.9E-07	1.5E-10	--
a-HCH (a-BHC)		8.0E-03	0.01	5	1.2E-07	2.8E-07	--	3.4E-5	5	2.1E-09	1.5E-07	--	1.8E-5
b-HCH (b-BHC)		2.0E-05	0.01	15	3.5E-07	8.3E-07	--	4.1E-2	15	6.2E-09	4.4E-07	--	2.2E-2
d-HCH (d-BHC)		2.0E-05	0.01	2	4.7E-08	1.1E-07	--	5.5E-3	2	8.3E-10	5.8E-08	--	2.9E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	50	1.2E-06	2.8E-06	--	9.2E-4	50	2.1E-08	1.5E-06	--	4.9E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	1800	1.3E-04	3.0E-04	--	1.5E-1	1800	2.2E-06	1.6E-04	--	7.9E-2
Mercury (as total, inroganic)		3.6E-04	0.01	120	2.8E-06	6.6E-06	--	1.9E-2	120	5.0E-08	3.5E-06	--	9.8E-3
				↑ RBSC Derived					↑ RBSC Derived				

ISZ 5

Risk Based Soil Concentrations
ISZ 5

COPC	RBSC (mg/kg)	Calculated Risk: Ingestion non-threshold	Ingestion threshold	dermal contact non-threshold	dermal contact threshold	Total Risk non-threshold	Total Risk threshold
hexachlorobenzene (HCB)	5		0.007		0.017		0.025
1,2-dichloroethane (EDC)	30	3.6E-08		1.5E-08		5.2E-08	
chloroform	5		0.00018		0.000076		0.00026
trichloroethene (TCE)	5		0.000049		0.000021		0.000070
1,1,2-trichloroethane	10		0.00059		0.00025		0.00083
chlorobenzene	600		0.0016		0.00069		0.0023
ethylbenzene	150		0.00036		0.00015		0.00052
xylenes	800		0.00107		0.00045		0.0015
PAHs (BaP equivalent)	36	1.8E-06		5.5E-06		7.3E-06	
1,4-dichlorobenzene	15		0.00033		0.00014		0.00047
1,2,3-trichloropropane	0.5	3.5E-07		1.5E-07		5.0E-07	
a-HCH (a-BHC)	2		0.000059		0.000025		0.000083
b-HCH (b-BHC)	8		0.094		0.040		0.13
d-HCH (d-BHC)	4		0.047		0.020		0.067
g-HCH (g-BHC or lindane)	8		0.00063		0.00026		0.00089
DDX (sum of DDT, DDE and DDD)	1900		0.22		0.16		0.38

7.9E-06	**0.61**	**Total Risk from Soil Ingestion and Dermal Contact**
6.4E-05	**0.24**	**Total Risk from Inhalation Exposures**
2.0E-06	**0.40**	**Allowable Risk via Inhalation assuming presence of 1,2,3-trichloropropane addressed**
9.9E-06	**1.0**	**Total Risk**

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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	**2.2E-6**	**3.8E-1**			**TOTAL**	**1.8E-8**	**9.4E-2**
hexachlorobenzene (HCB)		1.6E-04	5	5.0E-07	1.2E-06	--	7.3E-3	5	4.2E-09	2.9E-07	--	1.8E-3
1,2-dichloroethane (EDC)	1.2E-02		30	3.0E-06	7.0E-06	3.6E-8	--	30	2.5E-08	1.8E-06	3.0E-10	--
chloroform		6.5E-03	5	5.0E-07	1.2E-06	--	1.8E-4	5	4.2E-09	2.9E-07	--	4.5E-5
trichloroethene (TCE)		2.4E-02	5	5.0E-07	1.2E-06	--	4.9E-5	5	4.2E-09	2.9E-07	--	1.2E-5
1,1,2-trichloroethane		4.0E-03	10	1.0E-06	2.3E-06	--	5.9E-4	10	8.4E-09	5.9E-07	--	1.5E-4
chlorobenzene		8.6E-02	600	6.0E-05	1.4E-04	--	1.6E-3	600	5.0E-07	3.5E-05	--	4.1E-4
ethylbenzene		9.7E-02	150	1.5E-05	3.5E-05	--	3.6E-4	150	1.3E-07	8.8E-06	--	9.1E-5
xylenes		1.8E-01	800	8.1E-05	1.9E-04	--	1.1E-3	800	6.7E-07	4.7E-05	--	2.7E-4
PAHs (BaP equivalent)	5.0E-01		36	3.6E-06	8.5E-06	1.8E-6	--	36	3.0E-08	2.1E-06	1.5E-8	--
1,4-dichlorobenzene		1.1E-02	15	1.5E-06	3.5E-06	--	3.3E-4	15	1.3E-08	8.8E-07	--	8.2E-5
1,2,3-trichloropropane	7.0E+00		0.5	5.0E-08	1.2E-07	3.5E-7	--	0.5	4.2E-10	2.9E-08	2.9E-9	--
a-HCH (a-BHC)		8.0E-03	2	2.0E-07	4.7E-07	--	5.9E-5	2	1.7E-09	1.2E-07	--	1.5E-5
b-HCH (b-BHC)		2.0E-05	8	8.1E-07	1.9E-06	--	9.4E-2	8	6.7E-09	4.7E-07	--	2.3E-2
d-HCH (d-BHC)		2.0E-05	4	4.0E-07	9.4E-07	--	4.7E-2	4	3.4E-09	2.3E-07	--	1.2E-2
g-HCH (g-BHC or lindane)		3.0E-03	8	8.1E-07	1.9E-06	--	6.3E-4	8	6.7E-09	4.7E-07	--	1.6E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1900	1.9E-04	4.5E-04	--	2.2E-1	1900	1.6E-06	1.1E-04	--	5.6E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 5

