12 Toxicity Assessment

This section of the report provides toxicity assessments for the contaminants that are the subject of the site-specific risk assessment, as identified in Section 11. The assessment forms the third step in the site-specific risk assessment for ground contamination at the Former Gasworks site. The methodology that has been used was previously described in Section 2.4. These contaminants comprise PAHs (Section 12.1), benzene (Section 12.2), toluene (Section 12.3), ethylbenzene (Section 12.4), xylenes (Section 12.5) and petroleum hydrocarbons (Section 12.6).

12.1 PAHs

General

The NHMRC (2004) *"Australian Drinking Water Guidelines"* and the WHO (2000) *"Air Quality Guidelines for Europe"* advise that PAHs are widespread throughout the environment. They are formed in forest fires and in the combustion of fossil fuels and are present in emissions from coke ovens, gasworks, aluminium smelters and motor vehicles.

Food is the major source of intake of PAHs. Highest concentrations occur in smoked foods, leafy vegetables and the burnt fat of metals. Intake from foods is extremely variable but significantly higher than from drinking water.

Toxicology

Most of the toxicological literature deals specifically with benzo(a)pyrene (BaP). Few studies are available for the other PAHs. Some PAHs have been found to be carcinogenic by non-oral routes, but others are known to have low potential for carcinogenicity. Estimates of the relative potency of PAH indicator compounds are provided by the WHO INCHEM (1998) environmental health study and more recently by Fitzgerald (1998), which are summarised in **Table 30**.

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- Table 30 Toxicity Equivalence Factors for PAHs

Compound	Range of Relative Potencies
Benzo(a)pyrene	1
Dibenz(a,h)anthracene	4
Benz(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.1
Indo(1,2,3-c,d)pyrene	0.1
Anthracene	0.001
Benzo(g,h,i)perylene	0.1
Chrysene	0.1
Acenaphthene	0.001
Acenaphthylene	0.001
Fluoranthene	0.01
Fluorene	0.001
Naphthalene	0.001
Phenanthrene	0.001
Pyrene	0.001

Reference: Fitzgerald (1998)

BaP is absorbed principally through the gastrointestinal tract and the lungs. The rate of absorption increases with increased intake of polyunsaturated fatty acids. BaP is rapidly distributed to the organs and may be stored in mammary and adipose tissue. Metabolism occurs mainly in the liver.

The International Agency for Research on Cancer (IARC) has concluded that BaP is probably carcinogenic to humans (Group 2A).

Human Health Criteria

The WHO or Australian health authorities have not published health criteria that specify acceptable intakes for PAH substances. Recourse has therefore been made to the USEPA (2002) PRGs, which provide health criteria for PAHs in terms of Reference Doses for non-carcinogenic contaminants and Slope Factors (SF) for carcinogenic contaminants.

A Reference Dose (RfD) is defined (NEPC, 1999a) as *"an estimate (with uncertainty factors spanning perhaps an order of magnitude) of the daily exposure (mg/kg body weight/day) to the general human population (including sensitive sub-groups) that is likely to be without an appreciable risk of deleterious effects during a life time of exposure. It is derived from the NOAEL or the LOAEL by application of uncertainty factors that reflect various types of data used to*

estimate RfD and an additional modifying factor, which is based on professional judgement of the entire data base of the chemical (IRIS, 1996)".

RfDs are provided in units of mg/kg body weight-day while SFs are provided in 1/(mg/kg body weight-day).

The US EPA PRGs provide RfDs and SFs for ingestion (oral) soil (RfDo) and inhalation (RfDi) together with skin absorption factors for soil. A summary of these RfD/SF values for individual PAH compounds that are included in the US EPA PRG assessment is provided in **Table 31**.

- Table 31 US EPA Reference Doses & Slope Factors for PAHs

Compound	SFo 1/(mg/kg-day)	SFi 1/(mg/kg-day)	Skin Absorption Factor
Benzo(a)pyrene	7.3E+00		0.13
Dibenz(a,h)anthracene	7.3E+00		0.13
Benz(a)anthracene	7.3E-01		0.13
Benzo(b)fluoranthene	7.3E-01		0.13
Benzo(k)fluoranthene	7.3E-02		0.13
Indeno(1,2,3-c,d)pyrene	7.3E-01		
Chrysene	7.3E-03		0.13
Compound	**RfDo (mg/kg/day)**	**RFDi (mg/kg/day)**	**Skin Absorption Factor**
Anthracene	3.0E-01	3.0E-01	
Acenaphthene	6.0E-02	6.0E-02	
Acenaphthylene			
Fluoranthene	4.0E-02	4.0E-02	0.13
Fluorene	4.0E-02	4.0E-02	
Naphthalene	2.0E-02	8.6E-04	
Phenanthrene			
Pyrene	3.0E-02	3.0E-02	
PAHs (general)			0.13

A study by Fitzgerald (1998) that appears in the Fourth National Workshop on the Health Risk Assessment and Management of Contaminated Sites in Australia developed a guideline dose for BaP using actual tumour dose-response data from laboratory studies. The study recommended a guideline dose of 9.5μg/day for a 70kg adult and 1.8μg/day for a 2.5 year old child (13.2kg).

