



# National Environment Protection (Assessment of Site Contamination) Measure 1999

as amended

made under section 14(1) of the

*National Environment Protection Council Act 1994 (Cwlth), the National Environment Protection Council (New South Wales) Act 1995 (NSW), the National Environment Protection Council (Victoria) Act 1995 (Vic), the National Environment Protection Council (Queensland) Act 1994 (Qld), the National Environment Protection Council (Western Australia) Act 1996 (WA), the National Environment Protection Council (South Australia) Act 1995 (SA), the National Environment Protection Council (Tasmania) Act 1995 (Tas), the National Environment Protection Council Act 1994 (ACT) and the National Environment Protection Council (Northern Territory) Act 1994 (NT)*

**Compilation start date:** 16 May 2013

**Includes amendments up to:** *National Environment Protection (Assessment of Site Contamination) Amendment Measure 2013 (No. 1)*

This compilation has been split into 22 volumes

Volume 1: sections 1–6, Schedules A and B  
Volume 2: Schedule B1  
Volume 3: Schedule B2  
Volume 4: Schedule B3  
Volume 5: Schedule B4  
Volume 6: Schedule B5a  
Volume 7: Schedule B5b  
Volume 8: Schedule B5c  
Volume 9: Schedule B6

Prepared by the Office of Parliamentary Counsel, Canberra

---

Volume 10: Schedule B7 - Appendix 1  
Volume 11: Schedule B7 - Appendix 2  
Volume 12: Schedule B7 - Appendix 3  
Volume 13: Schedule B7 - Appendix 4  
Volume 14: Schedule B7 - Appendix 5  
**Volume 15: Schedule B7 - Appendix 6**  
Volume 16: Schedule B7 - Appendix B  
Volume 17: Schedule B7 - Appendix C  
Volume 18: Schedule B7 - Appendix D  
Volume 19: Schedule B7  
Volume 20: Schedule B8  
Volume 21: Schedule B9  
Volume 22: Endnotes

Each volume has its own contents

---

## About this compilation

### The compiled instrument

This is a compilation of the *National Environment Protection (Assessment of Site Contamination) Measure 1999* as amended and in force on 16 May 2013. It includes any amendment affecting the compiled instrument to that date.

This compilation was prepared on 22 May 2013.

The notes at the end of this compilation (the *endnotes*) include information about amending Acts and instruments and the amendment history of each amended provision.

### Uncommenced provisions and amendments

If a provision of the compiled instrument is affected by an uncommenced amendment, the text of the uncommenced amendment is set out in the endnotes.

### Application, saving and transitional provisions for amendments

If the operation of an amendment is affected by an application, saving or transitional provision, the provision is identified in the endnotes.

### Modifications

If a provision of the compiled instrument is affected by a textual modification that is in force, the text of the modifying provision is set out in the endnotes.

### Provisions ceasing to have effect

If a provision of the compiled instrument has expired or otherwise ceased to have effect in accordance with a provision of the instrument, details of the provision are set out in the endnotes.

National Environment Protection (Assessment of Site Contamination) Measure  
1999 National Environment Protection (Assessment of Site Contamination) Measure  
1999 National Environment Protection (Assessment of Site Contamination) Measure 1999  
National Environment Protection (Assessment of Site Contamination) Measure  
1999 National Environment Protection (Assessment of Site Contamination) Measure  
1999 National Environment Protection (Assessment of Site Contamination) Measure 1999  
National Environment Protection (Assessment of Site Contamination) Measure  
1999 National Environment Protection (Assessment of Site Contamination) Measure  
1999 National Environment Protection (Assessment of Site Contamination) Measure 1999  
National Environment Protection (Assessment of Site Contamination) Measure 1999  
National Environment Protection (Assessment of Site Contamination) Measure 1999  
National Environment Protection (Assessment of Site Contamination) Measure 1999

# Schedule B7



## APPENDIX A6

### The Derivation of Interim HILs for Volatile Organic Chlorinated Compounds

# Contents

## The derivation of interim HILs for volatile organic chlorinated compounds

		Page
<b>1</b>	<b>Trichloroethene (TCE)</b>	<b>1</b>
1.1	General	1
1.2	Previous HIL	1
1.3	Proposed Interim HIL	1
1.4	Significance of Exposure Pathways	2
1.4.1	Inhalation	2
1.4.2	Dermal absorption	2
1.4.3	Plant Uptake	2
1.4.4	Intakes from Other Sources – Background	2
1.5	Identification of Toxicity Reference Values	3
1.5.1	Classification	3
1.5.2	Review of Available Values/Information	3
1.5.3	Recommendation	7
1.6	Calculated Interim HILs	7
1.7	References	8
<b>2</b>	<b>1,1,1-Trichloroethane</b>	<b>10</b>
2.1	General	10
2.2	Previous HIL	10
2.3	Proposed Interim HIL	10
2.4	Significance of Exposure Pathways	10
2.4.1	Inhalation	10
2.4.2	Dermal absorption	10
2.4.3	Plant Uptake	11
2.4.4	Intakes from Other Sources – Background	11
2.5	Identification of Toxicity Reference Values	11
2.5.1	Classification	11
2.5.2	Review of Available Values/Information	11
2.5.3	Recommendation	12
2.6	Calculated Interim HILs	12
2.7	References	13
<b>3</b>	<b>Tetrachloroethene (PCE)</b>	<b>15</b>