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**9.3E-7**	**1.6E-1**			**TOTAL**	**1.6E-8**	**8.4E-2**
hexachlorobenzene (HCB)		1.6E-04	5	2.1E-07	5.0E-07	--	3.1E-3	5	3.7E-09	2.6E-07	--	1.6E-3
1,2-dichloroethane (EDC)	1.2E-02		30	1.3E-06	3.0E-06	1.5E-8	--	30	2.2E-08	1.6E-06	2.7E-10	--
chloroform		6.5E-03	5	2.1E-07	5.0E-07	--	7.6E-5	5	3.7E-09	2.6E-07	--	4.0E-5
trichloroethene (TCE)		2.4E-02	5	2.1E-07	5.0E-07	--	2.1E-5	5	3.7E-09	2.6E-07	--	1.1E-5
1,1,2-trichloroethane		4.0E-03	10	4.2E-07	9.9E-07	--	2.5E-4	10	7.5E-09	5.2E-07	--	1.3E-4
chlorobenzene		8.6E-02	600	2.5E-05	5.9E-05	--	6.9E-4	600	4.5E-07	3.1E-05	--	3.7E-4
ethylbenzene		9.7E-02	150	6.4E-06	1.5E-05	--	1.5E-4	150	1.1E-07	7.9E-06	--	8.1E-5
xylenes		1.8E-01	800	3.4E-05	7.9E-05	--	4.5E-4	800	6.0E-07	4.2E-05	--	2.4E-4
PAHs (BaP equivalent)	5.0E-01		36	1.5E-06	3.6E-06	7.6E-7	--	36	2.7E-08	1.9E-06	1.3E-8	--
1,4-dichlorobenzene		1.1E-02	15	6.4E-07	1.5E-06	--	1.4E-4	15	1.1E-08	7.9E-07	--	7.3E-5
1,2,3-trichloropropane	7.0E+00		0.5	2.1E-08	5.0E-08	1.5E-7	--	0.5	3.7E-10	2.6E-08	2.6E-9	--
a-HCH (a-BHC)		8.0E-03	2	8.5E-08	2.0E-07	--	2.5E-5	2	1.5E-09	1.0E-07	--	1.3E-5
b-HCH (b-BHC)		2.0E-05	8	3.4E-07	7.9E-07	--	4.0E-2	8	6.0E-09	4.2E-07	--	2.1E-2
d-HCH (d-BHC)		2.0E-05	4	1.7E-07	4.0E-07	--	2.0E-2	4	3.0E-09	2.1E-07	--	1.0E-2
g-HCH (g-BHC or lindane)		3.0E-03	8	3.4E-07	7.9E-07	--	2.6E-4	8	6.0E-09	4.2E-07	--	1.4E-4
DDX (sum of DDT, DDE and DDD)		2.0E-03	1900	8.1E-05	1.9E-04	--	9.4E-2	1900	1.4E-06	1.0E-04	--	5.0E-2
			↑ RBSC Derived					↑ RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 5**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**5.6E-6**	**2.1E-1**			**TOTAL**	**9.9E-8**	**1.1E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	5	1.2E-06	2.8E-06	--	1.7E-2	5	2.1E-08	1.5E-06	--	9.1E-3
1,2-dichloroethane (EDC)	1.2E-02		0.01	30	7.1E-07	1.7E-06	8.5E-9	--	30	1.2E-08	8.7E-07	1.5E-10	--
chloroform		6.5E-03	0.01	5	1.2E-07	2.8E-07	--	4.2E-5	5	2.1E-09	1.5E-07	--	2.2E-5
trichloroethene (TCE)		2.4E-02	0.01	5	1.2E-07	2.8E-07	--	1.2E-5	5	2.1E-09	1.5E-07	--	6.1E-6
1,1,2-trichloroethane		4.0E-03	0.01	10	2.4E-07	5.5E-07	--	1.4E-4	10	4.2E-09	2.9E-07	--	7.3E-5
chlorobenzene		8.6E-02	0.01	600	1.4E-05	3.3E-05	--	3.8E-4	600	2.5E-07	1.7E-05	--	2.0E-4
ethylbenzene		9.7E-02	0.01	150	3.5E-06	8.3E-06	--	8.5E-5	150	6.2E-08	4.4E-06	--	4.5E-5
xylenes		1.8E-01	0.01	800	1.9E-05	4.4E-05	--	2.5E-4	800	3.3E-07	2.3E-05	--	1.3E-4
PAHs (BaP equivalent)	5.0E-01		0.13	36	1.1E-05	2.6E-05	5.5E-6	--	36	1.9E-07	1.4E-05	9.7E-8	--
1,4-dichlorobenzene		1.1E-02	0.01	15	3.5E-07	8.3E-07	--	7.7E-5	15	6.2E-09	4.4E-07	--	4.1E-5
1,2,3-trichloropropane	7.0E+00		0.01	0.5	1.2E-08	2.8E-08	8.3E-8	--	0.5	2.1E-10	1.5E-08	1.5E-9	--
a-HCH (a-BHC)		8.0E-03	0.01	2	4.7E-08	1.1E-07	--	1.4E-5	2	8.3E-10	5.8E-08	--	7.3E-6
b-HCH (b-BHC)		2.0E-05	0.01	8	1.9E-07	4.4E-07	--	2.2E-2	8	3.3E-09	2.3E-07	--	1.2E-2
d-HCH (d-BHC)		2.0E-05	0.01	4	9.4E-08	2.2E-07	--	1.1E-2	4	1.7E-09	1.2E-07	--	5.8E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	8	1.9E-07	4.4E-07	--	1.5E-4	8	3.3E-09	2.3E-07	--	7.8E-5
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	1900	1.3E-04	3.1E-04	--	1.6E-1	1900	2.4E-06	1.7E-04	--	8.3E-2
				↑ RBSC Derived					↑ RBSC Derived				

ISZ 7

Risk Based Soil Concentrations
ISZ 7

COPC	RBSC (mg/kg)	Calculated Risk					
		Ingestion		dermal contact		Total Risk	
		non-threshold	threshold	non-threshold	threshold	non-threshold	threshold
hexachlorobenzene (HCB)	30		0.044		0.10		0.15
a-HCH (a-BHC)	600		0.018		0.0074		0.025
b-HCH (b-BHC)	20		0.23		0.099		0.33
g-HCH (g-BHC or lindane)	1000		0.078		0.033		0.11
DDX (sum of DDT, DDE and DDD)	900		0.11		0.074		0.18

0.0E+00	0.80	Total Risk from Soil Ingestion and Dermal Contact
2.0E-06	0.20	Allowable Risk via Inhalation
2.0E-06	1.0	Total Risk

Exposure to Chemicals via Ingestion of Soils

Identified Soil Zone 7

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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		**4.8E-1**			**TOTAL**		**1.2E-1**
hexachlorobenzene (HCB)		1.6E-04	30	3.0E-06	7.0E-06	--	4.4E-2	30	2.5E-08	1.8E-06	--	1.1E-2
a-HCH (a-BHC)		8.0E-03	600	6.0E-05	1.4E-04	--	1.8E-2	600	5.0E-07	3.5E-05	--	4.4E-3
b-HCH (b-BHC)		2.0E-05	20	2.0E-06	4.7E-06	--	2.3E-1	20	1.7E-08	1.2E-06	--	5.9E-2
g-HCH (g-BHC or lindane)		3.0E-03	1000	1.0E-04	2.3E-04	--	7.8E-2	1000	8.4E-07	5.9E-05	--	2.0E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	900	9.1E-05	2.1E-04	--	1.1E-1	900	7.5E-07	5.3E-05	--	2.6E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

Identified Soil Zone 7

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL		**2.0E-1**			**TOTAL**		**1.1E-1**
hexachlorobenzene (HCB)		1.6E-04	30	1.3E-06	3.0E-06	--	1.9E-2	30	2.2E-08	1.6E-06	--	9.8E-3
a-HCH (a-BHC)		8.0E-03	600	2.5E-05	5.9E-05	--	7.4E-3	600	4.5E-07	3.1E-05	--	3.9E-3
b-HCH (b-BHC)		2.0E-05	20	8.5E-07	2.0E-06	--	9.9E-2	20	1.5E-08	1.0E-06	--	5.2E-2
g-HCH (g-BHC or lindane)		3.0E-03	1000	4.2E-05	9.9E-05	--	3.3E-2	1000	7.5E-07	5.2E-05	--	1.7E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	900	3.8E-05	8.9E-05	--	4.5E-2	900	6.7E-07	4.7E-05	--	2.4E-2
			↑ RBSC Derived					↑ RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 7**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*FE*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL		**2.6E-1**			**TOTAL**		**1.3E-1**
hexachlorobenzene (HCB)		1.6E-04	0.10	30	7.1E-06	1.7E-05	--	1.0E-1	30	1.2E-07	8.7E-06	--	5.5E-2
a-HCH (a-BHC)		8.0E-03	0.01	600	1.4E-05	3.3E-05	--	4.1E-3	600	2.5E-07	1.7E-05	--	2.2E-3
b-HCH (b-BHC)		2.0E-05	0.01	20	4.7E-07	1.1E-06	--	5.5E-2	20	8.3E-09	5.8E-07	--	2.9E-2
g-HCH (g-BHC or lindane)		3.0E-03	0.01	1000	2.4E-05	5.5E-05	--	1.8E-2	1000	4.2E-07	2.9E-05	--	9.7E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	900	6.4E-05	1.5E-04	--	7.4E-2	900	1.1E-06	7.9E-05	--	3.9E-2
				RBSC Derived					RBSC Derived				

