Schedule B7
Appendix A4

TCE/PCE and products

Contents
TCE/PCE and products

1 Trichloroethene (TCE)

1.1 General

Several comprehensive reviews of trichloroethene (TCE) in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 1997; WHO 1985; EU 2004; CCME 2007; NICNAS 2009; US EPA 2009). The following provides a summary of the key aspects of TCE that are relevant to the derivation of interim HILs.

TCE is a colourless, non-flammable, volatile liquid, with a characteristic slightly sweet odour. Most people can begin to smell TCE in air at a concentration of 100 ppm (ATSDR 1997).

TCE was not thought to occur naturally in the environment, until the discovery in 1995 that several species of marine macro-algae and at least one species on microalgae produce the compound. The importance of this release and potential exposure route is not currently known. It is mainly used as an industrial solvent in a variety of industries, primarily metal degreasing and cleaning operations. TCE can also be found in some household products, including correction fluid, paint removers, adhesives, and spot removers. TCE has also been used as a carrier solvent for the active ingredients of insecticides and fungicides; as a solvent for waxes, fats, resins, and oils; and as an anaesthetic for medical and dental use. It has also been used to extract spice oleoresins and caffeine from coffee (ATSDR 1997; WHO 1985).

TCE was manufactured in Australia for approximately 30 years from the early 1950s to the early 1980s. At present, the Australian market demand for TCE is entirely met by imports of the chemical. TCE is used widely in both large and small industries mainly as a degreasing agent (NICNAS 2009).

If released into the environment, the following can be noted with respect to TCE (WHO 1985):

- Air: TCE is expected to remain in vapour phase. Removal is primarily through reaction with hydroxyl radicals to produce low levels of phosgene, dichloroacetyl chloride, formyl chloride and other degradation products. The half-life of TCE varies from 1 day to months.
- Soil and water: TCE is expected to volatilise from surface soils and water. TCE may leach through soil into groundwater where it may persist for years.
- Water: depending on conditions, reductive dehalogenation to vinyl chloride may occur. Under anaerobic conditions TCE can be intrinsically biodegraded to form drichloroethene (1,1-DCE and isomers of 1,2-DCE) and vinyl chloride).

1.2 Previous HIL

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

1.3 Proposed interim HIL

Review of available information in relation to the presence of TCE in soil indicates that the vapour inhalation pathway is the most significant/important. This pathway should be assessed on the basis of measured vapour data, in particular soil vapour data. There are significant limitations in the derivation of a soil HIL, in particular, the modelling of phase partitioning from soil to soil vapour and the field measurement of volatiles in soil; hence an interim HIL has been derived for soil vapour only.

The following presents the values adopted for the calculation of a soil vapour interim HIL. In addition, other information that is relevant to the assessment of TCE in soil (relevant to other pathways of exposure) is presented.

1.4 Significance of exposure pathways

Inhalation

TCE is a volatile compound and, as such, the derivation of the HIL has considered the vapour inhalation pathway as the most significant. The approach adopted for the quantification of potential vapour migration to outdoor air and intrusion indoors is outlined in Schedule B7. Due to limitations with the vapour modelling approach adopted, the HILs derived are considered interim.

The inhalation of particulates (outdoors and indoors) is considered essentially insignificant, compared with vapour inhalation.

Dermal absorption

Insufficient data is available on the dermal absorption of TCE from soil. Given the volatility of the compound dermal absorption is expected to be low; however, as there is insufficient data available to further assess dermal absorption from soil, a default value of 0.03 (3%) has been adopted for the volatile organic compounds (US EPA 1995).

Plant uptake

Limited data are available on the potential for TCE to be taken up by home-grown produce. According to ATSDR (1997), TCE has been detected in small amounts in fruits and vegetables, suggesting a potential for limited phytoaccumulation. Laboratory studies with carrot and radish plants and radioactively labelled TCE (Schroll et al. 1994) showed some uptake however it is noted that the experiment indicated that uptake occurred mainly through the foliage (from the air) as opposed to the roots in these plants (with subsequent translocation throughout the plant tissues). Scnabel et al. (1997) looked at the uptake of TCE in edible garden plants (carrots, spinach and tomatoes) and identified that TCE when taken up was transformed and bound to plant tissues in a form that was less toxic than the parent compound.

On the basis of the above, the use of the more commonly adopted equations for quantifying plant uptake (as presented in Schedule B7) that do not address uptake of volatiles (from air) rather than the root, or transformations within the plant, are not considered appropriate and relevant for the assessment of TCE.

It is expected that the potential for plant uptake will be of less significance in the derivation of a soil HIL, when compared with the assessment of vapour inhalation, and given the limitations involved in providing a meaningful evaluation of plant uptake, it has not been considered in the derivation of HILs.

Intakes from other sources – background

As TCE is highly volatile, background intakes will be dominated by inhalation exposures. Concentrations of TCE in industrial, urban and regional areas are available in Australia. Data collected (DEC 2003) from urban and regional areas in NSW report average concentrations of TCE of approximately 0.1 ppbv (0.0005 mg/m^3), close to the analytical limit of reporting with a most samples noted to be not detected, with a maximum concentration in Sydney CBD of 3.6 ppbv (0.019 mg/m^3). Concentrations in an industrial area in Brisbane (Hawas et al. 2001) have been reported with average and maximum concentrations of 0.0002 mg/m^3 (also close to the limit of reporting) and 0.0005 mg/m^3 respectively. Background air concentrations in Canada (CCME 2007) are considered to be approximately 0.0014 mg/m^3, consistent with the range reported by DEC (2003).

Background intakes (dominated by inhalation) were estimated by the WHO (DWG 2008) to be approximately 0.04 µg/kg/day for children and 0.01 µg/kg/day for adults. Based on average concentrations reported in NSW and in Brisbane, intakes by young children are estimated to be approximately 0.3 µg/kg/day. These intakes are essentially negligible when compared with the recommended toxicity reference values (TRVs) for non-carcinogenic effects.

1.5 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 1995) has classified TCE as Group 2A: probably carcinogenic to humans.

In addition, TCE was classified as a 'probable' human carcinogen (Category B2) by the US EPA for all routes of exposure based upon evidence from animal studies. This classification has been withdrawn pending further review and has not been finalised to date. However, the draft US EPA review (2009) characterised TCE as 'carcinogenic to humans' by all routes of exposure, based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer, and evidence of TCE carcinogenicity in liver and lymphoid tissues. The data is supported by animal studies.

Review of available values/information

Some epidemiological studies indicate a possible association between exposure to TCE and an increased cancer risk, with IARC (1995) noting elevated risk for cancer of the liver and biliary tract and a modestly elevated risk for non-Hodgkin's lymphoma in three cohort studies. In animals, TCE induces tumours at several sites and in different species. Tumours have been seen in mouse liver and lung and rat kidney and testies. On the basis of the available information, most current reviews by IARC (1995), WHO (DWG 2008), CCME (2007) and US EPA (2009) consider TCE to be carcinogenic (with responses tending to increase with dose) via all routes of exposure.

The potential mode of action (MOA) for TCE is reviewed and discussed in the current WHO DWG (2008) and draft US EPA (2009) review.

The WHO DWG (2008) review concluded that the MOA for tumour induction by TCE may be attributed to non-genotoxic processes (related to cytotoxicity, peroxisome proliferation and altered cell signalling); genotoxic processes, (such as the production of genotoxic metabolites (for example, chloral and DCVC[1])); or the production of reactive oxygen species related to peroxisomal induction in the liver. The potential role of several mutagenic or carcinogenic metabolites of TCE cannot be ignored. Hence, TCE appears to be at least weakly genotoxic and evaluation of carcinogenicity on the basis of a non-threshold approach is considered appropriate,as is undertaken in the current WHO DWG (2008) and WHO air quality guidelines (2000).

The draft US EPA (2009) review provides a detailed assessment of genotoxicity (of TCE and metabolites) and mutagenicity. With respect to genotoxicity, although it appears unlikely that TCE, as a pure compound, causes point mutations, there is, however, evidence for TCE genotoxicity with respect to other genetic end-points, such as micronucleus formation. In addition, several TCE metabolites have tested positive in genotoxicity assays. It is noted that uncertainties with regard to the characterisation of TCE genotoxicity remain, particularly because not all TCE metabolites have been sufficiently tested in the standard genotoxicity screening battery to derive a comprehensive conclusion.