<b>3.1</b>	<b>General</b>	<b>15</b>
<b>3.2</b>	<b>Previous HIL</b>	<b>15</b>
<b>3.3</b>	<b>Proposed Interim HIL</b>	<b>15</b>
<b>3.4</b>	<b>Significance of Exposure Pathways</b>	<b>15</b>
<b>3.4.1</b>	<b>Inhalation</b>	<b>15</b>
<b>3.4.2</b>	<b>Dermal absorption</b>	<b>15</b>
<b>3.4.3</b>	<b>Plant Uptake</b>	<b>16</b>
<b>3.4.4</b>	<b>Intakes from Other Sources – Background</b>	<b>16</b>
<b>3.5</b>	<b>Identification of Toxicity Reference Values</b>	<b>16</b>
<b>3.5.1</b>	<b>Classification</b>	<b>16</b>
<b>3.5.2</b>	<b>Review of Available Values/Information</b>	<b>17</b>
<b>3.5.3</b>	<b>Recommendation</b>	<b>20</b>
<b>3.6</b>	<b>Calculated Interim HILs</b>	<b>20</b>
<b>3.7</b>	<b>References</b>	<b>20</b>
<b>4</b>	<b>Cis-1,2-Dichloroethene (DCE)</b>	<b>22</b>
<b>4.1</b>	<b>General</b>	<b>22</b>
<b>4.2</b>	<b>Previous HIL</b>	<b>22</b>
<b>4.3</b>	<b>Proposed Interim HIL</b>	<b>22</b>
<b>4.4</b>	<b>Significance of Exposure Pathways</b>	<b>22</b>
<b>4.4.1</b>	<b>Inhalation</b>	<b>22</b>
<b>4.4.2</b>	<b>Dermal absorption</b>	<b>22</b>
<b>4.4.3</b>	<b>Plant Uptake</b>	<b>23</b>
<b>4.4.4</b>	<b>Intakes from Other Sources – Background</b>	<b>23</b>
<b>4.5</b>	<b>Identification of Toxicity Reference Values</b>	<b>23</b>
<b>4.5.1</b>	<b>Classification</b>	<b>23</b>
<b>4.5.2</b>	<b>Review of Available Values/Information</b>	<b>23</b>
<b>4.5.3</b>	<b>Recommendation</b>	<b>24</b>
<b>4.6</b>	<b>Calculated Interim HILs</b>	<b>24</b>
<b>4.7</b>	<b>References</b>	<b>25</b>
<b>5</b>	<b>Vinyl Chloride</b>	<b>26</b>
<b>5.1</b>	<b>General</b>	<b>26</b>
<b>5.2</b>	<b>Previous HIL</b>	<b>26</b>
<b>5.3</b>	<b>Proposed Interim HIL</b>	<b>26</b>
<b>5.4</b>	<b>Significance of Exposure Pathways</b>	<b>26</b>
<b>5.4.1</b>	<b>Inhalation</b>	<b>26</b>
<b>5.4.2</b>	<b>Dermal absorption</b>	<b>27</b>
<b>5.4.3</b>	<b>Plant Uptake</b>	<b>27</b>
<b>5.4.4</b>	<b>Intakes from Other Sources – Background</b>	<b>27</b>
<b>5.5</b>	<b>Identification of Toxicity Reference Values</b>	<b>27</b>

<b>5.5.1</b>	<b>Classification</b>	<b>27</b>
<b>5.5.2</b>	<b>Review of Available Values/Information</b>	<b>28</b>
<b>5.5.3</b>	<b>Recommendation</b>	<b>30</b>
<b>5.6</b>	<b>Calculated Interim HILs</b>	<b>30</b>
<b>5.7</b>	<b>References</b>	<b>31</b>
<b>6</b>	<b>Shortened forms</b>	<b>33</b>

# 1 Trichloroethene (TCE)

## 1.1 General

Several comprehensive reviews of trichloroethene (TCE) in the environment and its 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 2011). 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 recent discovery in 1995 that several species of marine macro-algae and at least one species of micro-algae produce the compound. The importance of this release and potential exposure route is not currently known. TCE 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 the 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, depending on conditions.
- Water – Depending on conditions, reductive dehalogenation to vinyl chloride may occur. Under anaerobic conditions TCE can be intrinsically biodegraded to form dichloroethene (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

### 1.4.1 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 the main text of 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.

### 1.4.2 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).

### 1.4.3 Plant Uptake

Limited data is 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, though 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). Schnabel 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 the text of 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.