ISZ 8

Risk Based Soil Concentrations
ISZ 8

COPC	RBSC	Calculated Risk					
		Ingestion		dermal contact		Total Risk	
	(mg/kg)	non-threshold	threshold	non-threshold	threshold	non-threshold	threshold
hexachlorobenzene (HCB)	15		0.022		0.052		0.074
1,2-dichloroethane (EDC)	10	1.2E-08		5.1E-09		1.7E-08	
chloroform	45		0.0016		0.00069		0.0023
trichloroethene (TCE)	15		0.00015		0.000062		0.00021
tetrachloroethene (PCE)	650		0.017		0.0070		0.023
benzene	25	8.8E-08		3.7E-08		1.3E-07	
toluene	200		0.00024		0.00010		0.00034
PAHs (BaP equivalent)	40	2.0E-06		6.1E-06		8.2E-06	
1,4-dichlorobenzene	200		0.0044		0.0019		0.0062
a-HCH (a-BHC)	500		0.015		0.0062		0.021
b-HCH (b-BHC)	8		0.094		0.040		0.13
d-HCH (d-BHC)	5		0.059		0.025		0.083
g-HCH (g-BHC or lindane)	400		0.031		0.013		0.045
dieldrin	50		0.12		0.050		0.17
DDX (sum of DDT, DDE and DDD)	400		0.047		0.033		0.080
pentachlorophenol	15		0.0012		0.0069		0.0081
mercury (as total, inroganic)	75		0.050		0.021		0.071

8.3E-06	**0.64**	**Total Risk from Soil Ingestion and Dermal Contact**
3.8E-07	**0.22**	**Total Risk from Inhalation Exposures**
2.0E-06	**0.40**	**Allowable Risk via Inhalation**
1.0E-05	**1.0**	**Total Risk**

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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	**2.1E-6**	**4.6E-1**			**TOTAL**	**1.8E-8**	**1.1E-1**
hexachlorobenzene (HCB)		1.6E-04	15	1.5E-06	3.5E-06	--	2.2E-2	15	1.3E-08	8.8E-07	--	5.5E-3
1,2-dichloroethane (EDC)	1.2E-02		10	1.0E-06	2.3E-06	1.2E-8	--	10	8.4E-09	5.9E-07	1.0E-10	--
chloroform		6.5E-03	45	4.5E-06	1.1E-05	--	1.6E-3	45	3.8E-08	2.6E-06	--	4.1E-4
trichloroethene (TCE)		2.4E-02	15	1.5E-06	3.5E-06	--	1.5E-4	15	1.3E-08	8.8E-07	--	3.7E-5
tetrachloroethene (PCE)		9.2E-03	650	6.5E-05	1.5E-04	--	1.7E-2	650	5.5E-07	3.8E-05	--	4.1E-3
benzene	3.5E-02		25	2.5E-06	5.9E-06	8.8E-8	--	25	2.1E-08	1.5E-06	7.3E-10	--
toluene		2.0E-01	200	2.0E-05	4.7E-05	--	2.4E-4	200	1.7E-07	1.2E-05	--	5.9E-5
PAHs (BaP equivalent)	5.0E-01		40	4.0E-06	9.4E-06	2.0E-6	--	40	3.4E-08	2.3E-06	1.7E-8	--
1,4-dichlorobenzene		1.1E-02	200	2.0E-05	4.7E-05	--	4.4E-3	200	1.7E-07	1.2E-05	--	1.1E-3
a-HCH (a-BHC)		8.0E-03	500	5.0E-05	1.2E-04	--	1.5E-2	500	4.2E-07	2.9E-05	--	3.7E-3
b-HCH (b-BHC)		2.0E-05	8	8.1E-07	1.9E-06	--	9.4E-2	8	6.7E-09	4.7E-07	--	2.3E-2
d-HCH (d-BHC)		2.0E-05	5	5.0E-07	1.2E-06	--	5.9E-2	5	4.2E-09	2.9E-07	--	1.5E-2
g-HCH (g-BHC or lindane)		3.0E-03	400	4.0E-05	9.4E-05	--	3.1E-2	400	3.4E-07	2.3E-05	--	7.8E-3
dieldrin		1.0E-04	50	5.0E-06	1.2E-05	--	1.2E-1	50	4.2E-08	2.9E-06	--	2.9E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	400	4.0E-05	9.4E-05	--	4.7E-2	400	3.4E-07	2.3E-05	--	1.2E-2
pentachlorophenol		3.0E-03	15	1.5E-06	3.5E-06	--	1.2E-3	15	1.3E-08	8.8E-07	--	2.9E-4
mercury (as total, inroganic)		3.6E-04	75	7.5E-06	1.8E-05	--	5.0E-2	75	6.3E-08	4.4E-06	--	1.2E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 8

