

# 4

## Generic soil acceptance criteria

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# Generic soil acceptance criteria

## 4.1 Introduction

This module covers the following:

- development of generic health-based soil acceptance criteria
- ecological considerations
- aesthetic considerations
- health effect summaries for selected gasworks contaminants

Additional information on the generic soil acceptance can be found in Section 4 of the Users' Guide, including:

- ▲ land uses (Section 4.2.1)
- ▲ hazard identification (Section 4.2.2)
- ▲ exposure assessment (Section 4.2.3)
- ▲ toxicity assessment (Section 4.2.4)
- ▲ risk characterisation (Section 4.2.5)
- ▲ derivation of generic soil acceptance criteria (Section 4.2.6)
- ▲ summary of the generic soil acceptance criteria (Section 4.2.7)
- ▲ ecological considerations (Section 4.2.8)
- ▲ aesthetic considerations (Section 4.2.9)
- ▲ application of generic soil acceptance criteria (Section 4.2.10)
- ▲ development of site-specific acceptance criteria (Section 4.4)

## 4.2 Development of generic health-based soil acceptance criteria

In developing soil acceptance criteria reference has been made to the information and methodologies from a range of sources, including:

- exposure assessment equations developed by the USEPA, particularly USEPA (1991) "Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part B, Development of Preliminary Remediation Goals"
- exposure factor information agreed in the developing of previous industry-based guidelines in New Zealand and information presented in other sources such as Langley (1993, 1996) and by the USEPA and WHO
- toxicological information and dose response factors established in New Zealand in the New Zealand Drinking Water Standards, and information presented by the WHO and USEPA
- precedents established in New Zealand regarding the level of acceptable risk.

Information on the toxicity and dose response factors for contaminants of concern at gasworks sites are presented in Appendix 5A.

### 4.2.1 Land uses

Health-based generic soil acceptance criteria are derived for the following land uses:

- Agricultural/Horticultural
- Standard Residential (50% of produce home grown)
- Standard Residential (10% of produce home grown)
- High Density Residential
- Commercial/Industrial
- Parkland/Recreational

## 4.2.2 Hazard identification

### 4.2.2.1 *Contaminants of concern*

See Section 4.2.2.1 of the Users' Guide for information on the contaminants of concern.

### 4.2.2.2 *Receptors*

See Section 4.2.2.2 of the Users' Guide for information on receptors.

## 4.2.3 Exposure assessment

The exposure assessment in risk assessment is a measure of the likely exposure of the receptors (site users). Exposure assessment involves:

- identification of complete exposure pathways
- estimation of contaminant concentrations in media in which receptors may be exposed
- estimation of dose likely to be experienced by each receptor

The overall approach adopted for exposure assessment when deriving the generic acceptance criteria is based on the USEPA protocol for the development of preliminary remediation goals (USEPA, 1991). This is consistent with the approach used for the development of soil acceptance criteria for the timber industry (MfE/MoH, 1993). The exposure factors adopted for the derivation of the acceptance criteria have been modified to reflect New Zealand conditions and policy. In addition, some of the fate and transport modelling components of this section differ from the approach adopted by the USEPA for the development of preliminary remediation goals.

Because of the importance of the inhalation of volatiles in deriving criteria for BTEX and other volatile contaminants found at gasworks sites, particular attention has been given to modelling the emission of volatiles from contaminated soil. As volatilisation depends on soil properties, assumptions have been made regarding soil properties and depth to the contamination. The generic acceptance criteria have been based on a sandy loam soil, with criteria developed for surface soil (<1 m) and subsurface soil (>1m).

Exposure assessment depends on assumptions about a range of exposure factors. In practice, there is uncertainty regarding the value of many exposure factors (e.g. the quantity of soil ingested by children), whereas other exposure factors vary through the population (e.g. body weight). Conservative assumptions are mainly used to account for this uncertainty and variability, thus ensuring protection of public health.

The use of conservative point estimates in calculations involving many such parameters, however, can result in a compounding conservatism. Further, information on the level of conservatism inherent in the acceptance criteria is lost. **Probabilistic techniques such as Monte Carlo analysis may be used to improve the assessment of uncertainty. These techniques have not been used routinely in the development of generic criteria to date although the potential exists for this in the future.**

The impact of soil contamination on groundwater quality is best assessed by direct measurement of groundwater quality and assessment in accordance with the principles set out in Module 3. Therefore soil acceptance criteria based on the protection of groundwater quality have not been developed.

#### **4.2.3.1 Exposure pathway analysis**

Soil contamination only poses a risk to a receptor, if there is a complete pathway between the source of contamination and the receptor. Where the exposure pathway is incomplete there is no risk.

An exposure pathway consists of the following elements:

- a source and mechanism for release
- storage and/or transport media
- an exposure point, where the receptor comes in contact with the contamination, and
- an exposure route (e.g. inhalation).

For example, where a former gasworks site is redeveloped for residential use, some relevant exposure pathways are likely to include:

- ingestion of contaminated soil that may be exposed in the vicinity of the house
- consumption of home grown produce, and
- inhalation of volatiles, particularly benzene, in indoor air as a result of soil contamination beneath the building.

Inhalation of particulates is a complete exposure pathway, but in most circumstances the contribution of this pathway to the overall exposure is negligible. The exception is exposure scenarios involving high concentrations of suspended particulates, limited exposure via other routes, and contaminants exhibiting low volatility and significantly higher toxicity via the inhalation route, (e.g. arsenic, hexavalent chromium). None of the contaminants considered in deriving acceptance criteria satisfy these conditions. On this basis, exposure via inhalation of particulates has not been considered further.

See Section 4.2.3.1 of the Users' Guide for the table of exposure pathways.

#### **4.2.3.2 Exposure concentration estimation**

Many of the contaminants found at gasworks sites are relatively mobile in the soil and exposure may occur by contact with media other than that originally contaminated. To derive acceptance criteria to protect human health, it is necessary to find the relationship between contaminant concentrations in soil and those in other media to which site users may be exposed. Estimating contaminant concentrations at the point of exposure is one of the most critical elements of the risk assessment, and a source of uncertainty.

To determine contaminant concentrations at the point of exposure it is necessary to either directly measure contaminant concentrations at the relevant point, or predict the fate and transport of contaminants. Clearly, direct measurement is preferred in most cases, but, often this is not possible or practical (e.g. houses have not yet been built on the former gasworks site). For most initial site assessments, it is assumed that contaminant concentrations will be measured in soil and groundwater, but not in other media such as ambient air or produce.

As part of the development of acceptance criteria, an estimate of the relationship between contaminant concentrations in different media is required for the following exposure pathways:

- **Inhalation of volatiles**

An estimate of the contaminant concentration in indoor air and outdoor air, based on the concentration in soil is required.

- **Consumption of home grown produce**

An estimate of the uptake of contaminants by produce, based on the contaminant concentrations in soil, is required.

### ***Volatilisation***

The relationship between contaminant concentrations in air within the breathing zone indoors and outdoors, and the concentration in soil is described using the Volatilisation Factor (VF), which is defined as follows:

$$\text{VF} = \text{Concentration in air (mg/m}^3\text{)} / \text{Concentration in soil (mg/kg)}$$

The Volatilisation Factor is a function of soil and contaminant properties, the depth and thickness of contamination and the building or outdoor air characteristics. Modelling the transport of volatile contaminants from soil to indoor and outdoor air is one of the most important factors in deriving the acceptance criteria for volatile contaminants, such as benzene. A range of models have been developed for assessing the transport of volatile contaminants, however considerable uncertainty remains and development work in this area continues. The fate and transport of volatile contaminants in the subsurface is complex, involving a wide range of processes, few of which are well understood. Most of the available models consider only a small subset of the fate and transport processes which actually occur, and are based on simplified conceptual models of contamination (e.g. uniform contaminant concentrations through the contaminated zone).

Limited validation of the volatilisation models suggest they significantly over predict the transport of contaminants to indoor or outdoor air, although further work is required to determine the reasons for this.

### ***Plant uptake***

The primary concern with the uptake of contaminants by plants is the presence of contaminants in produce consumed by humans. The relationship between contaminant concentrations in soils and edible plant materials is highly specific to the specific plant species. The relationship between contaminant concentrations in edible produce and the concentration in soil is described using the Plant Uptake Factor (PUF), which is defined as follows:

$$\text{PUF} = \frac{\text{Concentration in edible portion of plant (mg/kg)}}{\text{Concentration in soil (mg/kg)}}$$

A range of published correlations between plant and soil concentrations are available. Most correlations are empirical, assuming a linear relationship between the plant and soil concentrations, and defining the ratio between the plant and soil concentrations in terms of  $K_{ow}$  or  $K_{oc}$  and the organic carbon content of the soil.

The available plant uptake models usually overestimate the concentration of many gasworks related contaminants for the following reasons:

- most hydrocarbons are readily degraded in the soil, particularly under conditions favouring biological activity such as those found in vegetable gardens (e.g. regular watering, fertiliser)
- significant losses by volatilisation are expected to occur within a period of, for example, a year
- enhanced degradation of contaminants may be expected in the plant root zone, and

- the depth range of most interest in a vegetable garden context is the upper 200 to 300 mm, where losses by volatilisation and other mechanisms are likely to be most pronounced.

As acceptance criteria have been based on long term exposure to contamination (e.g. 30 years for carcinogenic contaminants), less weight has been attached to criteria based on plant uptake and consumption of home grown produce, given that the derivation assumes constant soil concentrations.

Information on the uptake of inorganic chemicals is limited and the standard correlations used for organic chemicals do not apply. At gasworks sites cyanide is generally present as complex cyanide which has relatively low bioavailability and therefore uptake by plants is expected to be limited. Because of this, plant uptake has not been considered in deriving criteria for cyanide.

#### **4.2.3.3 Exposure estimation**

Generic acceptance criteria for the protection of human health, have been based on an estimate of the reasonable maximum exposure (RME) for a particular scenario (USEPA, 1989a). The RME combines upper bound and average exposure factors so that the result represents an exposure scenario that is both protective and reasonable. It is not the absolute worst case but represents a reasonable maximum exposure. (USEPA, 1991b).

The approach to exposure assessment and the development of health-based acceptance criteria is based on the procedures developed by the USEPA (1989, 1992). Assumptions employed in the risk assessment are based on recommendations by the USEPA (1989a, 1990b, 1991b, 1991d), information presented in Langley (1993, 1996) and precedents established in similar guidance for the timber industry (MfE/MoH, 1993).

The estimated exposure (or intake) is normalised for time and body weight and is generally calculated as:

$$\text{Intake} = \frac{\text{Concentration} \times \text{Contact Rate} \times \text{Exposure Frequency} \times \text{Exposure Duration}}{\text{Body Weight} \times \text{Averaging Time}}$$

The above equation may be rearranged to give health-based acceptance criteria on a route-specific basis as follows:

$$\text{Acceptance criteria} = \frac{\text{Acceptable Intake} \times \text{Body Weight} \times \text{Averaging Time}}{\text{Contact Rate} \times \text{Exposure Frequency} \times \text{Exposure Duration}}$$

Where **Acceptable Intake = (Proportion of RfD assigned to contaminated soil) x (Reference Dose (RfD))**

The above equation may be further rearranged to account for multiple exposure routes.

#### **4.2.3.4 Exposure factors**

The exposure factors adopted for developing screening criteria are consistent with those adopted in the revised "Health and Environmental Guidelines for Selected Timber Treatment Chemicals" and are in accordance with Ministry of Health policy.

For developing soil screening criteria for agricultural and residential land use, two age groups have been considered:

- Adults
- Children (1 to 6 years)

In a residential use, children and adults may live at a given site. and children may often spend the majority of their childhood at one residence. Consequently it is assumed that the exposure period begins when the child is a toddler and continues through childhood to adult life. Therefore, adult exposure may notionally be considered to correspond to 6 to 30 years of age.

The establishment of criteria based on exposure from 6 months to 30 years will also protect adults exposed for 30 yrs. For those contaminants for which a non-threshold dose response model has been adopted, the lifetime average daily dose relevant for risk assessment reflects a weighted mean of childhood and adult exposures. Where a threshold dose response model

has been adopted, a year-averaged exposure is used to determine acceptance criteria, with children the limiting receptor group. The exposure parameters for children reflect those of a 2-year-old child as soil ingestion is generally greatest at this time, whereas the exposure parameters for residents greater than 7 years old reflect those for adults.

Exposure via most of the pathways considered in deriving acceptance criteria is assumed to be constant with time i.e. contaminant concentrations do not decrease with time. This approach results in a significant over-estimate of exposure in the case of inhalation of volatiles, as depletion of the contaminated soil results in decreasing indoor and outdoor air concentrations with time. It is therefore necessary to determine average indoor and outdoor air concentrations, based on an assumed averaging time.

See Section 4.2.3.4 of the Users' Guide for the summary table of exposure factors.

**4.2.3.5 Agricultural**

**Protection of human health**

The major exposure assumptions are summarised below based on published typical average and upper bound values:

- exposure duration = 30 yrs, assuming exposure from 0 to 30 yrs of age, 6 years as child, 24 years as an adult.

The exposure duration is based on the reasonable maximum time spent on the one site in a rural context based on USEPA (1989).

- exposure frequency = 350 d/y (USEPA, 1989b)

Studies have shown that a child is likely to spend less than 200 days/year playing outside. However, Hawley (1985) estimated that 80% of indoors dirt is derived from local soil, meaning a child may be exposed whenever they are on-site, not just outdoors.

- body weight:
 

child (1-6 yrs)	= 15kg	(USEPA, 1992)
adult (7-30 yrs)	= 70kg	(ANZECC, 1992)
- soil ingestion rate:
 

child (1-6 yrs)	= 100mg/d	(ANZECC, 1992)
adult (7-30 yrs)	= 25mg/d	
- inhalation rate:
 

child (1-6 yrs)	= 3.8m <sup>3</sup> /d	(Langley, 1993)
adult (7-30 yrs)	= 22m <sup>3</sup> /d	
- exposed skin surface area:
 

child (1-6 yrs)	= 2625 cm <sup>2</sup>	(Langley, 1993)
adult (7-30 yrs)	= 4700 cm <sup>2</sup>	
- soil adherence:
 

1 mg/cm <sup>2</sup> allowing for soil contact typical of farming activities	(USEPA, 1988)
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- ingestion of produce:
 

child (1-6 yrs)	= 0.13kg/d	(Langley, 1989b)
adult (7-30 yrs)	= 0.45kg/d	
- proportion of produce grown on site = 100% (MoH, 1995)

The assumed garden produce ingestion rates are based on the average daily consumption of fruit and vegetables derived from national dietary surveys, as presented in Langley (1993).

Dermal exposure is defined by the duration and frequency of exposure, body weight, the adherence of soil to exposed skin, the area of skin exposed, and the skin absorption factor. Soil adherence values consistent with those adopted in previous New Zealand guidelines were adopted as a default, although uncertainty remains.

The absorption of contaminants through skin is uncertain, particularly where contaminants are applied in the form of a soil mixture. Published information was reviewed in order to

develop estimates for the skin absorption factors as follows (ASTM, 1994, USEPA, 1992, GRI, 1988):

- **Standard Residential/Agricultural/Horticultural**

PAHs	1 %
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BTEX, Phenolics	5%
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- **Parkland/Recreational**

PAHs	0.5%
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BTEX, Phenolics	2.5%
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- **Commercial/Industrial**

PAHs	0.6 %
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BTEX, Phenolics	3 %
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The assumed values take into account the matrix effects associated with application of contaminants in soil. Higher values have been reported for BTEX compounds, but most reported information does not account for losses by volatilisation from a thin film of soil in skin, and therefore lower values may be justified.

### Protection of plant and livestock

The impact of ground contamination on plant life and livestock may involve the following factors:

- protection of human health of residents who may consume produce
- protection of plant life — phytotoxicity
- maintenance of acceptable levels of contaminants in produce and livestock for sale.

The suitability of fruit and vegetable produce for human consumption may be assessed by comparing predicted produce concentrations with published Maximum Residue Limits (MRL). In the absence of MRLs for most of the contaminants of concern, the suitability of produce for human consumption may be assessed using health risk assessment techniques assuming 100% of produce consumed is from a contaminated source.