### 1.4.4 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 in NSW (DEC 2003) from urban and regional areas in NSW report average concentrations of TCE of approximately 0.1 ppbv (0.0005 mg/m<sup>3</sup>), close to the analytical limit of reporting with most samples noted to be not detected, with a maximum concentration in the Sydney CBD of 3.6 ppbv (0.019 mg/m<sup>3</sup>). Concentrations in an industrial area in Brisbane (Hawas et. al. 2001) have been reported with average and maximum concentrations of 0.0002 mg/m<sup>3</sup> (also close to the limit of reporting) and 0.0005 mg/m<sup>3</sup> respectively. Background air concentrations in Canada (CCME 2007) are considered to be approximately 0.0014 mg/m<sup>3</sup>, consistent with the range reported by DEC (2003). Background intakes (dominated by inhalation) were estimated by WHO (2011) 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 comprise approximately 10% of the recommended inhalation TRVs for non-carcinogenic effects. It is noted that other sources found indoors (from a wide range of common products) are likely to be

present and may contribute more significantly to background exposures. These sources may need to be addressed on a site-specific basis.

## 1.5 Identification of Toxicity Reference Values

### 1.5.1 Classification

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

Review by US EPA (2011) characterised TCE as carcinogenic in humans by all routes of exposure. This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer. The human evidence of carcinogenicity from epidemiologic studies of TCE exposure is strong for non-Hodgkin Lymphoma but less convincing than for kidney cancer, and more limited for liver and biliary tract cancer. Less human evidence is found for an association between TCE exposure and other types of cancer, including bladder, oesophageal, prostate, cervical, breast, and childhood leukaemia. Further support is derived from positive results in multiple rodent bioassays, similar toxicokinetics between rodents and humans, mechanistic data supporting a mutagenic mode of action for kidney tumours.

### 1.5.2 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 testis. On the basis of the available information, most current reviews by IARC (1995), WHO (2011), CCME (2007) and US EPA (2011) 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 (2011) and US EPA (2011) review.

The WHO DWG (2011) 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 (e.g., chloral and DCVC<sup>1</sup>)); 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 (2011) and WHO Air Quality Guidelines (2000)).

The most recent US EPA review (2011) 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 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. However, the metabolites that have been tested, particularly DCVC, have predominantly resulted in positive data, supporting the conclusion that these compounds are genotoxic.

---

<sup>1</sup> DCVC is the abbreviation for the metabolite S-(1,2-dichlorovinyl)-L-cysteine.

The MoA relevant to specific target organs in laboratory animals has been reviewed by US EPA. Only in the case of the kidney is it concluded that the data is 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 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 2011) 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 in the following.

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

Source	Value	Basis/Comments
<b>Australian</b>		
ADWG (NHMRC 2011)	No health-based value derived	Not derived due to insufficient data.
<b>International</b>		
WHO (2011)	SF = 0.00078 (mg/kg/day) <sup>-1</sup> TDI = 0.00146 mg/kg/day	The WHO guideline of 0.02 mg/L is 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 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 multistage model. The oral TDI derived from a BMDL <sub>10</sub> of 0.146 mg/kg/day associated with reproductive/developmental effects in rats, and an uncertainty factor of 100.
WHO (2000 and 2010)	UR = 4.3x10 <sup>-7</sup> (µg/m <sup>3</sup> ) <sup>-1</sup>	Inhalation unit risk derived on the basis of Leydig-cell tumours in the testes of rats and a linear multistage model. Inhalation unit risk from rat study is supported by a similar unit risk of 9 x10 <sup>-7</sup> (µg/m <sup>3</sup> ) <sup>-1</sup> derived from increased incidence of hepatic tumours in a cohort study of occupationally exposed adults. The non-threshold approach was adopted by the WHO as TCE was considered genotoxic and carcinogenic.
EU (2004)	SF = 0.0019 (mg/kg/day) <sup>-1</sup>	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 NOAEL of 38