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**8.9E-7**	**1.9E-1**			**TOTAL**	**1.6E-8**	**1.0E-1**
hexachlorobenzene (HCB)		1.6E-04	15	6.4E-07	1.5E-06	--	9.3E-3	15	1.1E-08	7.9E-07	--	4.9E-3
1,2-dichloroethane (EDC)	1.2E-02		10	4.2E-07	9.9E-07	5.1E-9	--	10	7.5E-09	5.2E-07	9.0E-11	--
chloroform		6.5E-03	45	1.9E-06	4.5E-06	--	6.9E-4	45	3.4E-08	2.4E-06	--	3.6E-4
trichloroethene (TCE)		2.4E-02	15	6.4E-07	1.5E-06	--	6.2E-5	15	1.1E-08	7.9E-07	--	3.3E-5
tetrachloroethene (PCE)		9.2E-03	650	2.8E-05	6.4E-05	--	7.0E-3	650	4.9E-07	3.4E-05	--	3.7E-3
benzene	3.5E-02		25	1.1E-06	2.5E-06	3.7E-8	--	25	1.9E-08	1.3E-06	6.5E-10	--
toluene		2.0E-01	200	8.5E-06	2.0E-05	--	1.0E-4	200	1.5E-07	1.0E-05	--	5.3E-5
PAHs (BaP equivalent)	5.0E-01		40	1.7E-06	4.0E-06	8.5E-7	--	40	3.0E-08	2.1E-06	1.5E-8	--
1,4-dichlorobenzene		1.1E-02	200	8.5E-06	2.0E-05	--	1.9E-3	200	1.5E-07	1.0E-05	--	9.8E-4
a-HCH (a-BHC)		8.0E-03	500	2.1E-05	5.0E-05	--	6.2E-3	500	3.7E-07	2.6E-05	--	3.3E-3
b-HCH (b-BHC)		2.0E-05	8	3.4E-07	7.9E-07	--	4.0E-2	8	6.0E-09	4.2E-07	--	2.1E-2
d-HCH (d-BHC)		2.0E-05	5	2.1E-07	5.0E-07	--	2.5E-2	5	3.7E-09	2.6E-07	--	1.3E-2
g-HCH (g-BHC or lindane)		3.0E-03	400	1.7E-05	4.0E-05	--	1.3E-2	400	3.0E-07	2.1E-05	--	7.0E-3
dieldrin		1.0E-04	50	2.1E-06	5.0E-06	--	5.0E-2	50	3.7E-08	2.6E-06	--	2.6E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	400	1.7E-05	4.0E-05	--	2.0E-2	400	3.0E-07	2.1E-05	--	1.0E-2
pentachlorophenol		3.0E-03	15	6.4E-07	1.5E-06	--	5.0E-4	15	1.1E-08	7.9E-07	--	2.6E-4
mercury (as total, inroganic)		3.6E-04	75	3.2E-06	7.4E-06	--	2.1E-2	75	5.6E-08	3.9E-06	--	1.1E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 8**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06**	**NonThreshold**	**4.2E-08**	**NonThreshold**
		5.5E-06	**Threshold**	**2.9E-06**	**Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**6.2E-6**	**1.8E-1**			**TOTAL**	**1.1E-7**	**9.7E-2**
hexachlorobenzene (HCB)		1.6E-04	0.10	15	3.5E-06	8.3E-06	--	5.2E-2	15	6.2E-08	4.4E-06	--	2.7E-2
1,2-dichloroethane (EDC)	1.2E-02		0.01	10	2.4E-07	5.5E-07	2.8E-9	--	10	4.2E-09	2.9E-07	5.0E-11	--
chloroform		6.5E-03	0.01	45	1.1E-06	2.5E-06	--	3.8E-4	45	1.9E-08	1.3E-06	--	2.0E-4
trichloroethene (TCE)		2.4E-02	0.01	15	3.5E-07	8.3E-07	--	3.5E-5	15	6.2E-09	4.4E-07	--	1.8E-5
tetrachloroethene (PCE)		9.2E-03	0.01	650	1.5E-05	3.6E-05	--	3.9E-3	650	2.7E-07	1.9E-05	--	2.0E-3
benzene	3.5E-02		0.01	25	5.9E-07	1.4E-06	2.1E-8	--	25	1.0E-08	7.3E-07	3.6E-10	--
toluene		2.0E-01	0.01	200	4.7E-06	1.1E-05	--	5.6E-5	200	8.3E-08	5.8E-06	--	2.9E-5
PAHs (BaP equivalent)	5.0E-01		0.13	40	1.2E-05	2.9E-05	6.1E-6	--	40	2.2E-07	1.5E-05	1.1E-7	--
1,4-dichlorobenzene		1.1E-02	0.01	200	4.7E-06	1.1E-05	--	1.0E-3	200	8.3E-08	5.8E-06	--	5.4E-4
a-HCH (a-BHC)		8.0E-03	0.01	500	1.2E-05	2.8E-05	--	3.4E-3	500	2.1E-07	1.5E-05	--	1.8E-3
b-HCH (b-BHC)		2.0E-05	0.01	8	1.9E-07	4.4E-07	--	2.2E-2	8	3.3E-09	2.3E-07	--	1.2E-2
d-HCH (d-BHC)		2.0E-05	0.01	5	1.2E-07	2.8E-07	--	1.4E-2	5	2.1E-09	1.5E-07	--	7.3E-3
g-HCH (g-BHC or lindane)		3.0E-03	0.01	400	9.4E-06	2.2E-05	--	7.3E-3	400	1.7E-07	1.2E-05	--	3.9E-3
dieldrin		1.0E-04	0.01	50	1.2E-06	2.8E-06	--	2.8E-2	50	2.1E-08	1.5E-06	--	1.5E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	400	2.8E-05	6.6E-05	--	3.3E-2	400	5.0E-07	3.5E-05	--	1.7E-2
pentachlorophenol		3.0E-03	0.25	15	8.9E-06	2.1E-05	--	6.9E-3	15	1.6E-07	1.1E-05	--	3.6E-3
mercury (as total, inroganic)		3.6E-04	0.01	75	1.8E-06	4.1E-06	--	1.2E-2	75	3.1E-08	2.2E-06	--	6.1E-3
				RBSC Derived					RBSC Derived				

ISZ 9

Risk Based Soil Concentrations
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COPC	RBSC	Calculated Risk					
		Ingestion		dermal contact		Total Risk	
	(mg/kg)	non-threshold	threshold	non-threshold	threshold	non-threshold	threshold
tetrachloroethene (PCE)	5		0.00013		0.000054		0.00018
benzene	10	3.5E-08		1.5E-08		5.0E-08	
PAHs (BaP equivalent)	40	2.0E-06		6.1E-06		8.2E-06	
a-HCH (a-BHC)	500		0.015		0.0062		0.021
b-HCH (b-BHC)	20		0.23		0.099		0.33
g-HCH (g-BHC or lindane)	500		0.039		0.017		0.056
dieldrin	50		0.12		0.050		0.17
DDX (sum of DDT, DDE and DDD)	800		0.094		0.066		0.16
endosulfan II	1200		0.052		0.022		0.074

8.2E-06	**0.81**	**Total Risk from Soil Ingestion and Dermal Contact**
3.8E-08	**0.00015**	**Total Risk from Inhalation Exposures**
2.0E-06	**0.20**	**Allowable Risk via Inhalation**
1.0E-05	**1.0**	**Total Risk**

Exposure to Chemicals via Ingestion of Soils

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**2.0E-6**	**5.5E-1**			**TOTAL**	**1.7E-8**	**1.4E-1**
tetrachloroethene (PCE)		9.2E-03	5	5.0E-07	1.2E-06	--	1.3E-4	5	4.2E-09	2.9E-07	--	3.2E-5
benzene	3.5E-02		10	1.0E-06	2.3E-06	3.5E-8	--	10	8.4E-09	5.9E-07	2.9E-10	--
PAHs (BaP equivalent)	5.0E-01		40	4.0E-06	9.4E-06	2.0E-6	--	40	3.4E-08	2.3E-06	1.7E-8	--
a-HCH (a-BHC)		8.0E-03	500	5.0E-05	1.2E-04	--	1.5E-2	500	4.2E-07	2.9E-05	--	3.7E-3
b-HCH (b-BHC)		2.0E-05	20	2.0E-06	4.7E-06	--	2.3E-1	20	1.7E-08	1.2E-06	--	5.9E-2
g-HCH (g-BHC or lindane)		3.0E-03	500	5.0E-05	1.2E-04	--	3.9E-2	500	4.2E-07	2.9E-05	--	9.8E-3
dieldrin		1.0E-04	50	5.0E-06	1.2E-05	--	1.2E-1	50	4.2E-08	2.9E-06	--	2.9E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	800	8.1E-05	1.9E-04	--	9.4E-2	800	6.7E-07	4.7E-05	--	2.3E-2
endosulfan II		5.4E-03	1200	1.2E-04	2.8E-04	--	5.2E-2	1200	1.0E-06	7.0E-05	--	1.3E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

