Schedule B7
Appendix A2

PAHs and phenols

Contents
PAHs and phenols

1 Benzo(a)pyrene

1.1 General

Several comprehensive reviews of polycyclic aromatic hydrocarbons (PAHs) and benzo(a)pyrene (BaP) in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 1995; WHO 1998; CCME 2008). The following provides a summary of the key aspects of these compounds that are relevant to the derivation of a soil HIL.

PAHs are a large group of organic compounds with two or more fused aromatic rings made up of carbon and hydrogen atoms. PAHs are formed from incomplete combustion of organic materials such as processing of coal, crude oil, combustion of natural gas, refuse, vehicle emissions, heating, cooking and tobacco smoking as well as natural processes including carbonisation. The natural background level is due to PAH production in plant species. Because of such widespread sources, PAHs are present almost everywhere. Food is considered to be the major source of human exposure to PAH due to the formation of PAH during cooking or from atmospheric deposition of PAHs on grains, fruits and vegetables (WHO 1998).

There are several hundred PAHs, including derivatives of PAHs. The best known (and studied) is BaP. While there are hundreds of PAHs, typically only 16 individual PAHs are analysed in site contamination investigations. These individual PAHs have been identified as the most significant based on: the amount of information available on each individual PAH; the toxicity (suspected to be more harmful than other PAHs); the greater chance of being exposed to these PAHs; and the fact that of all the PAHs analysed, the 16 selected are the most commonly reported at elevated concentrations on contaminated sites.

The major sources of PAHs to soils at any given location invariably contribute a mixture of PAHs, not just single compounds. Various PAH source types can be distinguished based on the characteristic compositions of PAH mixtures and information on the site history, but the contaminated soil matrix is nonetheless challenging from an environmental risk assessment perspective, since in a PAH contaminated soil there is likely to be a diverse compositional range of non-carcinogenic and carcinogenic PAHs of varying potency.

The major approach advocated by regulatory agencies such as the NEPC (1999; Fitzgerald 1991, 1998), US EPA, California EPA (OEHHA), Netherlands (RIVM 2001), the UK (EA 2002) and Canada (CCME 2008) for assessing the human health risks of PAH-containing mixtures involves the use of toxicity equivalence factors (TEFs). This approach relates the toxicity of other (potentially carcinogenic) individual PAHs to that of BaP, the most widely studied PAH.

There are more than a dozen sets of equivalency numbers that have been proposed over the last two decades. The most recent review of TEF and their basis, presented by CCME (2008) suggests the use of TEF recommended by the World Health Organisation (WHO 1998), with minor modifications. This is a scheme based on the order of magnitude cancer potency.

Any finer-scale assertions about relative potency for more generic application are hard to justify given the current state of knowledge and confounding influences such as the route of exposure, or non-additive effects in complex PAH mixtures. It is not currently possible to develop different relative potency schemes across different exposure routes (oral, dermal, inhalation), owing to a lack of data. Hence, the TEF adopted have been applied for all routes of exposure for the carcinogenic PAHs assessed.

Application of the TEFs are relevant to the assessment of PAHs that are considered to be carcinogenic. Other PAHs that are not carcinogenic should be assessed separately on an individual basis.

The following table presents a summary of the TEFs adopted for the assessment of carcinogenic PAHs (CCME 2008):

PAH	IARC classification	US EPA classification	TEF
Benzo(a)anthracene	2B	B2	0.1
Benzo(a)pyrene	1	B2	1
Benzo(b+j)fluoranthene	2B	B2	0.1
Benzo(k)fluoranthene	2B	B2	0.1
Benzo(g,h,i)perylene*	3	D	0.01
Chrysene	2B	B2	0.01
Dibenz(a,h)anthracene	2A	B2	1
Indeno(1,2,3-cd)pyrene	2B	B2	0.1

Notes: 1/A= Human carcinogen, 2A/B2= Probable human carcinogen, 2B/C=Possible human carcinogen, 3/D= Not classifiable.
* Benzo(g,h,i)perylene included due to positive findings in genotoxicity studies (WHO 1998). Note there are insufficient data available to determine carcinogenicity.

The toxic effects of different PAH compounds in a mixture are additive. Experimental evidence suggests that this is a fair assumption (Fitzgerald 1991, 1998; CCME 2008).

The following relates to the approach used to assess BaP in the derivation of HILs (which can be used for the assessment of BaP alone or for carcinogenic PAHs using the above TEFs).

1.2 Previous HIL

The derivation of the previous HIL (HIL A = 1 mg/kg) for BaP is presented by Fitzgerald (1991) and NEPC (1999). In summary, the HIL was derived on the basis of the following:

- Intakes associated with daily exposure by children and adults living near or on soil containing 1 mg/kg BaP were assessed on the basis of —
  - dermal absorption, with 1% BaP absorbed via the skin
  - ingestion, with 100% bioavailability assumed
  - inhalation, over 24 hours, with 100% bioavailability assumed.
- In comparison to background intakes of BaP, intakes from soil at 1 mg/kg are low but higher intakes may be nearing a significant contribution. Adoption of 1 mg/kg was considered appropriate also due to the potential for further review by the US EPA where reference values for BaP may change.

Further review of BaP (and PAHs using TEFs) by Fitzgerald (1998) on the basis of a derived modified benchmark dose calculated a value of 5 mg/kg on the basis of soil ingestion only.

1.3 Significance of exposure pathways

Oral bioavailability

A study by Hansen et al. (2007) demonstrated bioavailability of PAHS in three different soil samples ranging from 14% to 40% using an *in vitro* bioavailability model that simulates gastric digestion. In addition, the Massachusetts DEP uses a relative absorption fraction of 28% for PAHs (MADEP 2008) in its risk assessment program. In addition, it is noted that BaP (and PAHs) present in bitumen fragments are largely immobile and typically have a low bioavailability.

However, as bioavailability is highly site and source-specific, insufficient data are available to adequately define a value that differs from the default approach of 100% oral bioavailability. It is noted that a site-specific assessment of bioavailability can be undertaken where required.

Dermal absorption

Review of dermal absorption of BaP has been conducted by MfE (2010). This review has identified the following, based on studies on animals and humans (rather than modelled as presented by CCME]2008]):

- As BaP is actively metabolised in the skin, it is relevant to include both the amount that passes through the skin and that which remains bound to the skin to estimate dermal uptake.
- The US EPA (2004) recommends a dermal absorption factor of 0.13 (13%), which is based on data from Wester et al. (1990). These authors indicate that 13.2% of BaP in soil was absorbed by rhesus monkeys over a 24-h period. However, they also indicate that a reduced amount (1.4%) was absorbed into human skin from soil over the same time period, although no partitioning into human plasma occurred, i.e. the BaP remained bound to the skin.
- Another study on the dermal absorption of BaP from soils also showed that a minimal amount (0.1%) of BaP was absorbed through pig skin and 1.7% and 3.5% remained bound to the skin when BaP in aged sandy and clay soils was applied to the skin (Abdel-Rahman et al. 2002). A higher amount (3.3% and 8.3% in clay and sandy soils, respectively) was absorbed when non-aged soil (i.e. freshly spiked) was applied to the skin.
- A more recent study with human skin showed greater absorption through the skin, with approximately 7% of BaP passing through when applied as freshly spiked soil (Moody et al. 2007). A further 7% remained bound to the skin.
- As ageing soils decrease the bioavailability of BaP, the dermal absorption data from freshly spiked soils can provide a 'worst-case' estimate of dermal absorption. The geometric mean of dermal absorption using freshly spiked soils from the above studies (including *in vivo* studies) is 6%, while using data for aged soils yields a geometric mean of 2.6% skin (Abdel-Rahman et al. 2002).