Source	Value	Basis/Comments
		<p>mg/kg/day was considered.</p> <p>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 <math>0.0019 \text{ (mg/kg/day)}^{-1}</math>. This value was used in the quantification of risk associated with exposure from oral, dermal and inhalation pathways for workers, consumers and environmental exposures.</p>
Health Canada (2005)	<p>SF = <math>0.000811 \text{ (mg/kg/day)}^{-1}</math></p> <p>UR = <math>1.2 \times 10^{-7} \text{ (}\mu\text{g/m}^3\text{)}^{-1}</math></p> <p>TDI = 0.00146 mg/kg/day</p>	<p>Oral slope factor derived on the basis of the same study noted in WHO (2011), however a slightly different value is quoted.</p> <p>Inhalation unit risk based on renal adenocarcinomas in rats following inhalation exposures for 104 weeks in males (a lower, less conservative value was derived for females).</p> <p>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.</p>
CCME (2007)	<p>SF = <math>0.000811 \text{ (mg/kg/day)}^{-1}</math></p> <p>UR = <math>6.4 \times 10^{-7} \text{ (}\mu\text{g/m}^3\text{)}^{-1}</math></p> <p>TDI = 0.00146 mg/kg/day</p> <p>TC = 0.04 mg/m<sup>3</sup></p>	<p>Slope factor based on same study noted by Health Canada (2005).</p> <p>Inhalation unit risk based on older evaluation from Health Canada where a TC<sub>05</sub> (concentration expected to cause a 5% incidence in cancer) of 0.082 mg/m<sup>3</sup> and extrapolation based on an excess lifetime cancer risk of <math>10^{-6}</math>.</p> <p>TDI and TC values also presented for non-carcinogenic end points.</p> <p>TDI as noted by WHO DWG</p> <p>TC adopted from the former US EPA 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<sup>3</sup> was identified, and an uncertainty factor of 1000 adopted.</p>
RIVM (2001)	<p>PTDI = 0.05 mg/kg/day</p> <p>PTC = 0.2 mg/m<sup>3</sup></p>	<p>Provision threshold values derived for TCE due to limited data and an assumption that the genotoxic mechanism for TCE (numerical chromosome aberration <i>in vivo</i>) exhibits a threshold. The basis for these values is not listed here as the evaluation is considered dated.</p>
ATSDR (1997)	No chronic MRLs derived	No chronic oral or inhalation MRL has been established.
New York State (NYS DH 2006)	GV = 0.005 mg/m <sup>3</sup>	<p>An air guideline value (GV) of 0.01 mg/m<sup>3</sup> 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 (<math>1 \times 10^{-6}</math> to <math>1 \times 10^{-4}</math>). The guideline value was then reduced by a factor of 2 based on the consideration of additional factors (data gaps, concern regarding methods for</p>

Source	Value	Basis/Comments
		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 <sup>3</sup> .
US EPA (2011)	SF = 0.05 (mg/kg/day) <sup>-1</sup> UR = 4x10 <sup>-6</sup> (µg/m <sup>3</sup> ) <sup>-1</sup> RfD = 0.0005 mg/kg/day RfC = 0.002 mg/m <sup>3</sup>	Oral slope factor based on PBPK model-based route-to-route extrapolation from the inhalation value based on human kidney cancer risks. The value is also supported by data from oral bioassays. Inhalation unit risk derived on the basis of non-Hodgkin's lymphoma, renal cell carcinoma and liver tumours from a human inhalation (epidemiology) studies, adjusted (by a factor of 4) to address potential risk of tumours at multiple sites. The value is derived from linear extrapolation from the point of departure (LEC01). It is noted that even with the consideration of the 4-fold factor, the inhalation unit risk value derived is within the range of values derived from a wide range of studies. Application of the ADAF for kidney cancer risks due to evidence supporting a mutagenic MoA is recommended. RfD based on critical effects of heart malformations (rats), adult immunological effects (mice) and developmental immunotoxicity (mice), which is further supported by an oral study for the toxic nephropathy (rats) and route extrapolation from an inhalation study. RfC based on route-extrapolation from and oral studies for the critical effects of heart malformations in rats and immunotoxicity in mice, and incorporation of uncertainty factors ranging from 10 to 100.

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.

Many of the reviews conducted by WHO (2011), CCME (2007), RIVM (2001) and ATSDR (1997) have considered limited and dated databases of information (as noted). The most recent comprehensive review of TCE toxicity has been conducted by US EPA (2011), where the most recent studies and health end points have been addressed. The more recent review by WHO (2010), in relation to inhalation toxicity, considered some of the more recent studies, though the review has not considered non-carcinogenic end points, and the key studies considered by US EPA (2011) for the derivation of the inhalation unit risk were not considered in the WHO (2010) review. On this basis it is considered appropriate that the more recent evaluation conducted by US EPA (2011) be used for the purpose of establishing soil vapour Interim HILs.

The US EPA review has concluded that there is sufficient weight of evidence that TCE operates through a mutagenic mode of action (MoA) for kidney tumours and there is a lack of TCE-specific quantitative data in relation to early lifetime susceptibility. Hence it is appropriate to consider increased susceptibility associated with early lifetime exposures through the adjustment of exposure factors. This adjustment, however is noted to be relevant to the kidney cancer component of the total risk (note the inhalation unit risk includes a factor of 4-fold to address the risk of tumours at multiple sites). The effect of considering these age-adjusted exposure factors to only the kidney cancer portion of the unit risk has been evaluated by US EPA and determined to be of minimal impacts to the total cancer risk, except when exposure only occurs during early life (if these effects occur). In addition to this evaluation, a number of uncertainties have been identified in relation to applying the age

adjustment factors for a more complex carcinogenic MoA, as identified for TCE. Hence, for the purpose of deriving HILs where long-term exposures are considered, no further adjustments to account for potential early lifetime susceptibility have been incorporated.