[1] DCVC is the abbreviation for the metabolite S-(1,2-dichlorovinyl)-L-cysteine.

However, the metabolites that have been tested, particularly DCVC, have predominantly resulted in positive data, supporting the conclusion that these compounds are genotoxic.

The MOA relevant to specific target organs in laboratory animals have been reviewed by the US EPA. Only in the case of the kidney is it concluded that the data are sufficient to support a particular MOA being operative. For the kidney, the predominance of positive genotoxicity data in the database of available studies of TCE metabolites together with toxicokinetic data supports the conclusion that a mutagenic MOA is operative in TCE-induced kidney tumours. Hence, a linear (non-threshold) approach is recommended for the quantification of carcinogenic effects.

There is some evidence that certain populations may be more susceptible to exposure to TCE. Because the weight of evidence supports a mutagenic MOA being operative for TCE carcinogenicity in the kidney, and there is an absence of chemical-specific data to evaluate differences in carcinogenic susceptibility, early-life susceptibility is recommended by the US EPA to be assumed and the age-dependent adjustment factors (ADAFs) should be applied.

On the basis of the above, it is reasonable to consider a non-threshold approach for the assessment of carcinogenicity in relation to TCE. It is noted that a number of guidelines (such as WHO DWG) have been derived on the basis of both carcinogenic and non-carcinogenic end-points, with non-carcinogenic end-points noted to be more sensitive for at least oral intakes. Hence, both non-threshold and threshold reference values available have been noted.

The following quantitative values are available for TCE from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2004)	No health-based value derived	Not derived due to insufficient data.
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	SF = 0.00078 $(mg/kg/day)^{-1}$ TDI = 0.00146 mg/kg/day	WHO DWG (2008) based on the lower value derived from carcinogenic and non-carcinogenic end-points. It is noted that the guideline derived on the basis of reproductive/developmental (threshold) effects was most conservative. The oral slope factor (SF) adopted is from Health Canada (range of values derived) and based on combined tubular cell adenomas and adenocarcinomas of the kidneys in rats following oral exposure to TCE for 103 weeks and a linear multi-stage model. The oral tolerable daily intake (TDI) derived from a $BMDL_{10}$ of 0.146 mg/kg/day associated with reproductive/developmental effects in rats and an uncertainty factor of 100.
WHO (2000)	UR = $4.3x10^{-7}$ $(\mu g/m^3)^{-1}$	Inhalation unit risk (UR) derived on the basis of Leydig-cell tumours in the testes of rats and a linear multistage model. The non-threshold approach was adopted by the WHO as TCE was considered genotoxic and carcinogenic.
EU (2004)	SF = 0.0019 $(mg/kg/day)^{-1}$	TCE gives rise to concern for humans owing to possible mutagenic and carcinogenic effects and because it is not possible to identify a threshold exposure level below which these effects would not be expressed. For non-carcinogenic effects the most sensitive threshold effect evaluated was associated with CNS disturbance following repeated dose where a no-observed-adverse-effect level (NOAEL) of 38 mg/kg/day was considered. The EU has presented a calculation of lifetime cancer risk based on the T25 method in relation to non-Hodgkin lymphoma. From an inhalation study in female mice a HT25 dose descriptor for humans was derived as 130 mg/kg/day. Following the approach presented the EU calculated increased cancer risk for TCE for all groups using an equivalent slope factor of 0.0019 $(mg/kg/day)^{-1}$. This value was used in the quantification of risk associated with exposure from oral, dermal and inhalation pathways for workers, consumers and environmental exposures.

International cont'd		
Health Canada (2005)	SF = 0.000811 (mg/kg/day)$^{-1}$ UR = 1.2x10^{-7} (µg/m^3)$^{-1}$ TDI = 0.00146 mg/kg/day	Oral SF derived on the basis of the same study noted in the WHO DWG (2008);however, a slightly different value is quoted. Inhalation UR based on renal adenocarcinomas in rats following inhalation exposures for 104 weeks in males (a lower, less conservative value was derived for females). Note that the derivation of drinking water guidelines has also considered the oral TDI noted in the WHO DWG which results in a lower guideline than is derived on the basis of the oral slope factor.
CCME (2007)	SF = 0.000811 (mg/kg/day)$^{-1}$ UR = 6.4x10^{-7} (µg/m^3)$^{-1}$ TDI = 0.00146 mg/kg/day TC = 0.04 mg/m^3	SF based on same study noted by Health Canada (2005). Inhalation UR based on older evaluation from Health Canada where a TC_{05} (concentration expected to cause a 5% incidence in cancer) of 0.082 mg/m^3 and extrapolation based on an excess lifetime cancer risk of 10^{-6}. TDI and TC (tolerable concentration) values also presented for non-carcinogenic end-points. TDI as noted by WHO DWG TC adopted from the former US EPA reference concentration (RfC) (currently withdrawn pending finalisation of the 2009 draft) associated with effects on the CNS, kidney, liver and endocrine system in inhalation studies where a point of departure (POD) of 38 mg/m^3 was identified and an uncertainty factor of 1000 adopted.
RIVM (2001)	PTDI = 0.05 mg/kg/day PTC = 0.2 mg/m^3	Provision threshold values derived for TCE due to limited data and an assumption that the genotoxic mechanism for TCE (numerical chromosome aberration *in vivo*) exhibits a threshold. The basis for these values is not listed here as the evaluation is considered dated.
ATSDR (1997)	No chronic MRLs derived	No chronic oral or inhalation MRL has been established.
US EPA (IRIS 2010)	No evaluation	Previous assessments have been withdrawn from IRIS pending review.
New York State (2006)	GV = 0.005 mg/m^3	An air GV of 0.01 mg/m^3 was derived for non-carcinogenic effects (CNS effects in humans) is based on review of all available studies and associated end-points. The lowest guideline value has been adopted and is noted to be protective of the general population including sensitive life stages of infants, children, the infirm and elderly. The GV resulted in carcinogenic risk estimates at the lower end of the risk range (1x10^{-6} to 1x10^{-4}). The guideline value was then reduced by a factor of 2 based on the consideration of additional factors (data gaps, concern regarding methods for evaluating risks to children and concerns regarding human carcinogenicity) in addition to background levels and analytical capabilities. The resulting air guideline derived was 0.005 mg/m^3.
US EPA (2009 draft)	SF = 0.05 (mg/kg/day)$^{-1}$ UR = 4x10^{-6} (µg/m^3)$^{-1}$ RfD = 0.0004 mg/kg/day RfC = 0.005 mg/m^3	Oral SF based on PBPK model-based route-to-route extrapolation from the inhalation value. Value also supports by values derived from oral bioassays. Inhalation UR derived on the basis of kidney cancers from a human inhalation study, adjusted for potential risk of tumours at multiple sites. The value derived is supported by UR estimates derived from multiple rodent bioassays. Application of the ADAF for kidney cancer risks due to evidence supporting a mutagenic MOA is recommended. RfD (reference dose) based on critical effects of heart malformations (rats), adult immunological effects (mice), developmental immunotoxicity (mice) and toxic nephropathy (rats). RfC (reference concentration) based on route-to-route extrapolation from oral studies for the critical effects of heart malformations, immunotoxicity and toxic nephropathy and an inhalation study for the critical effects of increased kidney weight.

For TCE, the health end-points associated with carcinogenic (non-threshold) and non-carcinogenic (threshold) effects are similar in sensitivity. Hence, it is appropriate that the derivation of a guideline consider all relevant end-points to ensure that the value derived is adequately protective of all effects. It is recommended that the non-threshold values adopted by the WHO (2000) and as referenced in the WHO DWG be adopted for the assessment of carcinogenic effects. The inhalation UR values derived from the WHO are consistent with the range derived for other key carcinogenic effects (including kidney tumours) by CCME (2007) and Health Canada (2005). It is recommended that the threshold values from the WHO DWG (2008, the most current and relevant review) and CCME (2007, considered most appropriate due to the lack of published peer reviewed final guidance for non-carcinogenic effects) be adopted for the assessment of non-carcinogenic effects.