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Ecological Health

The ANZECC (1992) guidelines report that concentrations of PAHs in aquatic ecosystems are generally highest in sediments, intermediate in aquatic biota and lowest in the water column. Studies have found that sorption to suspended particles and bed sediments is the primary removal mechanism for high-molecular weight PAHs, whereas volatilisation and transport were the primary mechanisms for low-molecular weight PAHs. Mixed microbial population in sediment water systems may degrade some PAHs, with degradation progressively decreasing with increasing molecular weight.

PAHs are known to affect survival, growth, metabolism and initiate tumour formation in many organisms. In birds and mammals, effects include reduced embryo survival, development effects, mutagenesis, carcinogenesis, and teratogenesis. Aquatic invertebrates (particularly crustaceans) are sensitive to PAHs. Sublethal effects include inhibited reproduction and emergence.

In fish, PAHs may be tumorigenic. However, most fish rapidly metabolise and excrete PAHs, thus limiting toxicity.

12.2 Benzene

General

The NHMRC (2004) *"Australian Drinking Water Guidelines"* and the WHO (2000) *"Air Quality Guidelines for Europe"* advise that benzene is present in petrol, motor vehicle emissions, is used as a cleaning solvent and is a by-product of the coal gasification process. When released to surface waters, benzene rapidly volatilises to the air. It is a colourless liquid at room temperature with a density of 0.87 g/cm^3 at 20°C.

Inhalation accounts for more than 99% of the exposure of the general population, whereas intake from food and water is minimal.

Toxicology

Benzene is rapidly and efficiently absorbed and widely distributed throughout the body. It is metabolised predominantly into phenol by the liver and also be bone marrow.

Human health data are mainly from studies where benzene had been inhaled. Exposure to high concentrations in air can cause death. Lower concentrations can induce toxic effects, with white blood cells being most sensitive. There is considerable evidence that occupational exposure to low benzene concentrations for periods as short as 12 months may result in leukaemia.

In animal studies, benzene caused leukaemia and other cancers when administered orally and by inhalation to rats and mice. It can also induce chromosome damage and gene mutation in mammalian cells. It was not found to be mutagenic in tests with bacteria.

The most significant adverse effects from prolonged exposure to benzene are haematotoxicity, genotoxicity and carcinogenicity. IARC (1987) has concluded that benzene is carcinogenic to humans (Group 1).

Human Health Criteria

Benzene is a genotoxic human carcinogen and there is no safe or acceptable concentration for it in drinking water. The NHMRC (2004) drinking water guideline value of 0.001mg/L is based on a recommendation made by the WHO that this concentration in drinking water would entail a maximum lifetime risk of one extra case of leukaemia per million people.

Reference Doses and Slope Factors values provided by the US EPA (2002) PRGs for benzene are:

- SFo = 5.5E-02 (mg/kg body wt-day) $^{-1}$
- RfDo = 3.0E-03 mg/kg body wt-day
- SFi = 2.9E-02 (mg/kg body wt-day) $^{-1}$
- RfDi = 1.7E-03 mg/kg body wt-day

12.3 Toluene

General

Toluene is an aromatic hydrocarbon. The NHMRC (2004) *"Australian Drinking Water Guidelines"* and the WHO (1986) *"INCHEM Environmental Health Criteria 52"* advise that toluene occurs naturally as a component of crude oil and is present in petrol. Toluene is produced in large quantities during petroleum refining and is a by-product in the manufacture of styrene and coke-oven preparations. It also occurs in natural gas and emissions from volcanoes, forest fires and cigarettes.

The general population is exposed to toluene mainly through inhalation of vapour in ambient air, cigarette smoking and to a minor extent, by ingestion of food or water contaminated with toluene.

Toxicology

In humans, toluene is readily absorbed from the gastrointestinal tract after ingestion and is distributed preferentially in adipose tissue, then the kidneys, liver and brain. It is rapidly metabolised by the liver to benzl alcohol, benzoic acid and to a lesser extent phenols.

Data on human health effects come mainly from inhalation studies. The predominant effects of acute exposure were impairment of the CNS and irritation of the mucous membranes, with fatigue and drowsiness being the most obvious symptoms.

Rats exposed to toluene vapour for 2 years exhibited decreased blood haeatocrit values at high toluene concentrations. No data are available on long-term oral toxicity. However, a 13-week gavage study using rats and mice reported increased liver weights at doses from 625 mg/kg body weight per day.

Toluene generally did not exhibit genotoxic activity in tests on bacteria, yeast cells and mammalian cells in vitro. IARC (www.monographs.iarc.fr) has concluded that toluene is not classifiable as to its carcinogenicity in humans (Group 3).

Human Health Criteria

The NHMRC (2004) guidelines advise that the no effect level for toluene, based on a 13-week oral study using rats, is 312 mg/kg body weight per day. The NHMRC (2004) drinking water guideline value is 0.8mg/L, which includes a safety factor of 1000 using the results of the animal study as a basis for human exposure. This health-based value exceeds the taste threshold of 0.025mg/L for toluene in water.