### 1.5.3 Recommendation

In relation to TCE, only soil vapour Interim HILs have been derived. Hence only the inhalation pathway has been quantified in the development of these HILs. On the basis of the discussion above, the following inhalation toxicity reference values (TRVs) have been adopted for TCE:

<b><u>Recommendation for TCE (quantitative inhalation toxicity values)</u></b>
<p><u>Carcinogenic end points evaluated on the basis of:</u>            Inhalation TRV (TRV<sub>I</sub>) = 0.004 (mg/m<sup>3</sup>)<sup>-1</sup> (US EPA 2011)</p>
<p><u>Non-Carcinogenic end points evaluated on the basis of:</u>            Inhalation TRV (TRV<sub>I</sub>) = 0.002 mg/m<sup>3</sup> (US EPA 2011)</p>
<p>Background intakes from other sources (as % of TRV):            BI<sub>I</sub> = 10% for inhalation</p>

### 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 the text of Schedule B7):

- The calculated interim soil vapour HIL for TCE on the basis of the adopted threshold TRVs noted above is 0.02 mg/m<sup>3</sup>.
- The calculated interim soil vapour HIL for TCE on the basis of the adopted non-threshold TRVs noted above is 0.06 mg/m<sup>3</sup>.

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 (refer to Appendix B for equations used to calculate the HILs and Appendix C for calculations):

<b>HIL Scenario</b>	<b>Interim Soil Vapour HIL# (mg/m<sup>3</sup>)</b>
Residential A	0.02
Residential B	0.02
Recreational C	0.4
Commercial D	0.08

# 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 0.1 for HILs A, B and D and an outdoor attenuation factor of 0.05 for HIL C.

## 1.7 References

- ATSDR 1997, *Toxicological Profile for Trichloroethylene*, available on website and current.
- CCME 2007, *Canadian Soil Quality Guidelines, Trichloroethylene, Environmental and Human health Effects, Scientific Supporting Document*, Canadian Council of Ministers of the Environment.
- DEC 2003, *Ambient Air Quality Research Project (1996-2001), Internal working paper no. 4, Ambient concentrations of heavy metals in NSW*, Department of Environment and Conservation (NSW).
- EU 2004, *European Union Risk Assessment Report, CAS No: 79-01-6, EINECS No: 201-167-4, Trichloroethylene*, Institute for Health and Consumer Protection, European Chemicals Bureau, Existing Chemicals, vol. 31.
- Hawas, O, Hawker, D, Chan, A, Cohen, D, Christensen, E, Golding, G & Vowles, P 2001, 'Characterisation and Identification of Sources of Volatile Organic Compounds in an Industrial Area in Brisbane', *16th Int. Clean Air Conf.*, Christchurch, New Zealand, 18–22 August 2002, pp. 307–313.
- Health Canada 2005, *Trichloroethylene, Guidelines for Canadian Drinking Water Quality, Supporting Documentation*.
- 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, International Agency for Research on Cancer, Lyon, France*.
- NEPC 1999, *Schedule B (7a), Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council, Australia.
- NICNAS 2000, *Trichloroethylene – Priority Existing Chemical Assessment Report No. 8*, National Industrial Chemicals Notification and Assessment Scheme.
- NHMRC 2011, *National water quality management strategy, Australian drinking water guidelines*, National Health and Medical Research Council, Australia.
- NYS DH 2006, *Trichloroethene Air Criteria Document, Final Report*, New York State Department of Health.
- RAIS 2010, *Risk Assessment Information System*, website and database maintained by the Oak Ridge Operations Office, available from: <http://rais.ornl.gov/>.
- RIVM 2001, *Re-evaluation of human-toxicological Maximum Permissible Risk levels*, National Institute of Public Health and the Environment, Bilthoven, Netherlands, available from: <http://www.rivm.nl/bibliotheek/rapporten/711701025.html>.
- Schnabel, WE, Dietz, AC, Burken, JG, Schnoor, JL & Alvarez, PJ 1997, 'Uptake and transformation of trichloroethylene by edible garden plants', *Water Research*, vol. 31, Issue 4, pp. 816–824.
- Schroll, R, Bierling, B, Cao, G, Dörfler, U, Lahaniati, M, Langenbach, T, Scheunert, I & Winkler, R 1994, 'Uptake pathways of organic chemicals from soil by agricultural plants', *Chemosphere*, vol. 28, pp. 297–303.
- US EPA (IRIS 2012), data and information available from the *Integrated Risk Information System*, an online database, available from <http://www.epa.gov/iris/>.

- US EPA 1995, *Technical Guidance Manual, Assessing Dermal Exposure from Soil*, US EPA Region 3, December 1995, available from: <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.
- US EPA 2006, *Derivation of RBCs for Carcinogens that Act Via a mutagenic Mode of Action and Incorporate Default ADAFs*, Memo, October 2006, corrected November 2006.
- US EPA 2011, *Toxicological Review of Trichloroethylene*, in support of Summary Information on the Integrated Risk Information System (IRIS), September 2011, EPA/635/R-09/011F.
- WHO 1985, *Environmental Health Criteria No 50 Trichloroethylene*, World Health Organization, Geneva.
- WHO 2000, *Air Quality Guidelines for Europe, 2<sup>nd</sup> edn*, World Health Organization, Geneva.
- WHO 2010, *WHO Guidelines for Indoor Air Quality, Selected Pollutants*, WHO Regional Office for Europe.
- WHO 2011, *Guidelines for drinking-water quality, 4<sup>th</sup> edn*, World Health Organization, Geneva, available from [http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/en/index.html](http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html).