Identified Source Zone 9

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**8.6E-7**	**2.3E-1**			**TOTAL**	**1.5E-8**	**1.2E-1**
tetrachloroethene (PCE)		9.2E-03	5	2.1E-07	5.0E-07	--	5.4E-5	5	3.7E-09	2.6E-07	--	2.8E-5
benzene	3.5E-02		10	4.2E-07	9.9E-07	1.5E-8	--	10	7.5E-09	5.2E-07	2.6E-10	--
PAHs (BaP equivalent)	5.0E-01		40	1.7E-06	4.0E-06	8.5E-7	--	40	3.0E-08	2.1E-06	1.5E-8	--
a-HCH (a-BHC)		8.0E-03	500	2.1E-05	5.0E-05	--	6.2E-3	500	3.7E-07	2.6E-05	--	3.3E-3
b-HCH (b-BHC)		2.0E-05	20	8.5E-07	2.0E-06	--	9.9E-2	20	1.5E-08	1.0E-06	--	5.2E-2
g-HCH (g-BHC or lindane)		3.0E-03	500	2.1E-05	5.0E-05	--	1.7E-2	500	3.7E-07	2.6E-05	--	8.7E-3
dieldrin		1.0E-04	50	2.1E-06	5.0E-06	--	5.0E-2	50	3.7E-08	2.6E-06	--	2.6E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	800	3.4E-05	7.9E-05	--	4.0E-2	800	6.0E-07	4.2E-05	--	2.1E-2
endosulfan II		5.4E-03	1200	5.1E-05	1.2E-04	--	2.2E-2	1200	9.0E-07	6.3E-05	--	1.2E-2
			↑ RBSC Derived					↑ RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment - **ISZ 9**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06**	**NonThreshold**	**4.2E-08**	**NonThreshold**
		5.5E-06	**Threshold**	**2.9E-06**	**Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal 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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**6.1E-6**	**1.7E-1**			**TOTAL**	**1.1E-7**	**9.2E-2**
tetrachloroethene (PCE)		9.2E-03	0.01	5	1.2E-07	2.8E-07	--	3.0E-5	5	2.1E-09	1.5E-07	--	1.6E-5
benzene	3.5E-02		0.01	10	2.4E-07	5.5E-07	8.3E-9	--	10	4.2E-09	2.9E-07	1.5E-10	--
PAHs (BaP equivalent)	5.0E-01		0.13	40	1.2E-05	2.9E-05	6.1E-6	--	40	2.2E-07	1.5E-05	1.1E-7	--
a-HCH (a-BHC)		8.0E-03	0.01	500	1.2E-05	2.8E-05	--	3.4E-3	500	2.1E-07	1.5E-05	--	1.8E-3
b-HCH (b-BHC)		2.0E-05	0.01	20	4.7E-07	1.1E-06	--	5.5E-2	20	8.3E-09	5.8E-07	--	2.9E-2
g-HCH (g-BHC or lindane)		3.0E-03	0.01	500	1.2E-05	2.8E-05	--	9.2E-3	500	2.1E-07	1.5E-05	--	4.9E-3
dieldrin		1.0E-04	0.01	50	1.2E-06	2.8E-06	--	2.8E-2	50	2.1E-08	1.5E-06	--	1.5E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	800	5.7E-05	1.3E-04	--	6.6E-2	800	1.0E-06	7.0E-05	--	3.5E-2
endosulfan II		5.4E-03	0.01	1200	2.8E-05	6.6E-05	--	1.2E-2	1200	5.0E-07	3.5E-05	--	6.5E-3
				↑ RBSC Derived					↑ RBSC Derived				

ISZ 10

Risk Based Soil Concentrations
ISZ 10

COPC	RBSC (mg/kg)	Calculated Risk					
		Ingestion		dermal contact		Total Risk	
		non-threshold	threshold	non-threshold	threshold	non-threshold	threshold
benzene	5	1.8E-08		7.4E-09		2.5E-08	
xylenes	200		0.00027		0.00011		0.00038
PAHs (BaP equivalent)	40	2.0E-06		6.1E-06		8.2E-06	
a-HCH (a-BHC)	150		0.0044		0.0019		0.0063
b-HCH (b-BHC)	23		0.27		0.11		0.38
g-HCH (g-BHC or lindane)	150		0.012		0.0050		0.017
DDX (sum of DDT, DDE and DDD)	2000		0.23		0.17		0.40

8.2E-06	**0.81**	**Total Risk from Soil Ingestion and Dermal Contact**
2.0E-06	**0.20**	**Allowable Risk via Inhalation**
1.0E-05	**1.0**	**Total Risk**

Exposure to Chemicals via Ingestion of Soils

Pharmaceutical Building - **ISZ 10**

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Soils by Workers		Exposure Calculations (RME) Ingestion of Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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*CFEF*ED / BW*AT	kg/kg/day	**1.0E-07**	**NonThreshold**	**8.4E-10**	**NonThreshold**
		2.3E-07	**Threshold**	**5.9E-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	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	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	**2.0E-6**	**5.2E-1**			**TOTAL**	**1.7E-8**	**1.3E-1**
benzene	3.5E-02		5	5.0E-07	1.2E-06	1.8E-8	--	5	4.2E-09	2.9E-07	1.5E-10	--
xylenes		1.8E-01	200	2.0E-05	4.7E-05	--	2.7E-4	200	1.7E-07	1.2E-05	--	6.7E-5
PAHs (BaP equivalent)	5.0E-01		40	4.0E-06	9.4E-06	2.0E-6	--	40	3.4E-08	2.3E-06	1.7E-8	--
a-HCH (a-BHC)		8.0E-03	150	1.5E-05	3.5E-05	--	4.4E-3	150	1.3E-07	8.8E-06	--	1.1E-3
b-HCH (b-BHC)		2.0E-05	23	2.3E-06	5.4E-06	--	2.7E-1	23	1.9E-08	1.4E-06	--	6.8E-2
g-HCH (g-BHC or lindane)		3.0E-03	150	1.5E-05	3.5E-05	--	1.2E-2	150	1.3E-07	8.8E-06	--	2.9E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	2000	2.0E-04	4.7E-04	--	2.3E-1	2000	1.7E-06	1.2E-04	--	5.9E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

Pharmaceutical Building - **ISZ 10**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = $\frac{SAs*AF*Abs*ET*ME*CF*EF*ED}{BW*AT}$	kg-hr/kg/day	**4.2E-08** **9.9E-08**	**NonThreshold** **Threshold**	**7.5E-10** **5.2E-08**	**NonThreshold** **Threshold**

Daily Intake from Soil = Concentration in Soil x Dermal Absorption 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	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**8.6E-7**	**2.2E-1**			**TOTAL**	**1.5E-8**	**1.2E-1**
benzene	3.5E-02		5	2.1E-07	5.0E-07	7.4E-9	--	5	3.7E-09	2.6E-07	1.3E-10	--
xylenes		1.8E-01	200	8.5E-06	2.0E-05	--	1.1E-4	200	1.5E-07	1.0E-05	--	6.0E-5
PAHs (BaP equivalent)	5.0E-01		40	1.7E-06	4.0E-06	8.5E-7	--	40	3.0E-08	2.1E-06	1.5E-8	--
a-HCH (a-BHC)		8.0E-03	150	6.4E-06	1.5E-05	--	1.9E-3	150	1.1E-07	7.9E-06	--	9.8E-4
b-HCH (b-BHC)		2.0E-05	23	9.8E-07	2.3E-06	--	1.1E-1	23	1.7E-08	1.2E-06	--	6.0E-2
g-HCH (g-BHC or lindane)		3.0E-03	150	6.4E-06	1.5E-05	--	5.0E-3	150	1.1E-07	7.9E-06	--	2.6E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	2000	8.5E-05	2.0E-04	--	9.9E-2	2000	1.5E-06	1.0E-04	--	5.2E-2
			RBSC Derived					RBSC Derived				