As noted by the US EPA, a mutagenic MOA is suspected for kidney tumours and hence the guidance has recommended that increased susceptibility associated with early lifetime exposures be addressed. No non-threshold values available for TCE are relevant for kidney tumours and have not been derived to specifically address early lifetime susceptibility and hence these issues may need to be addressed when characterising exposure to TCE.

Recommendation

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

Recommendation for TCE (quantitative toxicity values)

Carcinogenic end-points evaluated on the basis of:

Oral TRV = 0.00078 $(mg/kg/day)^{-1}$ (WHO DWG [2008]) for oral and dermal exposures

Inhalation TRV = 0.00043 $(mg/m^3)^{-1}$ (WHO 2000) for inhalation exposures

Adjustment of exposure to address early-lifetime susceptibility to carcinogenic end-points recommended

Non-Carcinogenic end-points evaluated on the basis of:

Oral TRV = 0.00146 mg/kg/day (WHO DWG [2008] and CCME [2007]) for oral and dermal exposures

Inhalation TRV = 0.04 mg/m^3 (CCME 2007) for inhalation exposures

Dermal absorption factor = 0.03 assumed for volatile compounds

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

1.6 Calculated interim HILs

Based on the evaluation presented above, a range of approaches has been identified for the quantification of exposure and toxicity. The following comments relate to the derivation of the interim soil vapour HIL A (also note the methodology and assumptions adopted as outlined in Schedule B7):

The calculated interim soil vapour HIL for TCE on the basis of the adopted threshold TRVs noted above is 2 mg/m^3.

The calculated interim soil vapour HIL for TCE on the basis of the adopted non-threshold TRVs noted above, with no additional consideration of early childhood exposures, is 6 mg/m^3.

The calculated interim soil vapour HIL for TCE on the basis of the adopted non-threshold TRVs noted above, with consideration of early childhood exposures (on the basis of the methodology provided by US EPA 2006), is 3 mg/m^3.

Based on the above, the most sensitive end-point for the derivation of the interim soil vapour HIL is the assessment of threshold (non-carcinogenic) effects.

On the basis of the above, the following interim soil vapour HILs have been derived for TCE:

HIL scenario	Interim soil vapour HIL# (mg/m^3)
Residential A	2
Residential B	2
Recreational C	15
Commercial D	15

Interim soil gas HILs are conservative soil gas concentrations that can be adopted for the purpose of screening sites where further investigation is required on a site-specific basis. They are based on the potential for vapour intrusion indoors using an indoor air to soil gas attenuation factor of 0.01 for scenarios A, B and D and an outdoor attenuation factor of 0.005 for scenario C.

1.7 References for TCE

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2 1,1,1-Trichloroethane

2.1 General

Several comprehensive reviews of 1,1,1-trichloroethane (1,1,1-TCA) in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 1997, 2006; WHO 1990). The following provides a summary of the key aspects of 1,1,1-TCA that are relevant to the derivation of interim HILs.

1,1,1-TCA is a synthetic chemical that does not occur naturally in the environment. It is a colourless, volatile liquid, with a characteristic sharp, sweet odour, and a vapour that is denser than air. It is slightly soluble in water, and is found in a number of solvents in a variety of domestic and industrial uses. 1,1,1-TCA is typically non-flammable under normal conditions; however, at higher vapour concentrations (10 %), it can burn when it contacts a spark (ATSDR 1997).

No natural sources of 1,1,1-TCA have been identified. 1,1,1-TCA is a chlorinated hydrocarbon which is manufactured from vinyl chloride by chlorination. 1,1,1-TCA had many industrial and household uses; however, its production has been limited to essential industrial use and is to be phased out due to its effects to the ozone layer (ATSDR 1997). It is widely used as a cleaning solvent, and is used to clean electrical equipment, motors, electronic components, printed circuit boards, photographic film and various metal and plastic parts. It is also used as a lubricant in metal-cutting oils and as a component in inks, correction fluid and drain cleaners (NHMRC 2004).

2.2 Previous HIL

No previous HIL is available for 1,1,1-TCA (NEPC 1999).

2.3 Proposed interim HIL

Review of available information in relation to the presence of 1,1,1-TCA in soil indicates that the vapour inhalation pathway is the most significant/important. This pathway should be assessed on the basis of measured vapour data, in particular, soil vapour data. There are significant limitations in the derivation of a soil HIL, in particular the modelling of phase partitioning from soil to soil vapour and the field measurement of volatiles in soil. Hence, an interim HIL has been derived for soil vapour only.

The following presents the values adopted for the calculation of a soil vapour interim HIL. In addition, other information that is relevant to the assessment of 1,1,1-TCA in soil (relevant to other pathways of exposure) is presented.

2.4 Significance of exposure pathways

Inhalation:

1,1,1-TCA is a volatile compound and, as such, the derivation of the HIL has considered the vapour inhalation pathway. The approach adopted for the quantification of potential vapour migration to outdoor air and intrusion indoors is outlined in Schedule B7. It is noted that the derived HIL is dominated by the assessment of these pathways of exposure. Due to limitations with the vapour modelling approach adopted, the HILs derived are considered interim.

The inhalation of particulates (outdoors and indoors) is considered essentially insignificant, compared with vapour inhalation.

Dermal absorption

Insufficient data is available on the dermal absorption of 1,1,1-TCA from soil. Given the volatility of the compound, dermal absorption is expected to be low; however, as there is insufficient data available to further assess dermal absorption from soil, a default value of 0.03 (3%) has been adopted for the volatile organic compounds (US EPA 1995).

Plant uptake

No data is available on the potential for 1,1,1-TCA to be taken up by home-grown produce. Given the volatility of this compound, the potential for plant uptake is expected to be similar to that of TCE, which was considered to be limited. As with the assessment presented for TCE, the use of the more commonly adopted equations for quantifying plant uptake (as presented in Schedule B7) that do not address uptake of volatiles (from air) rather than the root, or transformations within the plant, are not considered appropriate and relevant for the assessment of 1,1,1-TCA.

It is expected that the potential for plant uptake will be of less significance in the derivation of a HIL, when compared with the assessment of vapour inhalation, and given the limitations involved in providing a meaningful evaluation of plant uptake, it has not been considered in the derivation of HILs.

Intakes from other sources – background

As 1,1,1-TCA is highly volatile and not persistent, background intakes will be dominated by inhalation exposures. TCA has been reported in sampling undertaken in urban, suburban and industrial areas in NSW (DEC 2003) where the average concentration reported was 0.1 ppbv (0.5 µg/m^3) and the maximum reported in Beresfield of 0.3 ppbv (1.6 µg/m^3). Concentrations of 1,1,1-TCA in industrial air in Brisbane (Hawas et al. 2001) were similar (mean of 0.15 ppbv and maximum of 0.4 ppbv). These concentrations are lower than the average urban concentration assumed by ATSDR (2006) of 1 ppbv. Indoor air sources may also be significant; however, there are no estimates of exposure or intake from these sources.

Based on the recommended inhalation TRV for 1,1,1-TCA, these concentrations are essentially negligible.

2.5 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 1999) has classified 1,1,1-TCA as Group 3: not classifiable.

Review by the US EPA (2007) noted that for 1,1,1-TCA there is 'inadequate information to assess carcinogenic potential'.

Review of available values/information

There is insufficient data available to determine carcinogenicity of 1,1,1-TCA (WHO DWG, ATSDR 2006 and US EPA 2007). Review by the US EPA (2007) has noted that 1,1,1-TCA has been tested extensively for genotoxic potential. The chemical has shown little capacity to produce genotoxic effects in bacteria or fungi. Results in mammalian test systems *in vitro* and *in vivo* were more mixed, but still predominantly negative for assays other than cell transformation. The chemical has been shown to interact weakly with DNA. The overall weight of evidence suggests that 1,1,1-TCA is not considered genotoxic.