Livestock (e.g. cattle, sheep, poultry) may be exposed to contaminants in soil and contaminants may accumulate in edible portions of livestock, increasing exposure of consumers. In practice the organic contaminants of concern at gasworks sites, although lipophilic, are readily metabolised and therefore are unlikely to accumulate at significant levels in livestock. In contrast, many chlorinated organics are not readily metabolised and may accumulate within livestock. Cyanide and complex cyanide are not lipophilic and are less likely to accumulate. On this basis, exposure via the consumption of livestock products where livestock have been reared on contaminated land is unlikely to be a significant route of exposure and therefore criteria have not been derived for this pathway.

Criteria developed for the protection of human health in an agricultural context are expected to broadly protect livestock health, based on consideration of:

- the higher soil consumption/body weight ratio for cattle and other livestock compared to humans
- the shorter lifespan of livestock reducing concern associated with cancer and points, and
- a lower level of protection (i.e. not all sensitive individuals protected) required in the case of livestock.

Information on the protection of plant life is limited for most of the contaminants of concern at gasworks sites.

**4.2.3.6 Residential**

Soil guidelines have been developed on the basis of reasonable maximum exposure assumptions. The major exposure assumptions are summarised below:

- exposure duration = 30 years, assuming exposure from 0 to 30 years of age; 6 years as a ‘child’, 24 years as an ‘adult’.

The exposure duration is based on the reasonable maximum time spent on the one site in a rural residential context based on USEPA (1989).

- exposure frequency = 350 days/year (USEPA, 1989 b)

Studies have shown that a child is likely to spend less than 200 days/year playing outside, however, Hawley (1985) estimated that 80% of indoors’ diet is derived from local soil meaning a child may be exposed wherever on site, not just outdoors.

- body weight:
 

child (1-6 yrs)	=15 kg	(USEPA, 1992)
adult (7-30 yrs)	=70 kg	(ANZECC, 1992)
- soil ingestion rate:
 

child (1-6 yrs)	=100 mg/d	(ANZECC, 1992)
adult (7-30 yrs)	=25 mg/d	
- inhalation rate:
 

child (1-6 yrs)	=3.8 m <sup>3</sup> /d	(Langley, 1993)
adult (7-30 yrs)	=20 m <sup>3</sup> /d outdoors	(ASTM, 1994)
	15 m <sup>3</sup> /d indoors	
- exposed skin surface area:
 

child (1-6 yrs)	=2625 cm <sup>2</sup>	(Langley 1993)
adult (7-30 yrs)	=4700 cm <sup>2</sup>	
- soil adherence: 0.5 mg/cm<sup>2</sup> (USEPA, 1988)
- produce ingestion rate:
 

child (1-6 yrs)	=0.13 kg/d	(Langley, 1993)
adult (7-30 yrs)	=0.45 kg/d	
- proportion of produce grown on site:
 

rural residential	= 50%	(Langley, 1993)
urban	= 10%	

**4.2.3.7 Commercial/industrial**

Human health is the primary on-site concern with regard to ground contamination where an ongoing industrial use is proposed. Where off-site transport of contaminants via soil movement, groundwater or surface water is likely, off-site environmental or health impacts may be most important. Acceptance criteria based on human health have been developed on the basis of reasonable maximum exposure assumptions. The major exposure assumptions are summarised below:

- exposure duration = 20 yrs (USEPA, 1989 b) (reasonable maximum time in one job, corresponds to 90th percentile time since last job in the US) (Finley, 1994)
- soil ingestion rate = 25 mg/day (for workers not directly involved in excavation) (ANZECC, 1992)
- inhalation rate = 9.6 m<sup>3</sup>/d (based on 8 hour working day) (Langley, 1993)
- skin surface area = 4700 cm<sup>2</sup>, based on exposure of 24% of total adult body surface area (Langley, 1993)
- soil adherence = 1.0 mg/cm<sup>2</sup> (USEPA, 1989)

The protection of human health is the primary on-site concern with regard to soil contamination where commercial/industrial site use is proposed. Where contaminated areas are fully paved and the integrity of the paving is maintained, the exposure to non-volatile soil

contaminants should be eliminated. However, the effectiveness of pavement as a barrier to the exposure of workers to ground contamination is highly dependent on the integrity and design of the pavement and on the nature of the underlying soils. Spreading and other transport of contaminated soil from areas where contaminated soil is unpaved or from areas of failed pavement may mean that protection against worker exposure to contaminated soil is reduced. The migration of volatiles through pavement, and the subsequent exposure, must also be assessed.

The acceptable contaminant concentration in soil on a paved industrial site may be controlled by exposures associated with ongoing maintenance of subsurface services or other subsurface works. For example, exposure associated with subsurface maintenance works may be effectively mitigated by the use of an appropriate site management plan requiring the use of protective clothing and equipment whenever the pavement is broken by subsurface works, and the diligent clean-up of soil and repair of the damaged areas.

**4.2.3.8 Parkland/recreational**

There is potential for human exposure to soil contamination in recreational areas with children the key exposure concern. Off-site migration of contaminated soil or dust may also occur. For exposure by the inhalation route, where there are buildings on site, e.g. kiosk or storeroom, this is the key exposure concern, and the criteria for commercial/industrial land use have been used for this route (to be protective of any works spending the majority of their time indoors at the site). The major exposure assumptions are summarised below:

- exposure duration = 30 years, assuming exposure from 0 to 30 years of age; 6 years as a ‘child’, 24 years as an ‘adult’.
- exposure frequency = 350 days/year (USEPA, 1989 b)
- body weight:
 

child (1-6 yrs)	=15 kg	(USEPA, 1992)
adult (7-30 yrs)	=70 kg	(ANZECC, 1992)
- soil ingestion rate:
 

child (1-6 yrs)	=50 mg/d	(ANZECC, 1992)
adult (7-30 yrs)	=10 mg/d	
- inhalation rate:
 

child (1-6 yrs)	=1.1 m <sup>3</sup> /d	(Langley, 1993)
adult (7-30 yrs)	=2.4 m <sup>3</sup> /d	
- exposed skin surface area:
 

child (1-6 yrs)	=2625 cm <sup>2</sup>	(Langley 1993)
adult (7-30 yrs)	=4700 cm <sup>2</sup>	
- soil adherence: 1.0 mg/cm<sup>2</sup> (USEPA, 1988)

**4.2.3.9 Maintenance**

For each of the above site uses, with the possible exception of agricultural use, there is potential for significant human exposure to ground contamination associated with subsurface maintenance works e.g. repair and replacement of services. Whilst the duration of such works is generally much shorter than the other exposure scenarios considered, the rate of intake of various contaminants is likely to be much higher and such exposure may be significant where undertaken routinely by the same person.

In order to develop reasonable but protective soil guideline values goals for adult workers involved in subsurface maintenance, the following exposure factors have been assumed:

- exposure duration = 20 yrs, 90% upper bound for time spent in one job (USEPA, 1989b)
- soil ingestion rate = 100 mg/d (for workers directly involved in excavation) (GRI, 1988).
- exposure frequency = 50 d/yr
- inhalation rate = 10 m<sup>3</sup>/d (Langley, 1993)
- skin soil adherence = 1.5 mg/cm<sup>2</sup> (USEPA, 1988)

The above assessment assumes that maintenance workers wear normal work clothes. The use of appropriate personal protective equipment may reduce worker exposure allowing work within areas with contaminant concentrations above the proposed criteria.

## 4.2.4 Risk characterisation

### 4.2.4.1 Carcinogens (non-threshold)

See Section 4.2.5.1 of the Users' Guide for information on carcinogens.

### 4.2.4.2 Non-carcinogens

Where more than one species has the same health effect or where exposure to a species may occur by more than one route, the HQ for each combination is summed to give a hazard index, HI. In the absence of further information, it is common practice to consider exposure to each substance separately. Where it is likely that substances have an additive or synergistic effect, this can be taken into account and the toxicological assessment should not be undertaken independently of such effects.

There is some evidence that toluene, ethylbenzene and xylene may act in a similar manner, particularly in relation to neurological effects. It may be argued, therefore, that additive or synergistic effects should be considered. Similarly some of the PAHs may be expected to show similar effects. However for the purposes of deriving generic acceptance criteria, each of the contaminants has been considered separately, with the exception of the carcinogenic PAHs.

The toxicological model underlying the USEPA assessment approach for non-carcinogenic health effects assumes the effects and dose are not necessarily cumulative over a lifetime. The USEPA RfDs for chronic health effects have been developed for exposure durations of months to years. On this basis, a year average Chronic Daily Intake is used to estimate the HQ in equation 7.7.

As chronic health effects may be experienced by children exposed to a substance over a period of months to years, if exposures to children and adults are combined for the assessment of non-carcinogenic health effects over, say, the 30 year exposure duration for a residential scenario, the year-averaged CDI for children would be underestimated, as would the likelihood of adverse health effects.

In particular, the year-averaged CDI for children would be underestimated when the higher exposure rates experienced by children for, for example, 6 years, are combined with lower rates of exposure experienced by adults for a longer period of time, and expressed as a year-average over a period of, for example, 30 years. Consequently, the assessment of non-carcinogenic health effects for residential and agricultural land uses are based on a year-average CDI for the most sensitive group (or the group with the highest weight-standardised exposure rate), i.e. children, rather than averaging over the entire 30 year exposure.

See Section 4.2.5.2 of the Users' Guide for information on non-carcinogens.

## 4.2.3 Derivation of generic soil acceptance criteria

Contaminant concentrations corresponding to the target risk level have been estimated for each exposure route e.g. inhalation of indoor air, inhalation of outdoor air, ingestion of soil, consumption of home grown produce and dermal absorption.

It may be argued that the exposure associated with each exposure route may be considered to be additive, and therefore that the acceptance criteria should be based on the soil concentration corresponding to the target risk level based on the cumulative exposure from all exposure routes (this is readily undertaken based on acceptance criteria for each individual exposure route). The above position is based on the assumption that a contaminant acts by a similar mechanism, despite exposure occurring by different exposure routes. While this is true for some contaminants, many exceptions are noted.

In practice, one exposure route is frequently dominant (resulting in a route specific acceptance criterion that is much lower than for other exposure routes). Therefore the acceptance criteria may be determined by selecting the lowest of the route specific acceptance criteria. Where more than one exposure route is significant, the impact of the combined exposure has been considered, and a note is included to this effect.

See Section 4.2.7 of the Users' Guide for a summary of the generic soil acceptance criteria.

## 4.3 Ecological considerations

Ecological considerations are an essential part of any assessment of the impact of former gasworks sites. Where sensitive ecological receptors are located near the site, ecological impact can be the limiting consideration.

Most gasworks sites are not located within pristine environments for which a very high level of protection of the surrounding ecosystems is required. Rather, most sites are located within a modified environment and the primary requirements for ecological protection relate to the protection of off-site environment quality and the associated ecosystems, and protection of on-site environmental quality is required to protect functions relevant to the site use e.g. protection of native and imported plants in the context of a residential use.

Policy objectives regarding the level of protection to be given to on-site ecosystems in the context of other land uses must be decided before the development of ecological investigation level guidelines for sensitive land uses.

The following precedents have been established regarding the development of guideline values based on environmental protection:

- **Agricultural**  
Protection of plant and livestock health, protection of human health via the consumption of produce from contaminated areas.
- **Residential**  
Protection of plant life and the protection of human health via the consumption of produce from contaminated areas.
- **Commercial/industrial**  
No specific requirement for protection of the on-site ecosystems.

The underlying premise in these precedents is that protection of on-site ecosystems is only required to the extent necessary to facilitate use of the land (e.g. protection of plant life to allow normal gardening activities in a residential context).

For each land use there is a residual requirement for the protection of the off-site environment, including groundwater quality, although these considerations are not explicitly incorporated in the derivation of soil guideline values. Rather, such considerations must be addressed on a site-specific basis. Where on-site ecosystems need protection in excess of that outlined above, published information such as the Environmental Quality Objectives for the Netherlands (including the Intervention Values) may be useful. Some published ecologically-based environmental quality objectives are presented in Appendix 4B.

In considering the possible impact of soil contamination on the off-site environment, the first step involves identification of:

- possible sensitive ecological receptors associated with the site (e.g. adjoining wetland ecosystems)
- possible exposure pathways for migration of the contaminant from the source to the ecological receptor (e.g. leaching from soil to groundwater, migration in

groundwater and discharge to the wetland). **Possible exposure pathways should also be reviewed to ensure completeness.**

Where a sensitive ecological receptor and a complete or potentially complete exposure pathway is identified, a further, more detailed evaluation of ecological risk should be undertaken.

Not enough work has been carried out in establishing the ecological considerations associated with gasworks contaminants. This is a field of work which is developed in the area of contaminated sites, and it is hoped that more attention can be focused on this area in the future.

More information of ecological considerations can be found in Section 4.2.8 of the Users' Guide.

## 4.4 Aesthetic considerations

Aesthetic impacts or impairment of the aesthetic qualities of a site are an important consideration in the management of contaminated land. There are several examples of sites that have been considered to be 'safe' in terms of their possible impacts on human health and the environment, yet have been deemed to be unsuitable for a sensitive use because of aesthetic impacts.

On gasworks sites, specific aesthetic concerns include free tars or 'tar balls'. Phenolic compounds have also been responsible for tainting of potable water flowing through plastic pipes in contaminated soil. The complex cyanides present in gasworks wastes can stain the soil a distinctive blue.

Of the effects noted above, odour is possibly the most sensitive aesthetic effect and can be associated with contamination by relatively light hydrocarbon compounds or the heavier tar materials.

While it is not possible to completely define the constituents responsible for odour impacts at gasworks, possible sources include;

- light PAHs such as naphthalene
- phenolic compounds e.g. cresol, and
- sulphurous odours associated with spent oxides.

Some odour may also be noted where manufactured gas is trapped within the soil matrix.

Weathering can have an important effect on both the odour associated with contaminated soil and the specific contaminants associated with such odour. As contamination weathers, the lighter organic compounds (e.g. benzene, naphthalene) are lost due to volatilisation and biodegradation, leaving the less volatile and more recalcitrant compounds.

In the assessment of aesthetic impact a tension exists between:

- the need to assess sites individually due to the site-specific nature of odour and the aesthetic effects, and
- the convenience and objectivity of establishing threshold soil concentrations for the protection of aesthetic quality. Assessment of aesthetic impact on a site by site basis relies on the notoriously subjective assessment of odour.

In practice, aesthetic impact is readily assessed on a site-specific basis and therefore generic criteria based on aesthetic impact have not been developed.

In assessing possible aesthetic impacts of contaminated soil, the following criteria may be considered:

- no perceptible odour associated with the soil (in close proximity to the soil)
- no perceptible discolouration of the soil

- no impact on soil structure, and
- no sheen development if a soil sample is submerged in water.

Aesthetic considerations are important when assessing the significance of soil contamination in the context of a sensitive land use, however, these considerations are of much lesser importance for less sensitive land uses, e.g. industrial. Although residents at a site may reasonably expect that the aesthetic quality of the soil is protected, on industrial land other aesthetic impacts associated with activities at the site would make it unreasonable to seek a protection of a high level of aesthetic soil quality. In an industrial context, concern would be associated with possible off-site aesthetic impacts. However an off-site impact is unlikely to be associated with contaminated soil within the site unless there is bulk soil movement or excavation.

Although contaminated soil at depth may be of concern with regard to human health, depending on the concentration of benzene and other volatiles, there is less concern about aesthetic impacts due to soil contamination at depth. Aesthetic effects are most likely to be noticed in close proximity to the soil, such as during gardening activities, and therefore concern is focused on the surface soils rather than the sub surface soils, i.e. those soils with which residents are most likely to come in direct contact.

More information on aesthetic considerations can be found in Section 4.2.9 of the Users' Guide.