Dermal Exposure to Chemicals via Contact with Soil

US Methodology - Sensitivity Assessment PB Bldg - **ISZ 10**

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Soils by Workers		Exposure Calculations (RME) Dermal Contact with Soils by Intrusive/Excavation Workers	
Exposure Parameters					
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays	60	Assume intrusive works for 60 days of the year
Exposure Duration (ED)	years	30	Duration of exposure as per NEPM 1999 and USEPA 1989	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	10950	USEPA 1989 and CSMS 1996	365	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)
Adherance 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
Intake Factor = SAs*AF*FE*ME*CF*EF*ED / BW*AT	kg-hr/kg/day	**2.4E-06** **5.5E-06**	**NonThreshold** **Threshold**	**4.2E-08** **2.9E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability 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 and Dermal Data			Concentration	Daily Intake		Calculated Risk		Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	Dermal Absorption	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient	in Soil	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)	(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**6.1E-6**	**2.3E-1**			**TOTAL**	**1.1E-7**	**1.2E-1**
benzene	3.5E-02		0.01	5	1.2E-07	2.8E-07	4.1E-9	--	5	2.1E-09	1.5E-07	7.3E-11	--
xylenes		1.8E-01	0.01	200	4.7E-06	1.1E-05	--	6.3E-5	200	8.3E-08	5.8E-06	--	3.3E-5
PAHs (BaP equivalent)	5.0E-01		0.13	40	1.2E-05	2.9E-05	6.1E-6	--	40	2.2E-07	1.5E-05	1.1E-7	--
a-HCH (a-BHC)		8.0E-03	0.01	150	3.5E-06	8.3E-06	--	1.0E-3	150	6.2E-08	4.4E-06	--	5.5E-4
b-HCH (b-BHC)		2.0E-05	0.01	23	5.4E-07	1.3E-06	--	6.3E-2	23	9.6E-09	6.7E-07	--	3.3E-2
g-HCH (g-BHC or lindane)		3.0E-03	0.01	150	3.5E-06	8.3E-06	--	2.8E-3	150	6.2E-08	4.4E-06	--	1.5E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.03	2000	1.4E-04	3.3E-04	--	1.7E-1	2000	2.5E-06	1.7E-04	--	8.7E-2
				↑ RBSC Derived					↑ RBSC Derived				

Appendix H

Derivation of Risk-Based Groundwater Concentrations (off-site)

RBGC

COPC	RBGC (ug/L)	RBGC (mg/L)	Calculated Risk - Workers Inhalation Indoors		Calculated Risk - Intrusive Workers Direct Contact		Inhalation in Excavation		Total Risk	
			Non-threshold	Threshold	Non-threshold	Threshold	Non-threshold	Threshold	Non-threshold	Threshold
hexachlorobenzene (HCB)	60	0.06				0.10			0.0E+00	0.10
1,2-dichloroethane (EDC)	120000	120	1.9E-09		9.1E-08		1.9E-06	0.0024	2.0E-06	0.0024
vinyl chloride	6000	6	2.5E-09		1.6E-06		3.0E-07	0.00037	1.9E-06	0.00037
chloroform	100000	100	3.0E-08	0.0024		0.10	2.4E-07	0.0080	2.4E-07	0.11
trichloroethene (TCE)	200000	200	1.1E-06			0.10	5.2E-07	0.0032	5.2E-07	0.10
tetrachloroethene (PCE)	30000	30		0.0057		0.10		0.00008	0.0E+00	0.10
1,1,2-trichloroethane	3500	3.5		0.10		0.0045		0.00010	0.0E+00	0.0046
chlorobenzene	200000	200		0.10		0.063			0.0E+00	0.063
benzene	50000	50	8.3E-07		3.6E-07		1.7E-06	0.013	2.1E-06	0.013
toluene	6000	6		0.10		0.00092		0.00024	0.0E+00	0.0012
a-HCH (a-BHC)	44000	44				0.10			0.0E+00	0.10
b-HCH (b-BHC)	110	0.11				0.10			0.0E+00	0.10
d-HCH (d-BHC)	110	0.11				0.10			0.0E+00	0.10
g-HCH (g-BHC or lindane)	16000	16				0.10			0.0E+00	0.10
dieldrin	330	0.33				0.10		0.0063	0.0E+00	0.10
DDX (sum of DDT, DDE and DDD)	300	0.3				0.10			0.0E+00	0.10
		Total	**2E-06**	**0.3**				Total	**7E-06**	**1**

Review of Soil Gas Data and Shallow Groundwater Data
Locations on and off the site above groundwater impacts only (where possible)

	Maximum reported cocnentrations from monitoring data colected to 2008 - roughly paired soil gas and shallow groundwater data																		
COPC	**AS10/25**	**OS01/ OS08**	**Ratio SG:GW**	**AS8**	**BP101/ OS02**	**Ratio SG:GW**	**AS9**	**MW14/ MW6/ BP205**	**Ratio SG:GW**	**AS12/13/ 27**	**OS04/ OS05/ OS10**	**Ratio SG:GW**	**AS3**	**MW43/ MW31/ MW44**	**Ratio SG:GW**	**All off-site Subslab**	**OS03/ OS09**	**Ratio SG:GW**	**Maximum Ratio**
	µg/m^3	(ug/L)	(ug/m3 per ug/L)	µg/m^3	µg/L	(ug/m3 per ug/L)	µg/m^3	µg/L	(ug/m3 per ug/L)	µg/m^3	µg/L	(ug/m3 per ug/L)	µg/m^3	µg/L	(ug/m3 per ug/L)	µg/m^3	µg/L	(ug/m3 per ug/L)	(ug/m3 per ug/L)
depth (m)	1	2.3-3.5		1	6		1	5.1000		1.0000	2.9		1	3.3-3.7		0.15	3.5		
1,2-dichloroethane (EDC)							160	16100.0000	9.94E-03	25	82500	3.03E-04	1	5020	1.99E-04	1	2800	3.57E-04	0.010
vinyl chloride										2.6	30	8.67E-02							0.087
chloroform		19		130	100	1.30E+00	77	690.0000	1.12E-01	80	140	5.71E-01	7.7	23	3.35E-01	21	3870	5.43E-03	1.3
trichloroethene (TCE)				5.30	1940	0.0027	36	9820.00000	3.67E-03	111665.0000	3750	2.98E+01	1	240	4.17E-03	1.9	3130	6.07E-04	30
tetrachloroethene (PCE)	3.5	100	3.50E-02	3589	100	35.890	15	34	4.41E-01	80.000	9	8.89E+00	16	100	1.60E-01	18	100	1.80E-01	36
1,1,2-trichloroethane	42000	100	4.20E+02				1	11.000	9.09E-02	229.000	35	6.54E+00	13	100	1.30E-01				420
chlorobenzene	5	5240	9.54E-04	5	54900	0.00009	1	913.00000	1.10E-03	2529.0000	200	1.26E+01	8631	31	2.78E+02	26	11600	2.24E-03	278
benzene	1.6	41	3.90E-02	4.7	3800	1.24E-03	3.7	4510	8.20E-04				6	1	6.00E+00	3.6	1990	1.81E-03	6.0
toluene	12000	100	1.20E+02	16949	356	4.76E+01	53	100	5.30E-01	10967	3	3.66E+03	8035	100	8.04E+01	62	5	1.24E+01	3656

100 Not detected - value presented is LOR
0.087 Maximum ratio based on LOR for soil gas, hence maximum ratio may be an overestimate
1.3 Maximum ratio based on on LOR for shallow groundwater, hence ratio may be an underestimate

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

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	1	Calculated from layers
Soil Temperature	[C]	T	16.0000	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	1	Average depth to soil gas sample
Moisture Content	[g/g]	mocon	0.1200	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	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 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