On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for 1,1,1-TCA. 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 guideline established	No guideline established in current ADWG (NHMRC 2004) due to insufficient data.
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	TDI = 0.6 mg/kg/day	The WHO DWG (2008) has derived a guideline of 2 mg/L based on a TDI of 0.6 mg/kg/day based on a NOAEL of 600 mg/kg associated with liver and kidney effects from a short-duration oral study in rats and an uncertainty factor of 1000.
RIVM (1993)	MPC = 4.8 mg/m^3	Maximum permissible concentration (MPC) in air derived on the basis of a duration corrected NOAEL of 482 mg/m3 associated with liver effects in a 2-year rat inhalation study and an uncertainty factor of 100.
ATSDR (2006)	No chronic MRLs derived	
US EPA (IRIS 2010)	RfD = 2 mg/kg/day RfC = 5 mg/m^3	Oral RfD of 2 mg/kg/day derived on the basis of a benchmark approach with a BMDL10 of 2155 mg/kg/day associated with reduced body weight in a 90 day mouse study and an uncertainty factor of 1000 (including 3 for database deficiencies). RfC derived on the basis of a NOAEL (HEC) of 1553 mg/m^3 associated with liver effects in mice and rats and an uncertainty factor of 100.

Limited quantitative data is available for 1,1,1-TCA; hence, it is recommended that the TDI available from the WHO be adopted for the assessment of oral intakes and the inhalation value from the US EPA review (essentially the same as derived by RIVM) be adopted for the assessment of inhalation exposures.

Recommendation

On the basis of the information presented above the following TRVs have been adopted for 1,1,1-TCA in the derivation of HILs:

Recommendation for 1,1,1-TCA

Oral TRV = 0.6 mg/kg/day (WHO DWG [2008]) relevant to oral and dermal routes of exposure

Dermal absorption factor = 0.03 (or 3%) assumed for volatile compounds

Inhalation TRV = 5 mg/m^3 (US EPA IRIS [2010])

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

2.6 Calculated interim HILs

On the basis of the above, the following interim soil vapour HILs have been derived for 1,1,1-TCA:

HIL scenario	Interim soil vapour HIL# (mg/m^3)
Residential A	260
Residential B	260
Recreational C	1800
Commercial D	1800

Interim soil gas HILs are conservative soil gas concentrations that can be adopted for the purpose of screening sites where further investigation is required on a site-specific basis. They are based on the potential for vapour intrusion indoors using an indoor air to soil gas attenuation factor of 0.01 for scenarios A, B and D and an outdoor attenuation factor of 0.005 for scenario C.

2.7 References for 1,1,1-TCA

ATSDR 1997, *Toxicological profile for 1, 1, 1-trichloroethane*, United States Department of Health and Human Services, Atlanta, Georgia, USA.

ATSDR 2006, Toxicological profile for 1,1,1-trichloroethane, available online at: <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=432&tid=76>.

DEC 2003, 'Ambient concentrations of heavy metals in NSW', *Ambient air quality research project (1996-2001)*, Internal working paper no. 4, Department of Environment and Conservation, New South Wales.

Hawas, O, Hawker, D, Chan A, Cohen, D, Christensen, E & Golding, G 2001, *Characterisation and identification of sources of volatile organic compounds in an industrial area in Brisbane.*

IARC 1999, *Summaries and evaluations: 1,1,1-trichloroethane*, International Agency for Research on Cancer, vol. 71, p. 881.

NEPC 1999, 'Schedule B (7a), Guideline on health-based investigation levels', *National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council.

NHMRC & NRMMC 2004, *National water quality management strategy. Australian drinking water guidelines*, National Health and Medical Research Council & Natural Resource Management Ministerial Council, Australia.

RAIS 2010, Risk assessment information system, website and database maintained by the Oak Ridge Operations Office, available online at < http://rais.ornl.gov/>.

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 1995, Assessing dermal exposure from soil, technical guidance manual,. US EPA Region 3, available online at <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.

US EPA 2007, *Toxicological review of 1,1,1-trichloroethane, in support of summary information on the integrated risk information system* (IRIS).

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 1990, *1,1,1-trichloroethane*, Environmental health criteria 136, WHO 1990, available online at: <http://www.inchem.org/documents/ehc/ehc/ehc136.htm>.

WHO DWG 2008, 2009, *Guidelines for drinking water quality, 3rd edn, incorporating first and second addenda (2008) and rolling revisions current to 2009*, World Health Organisation, available online at <http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html>.

3 Tetrachloroethene (PCE)

3.1 General

Several comprehensive reviews of tetrachloroethene (PCE) in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 1997; WHO 2006; NICNAS 2001; US EPA 2008). The following provides a summary of the key aspects of PCE that are relevant to the derivation of interim HILs.

Tetrachloroethene, also known as perchloroethylene (PCE) and tetrachloroethylene, is a synthetic, colourless, volatile, non flammable liquid, with a characteristic sharp, sweet odour. It has a relatively low solubility in water and is commonly used as a dry-cleaning and metal degreasing solvent (ATSDR 1997). PCE manufacture in Australia ceased in 1991. Use in Australia has declined from 1995, consistent with declining use worldwide. PCE is primarily imported in its 'pure' form with approximately 80% used in the dry cleaning industry in Australia (NICNAS 2001).

PCE is widespread in the environment and is found in trace amounts in water, aquatic organisms, air, foodstuffs, and human tissue. The highest environmental levels of PCE are found in the commercial dry-cleaning and metal-degreasing industries. PCE may degrade in the environment to more toxic compounds, including vinyl chloride (WHO 2006).

3.2 Previous HIL

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

3.3 Proposed interim HIL

Review of available information in relation to the presence of PCE in soil indicates that the vapour inhalation pathway is the most significant/important. This pathway should be assessed based on measured vapour data, in particular soil vapour data. There are significant limitations in the derivation of a soil HIL, in particular the modelling of phase partitioning from soil to soil vapour and the field measurement of volatiles in soil; hence, an interim HIL has been derived for soil vapour only.

The following presents the values adopted for the calculation of a soil vapour interim HIL. In addition, other information that is relevant to the assessment of PCE in soil (relevant to other pathways of exposure) is presented.

3.4 Significance of exposure pathways

Inhalation

PCE is a volatile compound and, as such, the derivation of the HIL has considered the vapour inhalation pathway. The approach adopted for the quantification of potential vapour migration to outdoor air and intrusion indoors is outlined in Schedule B7. It is noted that the derived HIL is dominated by the assessment of these pathways of exposure. Due to limitations with the vapour modelling approach adopted, the HILs derived are considered interim.

The inhalation of particulates (outdoors and indoors) is considered essentially insignificant, compared with vapour inhalation.

Dermal absorption

Insufficient data is available on the dermal absorption of PCE from soil. Given the volatility of the compound, dermal absorption is expected to be low; however, as there is insufficient data available to further assess dermal absorption from soil, a default value of 0.03 (3%) has been adopted for the volatile organic compounds (US EPA,1995).

Plant uptake

Limited data are available on the potential for PCE to be taken up by home-grown produce. Some data is available on the effects of PCE vapours on plant growth with a predicted no effect concentration (PNEC) of 8.2 µg/m^3 identified. ATSDR (1997) also notes that food products can absorb PCE from the atmosphere over time; hence, some studies on the level of PCE in food products are expected to reflect this process, rather than plant uptake from the roots. Given the volatility of this compound, the potential for plant uptake is expected to be limited. As with the assessment presented for TCE, the use of the more commonly adopted equations for quantifying plant uptake (as presented in Schedule B7) that do not address uptake of volatiles (from air) rather than the root, or transformations within the plant, are not considered appropriate and relevant for the assessment of PCE.

It is expected that the potential for plant uptake will be of less significance in the derivation of a HIL, when compared with the assessment of vapour inhalation, and given the limitations involved in providing a meaningful evaluation of plant uptake it has not been considered in the derivation of HILs.

Intakes from other sources – background

As PCE is highly volatile and not persistent, background intakes will be dominated by inhalation exposures. Concentrations of PCE in industrial, urban and regional areas are available in Australia. Data collected (DEC 2003) from urban and regional areas in NSW report average concentrations of PCE of approximately 0.1 ppbv, or 0.0007 mg/m^3 with a maximum concentration in Sydney CBD of 1.6 ppbv, or 0.01 mg/m^3. Concentrations in an industrial area in Brisbane (Hawas et al. 2001) have reported average and maximum concentrations reported of 0.015 mg/m^3 and 0.085 mg/m^3 respectively. These concentrations are consistent with those reported in other cities in Australia (NICNAS 2001).