Review by MfE (2010) resulted in the adoption of a dermal absorption factor of 2.6%, the arithmetic mean of data from aged soil (Abdel-Rahman et al. 2002). In the derivation of soil HILs in this review, the higher arithmetic mean value of 6%,based on data from freshly spiked soil and noted by MfE (2010) as a worst-case value that is supported by studies from Wester et al. (1990), Abdel-Rahman et al. (2002) and Moody et a.l (2007), has been adopted to ensure the value is relevant for all source types.

Inhalation of dust

BaP (and other carcinogenic PAHs) are not considered sufficiently volatile to be of significance and inhalation exposures associated with particulates outdoors and indoors are expected to be of less significance than ingestion of soil. While likely to be negligible, potential inhalation exposures associated with dust have been considered in the HIL derived.

Plant uptake

CCME (2008) notes that concentrations of PAHs in uncooked produce depends principally on its source. Plants grown on PAH-contaminated soils, however, have only a limited ability to take in through the roots and translocate anthropogenic PAHs to the above-ground plant biomass – especially for higher molecular weight PAHs. One mode of plant contamination is via the deposition of PAH-containing fine particulates onto plant surfaces.

PAHs may be bound within soils (via lignification), mineralised (ultimately to co_2 and water) or metabolised outside or within the plant (CCME 2008). Higher molecular weight PAHs such as BaP (and other carcinogenic PAHs) are considered persistent and are strongly absorbed to the soil. Lipophilic organic compounds such as PAHs (and BaP), with a low solubility in water, high Henrys law constant and high kow($> 10^4$) are bound strongly to the root surface and/or soils and are not readily translocated within plants (Schnoor 1997). These generally tend to partition into the epidermis or outer layers of the root tissue (or peel) and remain there bound to lipids in cell walls; transfer into the inner root or xylem is very slow or nonexistent. CCME (2008) notes that the general consensus in the literature is that the root uptake pathway of organic contaminants such as hydrocarbons and PAH constituents from the soil by plants is extremely limited, particularly for the heavier PAHs such as BaP.

On the basis of the above, plant uptake has not been considered in the derivation of HIL A. However, it is noted that if plant uptake were considered (using the equations presented in Appendix B), intakes derived from this source are low and do not significantly contribute to the HIL (<1%).

Intakes from other sources – background

Intakes of BaP from sources other than soil have been considered by Fitzgerald (1991) to range from 0.166-1.6 µg/day (from US EPA 1980) with intakes derived from food identified as the most significant. While more detailed reviews are available on potential intakes of BaP (CCME 2008), background intakes are not considered in the derivation of an HIL for BaP as a non-threshold approach has been adopted.

1.4 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 1987) has classified BaP as 2A: probable human carcinogen.

The US EPA has classified BaP as B2: probable human carcinogen.

Review of available values/information

BaP has been shown to be carcinogenic via all routes of exposure. BaP is an indirect carcinogen, that is, its carcinogenicity results from its metabolites, primarily various epoxides, as opposed to BaP itself. Several different types of tumours have been observed as a result of exposure to BaP, although tumour development is closely related to route of administration, that is, dermal application induces skin tumours and oral administration induces gastric tumours. Exposure to BaP causes disruption to cellular genetic material; in particular, DNA adducts are formed as a result of exposure and BaP is considered to be a genotoxic carcinogen (WHO 1998).

In addition, BaP has been demonstrated to be a skin irritant and dermal sensitiser (WHO 1998).

The US EPA (2005) has identified that BaP (and carcinogenic PAHs assessed on the basis of a TEF) are considered to act via a mutagenic mode of action and recommends that susceptibility associated with early lifetime exposures be addressed. No non-threshold values available for BaP have been derived to specifically address early lifetime susceptibility and hence these issues may need to be addressed when characterising exposure to BaP.

On this basis, a peer-reviewed non-threshold reference value is recommended for BaP. The following non-threshold values are available from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2004)	Not available	Current guideline of 0.00001 mg/L established in ADWG (NHMRC 2004 and draft 2009) based on the consideration of health effects in relation to the limit of determination for analysis. The assessment provided by the WHO is noted.
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	SF = 0.5 $(mg/kg/day)^{-1}$ UR =$8.7x10^{-5}$ $(ng/m^3)^{-1}$	Oral slope factor (SF) derived (WHO DWG 2008) based on an oral carcinogenicity study and a two-stage birth-death mutation model. Inhalation unit risk (UR) derived (WHO 2000) based on observations in coke oven workers to mixtures of PAHs. It is noted that the composition of PAHs to which coke oven workers are exposed may differ from that present in ambient air, or derived from soil contamination. It is noted that an inhalation UR is in the same order of magnitude as that derived using a linear multistage model associated with lung tumours in a rat inhalation study from coal tar/pitch condensation aerosols.
MfE (2010)	SF = 0.233 $(mg/kg/day)^{-1}$	Review of the carcinogenic reference values available for oral intakes by MfE (2010) considered the range of values available and differences in approaches adopted for low dose extrapolation. The application of cross-species scaling appeared to be the most significant factor affecting the cancer potency estimates. It was recommended that cross-species scaling should not be applied, consistent with the approach outlined in NHMRC (1999). Review of available studies (14 risk estimates using 4 databases) resulted in the calculation of a geometric mean based on data without scaling which was recommended for use in the derivation of a soil guideline value.
UK (EA 2002)	Derived index doses from WHO evaluations	Oral index dose derived on the basis of WHO approach and a lifetime cancer risk of 10^{-5}. Inhalation index dose based on WHO approach and adopting an air guideline of 0.25 ng/m^3. The air guideline is equivalent to a lifetime cancer risk of $4x10^{-5}$.
RIVM (2001)	SF = 0.2 $(mg/kg/day)^{-1}$	Oral SF derived by RIVM based on a chronic oral carcinogenic rat study and linear multi-stage model. The study considered was more recent than considered by the WHO. No inhalation assessment is provided by RIVM.
CCME (2008)	SF = 2.3 $(mg/kg/day)^{-1}$	Oral SF derived from a less than lifetime diet study on inbred CFW-Swiss mice associated with incidence of papillomas and squamous cell carcinomas and linear extrapolation. This is the same study as used by the US EPA in the derivation of their oral SF. The CCME review also noted that dermal exposures and primary oral exposures result in different kinds of cancers. Health Canada is currently reviewing data with respect to the derivation of a dermal cancer slope factor, which may require consideration when peer-reviewed and published. The oral SF has been used to derive a soil guideline associated with exposures via oral, dermal and inhalation exposures.
OEHHA (CEPA 1999)	SF = 11.5 $(mg/kg/day)^{-1}$ UR =0.0011 to0.0033 $(ug/m^3)^{-1}$	Oral SF derived using the same model and study as reported by the US EPA (IRIS, 2010) and CCME (2008), with the upper end of the range of values adopted by OEHHA. Inhalation UR derived on the basis of a respiratory tract tumours in an inhalation study in hamsters and a linearised multi-stage model.
US EPA (IRIS 2010)	SF = 7.3 $(mg/kg/day)^{-1}$	Oral SF (last reviewed in 1994) derived on the basis of the same study considered by CCME (above) where a range of slope factors were derived (4.5 to 11.7 $(mg/kg/day)^{-1}$). The geometric mean was adopted as the recommended SF for derivation of a drinking water criteria. No assessment of inhalation toxicity is available.