Vapour Transport Calculations for COPC Identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
vinyl chloride	5.58E-4			5.58E-4
chloroform	5.52E-4			5.52E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
tetrachloroethene (PCE)	3.80E-4			3.80E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
chlorobenzene	3.89E-4			3.89E-4
benzene	4.66E-4			4.66E-4
toluene	4.60E-4			4.60E-4

Calculated Air Concentrations	RBGC (ug/L) (A)	Maximum SG:GW Volatilisation Factor (ug/m3 per ug/L) (B)	Vapour Phase Concentration at 1m depth (= A x B) (ug/m^3)	Vapour Phase Concentration at 1m depth (units conversion) (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)
1,2-dichloroethane (EDC)	120000	9.94E-03	1.19E+03	1.2E-09	6.7E-11	**4.0E-06**
vinyl chloride	6000	8.67E-02	5.20E+02	5.2E-10	2.9E-11	**1.7E-06**
chloroform	100000	1.30E+00	1.30E+05	1.3E-07	7.2E-09	**4.3E-04**
trichloroethene (TCE)	200000	2.98E+01	5.96E+06	6.0E-06	2.5E-07	**1.5E-02**
tetrachloroethene (PCE)	30000	3.59E+01	1.08E+06	1.1E-06	4.1E-08	**2.5E-03**
1,1,2-trichloroethane	3500	4.20E+02	1.47E+06	1.5E-06	6.2E-08	**3.7E-03**
chlorobenzene	200000	2.78E+02	5.57E+07	5.6E-05	2.2E-06	**1.3E-01**
benzene	50000	6.00E+00	3.00E+05	3.0E-07	1.4E-08	**8.4E-04**
toluene	6000	3.66E+03	2.19E+07	2.2E-05	1.0E-06	**6.1E-02**

Inhalation of Volatile Chemicals

Offsite Areas Above GW Plumes

General Data/ Equations	Units	Exposure Calculations (RME) Indoors (based on RBGCs)	
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	1.17	Inhalation rate indoors
Exposure Time (ET)	hours/day	10	Assume 10 hours spent indoors
Bioavailability (B)	-	100%	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume all time spent at inside building located above plume
Intake Factor = $\frac{InhR*ET*B*FI*EF*ED}{BW*AT}$	m^3/kg/day	**4.7E-02** **1.1E-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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.9E-6**	**3.1E-1**
1,2-dichloroethane (EDC)	9.8E-03		4.0E-06	1.9E-07	4.4E-07	1.9E-9	--
vinyl chloride	3.1E-02		1.7E-06	8.2E-08	1.9E-07	2.5E-9	--
chloroform	1.5E-03	2.0E-02	4.3E-04	2.0E-05	4.7E-05	3.0E-8	2.4E-3
trichloroethene (TCE)	1.5E-03		1.5E-02	7.0E-04	1.6E-03	1.1E-6	--
tetrachloroethene (PCE)		4.7E-02	2.5E-03	1.2E-04	2.7E-04	--	5.7E-3
1,1,2-trichloroethane		4.0E-03	3.7E-03	1.8E-04	4.1E-04	--	1.0E-1
chlorobenzene		1.4E-01	1.3E-01	6.1E-03	1.4E-02	--	1.0E-1
benzene	2.1E-02		8.4E-04	4.0E-05	9.2E-05	8.3E-7	--
toluene		6.7E-02	6.1E-02	2.9E-03	6.7E-03	--	1.0E-1

Calculated from RBGCs, ratio of measured SG:GW and modelling from SG to indoor air

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 from GW

Site Specific Physical Input Parameters	Units	Abbrev.	Value	Comments
Thickness of Vadose Zone	[m]	vd	0.01	Calculated from layers
Soil Temperature	[C]	T	16.0000	Assumed
Vadose Zone Layer 1 Characteristics				***Silty Clays***
Depth of Layer 1	[m]	vd1	0.01	Assume excavation to depth of soil gas data
Moisture Content	[g/g]	mocon	0.1200	Conservative value for silty clays
Organic Carbon Fraction	-	foc	0.01	Assumed for silty clays
Soil Bulk Density	[g/ml]	rhob	1.8	Assumed for silty clays
Density of Solids	[g/ml]	sd	2.65	default
Total Soil Porosity	[ml/ml]	theta	0.32	1 - (rhob/sd)
Volumetric Water Content	[ml/ml]	wacon	0.216	mocon*rhob
Volumetric Air Content	[ml/ml]	acon	0.105	theta-wacon

Receptor Specific Input Parameters	Units	Abbrev.	Value	Comments
Outdoor Air Characteristics				
Length of Contaminated Area	[m]	length	1	Assumed size of excavation
Width of Contaminated Area	[m]	width	10	Assumed size of excavation
Wind Speed Outdoors	[m/s]	wspd	0.5	Low wind speed in excavation
Height of Outdoor Mixing Zone	[m]	outboxh	1.5	Mixing zone for excavation

Vapour Transport Calculations for COPC identified	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)
1,2-dichloroethane (EDC)	5.62E-4			5.62E-4
trichloroethene (TCE)	4.17E-4			4.17E-4
1,1,2-trichloroethane	4.25E-4			4.25E-4
toluene	4.60E-4			4.60E-4
1,2,3-trichloropropane	4.07E-4			4.07E-4
naphthalene	3.33E-4			3.33E-4

Calculated Air Concentrations	Vapour Phase Concentration at 1m depth (based on SG;GW ratio and RBGCs) (ug/m^3)	Vapour Phase Concentration at 1m deoth (units conversion) (g/cm^3)	Calculated Emission Rate from Surface of Ground ($g/s/m^2$)	Measured Emission Rate from Surface of Ground ($g/s/m^2$)	Enclosed Space Concentration Farmer (mg/m^3)	Air Concentration in Excavation - no seepage (mg/m^3)	Air Concentration in Excavation - with seepage (mg/m^3)
1,2-dichloroethane (EDC)	1.19E+03	1.2E-09	6.7E-09			**8.9E-05**	**1.6E+01**
vinyl chloride	5.20E+02	5.2E-10	2.9E-09			**3.9E-05**	**8.0E-01**
chloroform	1.30E+05	1.3E-07	7.2E-07			**9.6E-03**	**1.3E+01**
trichloroethene (TCE)	5.96E+06	6.0E-06	2.5E-05			**3.3E-01**	**2.7E+01**
tetrachloroethene (PCE)	1.08E+06	1.1E-06	4.1E-06			**5.5E-02**	**4.0E+00**
1,1,2-trichloroethane	1.47E+06	1.5E-06	6.2E-06			**8.3E-02**	**4.7E-01**
chlorobenzene	5.57E+07	5.6E-05	2.2E-04			**2.9E+00**	**2.7E+01**
benzene	3.00E+05	3.0E-07	1.4E-06			**1.9E-02**	**6.7E+00**
toluene	2.19E+07	2.2E-05	1.0E-04			**1.3E+00**	**8.0E-01**

Calculated based on RBGCs and a VF of 0.133 (mg/m3 per mg/L GW) where GW seeps into the excavation (assumed to occur for some of the time only)

Inhalation of Volatile Chemicals (Evaluated using Occupational TWA Values)