## 4.5 References

- 1 ANZECC 1992 "Australian Water Quality Guidelines for Fresh and Marine Waters", Australian & New Zealand Environment & Conservation Council , November 1992.
- 2 ANZECC/NHMRC (1992) "Australian and New Zealand Guidelines for the Assessment and Management of Contaminated Land".
- 3 ASTM (1994) "Emergency Standard Guide for the Risk-Based Corrective Action Applied at Petroleum Release Sites (RBCA)".
- 4 Finley B, Proctor P, Scott N, Harrington, and Price P (1994) "Recommended Distributions for Exposure Factors Frequently Used in Health Risk Assessment" Risk Analysis, Vol. 14, No.4, pp 533-553.
- 5 Imray P and Langley A (1996) "Health-Based Soil Investigation Levels" Proc. 3rd Nat. Workshop on the Health Risk Assessment and Management of Contaminated Sites, South Australian Health Commission.
- 6 Langley A (1993) "Refining Exposure Assessment" Proc. 2nd Nat. Workshop on the Health Risk Assessment of Contaminated Sites, South Australian Health Commission, Canberra, August 1993.
- 7 Langley A and Sabordo L (1996) "Exposure Factors in Risk Assessment" Proc. 3rd Nat. Workshop on the Health Risk Assessment and Management of Contaminated Sites, South Australian Health Commission.
- 8 MfE/MoH (1993) "Draft Health and Environmental Guidelines for Selected Timber Treatment Chemicals".
- 9 Ministry of Health, "New Zealand Drinking-Water Standards for New Zealand", January 1995.
- 10 NEHF (1996) "Health-Based Soil Investigation Levels", National Environmental Health Forum.
- 11 NHMRC/ARMCANZ 1995 "Australian Drinking Water Guidelines" National Health and Medical Research Council/Agricultural and Resource Management Council of Australia and New Zealand, March 1996.
- 12 Shell (1994) "The Concepts of HESP, Reference Manual, Human Exposure to Soil Pollutants, Version 2.10a".
- 13 USEPA (1988) "Exposure Assessment Manual".
- 14 USEPA (1989a) "Exposure Factors Handbook" EPA/600/8-89/043.
- 15 USEPA (1991b) "Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual, Part B, Development of Preliminary Remediation Goals".
- 16 USEPA (1992) "Dermal Exposure Assessment: Principles and Applications", EPA/600/8-91/011B.
- 17 USEPA (1993) "Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons".

## Appendix 4A

# Health effects summaries for selected gasworks contaminants

## Introduction

The health effects associated with a range of contaminants encountered at former gasworks sites have been reviewed in order to:

- provide background information for assessing the significance of contamination
- review basis for establishing response factors for use in the assessment of risk and the derivation of acceptance criteria
- nominate dose response factors for use in the derivation of acceptance criteria.

Procedures for the development of dose response factors for carcinogenic chemicals in soil are currently under review by the National Health and Medical Research Council (NHMRC). The adopted dose response factors for carcinogenic chemicals are therefore subject to review following the release of guidance from the NHMRC.

## General principles

### Background

The assessment of the human toxicity of selected gasworks constituents has been based on published information relating to the observed effects of exposure of humans and animals to each of these compounds. The information available is limited in that:

- the observed effects are associated with exposure to the chemical of interest at a higher level than that of interest in nominating acceptance criteria for contaminated land. In setting acceptance criteria, attention is focused on a level of exposure that results in no appreciable risk or no effect. In contrast, most suitable studies focus on levels of exposure that result in an effect which is necessarily higher
- the duration of exposure may be less than is of interest when assessing the risk associated with contaminated land
- effects are observed in animals rather than humans, and there is some uncertainty as to the relevance of animal data in predicting likely effects in humans.

Human data are used preferentially in the assessment of chemical toxicity. However, when human data are not available, animal data has been used to extrapolate an exposure limit that is without an appreciable risk of an adverse effect in humans. When animal data are used, considerations are given to the suitability of the animal models for extrapolation to humans. The appropriate animal models would consider their relevance to humans such as xenobiotic metabolism and exposure routes. The approach is conservative and safety factors are used in deriving the exposure limits. Acceptable concentrations of the chemical in soil and groundwater may be estimated on the basis of the “acceptable” level of exposure.

The reported adverse effects in humans and animals associated with exposure to each of the selected gasworks constituents have been reviewed in order to develop an understanding of the range of health effects in humans that may be associated with exposure to these chemicals.

In developing health-based acceptance criteria, it is necessary to make a quantitative estimate of the relationship between exposure or dose and response. The relationship between exposure and response (i.e. dose response relationship) is frequently assessed separately for carcinogenic health effects and other health effects. Whereas one approach has been generally used for the assessment of non-carcinogenic health effects, a range of approaches have commonly been used to assess carcinogenic health effects.

The assessment of the toxicity of the gasworks constituents and, in particular, development of the quantitative dose response relationships, has been undertaken in accordance with the ANZECC/NHMRC (1992) "Australian and New Zealand Guidelines for the Assessment and Management of Contaminated Sites" (ANZECC Guidelines). The ANZECC Guidelines nominate that, where available, the WHO/FAO PTWIs or ADIs should be used as the basis for the development of Investigation Thresholds. In regard to the site-specific assessment of risk, the ANZECC Guidelines note that "where effects other than cancer are concerned, an acceptable daily intake has often been established by dividing the NOEL by a safety factor of 100".

Whilst no specific guidance is provided regarding the assessment of carcinogenic health effects, the ANZECC Guidelines provide some guidance in which two broad approaches are suggested for deriving the Investigation Thresholds. Firstly, a 'threshold' model is used to Investigation Thresholds where the WHO/FAO PTWIs or ADIs are available. Secondly, where the WHO/FAO PTWIs or ADIs are not available, a 'non-threshold' model using mathematical linear extrapolation (slope factors) from high to low doses is used.

The ANZECC Guidelines indicate that as part of toxicity assessment, reference should be made to:

- Toxicological Profiles prepared by the ATSDR, in collaboration with the USEPA
- Environmental Health Criteria, prepared by the World Health Organization
- IARC Monographs on the Evaluation of Carcinogenic Risks to Humans.

Although there is no clear consensus on the appropriate methodology for the assessment of genotoxic carcinogens and germ cell mutagens, a non-threshold model for the assessment of carcinogen has generally been adopted. This approach is consistent with the WHO approach in setting "Guidelines for Drinking Water Quality"(1993) and with the NZDWG which make a distinction between genotoxic and non-genotoxic carcinogens.

The NHMRC Working Party on Cancer Risk Assessment are developing guidance for cancer risk assessment. In the interim, an approach generally consistent with the NZDWG has been adopted in the selection of dose response factors for deriving soil and water acceptance criteria of specific chemicals will follow release of the guidelines. It is understood that the NHMRC Working Party are considering adoption of a benchmark dose approach, based on that outlined by WHO (1994) in EHC 170, for the assessment of carcinogenic contaminants in soil.

Reference has been made to the approaches adopted by a range of organisations (e.g. NHMRC, WHO, USEPA, ATSDR etc.) in developing dose response factors, given the lack of definitive guidance in the ANZECC Guidelines regarding the assessment of carcinogenic health effects in the context of site specific risk assessment.

### **Classification of carcinogens**

The International Agency for Research on Cancer (IARC) first developed a system for qualitatively categorising carcinogens in 1977. This system was based on weight-of-evidence data which involves assessment of all toxicity data originating from human, animal and in-vitro studies to ascertain if a chemical can be classified as carcinogenic. Assessment of all the data often indicates lack of adequate data for humans, hence if sufficient evidence of carcinogenicity in animals exists, then the chemicals are regarded as carcinogenic to humans as well. A classification system was also produced by the USEPA in the late 70s and was

modeled on the IARC system. Table 4A.1 shows the different carcinogenic classifications developed by the two agencies.

### Dose response factors

In order to quantify the health risks or likelihood of an adverse health effect associated with human exposure to various contaminants, a number of dose response factors, such as Reference Doses (RfD) Acceptable Daily Intakes (ADI), Benchmark Doses, and Cancer Potency Factor (CPF) or Cancer Slope Factors (CSF), have been defined by organisations such as the United States Environmental Protection Agency (USEPA) and the World Health Organisation (WHO).

In risk assessment, dose response factors are used to relate estimates of exposure or intake of contaminants to the likelihood of adverse health effects.

Dose response factors have been developed on the basis of human and animal studies, in order to relate an estimated intake of a contaminant to health risk. The available human data relating dose to response is limited for most chemicals, as discussed above, and therefore it is necessary to extrapolate from the available animal data to determine exposure levels that are consistent with no appreciable risk in humans. Such extrapolation may represent the single largest source of uncertainty and conservatism in the risk assessment process. Published dose response factors (e.g. WHO, USEPA) are generally conservative, incorporating a number of safety factors or uncertainty to account for the inherent uncertainties in the available data and the extrapolation process. The acceptable levels of exposure may have been used to determine health-based acceptance criteria for various environmental media (e.g. soil).

In assessing the dose response relationship in accordance with the approach adopted in the NZDWG, chemical contaminants and their associated health effects may be divided into two broad classes, as follows:

- Contaminants that exhibit a threshold:

For such contaminants it is proposed that a threshold dose exists below which there is no appreciable risk of critical adverse health effects.

A RfD or Acceptable Daily Intake (ADI) is an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive sub-populations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound. In developing a RfD or ADI, safety or uncertainty factors are used to modify the available experimental data (e.g. a No Observable Effect Level from an animal study) to account for (if applicable):

- extrapolation from animals to humans
- sensitive sub-populations
- extrapolation from a Lowest Observable Effect Level (LOEL) to a No Observable Effect Level (NOEL).

- Contaminants that exhibit no threshold:

For some contaminants and some health effects, it is assumed that there is no threshold dose below which there is no appreciable risk; rather the likelihood of a response increases as the dose increases (i.e. no dose is completely risk free). This approach is most commonly applied to carcinogens, particularly genotoxic carcinogens.

To quantify the risk associated with a given exposure, the Cancer Slope Factor (CSF) is used. The Cancer Slope Factor is a plausible upper-bound estimate (95th percentile) of the probability of a response per unit intake of a chemical over a lifetime. The CSF is used to estimate an upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a

potential carcinogen. The Cancer Slope Factor should be regarded as an upper bound estimate, rather than as an estimate of the actual risk.

The existence of a threshold (or lack of it) for some health effects, particularly cancer endpoints, is subject to considerable debate. If the NHMRC Working Party adopt an approach to the assessment of carcinogens based on the concept of a benchmark dose, then the distinction between threshold and non-threshold contaminants may be lessened.

The dose response relationship for various contaminants may depend on the route of exposure. Most of the available dose response data relates to the oral route, although some information is available regarding the inhalation route particularly from occupational studies or specific animal studies. The oral exposure route is the route of most concern for the contaminants. The available information has been combined to determine an acceptable daily intake or similar dose response factor for the combined exposure from all routes. This approach requires specific consideration of the bioavailability absorption, and metabolism of contaminants by each route.

Some important considerations extrapolating dose response data from route to route, include:

- lipid solubility. If a compound is highly lipid soluble it is more readily absorbed via the dermal route. Further lipid solubility affects the hepatic metabolism of contaminants
- does first pass metabolism of contaminants occur following oral exposure and, if so, are the metabolites active or inactive in terms of the outcome of interest (e.g. cancer)? If contaminants are immediately metabolised to an active intermediate following oral exposure, then extrapolation of dose response data from the oral route to other routes may be compromised.

A single dose response factor for the combined exposure via all routes will not be adopted where:

- the site of the effect is very close to the point of exposure
- there is marked difference in the sensitivity of animals and humans by exposure route (e.g. due to differing metabolic processes for each route).

Table 4A.1 IARC and EPA classification of carcinogenic risk to humans<sup>1</sup>

IARC			Evaluation of Agent Mixture or Occupation	EPA		
Classification Grouping	Evidence from <sup>(2)</sup>			Classification Grouping	Evidence from <sup>(2)</sup>	
	Humans	Animals	Other Relevant Data <sup>(3)</sup>		Humans	Animals
1	S			IS carcinogenic	A	S
2A or or	L L I/ND	S S	Supp Supp	is <b>PROBABLY</b> carcinogenic	B1 B2 or	L I ND S S
2B or or	L I/ND I	S L	Supp	is <b>POSSIBLY</b> carcinogenic	C	ND L
3	I/ND	L		is <b>NOT CLASSIFIABLE</b> as to its carcinogenicity	D	Inadequate evidence or no data available
4	No evidence for carcinogenicity			is <b>PROBABLY NOT</b> carcinogenic	E	No evidence for carcinogenicity

- Notes
- 1 Based on Table from Langley (1993)
  - 2 S - sufficient      Supp - supportive  
L - limited      ND - no data  
I - inadequate
  - 3 Other relevant data include structure - activity considerations, pharmacokinetics and metabolism, toxicity, genetic and related effects.

## Assessment of chemical mixtures

A significant limitation with regard to most toxicity assessments is that the available information generally relates only to exposure to a single chemical, whereas in practice exposure to a range of chemicals occurs simultaneously. The effect of simultaneous exposure to multiple chemicals is generally not well understood. The effects of such combined exposures may be synergistic, additive or antagonistic. An example of synergistic interaction between chemicals is found in one of the proposed mechanisms of cancer formation, where initiation and promotion of the tumour may require exposure to different agents, such that a tumour does not occur unless exposure to both chemicals occurs.

Some information may be obtained regarding the possible effects of simultaneous exposure to more than one chemical by considering the route of absorption, distribution, metabolism and target organ. Where chemicals affect different target organs and there is little or no interaction between the metabolism and distribution of the chemicals in the body, then there may be some justification for assuming the effects are independent.

Examples of groups of contaminants likely to be found together at former gasworks sites and which may act in a similar manner include (although differences may be apparent in some effects):

- carcinogenic PAHs
- non-carcinogenic endpoints associated with PAHs (both carcinogenic and non-carcinogenic)
- toluene and ethylbenzene

## Health effects summaries for individual chemicals

### Overview

The health effects associated with selected chemicals of concern at former gasworks sites have been discussed in terms of the following issues:

- Kinetics and metabolism
- Animal toxicity
- Genotoxicity and carcinogenicity
- Human toxicity
- Dose response.

The discussion of dose response includes nomination of dose response factors used in the derivation of soil and water acceptance criteria. The discussion of dose response factors may require revision following the release of the report on the assessment of carcinogenic chemicals from the NHMRC Working Party.

### Benzene

#### *Primary reference*

WHO (1993) "Environmental Health Criteria 150, Benzene" IPCS

#### *Kinetics and metabolism*

Benzene is well absorbed in humans and experimental animals following exposure via the oral and inhalation route, however dermal absorption is generally poor in humans. Benzene tends to accumulate in tissues with a high lipid content, and it crosses the placenta.

Benzene metabolism occurs mainly in the liver, is mediated primarily through the cytochrome P-450 IIE1 enzyme system, involving the formation of a series of unstable reactive metabolites. Experimental evidence suggests the formation of two putative toxic metabolites, benzoquinone and muconaldehyde, in rodents can be saturated. This may have important implications in establishing a dose-response relationship for benzene, as a higher

proportion of the benzene will be converted to toxic metabolites at low doses than at high doses.

Metabolism of benzene in the liver is responsible for the detoxification of benzene via the formation of etheral sulfate, glucuronides and glutathione conjugates. However metabolism of benzene in the liver also leads to the production of metabolites, such as hydroquinone, p-benzoquinone and muconaldehyde which appear to be associated with benzene toxicity in bone marrow. The metabolic products of benzene are primarily excreted in the urine.

### ***Animal toxicity***

The available evidence suggests benzene is of low acute toxicity in a range of animal species, with LD<sub>50</sub> values for rats following oral exposure ranging between 3000 and 8100 mg/kg body weight. Reported LC<sub>50</sub> values based on inhalation exposure range from 15 000 mg/m<sup>3</sup> (8 h) in mice to 44 000 mg/m<sup>3</sup> (4 h) in rats.

There is no evidence that benzene is associated with teratogenic effects at doses lower than those required to produce maternal toxicity, however foetal toxicity has been demonstrated.

### ***Genotoxicity and carcinogenicity***

In vitro tests indicate that benzene is not mutagenic, however, benzene, or its metabolites, have been shown to cause chromosomal aberrations in experimental animals and sister chromatid exchange (SCE) and micronuclei in polychromatic red blood cells.