There are a wide range of non-threshold reference values available for oral intakes of BaP. The most recent review, where the methodology used for low dose extrapolation was reviewed, was conducted by MfE (2010). The evaluation presented considered all the available and relevant studies noted in the above tables and identified an oral reference value based on the geometric mean. This value is considered appropriate for the derivation of HILs. However, it is noted that the reference document remains a draft at the time of preparation of this evaluation; hence, additional consideration of a finalised peer-reviewed reference value has also been presented.

Based on the available published peer-reviewed sources, the oral reference value available from the WHO DWG (2008) can also be considered (remains current and relevant) in the derivation of soil HILs. The WHO oral reference value is similar to the value derived by RIVM (2001) and has been adopted by the UK (EA 2002).

The data available on inhalation exposures is dominated by occupational studies associated with exposure to coke oven emissions or coal tar pitch aerosols. BaP is not volatile and hence the relevance of these studies to the assessment of dust issues derived from contaminated sites is not clear. It is therefore recommended that the WHO oral reference value be considered for the assessment of all pathways of exposure.

1.4.1 Note on dermal exposures

BaP is suggested to act largely as a point-of-contact carcinogen (Knafla et al. 2006), as opposed to systemically; hence, it is more appropriate to derive soil guideline values for the dermal route of exposure using a route-specific SF, as opposed to considering it an addition to oral exposure.

For most compounds, such data are not available; however, for BaP, Knafla et al. (2006) have derived a dermal SF for BaP of 25 $(mg/kg/day)^{-1}$. This study examined all relevant studies and ultimately derived an average SF from three mouse skin-painting studies. Review of this study by CCME (2008) and MfE (2010) have noted that the approach adopted requires further review and consideration before being adopted. In particular, it is noted that the approach is a relatively untested and greater uncertainties exist in the extrapolation of dermal data derived from animals to humans than for the oral or inhalation route (Knafla et al. 2006). These uncertainties, coupled with the conservative approach used to quantify dermal exposures, suggest that at this stage the dermal SF should not be considered in the derivation of current HILs.

In addition, no other international agency has currently adopted the use of a dermal slope factor; hence, this approach is not recommended for use in the derivation of HILs. It is noted that CCME (2008) indicate that Health Canada are currently developing a dermal slope factor for BaP and further consideration of such values should be undertaken once these reviews have been completed.

Recommendation

On the basis of the discussion above the following TRVs have been adopted for BaP in the derivation of HILs:

Recommendation for BaP and carcinogenic PAHs as BaP TEF

Oral TRV = 0.233 $(mg/kg/day)^{-1}$ (MfE 2010) for all routes of exposure

Value has been compared with 0.5 $(mg/kg/day)^{-1}$ (WHO DWG 2008) for all routes of exposure

Dermal absorption factor = 0.06 (or 6%) (MfE 2010)

BaP equivalents to be determined for carcinogenic and potential genotoxic PAHs only using TEFs presented by CCME (2008)

Note early lifetime exposures to BaP may need to be addressed in the quantification of exposure as per US EPA (2005).

1.5 Calculated HILs for BaP and carcinogenic PAHs (as BaP TEF)

It is noted that the discussion above has identified that further consideration of early lifetime exposures to BaP may need to be considered in the quantification of exposure (calculated as per US EPA 2006).

Other uncertainties have also been noted in the above discussion, particularly in relation to the selection of the oral TRV (where the value from MfE (2010) may also be considered, although it is a draft) and dermal exposures. With respect to the derivation of HIL A, the following can be noted:

- HIL A = 20 mg/kg on the basis of the recommended oral TRV from MfE (2010) (also adopted for dermal exposures) and no additional consideration of early-lifetime exposures
- HIL A = 10 mg/kg on the basis of the oral TRV from WHO DWG (2008) (also adopted for dermal exposures) and no additional consideration of early-lifetime exposures
- HIL A = 6 mg/kg on the basis of the recommended oral TRV from MfE (2010) (also adopted for dermal exposures) and consideration of early-lifetime exposures[1]
- HIL A = 3 mg/kg on the basis of the oral TRV from WHO DWG (2008) and consideration of early-lifetime exposures[1]
- HIL A = 0.3 mg/kg on the basis of the recommended oral TRV from MfE (2010), but consideration of the dermal slope factor presented by Knafla et al. (2006) and no consideration of early lifetime exposures. Note that the HIL is lower (0.1 mg/kg) if early lifetime exposures are assessed for oral intakes.

With consideration of the uncertainties (particularly in relation to the assessment of dermal exposures) identified and the effect of these on the derived HIL A value (noted above), it is recommended that the lower value derived on the basis of the WHO DWG (2008) oral TRV (also adopted for dermal exposures) with consideration of early-lifetime exposures, that results in the calculation of HIL A = 3 mg/kg, be adopted. Hence, the following HILs are recommended for BaP and carcinogenic PAHs (assessed as BaP TEF):

HIL scenario	HIL* (mg/kg)	Percentage contribution from exposure pathways			
		Ingestion of soil/dust	Ingestion of home-grown produce	Dermal absorption of soil/dust	Inhalation (dust)
Residential A	3	47%	--	53%	<1%
Residential B	4	18%	--	82%	<1%
Recreational C	4	31%	--	69%	<1%
Commercial D	40	18%	--	82%	<1%

-- Pathway not included in derivation of HIL

* Noted that as the dermal absorption pathway dominates the derivation of HILs A, B and C and the exposure assumptions differ little between these scenarios, the HIL remains essentially unchanged. Note derived HILs to 2 significant figures presented in brackets.
Elevated levels of BaP in relatively immobile sources, such as bitumen fragments, do not represent a significant health risk.