The average air concentration reported by DEC (2003) comprises less than 5% of the recommended inhalation TRV for PCE.

Other significant exposures by the general public are likely to occur through the use of dry cleaning. Variable concentrations of PCE in homes and where dry-cleaned clothes are stored and worn are reported by NICNAS (2001) and the WHO (2000). A study on the effect of wearing dry-cleaned clothes reported median personal air concentrations ranging of 0.032 mg/m^3 to 0.22 mg/m^3 depending on the garment. These exposures along with exposures to paint solvents and cleaning material containing PCE were considered potentially significant. No estimate of intake by the general public is provided in the NICNAS review. Median indoor air concentrations reported by WHO (2006) for homes not located in the same building as dry-cleaners was 0.004 mg/m^3 (note that concentrations indoors were much higher in buildings with a dry-cleaners with indoor air levels ranging from 0.05 to 6.1 mg/m^3). This value is also essentially negligible compared with the recommended inhalation TRV. While there is the potential for increased background intakes depending on consumer use of products and frequency of dry-cleaning, average intakes are considered negligible.

3.5 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 1995) has classified PCE as Group 2A: probably carcinogenic to humans based on limited evidence in humans and sufficient evidence in experimental animals.

PCE was classified as a 'probable' human carcinogen (Category B2) by the US EPA for all routes of exposure based upon evidence from animal studies. This classification has been withdrawn pending further review and has not been finalised to date.

Review of available values/information

Some epidemiological studies indicate a possible association between chronic exposure to PCE and an increased cancer risk; however, the evidence provided is considered to be inconclusive (US EPA 2008). This is mainly due to concurrent exposure to other petroleum solvents as well as PCE, confounding factors (smoking, alcohol, socio-economic status) and small numbers of cancers in the studies.

An association between exposure to PCE (inhalation and ingestion) and an increased risk of cancer (mononuclear cell leukaemia and hepatic tumours) in animals has been suggested. Review of PCE by WHO (2000) indicates that PCE is a non-genotoxic animal carcinogen. Review of the possible mechanisms of tumour formation by PCE in animals suggests that the tumours observed may have little relevance for humans. This is subject to some debate; however, reviews by WHO (2006) and US EPA (2008) have noted that in the absence of suitable supporting evidence to the contrary, it must be concluded that the cancers produced by PCE in rodents are of potential relevance to humans.

From the weight of evidence, PCE does not appear to have significant genotoxic potential; however, some of the possible metabolites are recognised Ames bacterial mutagens (WHO 2000 and 2006, RIVM 2001). Review of the available studies by the WHO (2006) suggests that non-genotoxic mechanisms have been recognised for the formation of kidney tumours in male rats and liver tumours in mice for some chemicals. The available data on MOA for PCE are limited, and the dose–response data related to these recognised mechanisms are not consistent with the dose–response relationships for cancer induction by PCE. The WHO (2006) has derived a threshold inhalation value for PCE that is considered protective of key end-points including carcinogenicity. Hence it may be considered appropriate that a threshold dose-response approach be adopted for PCE.

It is noted that the draft review of PCE by the US EPA (2008) suggests that PCE has been shown to induce some genotoxic effects. There are a number of limitations noted in the assessment presented by the US EPA; in particular, that the MOA for PCE inducing carcinogenesis is not yet fully characterised or understood, and that the role of genotoxicity in hepatocarcinogenicity is uncertain. Where the US EPA lacks certainty, the default position is to be conservative and, as such, they have suggested considering PCE having a mutagenic MOA, where a non-threshold approach is recommended for the assessment of carcinogenicity and mutagenicity. The assessment of PCE should be updated should additional data become available that supports the US EPA review.

The following quantitative values are available for PCE from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2004)	TDI = 0.014 mg/kg/day	The current ADWG (NHMRC 2004) have derived a guideline of 0.05 mg/L for PCE based on a no-observed-effect level (NOEL) of 14 mg/kg/day from a 90-day drinking water study in rats and mice and an uncertainty factor of 1000. The uncertainty factor includes an additional 10 fold factor to address possible carcinogenicity.
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	TDI = 0.014 mg/kg/day	WHO DWG TDI based on the same study and uncertainty factor as noted in the ADWG (NHMRC 2004).
WHO (2006)	TC = 0.2 mg/m^3 TDI = 0.05 mg/kg/day	TC in air derived on the basis of the most sensitive endpoint, namely neurotoxicological effects, based on a LOAEC (adjusted) of 20 mg/m^3 from an occupational inhalation study (mean exposure of 10 years) and an uncertainty factor of 100. The TC derived lower that that from other key end-points such as kidney and liver effects and reproductive/developmental effects. Potential carcinogenic effects have been assessed on the basis of a benchmark dose (BMD) approach with a $BMCL_{10}$ of 20 mg/m^3 and if a multi-stage model were considered the TC of 0.2 mg/m^3 would be associated with a risk of 1 x10^{-3}. The assessment presented by WHO (2006) is an update of the earlier assessment presented in the WHO Air Quality Guidelines (2000) where a TC of 0.25 mg/m^3 was derived. Concern about potential carcinogenic effects are noted (and should be addressed through an in-depth risk assessment) in the guideline. TDI derived using a PBPK model using the inhalation TC data.
RIVM (2001)	TDI = 0.016 mg/kg/day TC = 0.25 mg/m^3	TDI derived on the basis of a NOAEL of 16 mg/kg/day associated with liver effects in a 4-week oral study in rats and an uncertainty factor of 1000. TC adopted based on older WHO (2000) evaluation derived from a lowest-observed-adverse-effect level (LOAEL) (adjusted) of 23 mg/m^3 from an occupational inhalation study and an uncertainty factor of 100.
Health Canada (1993)	TDI = 0.014 mg/kg/day TC = 0.36 mg/m^3	TDI derived on the same basis as noted for the WHO DWG and ADWG. TC derived from a LOAEL of 363 (adjusted) mg/m^3 associated with multiple effects in mice and an uncertainty factor of 1000.
ATSDR (1997)	No chronic oral MRL Inhalation MRL =0.24 mg/m^3	Nor chronic oral MRL has been established. The chronic inhalation MRL has been derived on the basis of a LOAEL (adjusted) of 24 mg/m^3 associated with neurobehavioural effects in an occupational inhalation study and an uncertainty factor of 100.
USEPA (IRIS 2010)	RfD = 0.01 mg/kg/day	Oral reference dose (RfD) (last reviewed in 1988) derived on the basis of a NOAEL of 14 (adjusted) associated with liver effects in mice and an uncertainty factor of 1000 (and the RfD rounded).
USEPA (2008 draft)	RfD = 0.004 mg/kg/day RfC = 0.02 mg/m^3	RfD derived based on a point of departure of 1.1 mg/kg/day obtained from route extrapolation of the study used to derive the RfC, noted to be similar to that obtained for liver effects in an oral mouse study and an uncertainty factor of 300. RfC derived on the basis of a LOAEL of 4.8 mg/m^3 associated with CNS effects from residents exposed to PCE living in close proximity to a dry-cleaning establishment and an uncertainty factor of 300. The draft USEPA review has also presented non-threshold values for the assessment of carcinogenicity with a unit risk in the range 2x10^{-6} to 2x10^{-5} (µg/m^3)$^{-1}$ derived from rat leukaemia data and different pharmacokinetic models. An oral SF is also derived, based on route extrapolation from the inhalation study.

For oral exposures, it is recommended that the TDI adopted in the derivation of the ADWG (NHMRC 2004) and WHO DWG (2008) be adopted. This value is consistent with threshold values derived from oral studies from RIVM (2001), Health Canada, and US EPA (IRIS, 2010). The draft US EPA (2008) value is derived based on route extrapolation from an inhalation study.

For inhalation exposures, it is recommended that the TC derived by the more recent WHO (2006) review be adopted. This is consistent with that derived by other agencies, with the exception of the draft US EPA value which is 10 times lower.