## 2 1,1,1-Trichloroethane

### 2.1 General

Several comprehensive reviews of 1,1,1-trichloroethane (1,1,1-TCA) in the environment and its toxicity to humans are available and should be consulted for more detailed information not presented in this summary (ATSDR 1997; ATSDR 2006; WHO 1990). The following provides a summary of the key aspects of 1,1,1-TCA that is 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 on 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 2011).

### 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

#### 2.4.1 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.

#### 2.4.2 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 though, 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).

### **2.4.3 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 the text of 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 an 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.

### **2.4.4 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 ( $0.5 \mu\text{g}/\text{m}^3$ ) and the maximum reported in Beresfield was 0.3 ppbv ( $1.6 \mu\text{g}/\text{m}^3$ ). 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.

It is noted that other sources found indoors (from a wide range of common products) are likely to be present and may contribute more significantly to background exposures. These sources need to be addressed on a site-specific basis.

## **2.5 Identification of Toxicity Reference Values**

### **2.5.1 Classification**

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

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

### **2.5.2 Review of Available Values/Information**

There is insufficient data available to determine carcinogenicity of 1,1,1-TCA (WHO 2011, ATSDR 2006 and US EPA 2007). Review by 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 but the following are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
<b>Australian</b>		
ADWG (NHMRC 2011)	No guideline established	No guideline established in current ADWG (NHMRC 2011) due to insufficient data.
<b>International</b>		
WHO (2011)	TDI = 0.6 mg/kg/day	No guideline is established as 1,1,1-TCA concentrations in drinking water are well below those of health concern. The review notes that a health-based guideline of 2 mg/L can be derived 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 <sup>3</sup>	Maximum permissible concentration (MPC) in air derived on the basis of a duration corrected NOAEL of 482 mg/m <sup>3</sup> 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 2012)	RfD = 2 mg/kg/day RfC = 5 mg/m <sup>3</sup>	Oral reference dose (RfD, last reviewed in 2007) 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 (last reviewed in 2007) derived on the basis of a NOAEL (HEC) of 1553 mg/m <sup>3</sup> associated with liver effects in mice and rats, and an uncertainty factor of 100.

In relation to inhalation exposures (the only pathway considered in development of soil vapour Interim HILs) the most recent review conducted by US EPA (which is consistent with the older review from RIVM) has been adopted.

### 2.5.3 Recommendation

In relation to 1,1,1-TCA, only soil vapour Interim HILs have been derived. Hence only the inhalation pathway has been quantified in the development of these HILs. On the basis of the discussion above, the following inhalation toxicity reference values (TRVs) have been adopted for 1,1,1-TCA:

<p><b><u>Recommendation for 1,1,1-TCA</u></b>            Inhalation TRV (TRV<sub>I</sub>) = 5 mg/m<sup>3</sup> (US EPA)            Background intakes from other sources (as % of TRV):                Bli = 0% for inhalation</p>
--

### 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 (refer to Appendix B for equations used to calculate the HILs and Appendix C for calculations):

HIL Scenario	Interim Soil Vapour HIL# (mg/m <sup>3</sup> )
Residential A	60
Residential B	60
Recreational C	1200
Commercial D	230

# 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 0.1 for HILs A, B and D and an outdoor attenuation factor of 0.05 for HIL C.

## 2.7 References

- 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*, July 2006, available from <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=432&tid=76>.
- DEC 2003, *Ambient Air Quality Research Project (1996-2001), Internal working paper no. 4, Ambient concentrations of heavy metals in NSW*, Department of Environment and Conservation (NSW), 2003.
- Hawas, O, Hawker, D, Chan, A, Cohen, D, Christensen, E, Golding, G & Vowles, P 2001, 'Characterisation and Identification of Sources of Volatile Organic Compounds in an Industrial Area in Brisbane', *16th Int. Clean Air Conf.*, Christchurch, New Zealand, 18–22 August 2002, pp. 307–313.
- IARC 1999, *Summaries & Evaluations, 1,1,1-Trichloroethane*, vol. 71, (1999), pp. 881, International Agency for Research on Cancer.
- NEPC 1999, *Schedule B (7a), Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council, Australia.
- NHMRC 2011, *National water quality management strategy, Australian drinking water guidelines*, National Health and Medical Research Council, Australia.
- RIVM 1993, *Derivation of Maximum Permissible Concentrations for Volatile Compounds in Air*, National Institute of Public Health and the Environment, Bilthoven, Netherlands, Report no. 679101 010, October 1993.
- US EPA (IRIS2012), data and information available from the *Integrated Risk Information System*, an online database, available from <http://www.epa.gov/iris/>.
- US EPA 1995, *Technical Guidance Manual, Assessing Dermal Exposure from Soil*, US EPA Region 3, December 1995, available from: <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), August 2007.
- WHO 1990, *1,1,1-Trichloroethane, Environmental Health Criteria 136*, available from <http://www.inchem.org/documents/ehc/ehc/ehc136.htm>.