Reference Doses and Slope Factors values provided by the US EPA (2002) PRGs for toluene are:

- SFo = not specified
- RfDo = 2.0E-01 mg/kg body wt-day
- SFi = not specified
- RfDi = 1.1E-01 mg/kg body wt-day

12.4 Ethylbenzene

General

Ethylbenzene is an aromatic hydrocarbon. The NHMRC (2004) *"Australian Drinking Water Guidelines"* and the WHO (1996) *"INCHEM Environmental Health Criteria 186"* advise that ethylbenzene occurs naturally as a component of crude oil and is present in petrol in small quantities. It is also used commercially in paints, insecticides and is a constituent of coal tars, asphalt and naphtha. Ethylbenzene is a non-persistent chemical, being degraded primarily by photo-oxidation and biodegradation. Volatilization to the atmosphere is rapid. It is a colourless liquid at room temperature with a sweet gasoline-like odour. It is lighter than water and has a density of 0.866g/cm^3 at 25°C.

Human exposure to ethylbenzene occurs mainly by inhalation; 40-60% of inhaled ethylbenzene is retained in the lung.

Toxicology

Ethylbenzene is readily absorbed from the human gastrointestinal tract. It can be stored in fat and is extensively metabolised, mainly to mandelic and phenylglyoxylic acids and excreted in the urine. It can cross the placenta. No data are available on the health effects in humans after oral exposure and inhalation data are limited to short-term studies.

A 6-month gavage study using rats reported enlargement of the liver and kidney at high doses. Liver effects were also observed in a number of inhalation studies. No long-term studies are available.

Studies on the mutagenic activity of ethylbenzene to bacteria, insects and mammalian cells have reported negative results. IARC (www.monographs.iarc.fr) has concluded that ethylbenzene is possibly carcinogenic to humans (Group 2B).

Human Health Criteria

Ethylbenzene has low acute and chronic toxicity for both animals and humans. It is toxic to the central nervous system and is an irritant of mucous membranes and the eyes. The WHO (1996) report that the threshold for these effects in humans after short single exposures was estimated to be about 430-860 mg/m3 (100-200ppm).

The NHMRC (2004) guidelines advise that the no effect level for ethylbenzene, based on a 6-month gavage study using rats, is 136mg/kg body weight per day. The NHMRC (2004) drinking water guideline value is 0.3mg/L, which includes a safety factor of 1000 using the results of the animal study as a basis for human exposure. This health-based value exceeds the taste threshold of 0.003mg/L for ethylbenzene in water.

Reference Doses and Slope Factors values provided by the US EPA (2002) PRGs for ethylbenzene are:

- SFo = 3.85E-03 (mg/kg body wt-day) $^{-1}$
- RfDo = 1.0E-01 mg/kg body wt-day
- SFi = 3.85E-03 (mg/kg body wt-day) $^{-1}$
- RfDi = 2.9E-01 mg/kg body wt-day

12.5 Xylenes

General

Xylene is an aromatic hydrocarbon which exists in three isomeric forms: ortho, meta and para. The NHMRC (2004) *"Australian Drinking Water Guidelines"* and the WHO (1997) *"INCHEM Environmental Health Criteria 186"* advise that xylenes occur as a component of crude oil and are present in petrol, but in small quantities. It is also produced in the petroleum refining process and is used in the manufacture of insecticides, pharmaceuticals, detergents, paints, adhesives and other products. Xylenes are readily biodegraded in surface waters and they volatilise to air very quickly. It is a colourless liquid at room temperature with an aromatic odour. It is lighter than water and has a density of between 0.860 and 0.876g/cm^3 at 25°C.

The majority of xylene released into the environment enters the atmosphere directly. In the atmosphere the xylene isomers are readily degraded, primarily by photooxidation. Volatilisation to the atmosphere from water is rapid for all three isomers. In soil and water, the meta and para isomers are readily biodegraded under a wide range of aerobic and anaerobic conditions, but the ortho isomer is more persistent. The limited evidence available suggests that bioaccumulation of the xylene isomers by fish and invertebrates is low.

Toxicology

Xylenes are readily absorbed after inhalation and metabolised almost completely to methyl benzoic acid. More than 90% is biotransformed to methylhippuric acid, which is excreted in urine. Xylene does not accumulate significantly in the human body. It can cross the placenta. No data are available on human absorption after ingestion, or on health effects of oral exposure in humans.

Acute exposure to high concentrations of xylene can result in CNS effects and irritation in humans. However, there have been no long-term controlled human studies or epidemiological studies. The chronic toxicity appears to be relatively low in laboratory animals. There is suggestive evidence, however, that chronic CNS effects may occur in animals at moderate concentrations of xylene. A 2-year gavage study using rats and mice reported decreased growth at high doses but no xylene-related lesions.

There was no evidence of carcinogenicity in oral and skin administration studies using rats and mice, and xylenes were not mutagenic in tests using bacteria and mammalian cells. IARC (www.monographs.iarc.fr) has concluded that xylene is not classifiable as to carcinogenicity in humans (Group 3).