Recommendation

On the basis of the information presented above the following TRVs have been adopted for PCE in the derivation of HILs:

Recommendation for PCE

Oral TRV = 0.014 mg/kg/day (NHMRC 2004 and WHO DWG[2008]) relevant to oral and dermal pathways of exposure

Dermal absorption factor = 0.03 (or 3%) assumed for volatile compounds

Inhalation TRV = 0.2 mg/m^3 (WHO 2006) for inhalation exposures

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

3.6 Calculated interim HILs

On the basis of the above, the following interim soil vapour HILs have been derived for PCE:

HIL scenario	Interim soil vapour HIL# (mg/m^3)
Residential A	10
Residential B	10
Recreational C	70
Commercial D	70

Interim soil gas HILs are conservative soil gas concentrations that can be adopted for the purpose of screening sites where further investigation is required on a site-specific basis. They are based on the potential for vapour intrusion indoors using an indoor air to soil gas attenuation factor of 0.01 for scenarios A, B and D and an outdoor attenuation factor of 0.005 for scenario C.

3.7 References for PCE

ATSDR 1997, Toxicological profile for tetrachloroethylene, available online at: <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=265&tid=48>.

DEC 2003, 'Ambient concentrations of heavy metals in NSW', *Ambient air quality research project (1996-2001)*, Internal working paper no. 4, Department of Environment and Conservation, New South Wales.

Health Canada 1993, Tetrachloroethylene, Priority substances list, Assessment report.

Hawas, O, Hawker, D, Chan A, Cohen, D, Christensen, E & Golding, G 2001, *Characterisation and identification of sources of volatile organic compounds in an industrial area in Brisbane.*

IARC 1995, 'Dry cleaning some chlorinated solvents and other industrial compounds', *IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans*, vol. 63, Lyon, International Agency for Research on Cancer, Lyon.

NEPC 1999, 'Schedule B (7a), Guideline on health-based investigation levels, *National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council.

NICNAS 2001, *Tetrachloroethylene – Priority existing chemical assessment report no. 15*, National Industrial Chemicals Notification and Assessment Scheme.

NHMRC & NRMMC 2004, *National water quality management strategy. Australian drinking water guidelines*, National Health and Medical Research Council & Natural Resource Management Ministerial Council, Australia.

RAIS 2010, Risk assessment information system, website and database maintained by the Oak Ridge Operations Office, available online at < http://rais.ornl.gov/>.

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 1995, Assessing dermal exposure from soil: technical guidance manual,. US EPA Region 3, available online at <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.

US EPA 2008, Toxicological review of tetrachloroethylene (perchloroethylene), in support of summary information on the integrated risk information system (IRIS), External review draft.

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 2000, *Air quality guidelines for Europe*, 2nd edn, World Health Organisation.

WHO 2006, *Concise international chemicals assessment document (CICAD) 68: tetrachloroethene*, World Health Organisation, available online at: <http://www.inchem.org/documents/cicads/cicads/cicad68.htm#1.0>.

WHO DWG 2008, 2009, *Guidelines for drinking water quality, 3rd edn, incorporating first and second addenda (2008) and rolling revisions current to 2009*, World Health Organisation, available online at <http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html>.

4 Cis-1,2-Dichloroethene (DCE)

4.1 General

Several comprehensive reviews of *cis*-1,2-dichloroethene (DCE) in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 1996; WHO DWG 2008). The following provides a summary of the key aspects of DCE that are relevant to the derivation of interim HILs.

DCE is a colourless, volatile liquid with a characteristic sharp harsh odour. It is one of two isomers of 1,2-DCE, the second being *trans*-1,2-DCE. It is highly flammable and extremely corrosive.

DCE is not known to occur naturally. It is most commonly used as a chemical intermediate to produce chlorinated solvents and chemical compounds. It is also used in rubber extraction, pharmaceutical manufacturing, as a refrigerant and in the extraction of oils from meats and fish. DCE has also historically been used as a solvent for a variety of waxes, resins, perfumes, dyes, lacquers, acetyl cellulose, thermoplastics and phenols (ATSDR 1996).

4.2 Previous HIL

No previous HIL is available for DCE (NEPC, 1999).

4.3 Proposed interim HIL

Review of available information in relation to the presence of DCE in soil indicates that the vapour inhalation pathway is the most significant/important. This pathway should be assessed on the basis of measured vapour data, in particular soil vapour data. There are significant limitations in the derivation of a soil HIL, in particular the modelling of phase partitioning from soil to soil vapour and the field measurement of volatiles in soil. Hence, an interim HIL has been derived for soil vapour only.

The following presents the values adopted for the calculation of a soil vapour interim HIL. In addition, other information that is relevant to the assessment of DCE in soil (relevant to other pathways of exposure) is presented.

4.4 Significance of exposure pathways

Inhalation

DCE is a volatile compound and, as such, the derivation of the HIL has considered the vapour inhalation pathway. The approach adopted for the quantification of potential vapour migration to outdoor air and intrusion indoors is outlined in Schedule B7. It is noted that the derived HIL is dominated by the assessment of these pathways of exposure. Due to limitations with the vapour modelling approach, adopted the HILs derived are considered interim.

The inhalation of particulates (outdoors and indoors) is considered essentially insignificant, compared with vapour inhalation.

Dermal absorption

Insufficient data are available on the dermal absorption of DCE from soil. Given the volatility of the compound, dermal absorption is expected to be low; however, as there is insufficient data available to further assess dermal absorption from soil, a default value of 0.03 (3%) has been adopted for the volatile organic compounds (US EPA 1995).

Plant uptake

No data is available on the potential for DCE to be taken up by home-grown produce. Given the volatility of this compound, the potential for plant uptake is expected to be limited. As with the assessment presented for TCE, the use of the more commonly adopted equations for quantifying plant uptake (as presented in Schedule B7) that do not address uptake of volatiles (from air) rather than the root, or transformations within the plant, are not considered appropriate and relevant for the assessment of DCE.

It is expected that the potential for plant uptake will be of less significance in the derivation of a HIL when compared with the assessment of vapour inhalation and, given the limitations involved in providing a meaningful evaluation of plant uptake, it has not been considered in the derivation of HILs.

Intakes from other sources – background

As DCE is highly volatile and not persistent, background intakes will be dominated by inhalation exposures. DCE is not considered to be a typical urban air contaminant and little data is available from data collected in Australian cities. *Cis*-1,2-DCE has been detected in volatile organic compound (VOC) sampling from Perth (WA DEP 2000) with average concentrations of 0.2 ppb (0.8 µg/m^3) and a maximum reported concentration of 2.1 ppb (8.3 µg/m^3). These values were comparable to average concentrations reported in air in the US and used by RIVM (2001) to estimate background intake of 1,2-DCE (both isomers) of approximately 0.13 µg/kg/day. Based on the recommended TRV for DCE, this intake is less than 5% and considered negligible (0%).

4.5 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC) has not classified DCE.

The US EPA has classified DCE as Group D: not classifiable.

Review of available values/information

There are no adequate data available to assess the carcinogenicity of DCE. Review of available genotoxicity studies by the WHO DWG (2008) provided equivocal results. Review by RIVM (2001) suggested that *cis*-1,2-DCE could be considered genotoxic *in vivo*, producing gene mutations and chromosome aberrations. However, no carcinogenic toxicity values have been derived for the *cis*- isomer. A more recent review of genotoxicity provided by the US EPA (2009 draft) suggested that overall, data for 1,2-DCE (both isomers) are not positive for genotoxicity an mutagenicity. The positive results (considered by RIVM) are considered inconsistent by the US EPA and need further confirmation. On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for DCE. 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)	TDI = 0.017 mg/kg/day for *trans*- isomer	The Australian Drinking Water Guidelines (NHMRC 2004) have derived a drinking water guideline of 0.06 mg/L for 1,2-DCE (both isomers) following guidance from the WHO (refer below).
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	TDI = 0.017 mg/kg/day for *trans*- isomer	The WHO DWG(2008) has derived a guideline of 0.05 mg/L based on a TDI of 0.017 mg/kg/day associated with a NOAEL of 17 mg/kg from a 90 day study in mice administered *trans*-1,2-DCE in drinking water and an uncertainty factor of 1000. This guideline is relevant to the sum of both *cis*- and *trans*- isomers, however this is due to the WHO adopting a conservative approach where there is no data available for the derivation of a *cis*- isomer value.
RIVM (2001)	TDI = 0.006 mg/kg/day TC = 0.03 mg/m^3	A TDI of 0.006 mg/kg/day has been established for *cis*-1,2-DCE based on a NOAEL of 32 mg/kg/day from a 90 day oral rat study (using the *cis*- isomer) and an uncertainty factor of 5000. Inhalation TC were derived for *cis*-1,2-DCE using route extrapolation from the oral study, resulting in a TC of 0.03 mg/m^3
ATSDR (1996)	No chronic MRLs derived	
US EPA (IRIS 2010) – currently withdrawn pending review	RfD = 0.02 mg/kg/day for *trans*- isomer	Oral reference dose (RfD) of 0.02 mg/kg/day for *trans*-1,2-DCE on the basis of a 90 day mouse study with drinking water (same study as used by WHO).
US EPA (2009 draft)	RfD = 0.006 mg/kg/day for *cis*- isomer	RfD derived on the bases of a $BMDL_{10}$ of 18.6 mg/kg/day from a 90 day study in mice (same study as considered by RIVM) with an uncertainty factor of 3000.