1.6 Calculated HILs for total PAHs

The derived HILs above relate to BaP and carcinogenic PAHs calculated on the basis of a BaP TEF (refer to Schedule B7). However, there are several hundred PAHs, including derivatives of PAHs of which typically only 16 individual PAHs are analysed in site contamination investigations. These individual PAHs have been identified as the most significant based on: the amount of information available on each individual PAH; the toxicity (suspected to be more harmful than other PAHs); the greater chance of being exposed to these PAHs; and the fact that, of all the PAHs analysed, the 16 selected are the most commonly reported at contaminated sites.

[1] Based on guidance available from US EPA (2005), early lifetime exposures have been accounted for by the application of adjustment factors (ADAFs) to calculate the risk for different life stages: risk during the first 2 years of life (ADAF = 10), risk for ages 2 through to less than 16 years (ADAF = 3), and the risk for ages 16 through to 70 years (ADAF = 1). The total calculated risk for a lifetime is the sum of risk over all life stages.

Hence, to assist in the assessment of contaminated sites, it is relevant to also consider total PAHs. Of the PAHs reported, these will comprise BaP and carcinogenic PAHs and other non-carcinogenic PAHs where the following can be noted with respect to the derivation of HILs:

- BaP and carcinogenic PAHs assessed as BaP TEF should be assessed on the basis of the above HILs
- naphthalene is the most significant volatile PAH and therefore the assessment of this compound should address all significant pathways of concern, including vapour inhalation (not addressed in the HIL for total PAHs). The presence of this compound in soil should be assessed on the basis of relevant guidelines such as the health screening levels (HSLs) (Friebel & Nadebaum, to be finalised)
- the remaining PAHs are considered non-carcinogenic and include acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, phenanthrene and pyrene. Rather than review the toxicity of each individual non-carcinogenic PAH, the published relative potencies to BaP (or TEFs) available for these PAHs (WHO 1998; CCME 2008) suggest that individual non-carcinogenic PAHs are at least 100 to 1000 times less toxic/potent than BaP. On this basis, a factor of 100 has been applied to the calculated BaP HILs to establish HILs for total PAHs. Review of soil guidelines developed by the US EPA (regional screening levels, 2010) indicates that, based on consideration of the same pathways of exposure (soil ingestion, dermal contact and inhalation of particulates), health-based guidelines for non-carcinogenic PAHs are at least 10,000 times higher than the BaP guideline. Hence, the adoption of a factor of 100 as an additive total for other non-carcinogenic PAHs is considered reasonable
- the HILs for total PAHs are only relevant provided carcinogenic PAHs meet the BaP HILs and naphthalene also meets the relevant HSLs.

On the basis of the above, the following HILs are recommended for total PAHs (provided carcinogenic PAHs meet the BaP HIL and naphthalene meets the relevant HSL):

HIL scenario	**HIL (mg/kg)**
Residential A	300
Residential B	400
Recreational C	400
Commercial D	4000

1.7 References for PAHs

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2 Phenol

2.1 General

Several comprehensive reviews of phenol in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 2008; WHO 1994; Health Canada 2000; EA 2009). The following provides a summary of the key aspects of phenol that are relevant to the derivation of a soil HIL.

Phenol is a colourless to white to pale pink crystalline solid at room temperature and ambient pressure. Phenol has a distinctive aromatic, somewhat 'sickening', sweet and acrid odour. Phenol is soluble in water and miscible with most organic solvents (for example, acetone and benzene) (ATSDR 2008). Many substituted phenols exist, for example dimethyl and trimethylphenols. These have different toxicities from phenol (ATSDR 2008). The widely varying toxicities and difficulty of making a generic assumption on the likely composition of phenol mixtures mean presenting an HIL representing 'total phenols' is considered impractical.

Therefore if substituted phenols may be present, these should be analysed and assessed as separate compounds, rather than on the basis of the phenol HIL.

Phenol can occur naturally in the environment as a product of organic matter decomposition and combustion of wood. Phenol is manufactured for use in phenolic resins, disinfectant and antiseptic and as an intermediate in organic synthesis (ATSDR 2008). Anthropogenic sources of phenol into the environment include vehicle exhaust and waste streams associated with its manufacture. Predominantly, phenol is released as air emission resulting from venting. Phenol can also be released in the metabolic processes in which it occurs as an intermediate. For example, phenol can be produced from the degradation of organic wastes containing benzene, an organic compound found extensively in the environment. Its primary occurrence as a soil contaminant is in former gas works and coking works sites (ATSDR 2008).

2.2 Previous HIL

The derivation of the previous HIL (HIL A = 8500 mg/kg) for phenol is presented by Turczynowicz (1993) and NEPC (1999). In summary, the HIL was derived on the basis of the following:

- Background intakes were considered in the derivation of the previous HIL with the intakes from food, water and ambient air considered where available. Due to the lack of available data, the quantification of intakes was limited; hence, intakes from contaminated soil was taken to be 25% of the adopted ADI to address these limitations.
- A reference dose (RfD) of 0.6 mg/kg/day referenced from the US EPA, based on a no-observed-adverse-effect level (NOAEL) of 60 mg/kg/day and uncertainty factor of 100 was considered.
- Dermal absorption of phenol was considered to be 12%.
- Oral bioavailability of phenol was considered to be 100%.
- Based on intakes derived from soil (ingestion, dermal absorption and dust inhalation) a HIL of 8500 mg/kg was calculated.

2.3 Significance of exposure pathways

Oral bioavailability

Insufficient data is available to adequately define the bioavailability of phenol in the range of contaminated sites that may need to be considered in Australia. On this basis, a default approach of assuming 100% oral bioavailability has been adopted in the derivation of a HIL. It is noted that a site-specific assessment of bioavailability can be undertaken where required.

Dermal absorption

ATSDR (2008) notes that phenol is readily absorbed through the skin, and the skin is considered the primary route of entry during occupational exposure (when considered as a product rather than in soil). Dermal absorption of phenol from soil suggested dermal absorption occurred and maximum phenol penetration was within 2 and 4 hours after application.

No compound-specific dermal absorption value is available for phenol and hence the default value of 0.1 (10%) for semi-volatile compounds available from US EPA (2004) has been adopted.

Inhalation of dust

Phenol is not considered sufficiently volatile to be of significance and inhalation exposures associated with particulates outdoors and indoors are expected to be of less significance than ingestion of soil. While likely to be negligible, potential inhalation exposures associated with dust have been considered in the HIL derived.

Plant uptake

Phenols occur naturally in plants and soils. Since phenol and phenolics are relatively water soluble, they are present in the soil solution and are easily taken up by plants via root absorption and stored in different parts of the plant (CCME 1999). Although it has been shown that plants readily uptake phenol, bioaccumulation does not take place due to a high rate of respiratory decomposition of phenol to CO2. The potential for the uptake of phenol into home-grown produce has been considered in the derivation of HIL A.