Benzene has been associated with several types of neoplasms in rats and mice following oral or inhalation exposure, including various types of epithelial neoplasms, e.g., Zymbal gland, liver, mammary tissue and nasal cavity neoplasms, and some lymphomas and leukaemias.

The evidence of carcinogenic health effects associated with benzene resulting from observation of occupationally exposed populations is presented in the following section.

Benzene has been classified as a Group 1 chemical (confirmed human carcinogen) by IARC.

### ***Human toxicity***

The most significant adverse effects from short- or long-term exposure to benzene are haematotoxicity, i.e. bone marrow suppression, immunotoxicity, genotoxicity and carcinogenicity.

Benzene is a well-established human carcinogen. Epidemiological studies of benzene-exposed workers have demonstrated a causal relationship between benzene exposure and the production of myelogenous leukaemia. A relationship between benzene exposure and the production of lymphoma and multiple myeloma remains to be clarified.

There is at present no adequate animal model for benzene-induced leukaemia in humans which limits the ability of researchers to conduct experiments that may assist in understanding the metabolism and mechanisms of action. The limited metabolic data suggests that several reactive metabolites of benzene are formed and these can form adducts both with DNA and protein. The failure to produce leukaemia in animals may be due to inadequate formation of leukaemogenic metabolites or the need to produce bone marrow damage prior to the induction of leukaemia.

Continuous exposure to benzene over a period of 10 years or more is expected to result in some toxicity, for both high and low doses. A high level of both bone marrow depression and aplastic anaemia may be seen at the higher doses although some damage would also be observed at lower doses.

The neurotoxicity and immunotoxicity of benzene has not been well studied in experimental animals or humans.

The risk of adverse health effects, particularly leukemia, associated with low-level benzene exposure has not been clearly established. Studies of workers exposed to relatively low concentrations of benzene (TWA: < 3.2-32 mg/m<sup>3</sup>, < 1-10 ppm) revealed no alteration in cell-cycle kinetics and in sister chromatid exchange rate, which are possible markers of

carcinogenic process. Only a marginal increase in chromosomal aberrations (chromatid deletions and gaps) was noted at the low levels of exposure outlined above.

### **Dose response**

A guideline value of 0.01 mg/litre was recommended by WHO (WHO, 1984) for benzene in drinking-water based on data for the production of leukaemia after inhalation exposures in humans and using a linear multistage extrapolation model and a life-time risk level of 1 in 100,000. This risk specific dose would correspond to a slope factor of  $0.035 \text{ (mg/kg/day)}^{-1}$ .

Benzene is considered a non-threshold toxicant by the USEPA due to its carcinogenicity. An oral slope factor value of  $0.029 \text{ (mg/kg/day)}^{-1}$  has been derived based on the observance of leukemia from occupational exposure by inhalation.

In derivation of the NZDWS, the Ministry of Health adopted an acceptable daily intake of  $0.29 \text{ } \mu\text{g/kg/day}$ , based on the WHO guideline, which corresponds to slope factor of  $0.035 \text{ (mg/kg/day)}^{-1}$ .

*For the purposes of deriving soil and water acceptance criteria, a slope factor of  $0.029 \text{ (mg/kg/day)}^{-1}$  has been adopted.*

## **Toluene**

### **Primary reference**

WHO (1986) "Environmental Health Criteria 52, Toluene" IPCS

### **Kinetics and metabolism**

Studies on humans and animals have shown that toluene is readily absorbed from the respiratory tract with 40 to 60% uptake reported in humans. Liquid toluene is also rapidly absorbed through the skin ( $14 \text{ to } 23 \text{ mg/cm}^2/\text{h}$ ), although absorption from the gastrointestinal tract appears to be slower.

Following absorption, toluene is rapidly distributed, with highest levels observed in adipose tissue followed by bone marrow, adrenal glands, kidneys, liver, brain, and blood. The relationship between arterial blood and alveolar air concentration has been found to exhibit a close linear correlation. Therefore, measuring the toluene concentration in alveolar air during exposure, allows the estimation of the arterial blood concentration.

Some 60 to 75% of absorbed toluene is metabolised to benzoic acid by the microsomal mixed-function oxidase system, with subsequent conjugation with glycine to form hippuric acid. It is eliminated in this form through the kidneys. Approximately 10 to 20% of the absorbed toluene is excreted as benzoyl glucuronide. Small amounts of toluene undergo ring hydroxylation to form o-, m-, and p-cresol, which are excreted in the urine as sulfate or glucuronide conjugates. A proportion of the absorbed toluene (20 to 40%) is eliminated unchanged in expired air. After a single exposure, the elimination of toluene and its metabolites is almost complete in 24 hours. The half-life of toluene in subcutaneous adipose tissue has been estimated to be between 0.5 and 2.7 days.

Toluene has been shown to affect biotransformation of several solvents, altering the likelihood and severity of associated adverse health effects. Toluene decreased n-hexane metabolism and neurotoxicity, and benzene metabolism and effects on the haematopoietic system. However, toluene has been associated with increased hepatotoxicity resulting from exposure to carbon tetrachloride.

### **Animal toxicity**

Acute inhalation data suggests that the sensitivity of various species to toluene decreases as follows: rabbit, guinea-pig, mouse, and rat. Inhalation  $\text{LC}_{50}$  values have been reported in the range of approximately  $20\,000 \text{ to } 26\,000 \text{ mg/m}^3$  for mice and  $45\,000 \text{ mg/m}^3$  for rats.

The reported oral  $\text{LD}_{50}$  for toluene in rats is between 2.6 and 7.5 g/kg body weight, depending on the strain, age, and differences in sex. Toluene is a slight dermal irritant and a moderate

eye irritant in animals and humans. The acute dermal toxicity of toluene appears to be quite low (rabbit: LD<sub>50</sub> 14.1 ml/kg body weight).

No effect was observed in short-term and long-term inhalation studies on experimental animals using toluene, at concentrations up to 375 mg/m<sup>3</sup> for a period of 24 months. In oral exposure studies, administration toluene at a rate of 590 mg/kg body weight/day, for 6 months did not produce any observable adverse effects. At low doses the target organs in rats appear to include the kidneys and testes, while at higher doses liver changes and effects on the central nervous system are observed.

Numerous studies using pure toluene have failed to demonstrate adverse effects on the blood.

Toluene can affect the central nervous system (CNS), but not the peripheral nervous system (PNS), although this is usually observed at high doses.

Toluene does not appear to be teratogenic in mice, rats, or rabbits, however fetotoxic effects were observed in rats at doses that were non-toxic to the dams (e.g. toluene concentrations up to 1000 mg/m<sup>3</sup>), and spontaneous abortion occurred in rabbits exposed to 1000 mg/m<sup>3</sup> during the period of organogenesis (which includes the period of organ development).

Oral exposure to toluene has been associated with teratogenic effects in CD-1 mice.

Exposure of CD-1 mice to toluene at 870 mg/kg body weight for days 6 to 15 significantly increased the incidence of cleft palate. No observable teratogenic effect was associated with exposure to toluene at a rate of 430 mg/kg body weight.

### ***Genotoxicity and carcinogenicity***

In general, very little evidence has been reported suggesting genotoxic or carcinogenic effects associated with exposure to toluene, and therefore toluene is normally regarded as non-carcinogenic.

Skin-painting studies on mice, where toluene was used as a vehicle control, and one inhalation study on rats exposed to toluene (112.5 to 1125 mg/m<sup>3</sup>, 6 h/day, 5 days/week, for 24 months) did not report evidence of carcinogenic effects.

The results of studies on the mutagenic effects of toluene in microbial, mammalian-cell, or whole-organism test systems have, in most cases, been negative. Positive findings were reported in 5 studies using in vivo mammalian assays. However, in these studies the purity of the toluene used was not stated and the possibility of impurities contributing to the observed effect cannot be discounted.

Toluene has not been classified as a possible, probable or confirmed human carcinogen by either the USEPA or IARC.

### ***Human toxicity***

Information on the toxicity of toluene in humans has been primarily derived from individuals exposed to toluene via inhalation either in occupational settings or during episodes of intentional abuse of solvent mixtures containing toluene.

The primary effect of acute exposure to toluene is on the central nervous system (CNS). The effect may be depressant or stimulatory, with euphoria in the induction phase, and may lead to convulsion or coma.

Single, short-term exposures to toluene (750 mg/m<sup>3</sup> for 8 h) have been associated with transient eye and respiratory tract irritation at 1500 mg/m<sup>3</sup>.

Repeated occupational exposures to toluene over a period of years at concentrations in the range 750 to 1500 mg/m<sup>3</sup> have resulted in some evidence of neurological effects. Inhalation of toluene was reported to be an important cause of brain diseases in children (aged 8 - 14 years), possibly leading to permanent neurological damage.

Transient abnormalities of hepatic enzyme activities have been found in abusers of toluene mixtures, but significant permanent hepatic damage has not been observed. Renal damage in

glue-sniffers have been reported but there is no evidence that toluene results in adverse effects on the blood or the heart.

Epidemiological information regarding the effects of exposure to toluene is limited (frequently information is confounded by concurrent exposure to a range of chemicals).

### ***Dose response***

The USEPA has nominated the following RfDs for toluene;

- 0.2 mg/kg/day by oral route, with a safety factor of 1000, based on NOAEL for effects on liver and kidneys in rats; and
- 0.4 mg/m<sup>3</sup> by inhalation with a safety factor of 300, based on LOAEL for neurological effects observed in a small population of workers.

In derivation of the NZDWS, the Ministry of Health adopted an acceptable daily intake of 0.22 mg/kg/day based on hepatotoxicity in mice from a 15-week gavage study and an uncertainty factor of 1000. This approach is consistent with that adopted by the WHO in the derivation of a drinking water guideline value.

***For the purposes of deriving soil and water acceptance criteria, a Reference Dose of 0.2 mg/kg/day has been adopted for the oral route, and a Reference Concentration of 0.4 mg/m<sup>3</sup> has been adopted for the inhalation route.***

## **Ethylbenzene**

### ***Primary reference***

WHO (1996) “Guidelines for Drinking-Water Quality-Health criteria and other supporting information”.

### ***Kinetics and metabolism***

Ethylbenzene is readily absorbed by oral, inhalation or dermal routes. Once absorbed, the distribution and excretion are rapid. In humans, storage of ethylbenzene in fat has been reported, and the compound has been observed to cross the placental barrier. Biotransformation in humans to mandelic acid and phenylglyoxalic acid is almost complete, both the metabolites being excreted in the urine. In animals, the metabolism of ethylbenzene differs from that in humans in that benzoic acid is the major metabolite together with mandelic acid. Urinary excretion of metabolites is rapid and is complete within 24 hours.

### ***Animal toxicity***

In a 6-month oral study in rats, doses of 400 mg/kg and above produced effects on liver and kidneys, with a NOAEL of 136 mg/kg. Liver effects were also observed in a number of inhalation studies with the LOAEL at 1305 mg/m<sup>3</sup> and NOAEL at 218 or 430 mg/m<sup>3</sup>.

Although teratogenicity studies have been carried out in rats and rabbits, via the inhalation route, no definite conclusion could have been drawn.

### ***Genotoxicity and carcinogenicity***

Studies on the mutagenic activity of ethylbenzene to bacteria, insects, mammalian cells (in vitro) and intact mammals have shown ethylbenzene to be devoid of mutagenic activity.

No carcinogenicity data on ethylbenzene are available.

### ***Human toxicity***

Ethylbenzene is mildly toxic to humans following skin contact or inhalation, and has been associated with systemic effects in humans. Ethylbenzene has been associated with irritation of the eyes, skin, nose, throat and respiratory tract at concentrations in the order of 0.2% (v/v). The lowest reported acutely toxic concentration (TC<sub>10</sub>) of ethylbenzene by inhalation for human is 100 ppm.

Ethylbenzene has been classified by the USEPA as a Class D chemical i.e. not classifiable as to human carcinogenicity due to inadequate human and animal evidence.

### **Dose response**

The USEPA has nominated the following dose response factors for ethylbenzene:

- an oral RfD of 0.1 mg/kg day with a safety factor of 1000, based on NOAEL by oral route for liver and kidney toxicity observed in rats
- an inhalation RfC of 1 mg/m<sup>3</sup>, with a safety factor of 300, based on NOAEL for developmental toxicity in rats and rabbits.

In derivation of the NZDWS, the Ministry of Health adopted an acceptable daily intake of 0.1 mg/kg/day based on hepatotoxicity and nephrotoxicity in rats reported as part of a limited 6 month study. A safety/uncertainty factor of 1000 was adopted reflecting limitations in the animal data used.

*For the purposes of deriving soil and water acceptance criteria, the Reference Dose of 0.1 mg/kg/day has been adopted for oral route, and a Reference Concentrations of 1 mg/m<sup>3</sup> has been adopted for inhalation route.*

### **Xylenes**

#### **Primary reference**

WHO (1996) “ Guidelines for Drinking-Water Quality- Health criteria and other supporting information”.

#### **Kinetics and metabolism**

Xylene is readily absorbed following inhalation and is also absorbed to some extent via the skin. However, there are no data available on human absorption after ingestion. Xylene is rapidly distributed following uptake. Once absorbed, xylenes is rapidly metabolised almost completely to methyl benzoic acid which is excreted in the urine as hippuric acid. Xylenes have been found to cross the placental barrier and are stored in adipose tissue in both animals and humans. The elimination half-life of xylenes from subcutaneous fat in humans ranges from 25 to 128 hours.

#### **Animal toxicity**

A 2-year feeding study has been carried out in rats and mice. In rats, decreased growth at high dose of 500 mg/kg/day with no observable compound related histological lesions. The NOAEL for rats was 250 mg/kg/day. Although embryotoxicity and developmental toxicity has been observed in mice, the observations were not conclusive due to the concurrent maternal toxicity.

Exposure to xylene by inhalation caused liver enzyme induction at high concentration ( $\geq 217$  mg/m<sup>3</sup>). No developmental toxicity has been observed in rodents due to inhalational exposure of xylene.

#### **Genotoxicity and carcinogenicity**

The mutagenicity studies of xylene in bacteria and mammalian cells, both in vitro and in vivo, have shown negative results. Xylene did not cause carcinogenicity at oral doses up to 500 mg/kg/day in rats and up to 1000 mg/kg/ in mice.

Xylene has been classified as a Class D chemical by the USEPA, i.e. it is not classifiable as to its human carcinogenicity due to inadequate human and animal evidence.

#### **Human toxicity**

In humans, exposure to xylene vapour has been associated with irritation of the eyes, nose and throat and some light-headedness at concentrations in excess of 200 ppm. Neurobehavioural effects were also reported after a 5-6 hours of exposure to xylenes at a concentration in the order of 100 ppm.

#### **Dose response**

The USEPA has nominated an oral RfD for xylenes of 2.0 mg/kg/day, incorporating a safety factor of 100 and based on the NOAEL of 179 mg/kg/day for decreased body weight and increased mortality in rats from a 103 week gavage study.

In derivation of the NZDWS, the Ministry of Health adopted an acceptable daily intake of 0.18 mg/kg/day based on the same study in rats but an uncertainty factor of 1000.

For comparison, a tolerable daily intake of 0.01 mg/kg/day was used in the derivation of soil acceptance criteria by the Dutch agencies.

*For the purpose of deriving soil and water criteria, the oral Acceptable Daily Intake of 0.18 mg/kg/day has been adopted.*

## **Polycyclic aromatic hydrocarbons**

### **Primary reference**

WHO (1996) “ Guidelines for Drinking-Water Quality-Health criteria and other supporting information”.

### **Introduction**

Polycyclic aromatic hydrocarbons (PAHs) is a large class of chemicals with two or more fused aromatic rings structure. PAHs occur in the environment as complex mixtures of which only a few components have been adequately characterised. Most of the available literature on PAHs is concerned with benzo[a]pyrene B[a]P the most abundant naturally occurring and anthropogenic PAH, and only limited information is available on the relative toxicity of the PAHs.