WHO 2011, *Guidelines for drinking-water quality, 4<sup>th</sup> edn*, World Health Organization, Geneva,  
available from  
[http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/en/index.html](http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html).

## **3 Tetrachloroethene (PCE)**

### **3.1 General**

Several comprehensive reviews of tetrachloroethene in the environment and its 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 2012). The following provides a summary of the key aspects of PCE that is 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**

#### **3.4.1 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.

#### **3.4.2 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 though, 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).

### 3.4.3 Plant Uptake

Limited data is 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  $8.2 \mu\text{g}/\text{m}^3$  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 the text of 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 an 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.

### 3.4.4 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 in NSW (DEC 2003) from urban and regional areas in NSW report average concentrations of PCE of approximately 0.1 ppbv, or  $0.0007 \text{ mg}/\text{m}^3$  (<5% of inhalation TRV) with a maximum concentration in the Sydney CBD of 1.6 ppbv, or  $0.01 \text{ mg}/\text{m}^3$  (5% of inhalation TRV). A study of concentrations in an industrial area in Brisbane (Hawas et. al. 2001) has reported average and maximum concentrations of  $0.015 \text{ mg}/\text{m}^3$  (7.5% of inhalation TRV) and  $0.085 \text{ mg}/\text{m}^3$  (42% of inhalation TRV) respectively. These concentrations are consistent with those reported in other cities in Australia (NICNAS 2001).

Other significant exposures of 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 WHO (2000). A study on the effect of wearing dry-cleaned clothes reported median personal air concentrations ranging from  $0.032 \text{ mg}/\text{m}^3$  to  $0.22 \text{ mg}/\text{m}^3$ , depending on the garment. These exposures, together 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 concentration reported by WHO (2006) for homes not located in the same building as dry-cleaners was  $0.004 \text{ mg}/\text{m}^3$  (note that concentrations indoors were much higher in buildings with a dry-cleaner with indoor air levels ranging from 0.05 to  $6.1 \text{ mg}/\text{m}^3$ ). 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 low, with a conservative average intake of 10% assumed in the derivation of HILs.

It is noted that other sources found indoors (from a wide range of common products) are likely to be present and may contribute more significantly to background exposures. These sources need to be addressed on a site-specific basis.

## 3.5 Identification of Toxicity Reference Values

### 3.5.1 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.

Review of PCE by US EPA (2012) classified it as '*Likely to be Carcinogenic to Humans*' by all routes of exposure, based on suggestive evidence of carcinogenicity in epidemiologic studies and conclusive evidence that the administration of PCE, either by ingestion or by inhalation to sexually mature rats and mice, increases tumour incidence.

### 3.5.2 Review of Available Values/Information

Some epidemiological studies indicate a possible association between chronic exposure to PCE and an increased cancer risk. Review of these studies has indicated that the evidence provided is inconclusive (US EPA 2012). 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, though recent reviews by WHO (2006) and US EPA (2012) 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; WHO 2006, RIVM 2001). Review of the available studies by 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. 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.

Review of PCE by US EPA (2012) suggests that PCE has been shown to induce some genotoxic effects. There are a number of limitations noted in the assessment presented by US EPA, in particular, the fact that the MoA for PCE that induces carcinogenesis is not yet fully characterised or understood and that the role of genotoxicity in hepatocarcinogenicity is uncertain. Where US EPA lacks certainty, the default position is to be conservative and, as such, it has suggested considering PCE having a mutagenic MoA, where a non-threshold approach is recommended for the assessment of carcinogenicity and mutagenicity. This is not consistent with the approach adopted in this assessment (consistent with NHMRC 1999 guidance). 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
<b>Australian</b>		
ADWG (NHMRC 2011)	TDI = 0.014 mg/kg/day	The current ADWG (NHMRC 2011) have derived a guideline of 0.05 mg/L for PCE based on a 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.
<b>International</b>		
WHO (2011)	TDI = 0.014 mg/kg/day	WHO DWG TDI based on the same study and uncertainty factor as noted in the ADWG (NHMRC 2011).
WHO (2006 and 2010)	TC = 0.2 mg/m <sup>3</sup> TC = 0.25 mg/m <sup>3</sup> TDI = 0.05 mg/kg/day	TC in air derived on the basis of the most sensitive end point, namely neurotoxicological effects, based on a mean LOAEC (adjusted) of 20 mg/m <sup>3</sup> from an occupational inhalation study (mean exposure of 10 years) (Seeber 1989), and an uncertainty factor of 100. The TC derived is lower than 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 approach with a BMCL <sub>10</sub> of 20 mg/m <sup>3</sup> and if a multistage model were considered the TC of 0.2 mg/m <sup>3</sup> would be associated with a risk of 1 x10 <sup>-3</sup> . 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 <sup>3</sup> was derived based on a LOAEL of 102 mg/m <sup>3</sup> in dry-cleaning workers, with adjustment for exposure duration (to LOAEL of 24.3 mg/m <sup>3</sup> ) (Mutti et al. 1993), and an uncertainty factor of 100. Further review of PCE by WHO (2010) re-confirmed the guideline of 0.25 mg/m <sup>3</sup> .
RIVM (2001)	TDI = 0.016 mg/kg/day TC = 0.25 mg/m <sup>3</sup>	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 LOAEL (adjusted) of 23 mg/m <sup>3</sup> 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 <sup>3</sup>	TDI derived on the same basis as noted for the WHO DWG and ADWG. TC derived from a LOAEL of 363 (adjusted) mg/m <sup>3</sup> associated with multiple effects in mice, and an uncertainty factor of 1000.
ATSDR (1997)	No chronic oral MRL Inhalation MRL = 0.24 mg/m <sup>3</sup>	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 <sup>3</sup> associated with neurobehavioural effects in an occupational