This has been undertaken on the basis of the equations presented in Appendix B with the following parameters and plant uptake factors estimated:

Parameter	Value	Reference/Comment
Parameters		
Koc	187 (cm^3/g)	RAIS (2010)
log Kow	1.46	RAIS (2010)
Diffusivity in water	$1.03x10^{-5}$ (cm^2/s)	RAIS (2010)
Calculated plant uptake factors (mg/kg produce fresh weight per mg/kg soil)		
Green vegetables	0.204	calculated
Root vegetables	0.307	calculated
Tuber vegetables	0.244	calculated
Tree fruit	0.00098	calculated

It is noted that plants can metabolise phenol readily; hence, exposure through eating food derived from plants grown in phenol-containing soil is probably minimal and the above is likely to be conservative.

Intakes from other sources – background

Background intakes of phenol were estimated in the supporting documentation for the current HIL (Turczynowicz 1993). Due to the lack of available data, the quantification of intakes was limited; hence, intakes from contaminated soil was taken to be 25% of the adopted ADI to address these limitations.

No data is available on potential intakes of phenol in Australia from food, water, consumer products and air. Estimates of background intakes by RIVM (2001) suggest intake may be dominated by inhalation exposures and background intakes may comprise 1 µg/kg/day. A more detailed review of background intakes by UK (EA 2009) considered intakes from food (dominated by the use of phenol as a flavouring additive), water (insignificant compared with food intakes), and air and consumer products where the total intake was estimated to be approximately 390 µg/day (350 µg/day from oral sources and 40 µg/day from inhalation sources). These are higher than estimated by Health Canada (2000) where intakes by young children (0.5-4 years) were estimated to be 0.27 to 0.66 µg/kg/day; these are more consistent with intakes estimated by RIVM (2001).

If the more conservative estimates of background intakes available from the UK (EA 2009) were considered, for a child these would comprise approximately 10% of the recommended oral TRV and 25% of the recommended inhalation TRV. A conservative assumption that background intakes comprise approximately 30% (with rounding) of the TRV can be assumed.

2.4 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 1999) has classified phenol as Group 3: not classifiable.

It is also noted that the US EPA (last reviewed in 2002) has classified phenol as Group D: not classifiable.

Review of available values/information

Data of genotoxicity and carcinogenicity of phenol are inconclusive. Available data (RIVM 2001) in experimental animals suggest phenol can act as a tumour promoter. ATSDR (2008) noted that 'under certain conditions, especially at high doses, phenol has the potential to be genotoxic. However at the exposure levels likely to occur near hazardous waste sites, phenol is not anticipated to be genotoxic'. Hence, phenol (at least at concentrations expected at contaminated site) is not considered genotoxic. On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for phenol.

Few quantitative toxicity values are available; however, the following threshold values are available from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG	No evaluation available	
OCS (2008)	No evaluation available	
International		
WHO DWG	No evaluation available	
WHO (1994)	TDI = 0.06 to 0.2 mg/kg/day	Based on the range of NOAEL values associated with kidney and developmental effects in rats with the application of an uncertainty factor of 200 to get a range which is the recommended upper limit of the TDI. Some uncertainty is noted with respect to genotoxic potential and hence the evaluation provided is recommended to be periodically reviewed.
RIVM (2001)	TDI = 0.04 mg/kg/day TC = 0.02 mg/m^3 (tolerable concentration)	TDI based on a NOAEL of 40 mg/kg/day associated developmental effects in rats and an uncertainty factor of 900 (and the TDI rounded). TC is provisional (due to the poor database) and based on a NOAEC of 20 mg/m^3 associated with adverse effects in various experimental animals after subchronic inhalation exposure and an uncertainty factor of 1000.
Health Canada (2000)	TDI = 0.12 mg/kg/day	TDI based on review of the available database and consideration that developmental effects are the most sensitive endpoints (noting other endpoints have limited data). Value derived on the basis of a NOAEL of 12 mg/kg/day for kidney effects (noted to be lower than that from developmental effects) in rats and an uncertainty factor of 100. Value derived is considered conservative.
EC (2006)	No ADI/TDI derived (acceptable daily intake/tolerable daily intake)	No ADI/TDI derived however critical data points were identified for systemic toxicity where an oral lowest-observed-adverse-effect level (LOAEL) of 1.8 mg/kg/day (based on reduced blood cell count in mice), inhalation LOAEL of 21 mg/m^3 (based on possible liver injury in exposed workers) and a dermal NOAEL of 1.18% (equivalent to 130 mg/kg/day) were identified. A NOAEL for developmental toxicity of 93 mg/kg/day was identified from a 2-generation rat study.
UK (EA 2009)	TDI = 0.7 mg/kg/day TC = 0.035 mg/m^3	TDI based on review of current studies and evaluations. The TDI is based on a NOAEL of 70 mg/kg/day associated with a 2-generation drinking water rat study and an uncertainty factor of 100. The study chosen is considered more appropriate that that considered by the US EPA, WHO and RIVM as it was of longer duration and associated with drinking water administration (note that phenol exhibited a higher degree of toxicity when given by stomach tube/gavage than when administered via drinking water). Inhalation value derived on the basis of a LOAEL of 21 mg/m^3 (same as identified by EC 2006) associated with potential liver effects in occupationally exposed workers and an uncertainty factor of 600. It is note that the review undertaken considers that the critical effects associated with inhalation exposures to phenol is likely to be it mutagenic potential, and a non-threshold approach may be appropriate, however no evaluations are available. Also noted that despite significant limitations in the available data it appears that phenol is more potential via inhalation than when ingested.
ATSDR (2008)	No chronic MRL derived	Oral MRL based on a LOAEL of 1 mg/kg/day associated thyroid effects in mink and an uncertainty factor of 1000 (same study as considered by RIVM).
US EPA (IRIS 2010)	RfD = 0.3 mg/kg/day	RfD (last reviewed in 2002) based on a benchmark dose approach where a BMDL of 93 mg/kg/day associated with decreased maternal weight gain in a short duration developmental rat study was derived and an uncertainty factor of 300 considered. The previous evaluation by the US EPA considered an oral RfD of 0.6 mg/kg/day, adopted in the derivation of the current HIL (Turczynowicz 1993).

While a number of limitations have been identified by the UK (EA 2009) review in the available data with respect to the quantification of phenol toxicity, the oral value recommended is based on the most recent review where a number of the database deficiencies have been more fully reviewed. This value has been adopted in the derivation of soil HILs.

Few inhalation values are available and hence, the threshold value derived by the UK (EA 2009) is recommended as it is based on a more recent review. As inhalation exposures appear to be more toxic than oral exposures, the consideration of separate toxicity values for oral and inhalation routes of exposure (even if the inhalation route of exposure is not as significant for the characterisation of contaminated soil issues) is appropriate.