It is recommended that the threshold values derived from studies associated with the *cis*-isomer (as recommended by RIVM (2001), consistent with that derived by US EPA (2009 draft)), be adopted for the assessment of oral/dermal and inhalation routes of exposure associated with DCE.

Recommendation

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

Recommendation for *cis*-1,2-DCE

Oral TRV = 0.006 mg/kg/day (RIVM 2001; US EPA 2009) relevant to oral and dermal exposures

Dermal absorption factor = 0.03 (or 3%) assumed for volatile compounds

Inhalation TRV = 0.03 mg/m^3 (RIVM 2001)

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

4.6 Calculated interim HILs

On the basis of the above, the following interim soil vapour HILs have been derived for DCE:

HIL scenario	Interim soil vapour HIL# (mg/m^3)
Residential A	2
Residential B	2
Recreational C	10
Commercial D	10

Interim soil gas HILs are conservative soil gas concentrations that can be adopted for the purpose of screening sites where further investigation is required on a site-specific basis. They are based on the potential for vapour intrusion indoors using an indoor air to soil gas attenuation factor of 0.01 for scenarios A, B and D and an outdoor attenuation factor of 0.005 for scenario C.

4.7 References for DCE

ATSDR 1996, *Toxicological profile for 1,2-dichloroethene*, available online at <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=464&tid=82>.

NEPC 1999, 'Schedule B (7a), Guideline on health-based investigation levels, *National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council.

NHMRC & NRMMC 2004, *National water quality management strategy. Australian drinking water guidelines*, National Health and Medical Research Council & Natural Resource Management Ministerial Council, Australia.

RAIS 2010, Risk assessment information system, website and database maintained by the Oak Ridge Operations Office, available online at < http://rais.ornl.gov/>.

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 1995, Assessing dermal exposure from soil: technical guidance manual, US EPA Region 3, available online at <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.

US EPA 2009, *Toxicological review of cis-1,2-dichloroethylene and trans-1,2-dichloroethylene, in support of summary information on the integrated risk information system* (IRIS), External review draft.

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

WA DEP 2000, *Volatile organic compounds monitoring in Perth, baseline air toxics project*, Western Australia Department of Environmental Protection.

WHO DWG 2008, 2009, *Guidelines for drinking water quality, 3rd edn, incorporating first and second addenda (2008) and rolling revisions current to 2009*, World Health Organisation, available online at <http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html>.

5 Vinyl chloride

5.1 General

Several comprehensive reviews of vinyl chloride in the environment and toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 2006; WHO 1999; IARC 2008). The following provides a summary of the key aspects of vinyl chloride that are relevant to the derivation of interim HILs.

Vinyl chloride is a colourless, flammable gas, with a characteristic slightly sweet odour. It has a high vapour pressure, a high value for Henry's law constant, a relatively low solubility in water, and is heavier than air. It is also soluble in most organic solvents. Under pressure, vinyl chloride is easily liquefied, and is commonly stored and transported as a liquid and made into polyvinylchloride (PVC) (ATSDR, 2006).

Vinyl chloride is not known to occur naturally. Vinyl chloride is predominantly used in the plastics industry, in the production of polyvinyl chloride (PVC). PVC is used in numerous industries including packaging, building, electrical appliances, medical care, agriculture, automobiles and toys. Vinyl chloride is also used in limited quantities as a refrigerant and an intermediate in the production of chlorinated compounds (WHO 1999).

Vinyl chloride is a degradation product of PCE/TCE/1,2-DCE and 1,1-DCE and its presence in the environment may not be due to a primary source; rather, it may be due to degradation of other chlorinated sources.

5.2 Previous HIL

No previous HIL is available for vinyl chloride (NEPC 1999).

5.3 Proposed interim HIL

Review of available information in relation to the presence of vinyl chloride in soil indicates that the vapour inhalation pathway is the most significant/important. This pathway should be assessed on the basis of measured vapour data, in particular soil vapour data. There are significant limitations in the derivation of a soil HIL, in particular the modelling of phase partitioning from soil to soil vapour and the field measurement of volatiles in soil. Hence, an interim HIL has been derived for soil vapour only.

The following presents the values adopted for the calculation of a soil vapour interim HIL. In addition, other information that is relevant to the assessment of vinyl chloride in soil (relevant to other pathways of exposure) is presented.

5.4 Significance of exposure pathways

Inhalation

Vinyl chloride is a volatile compound and, as such, the derivation of the HIL has considered the vapour inhalation pathway. The approach adopted for the quantification of potential vapour migration to outdoor air and intrusion indoors is outlined in Schedule B7. It is noted that the derived HIL is dominated by the assessment of these pathways of exposure. Due to limitations with the vapour modelling approach adopted, the HILs derived are considered interim.

It is noted that there is the potential for vinyl chloride to undergo biodegradation within the soil profile. Available data (Scheutz 2002) suggests that the degradation of vinyl chloride is complex involving both anaerobic and aerobic processes. Vinyl chloride is rapidly degraded in the presence of oxygen and is considered one of the least stable chlorinated chemical in soil gas. NJ DEP (2005) notes that due to these processes vinyl chloride is seldom found in soil gas above a contaminated source. Hence, while the potential for vapour migration to be significant has been modelled and considered in the HILs, due to the potential for degradation this approach is expected to be conservative for vinyl chloride.

The inhalation of particulates (outdoors and indoors) is considered essentially insignificant, compared with vapour inhalation.

Dermal absorption

Insufficient data is available on the dermal absorption of vinyl chloride from soil. Given the volatility of the compound, dermal absorption is expected to be low; however, as there is insufficient data available to further assess dermal absorption from soil a default value of 0.03 (3%) has been adopted for the volatile organic compounds (US EPA 1995).

Plant uptake:

No data are available on the potential for vinyl chloride to be taken up by home-grown produce. It is noted that vinyl chloride can be absorbed by produce packaged in PVC plastic. Concentrations reported in these products are not associated with plant uptake from soil. Given the volatility of this compound, the potential for plant uptake is expected to be limited. As with the assessment presented for TCE, the use of the more commonly adopted equations for quantifying plant uptake (as presented in Schedule B7) that do not address uptake of volatiles (from air) rather than the root, or transformations within the plant, are not considered appropriate and relevant for the assessment of vinyl chloride.

It is expected that the potential for plant uptake will be of less significance in the derivation of a HIL, when compared with the assessment of vapour inhalation, and given the limitations involved in providing a meaningful evaluation of plant uptake it has not been considered in the derivation of HILs.

Intakes from other sources – background

As vinyl chloride is highly volatile and not persistent, background intakes will be dominated by inhalation exposures. Concentrations of vinyl chloride in industrial, urban and regional areas are available in Australia. Data collected in NSW (DEC 2003) from urban and regional areas in NSW note that vinyl chloride was rarely detected (<1% of samples) with the maximum reported from the Sydney CBD of 0.3 ppbv (0.0008 mg/m^3). Vinyl chloride was not detected in ambient air sampling undertaken in Perth (WA DEP 2000). In addition, vinyl chloride has not been detected in drinking water and low levels are expected in food (NHMRC 2004). Low levels have been historically reported in some consumer products. Background intakes expected from vinyl chloride are expected to be low, with conservative intakes estimated by Health Canada (1992) of approximately 0.005 mg/kg/day and RIVM (2001) of approximately 0.00006 mg/kg/day (predominantly from inhalation). It is noted that, as the most sensitive end-point is carcinogenicity, which is assessed on the basis of a non-threshold approach, background intakes are not used in the derivation of the HIL.