The concern over PAH contamination in the environment relates mainly to the carcinogenic and mutagenic activity of some of these compounds. B[a]P benzo[a]pyrene is an indicator compound due to its carcinogenicity. For the purposes of this assessment, PAHs classified by the USEPA as Class D chemicals have been regarded as non-carcinogenic PAHs. Other PAHs may be grouped with B[a]P because of uncertainties in their carcinogenicity and because they accumulate or bioconcentrate in living tissue.

### **Kinetics and metabolism**

Absorption of PAHs mainly occurs following oral and inhalation exposure and rapidly distributed to the various organs and tissues. PAHs can also be absorbed following dermal exposure. The rate of absorption of the different PAHs is influenced by their lipid solubility. PAHs is highly lipophilic and may be stored in the breast and fat tissues. B[a]P has been shown to cross the placenta and is distributed in the developing foetus.

The metabolism of B[a]P occurred primarily in the liver involving oxidation and hydroxylation by the mixed-function oxygenases (MFOs) and detoxication by glucuronosyl-, sulfo- or glutathione transferases.

### **Animal toxicity**

The reported oral LD50s for PAHs range from 40 to 18 000 mg/kg of bodyweight. No treatment-related effects were observed in mice given anthracene by gavage at doses up to 1000 mg/kg/day for at least 90 days. Subchronic oral administration of naphthalene (50 mg/kg/day) has been associated with decreased body weight in rats. Mice subchronically exposed to fluoranthene at doses developed adverse effects in the kidney, liver and haematological system. Haematological and kidney effects have also been observed in mice following exposure to fluorene (125-500 mg/kg/day) and pyrene (127-917 mg/kg/day), respectively. Slight morphological changes in the liver and kidney of rats following oral exposure to acenaphthene for 40 days have been reported.

Reproductive effects were observed in offspring of mice given oral doses of B[a]P with reduction of fertility at doses as low as 10 mg/kg/day.

### **Genotoxicity and carcinogenicity**

B[a]P has been shown to be mutagenic in bacteria and in cultured human lymphoblastoid cells, after metabolic activation. It is considered that the diol-epoxides metabolites of B[a]P is considered to be more potent than the parent compound. B[a]P has also caused sister chromatid exchanges in *in vivo* and *in vitro* test systems.

PAHs have been shown in animals to affect proliferating tissues such as bone marrow, lymphoid organs, gonads and intestinal epithelium.

Many PAHs mixtures have been associated with increased incidences of cancer. Of the 16 PAHs identified by the USEPA in their primary pollutants list, seven are classified as probable human carcinogens (B2) ie. benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene. The basis for the carcinogenic classification of these compounds is varied. For example, no human data is available for chrysene, however it has been found to produce skin carcinomas and malignant lymphoma in mice. Benzo(a)pyrene has been shown to be carcinogenic to rodent and non rodent species following exposure by all three major pathways.

In humans, the evidence of carcinogenicity mainly comes from studies of workers who are exposed to mixtures containing PAHs in their occupations which involved processes such as coke production, oil refining or coal gasification. As inhalation and dermal exposures are the common exposure routes, cancers associated with exposure to the PAH-containing mixtures in humans are also commonly found in the lungs and skin.

### **Human toxicity**

Studies on human health effects of PAHs are limited. Skin lesions have been observed in human subjects skin-painted with benzo(a)pyrene.

Death caused by acute haemolytic anaemia due to accidental poisoning by naphthalene has also been reported. Although no human healths have been reported following exposure to other PAHs, it can be assumed that acute exposure to sufficiently high doses of PAHs can be lethal based on the observation of death in animals following oral exposure.

As indicated earlier, occupational studies indicate an increased incidence of cancers associated with exposure to PAH-containing mixtures in workers. Epidemiological studies have also indicated increased incidence of lung cancer in humans exposed to PAH-containing mixtures, ie. coke-oven emissions and cigarette smoke. However, it is not possible to evaluate the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans due to the complexity of the mixtures and the presence of other carcinogens.

### **Dose response**

#### ***Non-carcinogenic PAHs/Size***

The USEPA derived chronic oral RfDs for the non-carcinogenic PAHs as follows:

- 0.06 mg/kg/day for acenaphthene based upon NOAEL of 175 mg/kg/day with critical effect of hepatotoxicity in mice exposed by gavage for 90 days
- 0.3 mg/kg/day for anthracene based upon NOEL of 1000 mg/kg/day in mice exposed by gavage for 90 days
- 0.04 mg/kg/day for fluoranthene based upon NOAEL of 125 mg/kg/day with critical effects of nephropathy, liver weight changes and haematological alterations in mice exposed by gavage for 90 days
- 0.04 mg/kg/day for fluorene based upon NOAEL of 125 mg/kg/day with critical effects of decreased red blood cell count, packed cell volume and haemoglobin concentration in mice exposed by gavage for 13 weeks
- 0.03 mg/kg/day for pyrene based upon NOAEL of 75 mg/kg/day with critical effect of renal toxicity in mice exposed by gavage for 13 weeks.

The chronic RfDs for acenaphthene, fluoranthene, fluorene and pyrene were all derived using an uncertainty factor of 3,000. These values are adopted in this assessment for the derivation of the soil acceptance criteria.

For naphthalene, the chronic reference dose of  $4 \times 10^{-3}$  mg/kg/day used for risk calculation in the Health Risk Assessment for Soils Contaminated with Fuel hydrocarbons: Petrol in Australia (Lindon P, 1991), which based on decrease body weight gain in rats (HEAST, 1991), was adopted in this assessment for the derivation of the soil acceptance criteria.

There is currently no RfD value established for phenanthrene which is still under review by the USEPA. However, a oral RFD of  $3 \times 10^{-2}$  was available from the 1993 IRIS database and is adopted for this assessment.

The NZDWS and NZDWG in considering PAHs only present a health based guideline value for benzo(a)pyrene, based on a cancer endpoint. The non-carcinogenic PAHs are generally not a limiting consideration.

### *Carcinogenic PAHs*

The carcinogenic potency of these compounds is most commonly determined using data from animal studies, largely due to the lack of human studies from which the observed effects may be directly attributed to a specific PAH compound or group of compounds. The dose associated with a particular increased lifetime cancer risk, or the slope of the risk-dose relationship (slope factor) is estimated using the available human and animal data.

In general, the risk estimates for benzo(a)pyrene have been calculated from two studies in different species of rodents, orally exposed to benzo(a)pyrene, where forestomach cancer was observed (Neal and Rigdon, 1967; Brune *et al.*, 1981). The data set were separately fitted to the Linearised Multistage (LMS) model to provide a low-dose extrapolation. A 95% upper confidence limit is applied to determine an upper bound for the slope of the line (Slope Factor) derived by the LMS model. The cancer slope factor of  $7.3 \text{ (mg/kg/day)}^{-1}$  for benzo(a)pyrene was based on the geometric mean of risk estimates calculated from these studies.

To streamline the assessment of the carcinogenic PAHs, a relative potency approach has been developed to estimate cancer potency of the other carcinogenic PAHs based on their relative potency to benzo(a)pyrene. The toxicity equivalency factor (TEF), based on carcinogenicity, nominated by various organisations are shown in Table 4A.2. The TEFs nominated by the USEPA in provisional guidance (USEPA, 1993) are suggested for use in the assessment of gasworks sites. TEFs may be used to express the relative potency of a mixture of carcinogenic PAHs in terms of equivalent benzo(a)pyrene concentration.

Using the TEFs, calculated oral slope factors for the carcinogenic PAHs range from  $7.3 \text{ (mg/kg/d)}^{-1}$  for benzo(a)pyrene to  $0.073 \text{ (mg/kg/d)}^{-1}$  for chrysene.

In derivation of the NZDWS, the Ministry of Health adopted a tolerable daily intake for benzo(a)pyrene of  $0.00002 \text{ mg/kg/day}$ , corresponding to an excess life-time cancer risk of 1 in 100 000 (or a Slope Factor of 0.5) based on a quantitative risk assessment conducted using the two-stage birth-death mutation model.

***The USEPA derived slope factor for benzo(a)pyrene of  $7.3 \text{ (mg/kg/d)}^{-1}$  has been adopted for the derivation of acceptance criteria. The TEFs nominated by the USEPA should be used in assessing the risk associated with carcinogenic PAH mixtures.***

**Table 4A.2 Toxic equivalence factors (TEFS) for carcinogenic PAHs**

Chemical	US	Californian	Dutch (RIVM) <sup>3</sup>	Health	Adopted
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	EPA <sup>1</sup>	EPA <sup>2</sup>		Canada <sup>4</sup>	
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.0
Benzo(a)anthracene	0.1	0.1	0.1		0.1
Benzo(b)fluoranthene	0.1	0.1		0.06	0.1
Benzo(k)fluoranthene	0.01	0.1	0.1	0.04	0.1
Chrysene	0.01		1.0		0.01
Dibenz(a,h)anthracene	1.0	0.4			1.0
Indeno(1,2,3-cd)pyrene	0.1	0.1	0.1	0.12	0.1

<sup>1</sup> USEPA (1993) "Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons".

<sup>2</sup> California Environmental Protection Act (1994).

<sup>3</sup> RIVM Netherlands (1991) "Voorstel voor de humaan-toxicologische onderbouwing van C-(toetsings)waarden". Report no. 725201005.

<sup>4</sup> Canadian Environmental Protection Act (1994) "Polycyclic aromatic hydrocarbons".

<sup>5</sup> USEPA Carcinogen Classification System. Class B2 denotes probable human carcinogen based on limited (or no) human data and sufficient animal data.

## Phenol

### *Primary reference*

WHO (1994) "Environmental Health Criteria 161, Phenol" IPCS

### *Kinetics and metabolism*

Phenol is readily absorbed by all routes of exposure and is rapidly distributed to all tissues. The liver, the lung, and the gastrointestinal mucosa are the most important sites for phenol metabolism. The relative importance of each of these sites depends on route of administration and dose.

Absorbed phenol forms conjugates with glucuronic acid and sulfuric acid and, to a lesser extent, hydroxylates into catechol and hydroquinone. Phosphate conjugation also occurs. The formation of reactive metabolites (4,4-biphenol and diphenoquinone) has been demonstrated in vitro studies with human white blood cells (ie. activated neutrophils and leucocytes). The relative amounts of glucuronide and sulfate conjugates vary with dose and animal species. A shift from formation of sulfate conjugated to formation of glucuronide conjugates was observed in rats after increasing dosage.

Urinary excretion is the major route of phenol elimination in animals and humans. The rate of urinary excretion varies with dose, route of administration, and species. Excretion in faeces and elimination in expired air are relatively minor routes..

Benzene and phenol derivatives may, in vivo conversion, represent a source of phenol exposure from within the body.

### *Animal toxicity*

Phenol exhibits moderate acute toxicity in mammals. Oral LD<sub>50</sub> values in rodents range from 300 to 600 mg/kg body weight. Dermal LD<sub>50</sub> values for rats and rabbits range from 670 to 1400 mg/kg body weight, respectively, while the 8-h LC<sub>50</sub> for rats by inhalation is more than 900 mg/m<sup>3</sup>.

There is evidence that phenol is not associated with skin sensitisation.

The most important effects reported in short-term animal studies were neurotoxicity, liver and kidney damage, respiratory effects and growth retardation. In a limited 14-day study involving rats, an oral no-observed-adverse-effect level (NOAEL) of 12 mg/kg per day was reported, based on kidney effects.

No adequate studies examining the reproductive toxicity of phenol were identified. Phenol has been identified as a developmental toxicant in studies with rats and mice. In two multiple dose rat studies, NOAEL values of 40 mg/kg per day (the lowest-observed-adverse-effect level (LOAEL) was 53 mg/kg per day) and 60 mg/kg per day (the LOAEL was 120 mg/kg per

day) have been reported. In the mouse study, a NOAEL of 140 mg/kg per day (the LOAEL was 280 mg/kg per day) was identified.

Phenol and some of its metabolites can be cytotoxic as they have been demonstrated to covalently bind to tissue and plasma proteins.

### ***Genotoxicity and carcinogenicity***

The majority of bacterial mutagenicity tests have reported negative results for phenol, however mutations, chromosomal damage and DNA effects have been observed in mammalian cells in vitro. Induction of micronuclei in bone marrow cells of mice has been observed in some studies at high doses.

In carcinogenicity studies conducted with male and female rats and mice receiving phenol in their drinking-water, malignancies (e.g., C-cell thyroid carcinoma, leukaemia) were only seen in low-dose male rats. Two-stage carcinogenicity studies have shown that phenol, applied repeatedly to mouse skin, has promoting activity.

No evidence of carcinogenicity has been reported for phenol in human studies, although animal studies have indicated it may be a promoter and/or weak skin carcinogen in some species of mice. Phenol has not been classified as a human carcinogen (confirmed, probable or possible) by the USEPA (Class D)..

### ***Human toxicity***

Most of the information regarding adverse effects in humans associated with phenol exposure relates to acute rather than chronic exposure.

Clinical symptoms observed in humans following acute exposure include neuromuscular hyperexcitability and severe convulsions, necrosis of skin and mucous membranes of the throat, and effects on lungs, nerve fibres, kidneys, liver, and the pupil response to light.

Gastrointestinal irritation has been reported following ingestion of phenol. Local effects following dermal exposure range from painless blanching or erythema to corrosion and cell death.

Systemic effects associated with exposure to phenol include cardiac dysrhythmias, metabolic acidosis, hyperventilation, respiratory distress, acute renal failure, renal damage, dark urine, methaemoglobinaemia, neurological effects (including convulsions), cardiovascular shock, coma and death. The lowest reported dose resulting in a human death was 4.8 g by ingestion; death occurred within 10 min.

The potential for poisoning through inhalation of phenol vapours has long been recognised, however no cases of death following this route of exposure have been reported. Symptoms associated with inhalation of phenol included anorexia, weight loss, headache, vertigo, salivation and dark urine.

There is no evidence that Phenol is a sensitising agent.

### ***Dose response***

The lowest NOAEL values identified in animal experiments are for kidney and developmental effects, and in rats lie within the range of 12-40 mg/kg body weight per day. Using an uncertainty factor of 200, exposure in the range 60 to 200 µg/kg body weight per day is recommended as the upper limit for the total daily intake (TDI). Based on the upper-limit estimate for human daily intake of 100 µg/kg body weight per day, it is concluded that on average the general population exposure to phenol from all sources is below this range.

The USEPA have nominated a Reference Dose for phenol of 0.6 mg/kg/day, based on the NOAEL of 60 mg/kg/day in a developmental study in rats and an uncertainty factor of 100.

The Ministry of Health has not set a guideline value for the NZDWS.

*The reference dose of 0.6 mg/kg/day has been adopted for deriving the acceptance criteria.*

## **Cresols**

**Primary reference**

WHO (1995) "IPCS Environmental Health Criteria 168, Cresols"

Cresols are also known as methylphenols and have 3 possible isomers (ortho, meta, and para).

**Kinetics and metabolism**

Cresols are rapidly absorbed following oral or dermal exposure, and are distributed to all major organs. Following absorption cresols are largely metabolised through the glucuronidation and sulfation processes and eliminated as conjugates in the urine.

Significant quantities of cresols are also excreted in the bile, however, most of the cresols excreted in the bile are hydrolysed by the gut bacteria and reabsorbed.

In humans, endogenous p-cresol production occurs by anaerobic gut bacteria from tyrosine, and amino acids. Thus, it has been reported that an average of 50 mg of p-cresol is excreted in urine daily by healthy adults.

**Animal toxicity**

The available information indicates that all three isomers of cresols are toxic to rodents in dose-related manners with mice being more sensitive than rats. Systemic toxicity and death can result from all routes of exposure, although acute toxicity following exposure to cresol vapours is less likely due to the low vapour pressure of these compounds.

Cresols are strong skin and eye irritants. Oral and inhalational exposure to cresols has been associated with reproductive toxicity in female mice and rats, however no major compound-related reproductive toxicity has been reported in studies involving male rodents. O- and p-cresols cause mild fetotoxicity in the rats and rabbits, however only minor developmental effects have been reported.

**Genotoxicity and carcinogenicity**

The three cresol isomers have produced positive results in genetic toxicity studies both individually and in cresol mixtures. Cresols have been classified as possible human carcinogens (Class C) by the USEPA based on an increased incidence of skin papillomas in mice as part of a tumour initiation-promotion study.