Recommendation

On the basis of the discussion above, the following toxicity reference values (TRVs) have been adopted for phenol in the derivation of HILs:

Recommendation for phenol
Oral TRV = 0.7 mg/kg/day (EA, 2009) relevant to oral and dermal routes of exposure
Dermal absorption factor = 0.1 (or 10%) (US EPA 2004)
Inhalation TRV = 0.035 mg/m^3 (EA, 2009) relevant to inhalation routes of exposure
Intakes allowable from soil (as % of TRV) = 70%

2.5 Calculated HILs

On the basis of the above, the following HILs have been derived for phenol:

HIL scenario	HIL (mg/kg)	Percentage contribution from exposure pathways			
		Ingestion of soil/dust	Ingestion of home-grown produce	Dermal absorption of soil/dust	Inhalation (dust)
Residential A	3000	4%	91%	5%	<1%
Residential B	50000	17%	--	80%	3%
Recreational C	45000	30%	--	69%	1%
Commercial D	250000	11%	--	86%	3%

-- Pathway not included in derivation of HIL

2.6 References for Phenol

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WHO 1994, *Phenol*, Environmental health criteria document 161, International Programme on Chemical Safety, World Health Organisation, Geneva, available online at: <http://www.inchem.org/documents/ehc/ehc/ehc161.htm>.

3 Pentachlorophenol (PCP)

3.1 General

Several comprehensive reviews of pentachlorophenol (PCP) in the environment and its toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 2001; WHO 1987). The following provides a summary of the key aspects of PCP that are relevant to the derivation of a soil HIL.

Pure pentachlorophenol is a colourless, white or light tan crystalline solid (WHO 1987; ATSDR 2001). It has a characteristic 'phenolic' odour at high temperatures but it is relatively odourless at room temperature. Pentachlorophenol is moderately volatile at ambient temperature and insoluble in water (WHO 1987; ATSDR 2001). Technical grade pentachlorophenol is typically 86% pure and is dark gray to brown in colour as a result of the polychlorinated phenol impurities. It is typically manufactured in the form of dust, beads or flakes (ATSDR 2001).

Pentachlorophenol is an effective biocide and had wide applications in the commercial and agricultural industries as an insecticide (termiticide), fungicide, herbicide, molluscicide and algicide. The primary use of the compound was for wood preservation. In the United States the use of wood products treated with pentachlorophenol in domestic settings was banned; however, the compound is still used to preserve power line poles, railroad sleepers, wharf pilings, cross arms and fence posts (ATSDR 2001). Pentachlorophenol was also historically used as a disinfectant, as an ingredient in antifouling paint, as an insecticide or herbicide in domestic environments, in the textile industry, leather industry, in mineral oil and in glue (WHO 1987; ATSDR 2001).

Pentachlorophenol is no longer registered as the active ingredient in any chemical in Australia.

Review of the toxicity of PCP is complicated by the relatively large database on the toxicity of technical-grade PCP and the comparatively small database on pure PCP. Technical-grade PCP has been shown to contain a large number of impurities, including tetrachlorophenols and, to a much lesser extent, polychloro-dibenzodioxins, polychlorodibenzofurans, polychlorodiphenyl ethers, polychloro-phenoxy phenols and chlorinated hydrocarbons. These impurities, in particular the polychloro-dibenzodioxins and furans, are indicated to be responsible for at least some of the observed toxicity of the technical-grade PCP (MfE 2010). Notwithstanding, specific haematopoietic cancer risks are observed with PCP exposure which are not likely to be due to dioxins or other chlorophenol contaminants (Cooper & Jones 2008).

3.2 Previous HIL

No previous HIL has been derived from PCP (NEPC 1999).

3.3 Significance of exposure pathways

Oral bioavailability

Insufficient data are available to adequately define the bioavailability of PCP in the range of contaminated sites that may need to be considered in Australia. On this basis, a default approach of assuming 100% oral bioavailability has been adopted in the derivation of an HIL. It is noted that a site-specific assessment of bioavailability can be undertaken where required.

Dermal absorption

PCP is rapidly absorbed across the skin, and therefore dermal exposure potentially represents a significant route of exposure. The US EPA (2004) has identified a dermal absorption fraction of 0.25 (25%), based on a study by Wester et al. (1993) for PCP in soil. The study found that *in vivo* absorption in monkeys of PCP in soil was similar to PCP in acetone, with 24% of PCP absorbed over a 24-hour period. Few other studies are available where quantitative values are available and hence the dermal absorption value of 0.24 (24%) from Wester et al. (1993) has been used in the derivation of HILs for PCP.

Inhalation of dust

PCP is not considered sufficiently volatile to be of significance and inhalation exposures associated with particulates outdoors and indoors are expected to be of less significance than ingestion of soil. While likely to be negligible, potential inhalation exposures associated with dust have been considered in the HIL derived.

Plant uptake

In a review paper, McAllister et al. (1996) reported that available data on the plant uptake and transformation of PCP are inconsistent among studies and are inconclusive with regard to the abilities of specific plants to take up the compound. It was observed that the biodegradation of PCP by microorganisms and its adsorption to soil limit the availability of the compound for plant uptake (ATSDR 2001).

Further review by MfE (2010) considered that plant uptake of PCP is not a significant pathway of exposure given that PCP is known to be metabolised by plants (resulting in an over prediction of plant uptake by the models available), bioconcentration factors relevant to plant uptake are low, and recent papers relating to PCP and plants where uptake is noted are associated with phytoremediation through enhanced microbial activity at plant roots.

On the basis of the above, plant uptake of PCP is not considered significant. In addition, the application of general plant uptake equations is not considered appropriate.

Intakes from other sources – background

Limited information is available on background exposures to PCP by the general population (PCP intakes have not been addressed in the Australian Total Diet Surveys (ATDSs). PCP is no longer used in Australia and, while it is persistent, background levels are expected to be low. Dietary intakes are expected to be the most significant background source (ATSDR 2001). Total intakes of PCP (dominated by food intakes) have been estimated to be between 0.1 and 6 µg/day (WHO 1987) and 5-35 µg/day (WHO DWG 2008); however, these estimates are based on older data.

ATSDR (2001) notes that intakes estimated from a US total diet survey (1982-1984) suggested intakes for 2-year-old children were up to 48.5 ng/kg/day (about 0.6 µg/day). Estimates from a later total diet survey (1986-1991) suggested lower intakes by children aged 2 years of 1.4 ng/kg/day (about 20 ng/day). Intakes from the later study are consistent with background intakes estimated by RIVM (2001). These intakes are essentially negligible compared with the recommended oral TRV. Hence intakes from other sources have been considered to be negligible.

3.4 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 1991) has classified PCP as Group 2B: possibly carcinogenic to humans.

It is also noted that the US EPA has classified PCP as Group B2: probable human carcinogen.