5.5 Identification of toxicity reference values

Classification

The International Agency for Research on Cancer (IARC 2008) has classified vinyl chloride as Group 1: carcinogenic to humans.

Vinyl chloride is also classified as a known human carcinogen (Category A) by the US EPA for the inhalation route of exposure, and by analogy for the oral route of exposure. It is also considered highly likely to be carcinogenic by the dermal route.

Review of available values/information

Exposure to vinyl chloride via inhalation has been associated with increase in liver cancer including a rare form of angiosarcoma and biliary tract cancer. Other studies have indicated increase incidence of CNS and brain cancer. While most data is associated with inhalation exposures, ingestion studies suggest evidence of carcinogenicity via oral exposure (WHO 1999; ATSDR 2006).

Vinyl chloride has been identified as genotoxic and mutagenic (WHO 1999; ATSDR 2006; US EPA 2000). The US EPA (2000) review notes that vinyl chloride toxicity occurs via a genotoxic pathway (identified from a number of lines of evidence) that is understood in some detail. On this basis, the assessment of carcinogenicity on the basis of a non-threshold (linear) approach is appropriate.

The US EPA (2000) review also noted that chemically induced human liver carcinogenicity is associated with mutational alteration of multiple genes, consistent with a mutagenic mode of action. In addition, several studies of partial lifetime exposure suggest that the lifetime cancer risk depends on age at exposure, with higher lifetime risks attributable to exposures at younger ages. This is also noted by the WHO (2000 and in the WHO DWG 2008). Consistent with US EPA guidance, the derivation of non-threshold values for vinyl chloride has incorporated factors that address early life susceptibility and hence, if the US EPA non-threshold values are adopted (also considered in the WHO values), no additional adjustment is required in the quantification of exposure. It is noted, however, that the application of the US EPA values for exposures by adults only (such as workers) needs to adopt the most correct values that do not include early-life susceptibility.

The most sensitive end-point for vinyl chloride (particularly inhalation which will dominate the derivation of a HIL) is carcinogenicity (noting that in the derivation of the ADWG both carcinogenic and non-carcinogenic effects were considered as sensitive for the oral pathway). Hence, the selection of appropriate non-threshold values for the assessment of vinyl chloride exposure is relevant.

The following quantitative non-threshold values are available for vinyl chloride from Level 1 Australian and international sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2004)	Adopted WHO non-threshold approach.	Current guideline derived on the basis of the WHO non-threshold value and additional consideration of non-carcinogenic effects with a TDI of 0.00013 mg/kg/day
OCS (2008)	No evaluation available	
International		
WHO DWG (2008)	SF = 2.3 $(mg/kg/day)^{-1}$ (for exposures from birth) SF = 1.15 $(mg/kg/day)^{-1}$ (for exposures as adults)	WHO DWG (2008) derived on the basis of linear extrapolation from dose response data for all liver tumours from an oral exposure study in rats and assuming a doubling of the risk of exposure from birth (incorporating the 2-fold uncertainty identified by the US EPA (2000) review to address early life sensitivity. Exposures by workers (only adults) can be calculated on the basis of a slope factor that is 2 times lower.
WHO (2000)	UR = $1x10^{-6}$ $(\mu g/m^3)^{-1}$	Inhalation UR derived on the basis of occupational exposures studies associated with haemangiosarcoma and a linear multistage model. The value derived is noted to be limited as it does not address early life sensitivity identified in newborn animals (relevant to exposures by children to 10 years).
Health Canada (1992)	SF = 0.26 $(mg/kg/day)^{-1}$	SF based on the upper value from a free extrapolation method associated with hepatocellular angiosarcomas in female rats. The evaluation is older than that considered by the WHO and US EPA and does not include any consideration of early life sensitivity.
RIVM (2001)	SF = 0.17 $(mg/kg/day)^{-1}$ UR = $2.8x10^{-5}$ $(\mu g/m^3)^{-1}$	SF derived on the basis of hepatocellular carcinomas, angiosarcomas and neoplastic nodules in female rats as markers for carcinogenic response, and a linear extrapolation model. Inhalation UR derived on the basis liver effects in an inhalation study on female rats and mice and an extrapolation model. No consideration of early-life sensitivity was considered by RIVM. Threshold values were also derived for non-carcinogenic effects with a TDI = 0.0013 mg/kg/day which is based on the same study as considered in the ADWG, but with a less conservative uncertainty factor of 100. An inhalation TC = 0.056 mg/m^3 was derived based on an inhalation study. RIVM notes that the carcinogenic end-points are most sensitive.
ATSDR (2006)	No quantitative assessment of carcinogenic effects	ATSDR does not provide quantitative estimates of carcinogenic effects. However for non-carcinogenic effects a chronic oral MRL = 0.003 associated with non-neoplastic effects in livers from a chronic oral rat study was derived.

Source	Value	Basis/Comments
US EPA (IRIS 2010)	SF = 1.5 (mg/kg/day)$^{-1}$ (for exposures over lifetime) SF = 0.75 (mg/kg/day)$^{-1}$ (for exposures as adult) UR = 8.8x10^{-6} (µg/m^3)$^{-1}$ for exposures over lifetime) UR = 4.4x10^{-6} (µg/m^3)$^{-1}$ for exposures as adult)	SF derived on the basis of hepatocellular carcinomas, angiosarcomas and neoplastic nodules in female rats as markers for carcinogenic response, a PBPK model to estimate human equivalent dose and linearised multistage model. Based on animal evidence of age-dependent sensitivity an additional 2-fold uncertainty has been included to address early-life sensitivity in exposures from birth. Inhalation UR derived on the basis liver angiosarcomas, angiomas, hepatomas and neoplastic nodules in an inhalation study on female rats and mice and an extrapolation model. Based on animal evidence of age-dependent sensitivity an additional 2-fold uncertainty has been included to address early-life sensitivity in exposures from birth. The US EPA review also identified threshold values for the assessment of non-carcinogenic effects with an oral RfD = 0.003 mg/kg/day (same as derived by ATSDR) and an RfC = 0.1 mg/m^3 based on route-extrapolation from the oral value.

Both the WHO and USEPA recognise age sensitivity is important with respect to the assessment of exposure to vinyl chloride and hence it is appropriate to adopt toxicity values that take these issues into consideration. On this basis, the non-threshold reference values available in the current WHO DWG (2008) for oral exposure and presented by the US EPA for inhalation exposure are recommended for use in the derivation of HILs.

Recommendation

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

Recommendation for vinyl chloride (quantitative toxicity values)

Carcinogenic end-points most sensitive and evaluated on the basis of:

Oral TRV = 2.3 (mg/kg/day)$^{-1}$ (WHO DWG [2008]) for oral and dermal exposures from birth (HIL A, B and C)

Oral TRV = 1.15 (mg/kg/day)$^{-1}$ (WHO DWG [2008]) for oral and dermal exposures as adults (HIL D)

Inhalation TRV = 0.0088 (mg/m^3)$^{-1}$ (US EPA [IRIS 2010]) for inhalation exposures from birth (HIL A, B and C)

Inhalation TRV = 0.0044 (mg/m^3)$^{-1}$ (US EPA [IRIS 2010]) for inhalation exposures as adults (HIL D)

Dermal absorption factor = 0.03 assumed for volatile compounds

5.6 Calculated interim HILs

On the basis of the above the following interim soil vapour HILs have been derived for vinyl chloride:

HIL scenario	Interim soil vapour HIL# (mg/m^3)
Residential A	0.3
Residential B	0.3
Recreational C	2
Commercial D	2

Interim soil gas HILs are conservative soil gas concentrations that can be adopted for the purpose of screening sites where further investigation is required on a site-specific basis. They are based on the potential for vapour intrusion indoors using an indoor air to soil gas attenuation factor of 0.01 for scenarios A, B and D and an outdoor attenuation factor of 0.005 for scenario C.

5.7 References for vinyl chloride

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6 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