**Human toxicity**

Oral exposure to cresols in humans mainly affects the blood and kidneys, although effects on the lungs, liver, heart and central nervous system have also been reported. Acute dermal exposure has been associated with skin burns, scarring and systemic toxicity. High level, acute exposure to cresols may result in coma and death.

**Dose response**

The USEPA have nominated an RfD for cresol (the ortho and meta isomers) of  $5.0 \times 10^{-2}$  mg/kg/day, based on neutrotoxicity in rat studies and an uncertainty factor of 1000. An RfD for p-cresol has not been nominated by the USEPA in the Integrated Risk Information System (IRIS) database. A similar RfD ( $5.0 \times 10^{-3}$  mg/kg/day) had previously been nominated for p-cresol in the USEPA Health Effects Assessment Summary Tables (HEAST), however this was not ratified following review by the USEPA.

No Slope Factor has been nominated for cresols despite them being classified as a Class C chemical (possible human carcinogen).

The WHO has determined an NOAEL of 50 mg/kg/day for all three isomers based on the results of subchronic studies. WHO applied an uncertainty factor of 300, an ADI of 0.17mg/kg/day.

*For the purpose of deriving soil and groundwater criteria, the USEPA Reference Dose for o-, m-m p-Cresol of 0.05 mg/kg/day for cresols has been adopted.*

**Cyanide**

**Primary reference**

Turczynowicz L. (1993) "The Assessment and Management of Gasworks Sites" Proc. 2nd National Workshop on the Health Risk Assessment and Management of Contaminated Land, SA Health Commission.

WHO (1996) "Guidelines for Drinking-Water Quality-Health criteria and other supporting information".

**Kinetics and metabolism**

Cyanide is absorbed following inhalation and ingestion and via the eye and skin, although the rate of adsorption depends very heavily on the form of cyanide (eg. free cyanide compared to complex cyanide). Cyanide is rapidly distributed via the blood to all organs and tissues. Cyanide ions exhibit a high affinity for haemoglobins in the red blood cells and plasma proteins.

Metabolism of cyanide in the liver occurs via the enzyme rhodanase, converting cyanide to thiocyanate. In humans, the metabolism occurs within 20 min to 1 hour following exposure. Cyanide is excreted primarily in the urine in the form of thiocyanate.

**Animal toxicity**

The mechanism of cyanide toxicity is associated with the ability of cyanide to bind to heme moiety and proteins. Dissociation of hydrogen cyanide and cyanide salts in vivo releases cyanide ions that disrupt enzymes systems by complexing with heavy metal ions contained in the enzyme systems. For example, cyanide (CN<sup>-</sup>) forms a stable complex with ferric ion (Fe<sup>3+</sup>) in the cytochrome oxidase enzymes consequently inhibiting oxidase, the terminal oxidase in the mitochondrial respiratory chain and causing cytotoxic anoxia (oxygen depletion with the cell).

The target organs of cyanide include the central nervous system, cardiovascular and respiratory systems and the thyroid.

Developmental toxicity has been observed in rats orally exposed to cyanide, with a LOAEL of approximately 51.2 mg/kg CN<sup>-</sup> per day reported.

Effects on behavioural patterns and serum biochemistry were observed in pigs exposed for 6 months at 1.2 mg/kg.bw/day (nominated as a LOAEL), pigs may be more sensitive to cyanide than many of the other species tested.

**Genotoxicity and carcinogenicity**

Cyanide has not been shown to be genotoxic and has not been associated with carcinogenic effects in animals or humans.

**Human toxicity**

A human oral LD<sub>50</sub> of cyanide was estimated to be 1.52 mg/kg based on reported incidences of abuse. A dermal LD<sub>50</sub> of 100 mg/kg bw has also been estimated for HCN.

Chronic exposure to low levels of cyanide salts has been associated with enlargement of the thyroid gland in humans. Persistent neuropsychiatric effects resulting from one or more acute exposure episodes have also been observed.

Exacerbation of vitamin B12 deficiency and increased incidence of goitre in humans have been associated with exposure to cyanide.

**Complex cyanides**

There is limited information on the health effects associated with exposure to complex cyanides. In general, the toxicity of these complexes is expected to be low compared to the toxicity of free cyanides and related to the degree of dissociation forming free cyanide.

In a 90-day subchronic feeding study of rats using sodium ferrocyanide in the diet, a NOEL of 0.05 % (in the diet) was established, which equated to an intake of 25 mg/kg bw/day.

**Dose response**

The USEPA has nominated an RfD of 0.02 mg/kg/day for cyanide based on a chronic oral rat study, which reported a NOAEL of 10.8 mg/kg/day for decreased body weight, thyroid effects and nerve degeneration, with an uncertainty factor of 100 and modifying factor of 5 applied.

In derivation of the NZDWS, the Ministry of Health adopted a tolerable daily intake for cyanide (free) of 0.012 mg/kg/day, based on the LOAEL of 1.2 mg/kg/day for behavioural patterns and serum bichemistry from the subchronic study in pigs and application of an uncertainty factor of 100.

Turczynowicz (1993) indicated an ADI for complex cyanide of 0.025 mg/kg/day which was based on the 90-day subchronic study by the Gas Research Institute, using rats exposed to sodium ferrocyanide in diet. The ADI was derived from the NOAEL of 25 mg/kg/day for kidney effects, with a safety factor of 1000 applied.

*For the purposes of deriving soil and water acceptance criteria, an Acceptable Daily Intake of 0.01mg/kg/day has been adopted for free cyanide and 0.025 mg/kg/day for complex cyanide.*

**Table A4.3 Summary of dose response factors**

Contaminant	Carcinogenic Category <sup>(1)</sup>	Parameter <sup>(2)</sup>	Source			Adopted
			USEPA <sup>(3)</sup>	Australian <sup>(4)</sup>	NZ <sup>(5)</sup>	
<b>Non-carcinogenic PAHs</b>						
naphthalene		oral RfD	NA	4 x 10 <sup>-3</sup>		4 x 10 <sup>-3</sup>
acenaphthene	D	oral RfD	6 x 10 <sup>-2</sup>			6 x 10 <sup>-2</sup>
acenaphthylene	D	oral RfD	3 x 10 <sup>-2</sup>			3 x 10 <sup>-2</sup>
anthracene	D	oral RfD	3 x 10 <sup>-1</sup>			3 x 10 <sup>-1</sup> 3 x 10 <sup>-1</sup>
phenanthrene	D	oral RfD	3 x 10 <sup>-2</sup>			3 x 10 <sup>-2</sup>
fluoranthene	D	oral RfD	4 x 10 <sup>-2</sup>			4 x 10 <sup>-2</sup>
fluorene	D	oral RfD	4 x 10 <sup>-2</sup>			4 x 10 <sup>-2</sup>
pyrene	D	oral RfD	3 x 10 <sup>-2</sup>			3 x 10 <sup>-2</sup>
<b>Carcinogenic PAHs</b>						
benzo (a) pyrene	B2	oral SF	7.3		0.5	7.3
<b>Phenolics</b>						
phenol	D	oral RfD <sup>(8)</sup>	6 x 10 <sup>-1</sup>	6 x 10 <sup>-1</sup>	NA	6 x 10 <sup>-1</sup>
cresol (o, m)	C	oral RfD	5 x 10 <sup>-2</sup>		NA	5 x 10 <sup>-2</sup>
2,4-dimethylphenol		Oral RfD	0.02		NA	0.02
<b>BTEX</b>						
Benzene	A	oral SF inhal UR	0.029 0.000008		0.03	0.029
Toluene	D	oral RfD inhal UR	0.2 0.4		0.22	0.2
Ethylbenzene	D	oral RfD inhal RfC	0.1 1		0.1	0.1
Xylene	D	oral RfD	2		0.18	0.18
<b>Inorganics</b>						
Cyanide-Free Complex	D	oral RfD oral RfD	0.02	0.01 0.025	0.012	0.01 0.025

1 USEPA Carcinogen Classification System.

2 Units: oral SF, (mg/kg/day)<sup>-1</sup>; inhalation UR, (µg/m<sup>3</sup>)<sup>-1</sup>; oral RfD, mg/kg/day.

3 From USEPA Integrated Risk Information System (1993, 1995 and 1996).

4 Monograph Series "National Workshop on the Health Risk Assessment and Management of Contaminated Sites" (1991 and 1993).

5 Guidelines for Drinking Water Quality and Management in New Zealand (1995).

6 Refer to discuss of PAH Toxic Equivalent Factors in 4.1.2(c).

## Appendix 4B

# Ecologically-based investigation thresholds

Guidelines for ecological risk assessment in Australia which will incorporate ecological investigation levels, are currently being developed under ANZECC. The current ANZECC Guidelines present environmental investigation thresholds for a range of chemicals, some of which may be of concern at former gasworks sites. The ANZECC environmental investigation level guidelines have been developed based on consideration of phytotoxicity (protection of plant life), background concentrations (particularly for heavy metals) and other considerations depending on the contaminant. While environmental investigation guideline values have been developed for a range of metals, values have been nominated for few organic contaminants. The environmental investigation level guidelines nominated in the ANZECC Guidelines are presented in Table 4A.3.

The ANZECC Guidelines note that where an environmental investigation level guideline has not been nominated for a specific chemical, reference may be made to the Dutch B guidelines. The Dutch guidelines have since been revised and the ABC level guidelines have been replaced with Target and Intervention Values based on human health and ecological considerations. In the interim the average of the Target and Intervention Values (as used by the Dutch as an investigation threshold) has been proposed as an environmental investigation level guideline where the ANZECC Guidelines do not nominate a value. The Dutch Target and Intervention Values for gasworks related contaminants are presented in Table 4B.1 for information.

**Table 4B.1 Environmental soil quality guidelines (mg/kg)**

Substance	ANZECC Environmental Investigation Level	Dutch Target Values	Dutch Intervention Value
<b>Heavy Metals</b>			
Antimony Sb	20	-	-
Arsenic As	20	29	55
Cadmium Cd	3	0.8	12
Chromium Cr	50	100	380
Copper Cu	70	36	190
Lead Pb	300	85	530
Manganese Mn	500	-	-
Mercury Hg	1	0.3	10
Nickel Ni	60	35	210
Tin Sn	50	20	-
<b>Phenolic Compounds</b>			
Phenols	-	0.05	40
Cresols		DL <sup>1</sup>	5
<b>BTEX</b>			
Benzene	1	0.05	1
Toulene		0.05	130
Ethylbenzene		0.05	50
Xylene		0.05	25

Substance	ANZECC Environmental Investigation Level	Dutch Target Values	Dutch Intervention Value
<b>Polycyclic Aromatic Hydrocarbons (PAH)</b>			
PAH (total)		1	40
Benzene (a) pyrene		0.025	-
<b>Inorganics</b>			
Cyanide		1	20 free
		5	650 complex pH <5
		5	50 complex pH >5

1 DL denotes Direction Limit.

As part of the development of guidelines for ecological risk assessment, the ANZECC/NHMRC Technical Working Group on Contaminated Sites are developing ecological investigation levels, developed in accordance with the guidelines for ecological risk assessment. The ecological investigation levels will supersede the existing environmental investigation level guidelines. The guidelines for ecological risk assessment and the ecological investigation level guidelines are expected to be released in draft form in May, 1997.

The focus for ecological risk assessment and the derivation of ecological investigation thresholds has been sensitive land uses, such as residential, agricultural and various forms of open space. While there is a requirement for the protection of the off-site environment irrespective of land use, very limited protection of on-site ecosystems is usually required in the context of commercial and industrial land uses. In most cases, protection of the off-site environment (for example, via leaching of contaminants from soil into groundwater, followed by off-site transport) and human health on-site are the limiting considerations in the assessment of contaminated land where industrial or commercial use is proposed.

The resolution of policy objectives regarding the level of protection to be afforded to on-site ecosystems in the context of other land uses is a prerequisite for the development of ecological investigation level guidelines for sensitive land uses.

In the New Zealand context the following precedents have been established regarding the development of guideline values based on environmental protection.

# Appendix 4C

## Exposure equations

Specific forms of the general equations are presented in this attachment for the following exposure routes:

- ingestion of soil
- inhalation of volatiles
- dermal absorption
- consumption of home grown produce

### Ingestion of contaminated soil

The Chronic Daily Intake (CDI) may be determined by the following expression:

$$\text{CDI} = \frac{\text{C} \times \text{CF} \times \text{IR}_{\text{adj}} \times \text{EF} \times \text{MF}}{\text{AT}}$$

where C = concentration of species in the soil  
 CF = conversion factor =  $10^{-6}$  kg/mg  
 EF = exposure frequency  
 AT = averaging time = (ED x 365) days for non-carcinogens by convention or (70 years x 365) days for carcinogens, representing lifetime exposure, by convention (USEPA, 1989a)  
 MF = matrix factor, accounts for reduced bioavailability of contaminant due to binding to the soil matrix. In the absence of necessary information, MF usually taken as 1.0. (USEPA, 1989a)  
 $\text{IR}_{\text{adj}} =$  age adjusted ingestion rate  
 $= \frac{\text{ED}_i \times \text{IR}_i}{\text{BW}_i}$   
 where  $\text{ED}_i =$  exposure duration (yr) for age group 'i'  
 $\text{IR}_i =$  ingestion rate (mg/d) for age group 'i'  
 $\text{BW}_i =$  body weight (kg) for age group 'i'

The CDI determined is a weighted average, taking account of variation in body weight and ingestion rate with age.

### Inhalation of volatile contaminants

The Chronic Daily Intake (CDI) by inhalation of volatile may be determined by the following expression:

$$\text{CDI} = \frac{\text{IR} \times \text{C} \times \text{VF} \times \text{EF} \times \text{ED}}{\text{AT} \times \text{BW}}$$

where C = concentration of species in soil  
 VF = volatilisation factor  
 EF = exposure frequency  
 AT = averaging time = (ED x 365) days for non-carcinogens by convention or (70years x 365) days for carcinogens, a lifetime by convention  
 ED = exposure duration (yr)  
 IR = ingestion rate (mg/d)  
 BW = body weight (kg)

The significance of soil contamination by volatile components such as BTEX depends on the depth to the contaminated layer. Acceptance criteria based on the inhalation of volatiles have been derived for two assumed depths to the contaminated layer, as follows:

- **Surface soils, <1 m**

Surface contamination is of primary concern in health risk assessment due to the range of exposure routes that are likely to be complete. Normal digging activities, say, in a residential context are unlikely to extend beyond a depth of 1 m.

- **Sub-surface soils, >1 m**

The depth to contamination has an important impact on the rate of volatilisation of contaminants and on the relevant exposure pathways. Where contaminated soil is located at depths greater than 1 m it is assumed that normal users of the site are less likely to come in direct contact with contaminated soils. Hence Tier 1 Acceptance Criteria for this depth range do not consider ingestion of soil, dermal adsorption and home produce consumption.

In order to properly account for source depletion in volatilisation modelling it is necessary to make an assumption regarding the thickness of the contaminated zone. For the purposes of deriving acceptance criteria a thickness of 2 m has been assumed throughout.

Soil type has a significant impact on the rate at which contaminants may volatilise from soil, and particularly the rate at which vapours may diffuse through the soil column. Criteria for volatile contaminants may be developed for a range of soil types, reflecting the varying soil conditions likely to be encountered at gasworks sites. This approach was adopted in the development of guidelines for the oil industry. In order to streamline the presentation of acceptance criteria for gasworks sites, criteria have been developed for sand/sandy loam only. The assumed soil properties are relatively conservative, ie. they are likely to overestimate the emission of volatiles at most sites.

Table 4C.1 presents the assumed soil properties for use in volatilisation modelling.