Intrusive Workers - Off-Site GW

General Data/ Equations	Units	Exposure Calculations (RME) Intrusive Worker in Excavation	
Exposure Parameters			
Exposure Frequency (EF)	days/year	30	Assume groundwater intersected for half the time (total 60 days)
Exposure Duration (ED)	years	1	Assume works occur over a 1 year period
Body Weight (BW)	kg	70.0000	USEPA 1989 and CSMS 1996
Averaging Time - NonThreshold (ATc)	days	25550.00000	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	365.0000	USEPA 1989 and CSMS 1996
Inhalation Rate (InhR)	m^3/hr	2.2000	Inhalation rate equivalent to an adult walking at 6.4 km/hr, enHealth 2003
Exposure Time (ET)	hours/day	2.00000	Assume groundwater in excavation with groundwater seppage for 2 hours each day
Bioavailability (B)	-	1.00000	Assume 100% bioavailability via inhalation
Fraction Inhaled (FI)	-	100%	Assume 100% of time outdoors is spent above plume
Intake Factor = InhR*ET*B*FI*EF*ED / BW*AT	m^3/kg/day	**0.000**	**NonThreshold**
		0.00517	**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	
	Non-Threshold Slope Factor	Threshold TWA	in Air	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/m^3)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**3.1E-2**	**4.2E-2**
hexachlorobenzene (HCB)		2.9E-04				--	--
1,2-dichloroethane (EDC)	9.8E-03	5.7E+00	2.66E+00	2.0E-04	1.4E-02	1.9E-6	2.4E-3
vinyl chloride	3.1E-02	1.9E+00	0.1330	9.8E-06	6.9E-04	3.0E-7	3.7E-4
chloroform	1.5E-03	1.4E+00	2.22E+00	1.6E-04	1.1E-02	2.4E-7	8.0E-3
trichloroethene (TCE)	1.5E-03	7.7E+00	4.71E+00	3.5E-04	2.4E-02	5.2E-7	3.2E-3
tetrachloroethene (PCE)		4.8E+01	7.10E-01	5.2E-05	3.7E-03	--	7.7E-5
1,1,2-trichloroethane		7.9E+00	1.47E-01	1.1E-05	7.6E-04	--	9.7E-5
chlorobenzene		6.6E+00	6.84E+00	5.0E-04	3.5E-02	--	5.4E-3
benzene	2.1E-02	4.6E-01	1.12E+00	8.3E-05	5.8E-03	1.7E-6	1.3E-2
toluene		2.7E+01	1.25E+00	9.3E-05	6.5E-03	--	2.4E-4
a-HCH (a-BHC)		1.4E-02				--	--
b-HCH (b-BHC)		1.4E-02				--	--
d-HCH (d-BHC)		1.4E-02				--	--
g-HCH (g-BHC or lindane)		1.4E-02				--	--
DDX (sum of DDT, DDE and DDD)		1.4E-01				--	--
cis-1,2-dichloroethene		1.1E+02				--	--

Caculated based on groundwater seepage for 5 days, and no seepage for 25 days

Exposure to Chemicals via Incidental Ingestion of Water

Offsite GW

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Water by Intrusive/Excavation Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	5	Assume direct contact with goundwater occurs infrequently
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 in excavation
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**1.4E-08** **9.8E-07**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water 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	
	Non-Threshold Slope Factor	Threshold ADI, TDI or RfD	in Water	NonThreshold	Threshold	NonThreshold Risk	Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
					TOTAL	**1.4E-6**	**5.6E-2**
hexachlorobenzene (HCB)		1.6E-04	0.06	8.4E-10	5.9E-08	--	3.7E-4
1,2-dichloroethane (EDC)	1.2E-02		120	1.7E-06	1.2E-04	2.0E-8	--
vinyl chloride	2.3E+00		6	8.4E-08	5.9E-06	1.9E-7	--
chloroform		6.5E-03	100	1.4E-06	9.8E-05	--	1.5E-2
trichloroethene (TCE)		2.4E-02	200	2.8E-06	2.0E-04	--	8.2E-3
tetrachloroethene (PCE)		9.2E-03	30	4.2E-07	2.9E-05	--	3.2E-3
1,1,2-trichloroethane		4.0E-03	3.5	4.9E-08	3.4E-06	--	8.6E-4
chlorobenzene		8.6E-02	200	2.8E-06	2.0E-04	--	2.3E-3
benzene	3.5E-02		50	7.0E-07	4.9E-05	2.4E-8	--
toluene		2.0E-01	6	8.4E-08	5.9E-06	--	3.0E-5
a-HCH (a-BHC)		8.0E-03	44	6.2E-07	4.3E-05	--	5.4E-3
b-HCH (b-BHC)		2.0E-05	0.11	1.5E-09	1.1E-07	--	5.4E-3
d-HCH (d-BHC)		2.0E-05	0.11	1.5E-09	1.1E-07	--	5.4E-3
g-HCH (g-BHC or lindane)		3.0E-03	16	2.2E-07	1.6E-05	--	5.2E-3
dieldrin		1.0E-04	0.33	4.6E-09	3.2E-07	--	3.2E-3
DDX (sum of DDT, DDE and DDD)		2.0E-03	0.3	4.2E-09	2.9E-07	--	1.5E-4

RBGCs

Dermal Exposure to Chemicals via Contact with Water

Offsite GW

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with water by Intrusive/Excavation Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	5	Assume direct contact with goundwater occurs infrequently
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	365.0000	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	3300	Exposed durface area of construction worker as per USEPA 2001
Exposure Time (ET)	hr/day	2.0000	Assume contact with water for 2 hours per day
Conversion Factor (CF)	L/cm^3	0.00	Conversion of units
Intake Factor = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**9.2E-06** **6.5E-04**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability 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: Non-Threshold Slope Factor	Toxicity Data: Threshold ADI, TDI or RfD	Toxicity Data: Dermal Permeability	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
						TOTAL	**1.8E-6**	**1.1E+0**
hexachlorobenzene (HCB)		1.6E-04	4.1E-1	0.06	2.3E-07	1.6E-05	--	9.9E-2
1,2-dichloroethane (EDC)	1.2E-02		5.3E-3	120	5.9E-06	4.1E-04	7.0E-8	--
vinyl chloride	2.3E+00		1.1E-2	6	6.3E-07	4.4E-05	1.4E-6	--
chloroform		6.5E-03	8.9E-3	100	8.2E-06	5.7E-04	--	8.8E-2
trichloroethene (TCE)		2.4E-02	1.6E-2	200	3.0E-05	2.1E-03	--	8.7E-2
tetrachloroethene (PCE)		9.2E-03	4.8E-2	30	1.3E-05	9.3E-04	--	1.0E-1
1,1,2-trichloroethane		4.0E-03	6.4E-3	3.5	2.1E-07	1.4E-05	--	3.6E-3
chlorobenzene		8.6E-02	4.1E-2	200	7.5E-05	5.3E-03	--	6.1E-2
benzene	3.5E-02		2.1E-2	50	9.5E-06	6.7E-04	3.3E-7	--
toluene		2.0E-01	4.5E-2	6	2.5E-06	1.8E-04	--	8.9E-4
a-HCH (a-BHC)		8.0E-03	2.8E-2	44	1.1E-05	7.9E-04	--	9.9E-2
b-HCH (b-BHC)		2.0E-05	2.8E-2	0.11	2.8E-08	2.0E-06	--	9.9E-2
d-HCH (d-BHC)		2.0E-05	2.8E-2	0.11	2.8E-08	2.0E-06	--	9.9E-2
g-HCH (g-BHC or lindane)		3.0E-03	2.8E-2	16	4.1E-06	2.9E-04	--	9.6E-2
dieldrin		1.0E-04	4.5E-2	0.33	1.4E-07	9.5E-06	--	9.5E-2
DDX (sum of DDT, DDE and DDD)		2.0E-03	1.0E+0	0.3	2.8E-06	1.9E-04	--	9.7E-2

RBGCs