Review of available values/information

Studies on experimental animals have shown some carcinogenic potential associated with oral exposures to technical grade and mixtures of PCP. However, PCP has not demonstrated genotoxicity in *in vitro* and *in vivo* test systems and in occupationally exposed humans (RIVM 2001; NHMRC 2009). Review by ATSDR (2001) and IARC (1991) suggests PCP may exhibit weak clastogenic effects. Review by MfE (2010) suggested that the data on the genotoxicity of PCP are equivocal, with the strongest indication of genotoxicity (chromosomal effects) occurring in assays with rat microsomal protein (S9). The primary rodent metabolite, tetrahydrochloroquinone (TeHQ), is unambiguously genotoxic. TeHQ does not appear to be a major metabolite of PCP in humans. Furthermore, the majority of PCP appears to be excreted unchanged (ATSDR 2001).

On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for PCP.

Few quantitative toxicity values are available; however, the following threshold values are available from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2004)	TDI = 0.003 mg/kg/day	The current ADWG (NHMRC 2004) has derived a health-based guideline of 0.01 mg/L, from a TDI of 0.003 mg/kg/day (assuming 10% contribution from drinking water). No further detail is presented in the current ADWG documentation. The draft revision to the ADWG (NHMRC 2009) also adopts the TDI of 0.003 mg/kg/day, noted to be based on a no-observed-effect level (NOEL) of 3 mg/kg/day based on a 2-year rat study and an uncertainty factor of 1000 (10 for interspecies extrapolation, 10 for intraspecies variability extrapolation and an additional safety factor of 10 due to the limitations of the toxicological data available at the time the ADI was set).
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	No threshold value set	The current WHO DWG (2008) has derived a provisional guideline of 0.009 mg/L based on a US NTP study and a linear multi-stage model associated with tumour increases and an excess lifetime risk of 10^{-5} (review unchanged since 1993). It is noted that pentachlorophenol is included in the rolling revisions to the DWG, with no revisions currently available.
WHO (1987)	ADI = 0.003 mg/kg/day	References an ADI derived by the National Academy of Sciences which is based on a NOEL of 3 mg/kg/day from a long-term feeding study in rats and an uncertainty factor of 1000 (same study as considered in the ADWG).
RIVM (2001)	TDI = 0.003 mg/kg/day	TDI based on a LOAEL of 1 mg/kg/day associated thyroid effects in mink and an uncertainty factor of 300.
ATSDR (2001)	MRL = 0.001 mg/kg/day	Oral MRL based on a LOAEL of 1 mg/kg/day associated thyroid effects in mink and an uncertainty factor of 1000 (same study as considered by RIVM.
US EPA (IRIS 2010)	RfD = 0.03 mg/kg/day	RfD (last reviewed in 1987) based on a NOAEL of 3 mg/kg/day associated with liver and kidney effects in rats and an uncertainty factor of 100. The U SEPA has also derived a non-threshold oral SF not considered relevant here.

The threshold reference value adopted in the ADWG (NHMRC 2004, 2009) which is consistent with that derived by RIVM (2001) and ATSDR (2001) is recommended.

No dermal or inhalation specific studies or data are available. For the presence of PCP in soil it is considered appropriate to consider use of the available TDI for all pathways of exposures.

Recommendation

On the basis of the discussion above, the following TRVs have been adopted for PCP in the derivation of HILs:

Recommendation for pentachlorophenol

Oral TRV = 0.003 mg/kg/day (ADWG in NHMRC 2004 ,2009) relevant to all pathways of exposure

Dermal absorption factor = 0.24 (or 24%) (Wester et al. 1993)

Intakes allowable from soil (as % of TRV) = 100%

3.5 Calculated HILs

On the basis of the above, the following HILs have been derived for PCP:

HIL scenario	HIL (mg/kg)	Percentage contribution from exposure pathways			
		Ingestion of soil/dust	Ingestion of home-grown produce	Dermal absorption of soil/dust	Inhalation (dust)
Residential A	100	26%	--	74%	<1%
Residential B	150	8%	--	92%	<1%
Recreational C	140	15%	--	85%	<1%
Commercial D	700	5%	--	95%	<1%

-- Pathway not included in derivation of HIL

3.6 References for PCP

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4 Total cresols

4.1 General

Several comprehensive reviews of cresols in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 2008; WHO 1995). The following provides a summary of the key aspects of cresols that are relevant to the derivation of a soil HIL.

Cresols are a group of isomers comprising a single benzene ring, a hydroxyl group and a methyl group (C_7H_8O). There are three structural isomers including, m-cresol (2-methylphenol), p-cresol (3-methylphenol), and o-cresol (4-methylphenol). These isomers may occur separately or as a mixture (ATSDR 2008).

In their pure form, cresols are colourless solids, whilst mixtures are more commonly liquids. Cresols are semi-volatile compounds with moderate solubility in water and a medicinal type odour (ATSDR 2008). The abundance of p-cresols in the environment is significantly greater than that of the alternative isomers, as is the abundance of o-cresol relative to that of m- cresols. However, there is a greater amount of information and studies surrounding the health effects associated with m- and o-cresols. It should be noted that the behaviour of all three isomers in the environment is considered to be similar.

Cresols are both a naturally occurring and manufactured group of chemicals that may be used as solvents, disinfectants, deodorisers, wood preservatives and to make other chemicals (ATSDR 2008). O-cresol is used in the manufacture of several dye intermediates (ATSDR 2008). P-cresol is predominantly used in the manufacture of anti oxidants synthetic food flavours and fragrances, and m-cresol is used in the synthesis of many herbicides and insecticides (ATSDR 2008). Cresols occur in various plant oils including, peppermint, sandalwood, jasmine, Easter lily, ylang ylang, eucalyptus and camphor.

4.2 Previous HIL

No previous HIL is available for cresols (NEPC 1999).

4.3 Significance of exposure pathways

Oral bioavailability

Insufficient data is available to adequately define the bioavailability of cresols in the range of contaminated sites that may need to be considered in Australia. On this basis a default approach of assuming 100% oral bioavailability has been adopted in the derivation of a HIL. It is noted that a site-specific assessment of bioavailability can be undertaken where required.

Dermal absorption

Insufficient data is available on the dermal absorption of cresols from soil. Hence, the default values of 0.1 (10%) suggested by US EPA (2004) for semi-volatiles has been adopted in the derivation of HILs.

Inhalation of dust

Cresols are not considered sufficiently volatile to be of significance and inhalation exposures associated with particulates outdoors and indoors are expected to be of less significance than ingestion of soil. While likely to be negligible, potential inhalation exposures associated with dust have been considered in the HIL derived.

Plant uptake

No data is available on the potential for the uptake of cresols into edible fruit and vegetable crops. Limited data is also available on the potential or cresols to bioaccumulate. Cresols are soluble in water and based on Koc values referenced by OECD (2003) there is a low sorption potential for cresols. Hence, while specific data is lacking, there is the potential for cresols to be available in soil water to be taken up by plants. Hence, a conservative approach has been taken to consider the potential for the uptake of cresols into home-grown produce in the derivation of HIL A.