**Table 4C.1 Soil Properties for Volatilisation Modelling**

Soil Type	Air Filled Porosity (unitless)	Water Filled Porosity (unitless)	Total Porosity (unitless)	Organic Carbon Content (%)	Bulk Density (tonne/m <sup>3</sup> )	Capillary Fringe Thickness (m)
Sand, sandy loam, silty sand	0.26	0.12	0.38	0.3	1.9	0.05

### Dermal absorption from contaminated soil

The Chronic Daily Intake (CDI) for dermal absorption from contaminated soil may be determined using the following expression:

$$CDI = \frac{C \times AH_{adj} \times AR \times AF \times EF \times PC}{AT}$$

- where: C= concentration of species in the soil  
 AR= area of exposed skin (face, neck, forearms, hands)  
 AF= absorption factor  
 EF= exposure frequency  
 AT= averaging time = (ED x 365) days for non-carcinogens by convention or (70 years x 365) days for carcinogens, a lifetime by convention  
 $AH_{adj} = \frac{AH_i \times ED_i}{BW}$   
 where  $AH_i$  = soil adherence (mg/cm<sup>2</sup>) for age group 'i'  
 $ED_i$  = exposure duration (yr) for age group 'i'  
 $Bw_i$  = body weight (kg) for age group 'i'

The CDI determined is a weighted average, taking into account variation in body weight, skin area and exposure patterns with age.

## Ingestion of produce

The Chronic Daily Intake (CDI) for ingestion of produce may be estimated using the following expression:

$$\text{CDI} = \frac{\text{C} \times \text{PUF} \times \text{IP}_{\text{adj}} \times \text{EF} \times \text{Pg}}{\text{AT}}$$

where: CP = concentration of species in soil (mg/kg)

PUF = product uptake factor (unitless)

EF = exposure frequency (d/yr)

AT = averaging time = (ED x 365) days for non-carcinogens by convention or (70 yrs x 365) days for carcinogens by convention

Pg = proportion of produce grown on-site

IP<sub>adj</sub> = age adjusted ingestion rate for produce

$$= \frac{\text{IP}_i \times \text{ED}_i}{\text{BW}_i}$$

where: IP<sub>i</sub> = ingestion rate for produce (kg/d) for age group 'i'

ED<sub>i</sub> = exposure duration (yrs) for age group 'i'

BW<sub>i</sub> = body weight (kg) for age group 'i'

The CDI estimated is a weighted average taking into account variation in body weight and produce consumption with age.

The development of acceptance criteria based on exposure via the consumption of produce depends on estimation of the plant uptake factor, PUF.

An empirical formula has been derived by Travis and Arms (1988) to simulate contaminant uptake by plants. The octanol-water partition coefficient ( $K_{ow}$ ) is the key parameter for making these predictions.

The bioconcentration factor is the measure of a chemical's potential to accumulate in an organism. For vegetation, this is defined as is the ratio of the concentration in aboveground parts (mg of compound / kg of dry plant) to the concentration in soil (mg of compound / kg of dry soil). The geometric mean functional regression method is used to determine the proper correlation between bioconcentration factors and  $K_{ow}$ .

This yields the equation

$$\log B_v = 1.588 - 0.578 \log K_{ow}$$

where:  $B_v$  = Bioconcentration Factor for Vegetation

$K_{ow}$  = Octanol Water Partition Coefficient.

The bioconcentration factor ( $B_v$ ) for an organic in vegetation is inversely proportional to the square root of the octanol-water partition coefficient ( $K_{ow}$ ).

$B_v$  is based on the dry weight of vegetation and the PUF is based on the fresh weight of vegetation which is 80% moisture.

$$\text{PUF} = \frac{10^{(1.588 - 0.578 \times \log K_{ow})}}{5}$$

where 5 = conversion of  $B_v$  from dry weight to fresh weight.

**Table 4C.2 Health Risk Based Acceptance Criteria - Agricultural Site Use**

Site Use	Residential	Exposure Frequency	350 d/yr	Exposure Dur (1-6 yrs)	6 yrs
Receptor	Children resident on site for up to 30 yrs	Averaging Time (carc) (non-carc)	70 yrs 30 yrs	Exposure Dur (7-30 yrs)	24 yrs
Target Risk	0.00001	Age Adjusted Ingestion factor	48.57 mg.yr/kg.d	Ingestion Rate (1-6 yrs)	100 mg/d
Target HI	1	Age adjusted dermal exposure factor	2.7E+03	Ingestion Rate (7-30 yrs)	25 mg/d
		Body weight	15 kg	Skin Area (1-6 yrs) (sq.cm)	2625
		Body weight	70 kg	Skin Area (7-30 yrs)(sq.cm.)	4700
				Soil Adherennce (mg/sq.cm.)	1
				Produce Ingestion (1-6 yrs, kg)	0.13
				Produce Ingestion (7-30 yrs, kg)	0.45
				Proportion of produce from contaminated source	1
				Proportion root produce	0.5

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d))	RfD (mg/kg/d)	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>1</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.05		3.00E-01			3.0E-01	3.0E-01					4.7E+04	3.6E+04	3.25E+01
cresol (o)	0.05		2.50E-02			2.5E-02	2.5E-02					3.9E+03	3.0E+03	5.20E+00
cresol (m)	0.05		2.50E-02			2.5E-02	2.5E-02					3.9E+03	3.0E+03	5.27E+00
cresol (p)														
<b>BTEX</b>														
benzene	0.05	2.90E-02		3.4E-04	3.4E-04			5.2E+02	1.9E+02	2.65E-01				
toluene	0.05		1.00E-01			1.0E-01	1.0E-01					1.6E+04	1.2E+04	5.88E+01
ethylbenzene	0.05		5.00E-02			5.0E-02	5.0E-02					7.8E+03	6.0E+03	5.14E+01
xylene	0.05		9.00E-02			9.0E-02	9.0E-02					1.4E+04	1.1E+04	1.07E+02
<b>Non-carcinogenic PAHs</b>														
naphthalene	0.01		2.00E-03			2.0E-03	2.0E-03					3.4E+02	1.2E+03	1.71E+00
acenaphthene	0.01		3.00E-02			3.0E-02	3.0E-02					4.7E+03	1.8E+04	8.59E+01
anthracene	0.01		1.50E-01			1.5E-01	1.5E-01					2.3E+04	8.9E+04	8.70E+02
fluorene	0.01		2.00E-02			2.0E-02	2.0E-02					3.1E+03	1.2E+04	8.10E+01
phenanthrene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	8.9E+03	8.82E+01
pyrene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	8.9E+03	1.54E+02
fluoranthene	0.01		2.00E-02			2.0E-02	2.0E-02					3.1E+03	1.2E+04	3.23E+02
acenaphthylene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	8.9E+03	5.25E+01
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.01	7.30E+00		1.4E-06	1.4E-06			2.1E+00	3.8E-00	1.79E-01				
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					3.9E+02		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					9.8E+02		

**Table 4C.3 Health risk based acceptance criteria - Agricultural Site use**

<sup>1</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.

## Estimation of target soil concentration - produce based

Contaminant	Target produce concentration (mg/kg)		Koc	Kow	Uptake Factor	1/(Plant Uptake Factor)	Target Soil Concentration (mg/kg)	
	Carcinogenic	Non-carcinogenic					Carcinogenic	Non-carcinogenic
<b>Phenolics</b>								
phenol		3.61E+01	1.60E+01	2.88E+01	1.11E+00	9.01E-01		3.25E+01
cresol (o)		3.01E+00	1.03E+02	8.91E+01	5.78E-01	1.73E+00		5.20E+00
cresol (m)		3.01E+00	3.46E+01	9.12E+01	5.70E-01	1.75E+00		5.27E+00
cresol (p)								
<b>BTEX</b>								
benzene	1.22E-01		8.30E+01	1.32E+02	4.61E-01	2.17E+00	2.65E-01	
toluene		1.20E+01	3.02E+02	5.37E+02	2.05E-01	4.89E+00		5.88E+01
ethylbenzene		6.02E+00	1.10E+03	1.41E+03	1.17E-01	8.55E+00		5.14E+01
xylene		1.08E+01	2.40E+02	1.82E+03	1.01E-01	9.89E+00		1.07E+02
<b>Non-carcinogenic PAHs</b>								
napthalene		2.41E-01	1.29E+03	1.02E+03	1.41E-01	7.09E+00		1.71E+00
acenaphthene		3.61E+00	4.60E+03	8.32E+03	4.20E-02	2.38E+01		8.59E+01
anthracene		1.80E+01	1.60E+04	2.82E+04	2.07E-02	4.82E+01		8.70E+02
fluorene		2.41E+00	5.01E+03	1.51E+04	2.97E-02	3.37E+01		8.10E+01
phenanthrene		1.80E+00	2.29E+04	2.88E+04	2.05E-02	4.88E+01		8.82E+01
pyrene		1.80E+00	3.80E+04	7.59E+04	1.17E-02	8.54E+01		1.54E+02
fluoranthene		2.41E+00	4.17E+04	1.66E+05	7.44E-03	1.34E+02		3.23E+02
acenaphthylene		1.80E+00	4.79E+03	1.18E+04	3.44E-02	2.91E+01		5.25E+01
<b>Carcinogenic PAHs</b>								
benzo(a)pyrene	4.85E-04		3.89E+05	9.55E+05	2.71E-03	3.69E+02	1.79E-01	
<b>Inorganics</b>								
cyanide (free)		3.01E-01						0.00E+00
cyanide (complex)		7.52E-01						0.00E+00

**Table 4C.4 Health risk based acceptance criteria - Standard residential site use (10% produce consumed)**

Site Use	Residential	Exposure Frequency	350 d/yr	Exposure Dur (1-6 yrs)	6 yrs
Receptor	Children resident on site for up to 30 yrs	Averaging Time (carc) (non-carc)	70 yrs 30 yrs	Exposure Dur (7-30 yrs)	24 yrs
Target Risk	0.00001	Age Adjusted Ingestion factor	48.57 mg.yr/kg.d	Ingestion Rate (1-6 yrs)	100 mg/d
Target HI	1	Age adjusted dermal exposure factor	2.7E+03	Ingestion Rate (7-30 yrs)	25 mg/d
		Body weight	15 kg	Skin Area (1-6 yrs) (sq.cm)	2625
		Body weight	70 kg	Skin Area (7-30 yrs)(sq.cm.)	4700
				Soil Adherennce (mg/sq.cm.)	0.5
				Produce Ingestion (1-6 yrs, kg)	0.13
				Produce Ingestion (7-30 yrs, kg)	0.45
				Proportion of produce from contaminated source	0.1
				Proportion root produce	0.5

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d))	RfD (mg/kg/d)	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>2</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.05		3.00E-01			3.0E-01	3.0E-01					4.7E+04	7.2E+04	3.25E+02
cresol (o)	0.05		2.50E-02			2.5E-02	2.5E-02					3.9E+03	3.0E+03	5.20E+01
cresol (m)	0.05		2.50E-02			2.5E-02	2.5E-02					3.9E+03	3.0E+03	5.27E+01
cresol (p)														
<b>BTEX</b>														
benzene	0.05	2.90E-02		3.4E-04	3.4E-04			5.2E+02	1.9E+02	2.65E-01				
toluene	0.05		1.00E-01			1.0E-01	1.0E-01					1.6E+04	2.4E+04	5.88E+02
ethylbenzene	0.05		5.00E-02			5.0E-02	5.0E-02					7.8E+03	1.2E+04	5.14E+02
xylene	0.05		9.00E-02			9.0E-02	9.0E-02					1.4E+04	2.1E+04	1.07E+03
<b>Non-carcinogenic PAHs</b>														
naphthalene	0.01		2.00E-03			2.0E-03	2.0E-03					3.4E+02	2.4E+03	1.71E+01
acenaphthene	0.01		3.00E-02			3.0E-02	3.0E-02					4.7E+03	3.6E+04	8.59E+02
anthracene	0.01		1.50E-01			1.5E-01	1.5E-01					2.3E+04	1.8E+05	8.70E+03
fluorene	0.01		2.00E-02			2.0E-02	2.0E-02					3.1E+03	2.4E+04	8.10E+02
phenanthrene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	1.8E+04	8.82E+02
pyrene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	1.8E+04	1.54E+03
fluoranthene	0.01		2.00E-02			2.0E-02	2.0E-02					3.1E+03	2.4E+04	3.23E+03
acenaphthylene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	1.8E+04	5.25E+02
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.01	7.30E+00		1.4E-06	1.4E-06			2.1E+00	7.5E+00	1.79E-01				
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					3.9E+02		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					9.8E+02		

**Table 4C.5 Health risk based acceptance criteria - Standard residential site use (10% produce consumed)**

<sup>2</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.

## Estimation of target soil concentrations - produce based

Contaminant	Target produce concentration (mg/kg)		Koc	Kow	Uptake Factor	1/(Plant Uptake Factor)	Target Soil Concentration (mg/kg)	
	Carcinogenic	Non-carcinogenic					Carcinogenic	Non-carcinogenic
<b>Phenolics</b>								
phenol		3.61E+02	1.60E+01	2.88E+01	1.11E+00	9.01E-01		3.25E+02
cresol (o)		3.01E+01	1.03E+02	8.91E+01	5.78E-01	1.73E+00		5.20E+01
cresol (m)		3.01E+01	3.46E+01	9.12E+01	5.70E-01	1.75E+00		5.27E+01
cresol (p)								
<b>BTEX</b>								
benzene	1.22E+00		8.30E+01	1.32E+02	4.61E-01	2.17E+00	2.65E+00	
toluene		1.20E+02	3.02E+02	5.37E+02	2.05E-01	4.89E+00		5.88E+02
ethylbenzene		6.02E+01	1.10E+03	1.41E+03	1.17E+01	8.55E+00		5.14E+02
xylene		1.08E+02	2.40E+02	1.82E+03	1.01E-01	9.89E+00		1.07E+03
<b>Non-carcinogenic PAHs</b>								
naphthalene		2.41E+00	1.29E+03	1.02E+03	1.41E-01	7.09E+00		1.71E+01
acenaphthene		3.61E+01	4.60E+03	8.32E+03	4.20E-02	2.38E+01		8.59E+02
anthracene		1.80E+02	1.60E+04	2.82E+04	2.07E-02	4.82E+01		8.70E+03
fluorene		2.41E+01	5.01E+03	1.51E+04	2.97E-02	3.37E+01		8.10E+02
phenanthrene		1.80E+01	2.29E+04	2.88E+04	2.05E-02	4.88E+01		8.82E+02
pyrene		1.80E+01	3.80E+04	7.59E+04	1.17E-02	8.54E+01		1.54E+03
fluoranthene		2.41E+01	4.17E+04	1.66E+05	7.44E-03	1.34E+02		3.23E+03
acenaphthylene		1.80E+01	4.79E+03	1.18E+04	3.44E-02	2.91E+01		5.25E+02
<b>Carcinogenic PAHs</b>								
benzo(a)pyrene	4.85E-03		3.89E+05	9.55E+05	2.71E-03	3.69E+02		
<b>Inorganics</b>								
cyanide (free)		3.01E+00					1.79E+00	0.00E+00
cyanide (complex)		7.52E+00						0.00E+00

**Table 4C.6 Health risk based acceptance criteria - Standard residential site use (50% produce consumed)**

Site Use	Residential	Exposure Frequency	350 d/yr	Exposure Dur (1-6 yrs)	6 yrs
Receptor	Children resident on site for up to 30 yrs	Averaging Time (carc) (non-carc)	70 yrs 30 yrs	Exposure Dur (7-30 yrs)	24 yrs
Target Risk	0.00001	Age Adjusted Ingestion factor	48.57 mg.yr/kg.d	Ingestion Rate (1-6 yrs)	100 mg/d
Target HI	1	Age adjusted dermal exposure factor	2.7E+03	Ingestion Rate (7-30 yrs)	25 mg/d
		Body weight	15 kg	Skin Area (1-6 yrs) (sq.cm)	2625
		Body weight	70 kg	Skin Area (7-30 yrs)(sq.cm.)	4700
				Soil Adherennce (mg/sq.cm.)	0.5
				Produce Ingestion (1-6 yrs, kg)	0.13
				Produce Ingestion (7-30 yrs, kg)	0.45
				Proportion of produce from contaminated source	0.5
				Proportion root produce	0.5

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d))	RfD (mg/kg/d)	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>3</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.05		3.00E-01			3.0E-01	3.0E-01					4.7E+04	7.2E+02	6.51E+01
cresol (o)	0.05		2.50E-02			2.5E-02	2.5E-02					3.9E+03	6.0E+03	1.04E+01
cresol (m)	0.05		2.50E-02			2.5E-02	2.5E-02					3.9E+03	6.0E+03	1.05E+01
cresol (p)														
<b>BTEX</b>														
benzene	0.05	2.90E-02		3.4E-04	3.4E-04			5.2E+02	3.8E+02	2.65E-01				
toluene	0.05		1.00E-01			1.0E-01	1.0E-01					1.6E+04	2.4E+04	1.18E+02
ethylbenzene	0.05		5.00E-02			5.0E-02	5.0E-02					7.8E+03	1.2E+04	1.03E+02
xylene	0.05		9.00E-02			9.0E-02	9.0E-02					1.4E+04	2.1E+04	2.14E+02
<b>Non-carcinogenic PAHs</b>														
naphthalene	0.01		2.00E-03			2.0E-03	2.0E-03					3.1E+02	2.4E+03	3.41E+00
acenaphthene	0.01		3.00E-02			3.0E-02	3.0E-02					4.7E+03	3.6E+04	1.72E+02
anthracene	0.01		1.50E-01			1.5E-01	1.5E-01					2.3E+04	1.8E+05	1.74E+03
fluorene	0.01		2.00E-02			2.0E-02	2.0E-02					3.1E+03	2.4E+04	1.62E+02
phenanthrene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	1.8E+04	1.76E+02
pyrene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	1.8E+04	3.08E+05
fluoranthene	0.01		2.00E-02			2.0E-02	2.0E-02					3.1E+03	2.4E+04	6.47E+02
acenaphthylene	0.01		1.50E-02			1.5E-02	1.5E-02					2.3E+03	1.8E+04	1.05E+02
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.01	7.30E+00		1.4E-06	1.4E-06			2.1E+00	7.5E+00	3.58E-01				
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					3.9E+02		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					9.8E+02		

<sup>3</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.

**Table 4C.7 Health risk based acceptance criteria -Standard residential site use (50% produce consumed)**  
**Estimation of target soil concentrations - produce based**

Contaminant	Target produce concentration (mg/kg)		Koc	Kow	Uptake Factor	1/(Plant Uptake Factor)	Target Soil Concentration (mg/kg)	
	Carcinogenic	Non-carcinogenic					Carcinogenic	Non-carcinogenic
<b>Phenolics</b>								
phenol		7.22E+01	1.60E+01	2.88E+01	1.11E+00	9.01E-01		6.51E+01
cresol (o)		6.02E+00	1.03E+02	8.91E+01	5.78E-01	1.73E+00		1.04E+01
cresol (m)		6.02E+00	3.46E+01	9.12E+01	5.70E-01	1.75E+00		1.05E+01
cresol (p)								
<b>BTEX</b>								
benzene	2.44E-01		8.30E+01	1.32E+02	4.61E-01	2.17E+00	5.30E-01	
toluene		2.41E+01	3.02E+02	5.37E+02	2.05E-01	4.89E+00		1.18E+02
ethylbenzene		1.20E+01	1.10E+03	1.41E+03	1.17E-01	8.55E+00		1.03E+02
xylene		2.17E+01	2.40E+02	1.82E+03	1.01E-01	9.89E+00		2.14E+02
<b>Non-carcinogenic PAHs</b>								
napthalene		4.81E-01	1.29E+03	1.02E+03	1.41E-01	7.09E+00		3.41E+00
acenaphthene		7.22E+00	4.60E+03	8.32E+03	4.20E-02	2.38E+01		1.72E+02
anthracene		3.61E+01	1.60E+04	2.82E+04	2.07E-02	4.82E+01		1.74E+03
fluorene		4.81E+00	5.01E+03	1.51E+04	2.97E-02	3.37E+01		1.62E+02
phenanthrene		3.61E+00	2.29E+04	2.88E+04	2.05E-02	4.88E+01		1.76E+02
pyrene		3.61E+00	3.80E+04	7.59E+04	1.17E-02	8.54E+01		3.08E+02
fluoranthene		4.81E+00	4.17E+04	1.66E+05	7.44E-03	1.34E+02		6.47E+02
acenaphthylene		3.61E+00	4.79E+03	1.18E+04	3.44E-02	2.91E+01		1.05E+02
<b>Carcinogenic PAHs</b>								
benzo(a)pyrene	9.70E-04		3.89E+05	9.55E+05	2.71E-03	3.69E+02	3.58E-01	
<b>Inorganics</b>								
cyanide (free)		6.02E-01						0.00E+00
cyanide (complex)		1.50E+00						0.00E+00

**Table 4C.8 Health risk based acceptance criteria - High density residential site use**

Site Use	Residential high density	Exposure Frequency	350 d/yr	Exposure Duration (1-6 yrs)	6 yrs
Receptor	Children resident on site for up to 30 yrs	Averaging time (carc (non-carc))	70 yrs 30 yrs	Exposure duration (7-30 yrs)	24 yrs
Target risk	0.00001	Age adjusted ingestion factor	11.71 mg.yr/kg.d	Ingestion rate (1-6 yrs)	25 mg/d
Target HI	1	Skin area (1.6 yrs) (sq.cm.)	2625	Ingestion rate (7-30 yrs)	5 mg/d
		Skin area (7-30 yrs) (sq.cm.)	4700	Soil adherence (mg/sq.cm.)	0.1
		Body weight (1-6 yrs)	15 kg	Age adjusted dermal exposure factor	2.7E+03
		Body weight (7-30 yrs)	70 kg		

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d))	RfD (mg/kg/d)	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>4</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.05		3.00E-01			3.0E-01	3.0E-01					1.9E+05	3.6E+05	
cresol (o)	0.05		2.50E-02			2.5E-02	2.5E-02					1.6E+04	3.0E+04	
cresol (m)	0.05		2.50E-02			2.5E-02	2.5E-02					1.6E+04	3.0E+04	
cresol (p)														
<b>BTEX</b>														
benzene	0.05	2.90E-02		3.4E-04	3.4E-04			2.1E+03	1.9E+03					
toluene	0.05		1.00E-01			1.0E-01	1.0E-01					6.3E+04	1.2E+05	
ethylbenzene	0.05		5.00E-02			5.0E-02	5.0E-02					3.1E+04	6.0E+04	
xylene	0.05		9.00E-02			9.0E-02	9.0E-02					5.6E+04	1.1E+05	
<b>Non-carcinogenic PAHs</b>														
napthalene	0.01		2.00E-03			2.0E-03	2.0E-03					1.3E+03	1.2E+04	
acenaphthene	0.01		3.00E-02			3.0E-02	3.0E-02					1.9E+04	1.8E+05	
anthracene	0.01		1.50E-01			1.5E-01	1.5E-01					9.4E+04	8.9E+05	
fluorene	0.01		2.00E-02			2.0E-02	2.0E-02					1.3E+04	1.2E+05	
phenanthrene	0.01		1.50E-02			1.5E-02	1.5E-02					9.4E+03	8.9E+04	
pyrene	0.01		1.50E-02			1.5E-02	1.5E-02					9.4E+03	8.9E+04	
fluoranthene	0.01		2.00E-02			2.0E-02	2.0E-02					1.3E+04	1.2E+05	
acenaphthylene	0.01		1.50E-02			1.5E-02	1.5E-02					9.4E+03	8.9E+04	
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.01	7.30E+00		1.4E-06	1.4E-06			8.5E+00	3.8E+01					
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					1.6E+03		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					3.9E+03		

<sup>4</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.

**Table 4C.9 Health risk based acceptance criteria - Commercial site use**

Site Use	Commercial	Exposure Frequency	240 d/yr	Exposure Dur	20 yrs
Receptor	Industrial Adult Worker for 20 yrs	Averaging time (carc) (non-carc)	70 yrs 20 yrs	Ingestion rate	25 mg/d
Target risk	0.00001	Skin area (sq.cm.)	4700	Soil adherence (mg/sq.cm.)	1
Target HI	1	Body weight	70 kg		

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d) Oral	RfD (mg/kg/d) Oral	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>5</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.03		3.00E-01			3.0E-01	3.0E-01					1.3E+06	2.3E+05	
cresol (o)	0.03		2.50E-02			2.5E-02	2.05E-02					1.1E+05	1.9E+04	
cresol (m)	0.03		2.50E-02			2.5E-02	2.5E-02					1.1E+05	1.9E+04	
cresol (p)														
<b>BTEX</b>														
benzene	0.03	2.90E-02		3.4E-04	3.4E-04			5.1E+03	9.1E+02					
toluene	0.03		1.00E-01			1.0E-01	1.0E-01					4.3E+05	7.6E+04	
ethylbenzene	0.03		5.00E-02			5.0E-02	5.0E-02					2.1E+05	3.8E+04	
xylene	0.03		9.00E-02			9.0E-02	9.0E-02					3.8E+05	6.8E+04	
<b>Non-carcinogenic PAHs</b>														
naphthalene	0.006		2.00E-03			2.0E-03	2.0E-03					8.5E+03	7.6E+03	
acenaphthene	0.006		3.00E-02			3.0E-02	3.0E-02					1.3E+05	1.1E+05	
anthracene	0.006		1.50E-01			1.5E-01	1.5E-01					6.4E+05	5.7E+05	
fluorene	0.006		2.00E-02			2.0E-02	2.0E-02					8.5E+04	7.6E+04	
phenanthrene	0.006		1.50E-02			1.5E-02	1.5E-02					6.4E+04	5.7E+04	
pyrene	0.006		3.00E-02			1.5E-02	1.5E-02					1.3E+05	1.1E+05	
fluoranthene	0.006		2.00E-02			2.0E-02	2.0E-02					8.5E+04	7.6E+04	
acenaphthylene	0.006		1.50E-02			1.5E-02	1.5E-02					6.4E+04	5.7E+04	
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.006	7.30E+00		1.4E-06	1.4E-06			2.0E+01	1.8E+01					
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					1.1E+04		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					2.7E+04		

<sup>5</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.

**Table 4C.10 Health risk based acceptance criteria - Commercial site use (maintenance worker)**

Site Use	Commercial	Exposure frequency	50 d/yr	Exposure duration	20 yrs
Receptor	Worker for 20 yrs	Averaging time (carc)	70 yrs		
		(non-carc)	20 yrs	Ingestion rate	100 mg/d
Target risk	0.00001	Skin Area (sq.cm.)	4700	Soil adherence (mg/sq.cm.)	1.5
Target HI	1	Body weight	70 kg		

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d)) Oral	RfD (mg/kg/d) Oral	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>6</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.03		3.00E-01			3.0E-01	3.0E-01					1.5E+06	7.2E+05	
cresol (o)	0.03		2.50E-02			2.5E-02	2.5E-02					1.3E+05	6.0E+04	
cresol (m)	0.03		2.50E-02			2.5E-02	2.5E-02					1.3E+05	6.0E+04	
cresol (p)														
<b>BTEX</b>														
benzene	0.03	2.90E-02		3.4E-04	3.4E-04			6.2E+03	2.9E+03			5.1E+05	2.4E+05	
toluene	0.03		1.00E-01			1.0E-01	1.0E-01					2.6E+05	1.2E+05	
ethylbenzene	0.03		5.00E-02			5.0E-02	5.0E-02					4.6E+05	2.2E+05	
xylene	0.03		9.00E-02			9.0E-02	9.0E-02							
<b>Non-carcinogenic PAHs</b>														
naphthalene	0.06		2.00E-03			2.0E-03	2.0E-03					1.0E+04	2.4E+04	
acenaphthene	0.06		3.00E-02			3.0E-02	3.0E-02					1.5E+05	3.6E+05	
anthracene	0.06		1.50E-01			1.5E-01	1.5E-01					7.7E+05	1.8E+06	
fluorene	0.06		2.00E-02			2.0E-02	2.0E-02					1.0E+05	2.4E+05	
phenanthrene	0.06		1.50E-02			1.5E-02	1.5E-02					7.7E+04	1.8E+05	
pyrene	0.06		3.0E-02			3.0E-02	3.0E-02					1.5E+05	3.6E+05	
fluoranthene	0.06		2.00E-02			2.0E-02	2.0E-02					1.0E+05	2.4E+05	
acenaphthylene	0.06		1.50E-02			1.5E-02	1.5E-02					7.7E+04	1.8E+05	
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.006	7.30E+00		1.4E-06	1.4E-06			2.5E+01	5.8E+01					
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					1.3E+04		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					3.2E+04		

<sup>6</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.

**Table 4C.11 Health risk based acceptance criteria - Parkland/recreational site use**

Site Use	Parkland/recreational	Exposure Frequency	350 d/yr	Exposure Duration (1-6 yrs)	6 yrs
Receptor	Children resident on site for up to 30 yrs	Averaging time (carc)	70 yrs	Exposure duration (7-30 yrs)	24 yrs
		(non-carc)	30 yrs	Ingestion rate (1-6 yrs)	50 mg/d
Target risk	0.00001	Age adjusted ingestion factor	23.43 mg.yr/kg.d	Ingestion rate (7-30 yrs)	10 mg/d
Target HI	1	Skin area (1-6 yrs) (sq.cm.)	2625	Soil adherence (mg/sq.cm.)	1
		Skin area (7-30 yrs) (sq.cm.)	4700	Age adjusted dermal exposure factor	2.7E+03
		Body weight (1-6 yrs)	15 kg	Body weight (7-30 yrs)	70kg

Contaminant	Skin Absorption Factor	SF (1/(mg/kg/d))	RfD (mg/kg/d)	Acceptable CDI				Health Based Acceptance Criteria (mg/kg) <sup>7</sup>						
				Carcinogenic		Non-carcinogenic		Carcinogenic			Non-carcinogenic			
				Oral	Dermal	Oral	Dermal	Oral	Dermal	Produce	Oral	Dermal	Produce	
<b>Phenolics</b>														
phenol	0.025		3.00E-01			3.0E-01	3.0E-01					9.4E+04	7.2E+04	
cresol (o)	0.025		2.50E-02			2.5E-02	2.5E-02					7.8E+03	6.0E+03	
cresol (m)	0.025		2.50E-02			2.5E-02	2.5E-02					7.8E+03	6.0E+03	
cresol (p)														
<b>BTEX</b>														
benzene	0.025	2.90E-02		3.4E-04	3.4E-04			1.1E+03	3.8E+02					
toluene	0.025		1.00E-01			1.0E-01	1.0E-01					3.1E+04	2.4E+04	
ethylbenzene	0.025		5.00E-02			5.0E-02	5.0E-02					1.6E+04	1.2E+04	
xylene	0.025		9.00E-02			9.0E-02	9.0E-02					2.8E+04	2.1E+04	
<b>Non-carcinogenic PAHs</b>														
napthalene	0.005		2.00E-03			2.0E-03	2.0E-03					6.3E+02	2.4E+03	
acenaphthene	0.005		3.00E-02			3.0E-02	3.0E-02					9.4E+03	3.6E+04	
anthracene	0.005		1.50E-01			1.5E-01	1.5E-01					4.7E+04	1.8E+05	
fluorene	0.005		2.00E-02			2.0E-02	2.0E-02					6.3E+03	2.4E+04	
phenanthrene	0.005		1.50E-02			1.5E-02	1.5E-02					4.7E+03	1.8E+04	
pyrene	0.005		1.50E-02			1.5E-02	1.5E-02					4.7E+03	1.8E+04	
fluoranthene	0.005		2.00E-02			2.0E-02	2.0E-02					6.3E+03	2.4E+04	
acenaphthylene	0.005		1.50E-02			1.5E-02	1.5E-02					4.7E+03	1.8E+04	
<b>Carcinogenic PAHs</b>														
benzo(a)pyrene	0.005	7.30E+00		1.4E-06	1.4E-06			4.3E+00	7.5E+00					
<b>Inorganics</b>														
cyanide (free)			2.50E-03			2.5E-03	2.5E-03					7.8E+02		
cyanide (complex)			6.25E-03			6.3E-03	6.3E-03					2.0E+03		

<sup>7</sup> These criteria are based on 30 years, criteria for non-carcinogens are based on the most critical 6 years.