This has been undertaken on the basis of the equations presented in Appendix B with the following parameters and plant uptake factors estimated:

Parameter	Value	Reference/Comment
Parameters		
Koc	307 (cm^3/g)	RAIS (2010)
log Kow	1.95	RAIS (2010)
Diffusivity in water	$9.78x10^{-6}$ (cm^2/s)	RAIS (2010)
Calculated plant uptake factors (mg/kg produce fresh weight per mg/kg soil)		
Green vegetables	0.18	calculated
Root vegetables	0.255	calculated
Tuber vegetables	0.152	calculated
Tree fruit	0.00044	calculated

Intakes from other sources – background

Limited information is available on background exposures to cresols by the general population. Available reviews by ATSDR (2008), OECD SIDS (2003) and RIVM (2001) have not been able to quantify background intakes due to a lack of data. As data is lacking for background intakes of cresols, an estimate or default value can be assumed. Cresols are expected to be widely present in the environment and hence a value of 50% may be relevant where data are not available.

4.4 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC) has not classified cresols with respect to human carcinogenicity.

The US EPA has classified cresols as Group C: possible human carcinogen.

Review of available values/information

There are no adequate data available to assess carcinogenicity of cresols. One study suggests cresols may promote skin tumours. Genotoxicity of cresols has been evaluated (ATSDR 2008) and the weight of evidence suggests that 'cresols do not pose a genotoxic threat to humans under normal environmental exposure conditions'. On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for cresols.

Few quantitative toxicity values are available, however the following are available from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2004)	No evaluation available	
OCS (2008)	No evaluation available	
International		
WHO (1995)	ADI = 0.17 mg/kg/day	ADI derived by WHO (1995) on a NOAEL of 50 mg/kg/day from a subchronic study and a 300 fold uncertainty factor (which included an additional 10 fold factor to address the lack of chronic studies and possible genotoxic and promoting activity).
RIVM (2001)	TDI = 0.05 mg/kg/day TC = 0.17 mg/m^3	TDI based on a 90-day subchronic oral study TC based on route extrapolation from oral data.
OEHHA (2009)	REL = 0.6 mg/m^3	Chronic REL based on route extrapolation of the LOAEL and NOAEL derived from the study used to derive the current US EPA RfD for 2- and 3-methylphenol.
ATSDR (2008)	MRL = 0.1 mg/kg/day	Oral MRL based on a LOAEL associated with increased incidences of bronchiole hyperplasia of the lung and follicular degeneration of the thyroid gland from a 2-year dietary study in female mice.
US EPA (IRIS 2010)	RfD = 0.05 mg/kg/day	RfD derived for 2- and 3-methylphenol based on decreased body weights and neurotoxicity in a 90-day subchronic study in rats.

The threshold value derived by ATSDR (2008) is based on a chronic study not available at the time when the WHO (1995), RIVM (2001) or US EPA conducted their review (where threshed values were derived on the basis of subchronic studies). On this basis, the oral value (taken as an ADI) available from ATSDR (2008) is considered the most current and robust value for deriving a soil HIL.

No dermal or inhalation specific studies or data are available. For the presence of cresols in soil it is considered appropriate to consider use of the available ADI for all pathways of exposures.

Recommendation

On the basis of the discussion above the following TRVs have been adopted for cresols (as sum of all isomers) in the derivation of HILs:

Recommendation for cresols

Oral TRV = 0.1 mg/kg/day (ATSDR 2008) relevant to all pathways of exposure

Dermal absorption factor = 0.1 (or 10%) (US EPA 2004)

Intakes allowable from soil (as % of TRV) = 50%

4.5 Calculated HILs

On the basis of the above the following HILs have been derived for cresols:

HIL scenario	HIL (mg/kg)	Percentage contribution from exposure pathways			
		Ingestion of soil/dust	Ingestion of home-grown produce	Dermal absorption of soil/dust	Inhalation (dust)
Residential A	400	5%	89%	6%	<1%
Residential B	5500	18%	--	82%	<1%
Recreational C	4700	30%	--	69%	<1%
Commercial D	27000	12%	--	88%	<1%

-- Pathway not included in derivation of HIL

4.6 References for cresol

ATSDR 2008, *Toxicological profile for cresols*, Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia, USA, available online at <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=946&tid=196>.

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OECD SIDS 2003, M/P-Cresol Category, SIDS initial assessment report for SIAM 16.

OEHHA 2009, *Air toxicology and epidemiology, summary of current reference exposure levels and cancer potency factors*, available from Office of Environmental Health Hazard Assessment (OEHHA), online at <http://www.oehha.org/air/toxic_contaminants/index.html>.

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RIVM 2001, *Re-evaluation of human-toxicological maximum permissible risk levels*, National Institute of Public Health and the Environment, Bilthoven, The Netherlands, available online at <http://www.rivm.nl/bibliotheek/rapporten/711701025.html>.

US EPA 2004a, *Risk assessment guidance for Superfund, Volume I: Human health evaluation manual (Part E), Supplemental guidance for dermal risk assessment, Final*, EPA/540/R-99/005, OSWER 9285.7-02EP.

US EPA (IRIS 2010), Data and information from the integrated risk information system, an online database, available online at <http://www.epa.gov/iris/>.

WHO 1995, *Cresols*, Environmental health criteria 168, International Programme on Chemical Safety, World Health Organisation.

5 Shortened forms

ADI	Acceptable daily intake
ADWG	Australian Drinking Water Guidelines
ANZECC	Australia and New Zealand Environment and Conservation Council
ATDS	Australian Total Diet Survey
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark dose
BTEX	Benzene, toluene, ethylbenzene and total xylenes
CCME	Canadian Council of Ministers of the Environment
CICAD	Concise International Chemicals Assessment Document
CNS	Central nervous system
EHC	Environmental health criteria
EPA	Environment Protection Authority
FSANZ	Food Standards Australia New Zealand
HEC	Human equivalent concentration
HED	Human equivalent dose
HIL	Health investigation level
HSDB	Hazardous Substances Data Bank
HSL	Health screening level

IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
JECFA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	WHO/FAO Joint Meeting on Pesticide Residues
LOAEL	Lowest-observed-adverse-effect level
LOEL	Lowest-observed-effect level
MF	Modifying factor
MOA	Mode (or mechanism) of action
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NOAEL	No-observed-adverse-effect level
NOEL	No-observed-effect level
NSW DECC	New South Wales Department of Environment and Climate Change
OCS	Office of Chemical Safety
PAH	Polycyclic aromatic hydrocarbon
PTDI	Provisional tolerable daily intake
PTWI	Provisional tolerable weekly intake
RAIS	Risk Assessment Information System
RfC	Reference concentration
RfD	Reference dose
SF	Slope factor
TC	Tolerable concentration
TCE	Trichloroethene
TDI	Tolerable daily intake
TPH	Total petroleum hydrocarbons
TPHCWG	Total Petroleum Hydrocarbon Criteria Working Group
UF	Uncertainty factor
UR	Unit risk
USEPA	United States Environmental Protection Agency
VC	Vinyl chloride
VOC	Volatile organic compound
WHO	World Health Organisation
WHO DWG	World Health Organisation Guidelines for Drinking Water