Source	Value	Basis/Comments
		inhalation study, and an uncertainty factor of 100.
US EPA (2012)	RfD = 0.006 mg/kg/day RfC = 0.04 mg/m <sup>3</sup>	RfD derived based on route extrapolation from the inhalation studies. RfC derived on the basis of the midpoint of RfCs derived from 2 studies. An RfC of 0.015 mg/m <sup>3</sup> was derived from a LOAEL of 15 mg/m <sup>3</sup> associated with colour confusion in an adult occupational study (Cavalleri et al. 1994), and application of a 100-fold uncertainty factor. An RfC of 0.056 mg/m <sup>3</sup> was derived from a LOAEL of 56 mg/m <sup>3</sup> associated with cognitive and reaction time effects in an adult occupational study (Echeverria et al. 1995), and application of a 100-fold uncertainty factor. The derived value is consistent with that derived for liver effects from the study by Mutti et al. (1993), and 1000-fold uncertainty factor. The 100-fold uncertainty factor applied to these key studies includes a 10-fold factor to address database deficiencies in relation to characterising the hazard and dose response in the human population. The US EPA review also identified non-threshold values not considered relevant in this evaluation.

In relation to the identification of an appropriate inhalation TRV for use in the derivation of a soil vapour interim HIL, the following is noted from the above studies:

- The point of departure (LOAELs in this case) from key studies by WHO (2006; 2010) and US EPA (2012) are similar, ranging from 0.02 to 0.056 mg/m<sup>3</sup>;
- The key studies identified in the US EPA (2012) review were also considered in the WHO (2006 and 2010) reviews, with the WHO (2006) review determining that the study conducted by Cavalleri et al. (1994) (used by US EPA as the lower end of the range of two principal RfCs derived) provided results that were difficult to interpret and hence not suitable for the determination of a threshold criterion. The other principal study considered by US EPA was not used as a key study by WHO. Similarly, the key study adopted by WHO (2006), while initially identified by US EPA as an appropriate key study, was not considered due to concerns regarding discrepant results;
- The key difference between the WHO and US EPA reviews and derived inhalation TRVs is the application of uncertainty factors. The WHO reviews have consistently applied an uncertainty factor of 100 to address intra-species variability and the use of a LOAEL. US EPA (2012) has applied an additional factor of 10-fold to address database deficiencies in relation to characterising the hazard and dose–response in the human population (i.e. residents rather than workers). The WHO (2006) review considered the use of occupational studies to be conservative for the general population, as worker exposures are likely to include short duration peaks of higher concentrations. This approach (by WHO) is consistent with that adopted in the assessment of exposures by the general public, based on occupational studies, in Australia.
- Based on the above, both the WHO and US EPA reviews have considered the same key studies and database of information. However, the interpretation of uncertainty in relation to the use of occupational studies for establishing criteria for the general public differs. Where the range of potential RfCs (from suitable available studies) was considered by US EPA (including consideration of uncertainty factors), the inhalation

value of 0.2 mg/m<sup>3</sup> derived by WHO (2006) lies at the lower end of the range of criteria derived. Hence adopting the WHO (2006) inhalation TRV of 0.2 mg/m<sup>3</sup> is considered appropriate for the derivation of soil vapour Interim HILs.

### 3.5.3 Recommendation

In relation to PCE, only soil vapour Interim HILs have been derived. Hence, only the inhalation pathway has been quantified in the development of these HILs. On the basis of the discussion above, the following inhalation toxicity reference values (TRVs) have been adopted for PCE:

<p><b><u>Recommendation for PCE</u></b>            Inhalation TRV (TRV<sub>I</sub>) = 0.2 mg/m<sup>3</sup> (WHO 2006)            Background intakes from other sources (as % of TRV):                Bli = 10% for inhalation</p>
---

### 3.6 Calculated Interim HILs

On the basis of the above, the following interim soil vapour HILs have been derived for PCE (refer to Appendix B for equations used to calculate the HILs and Appendix C for calculations):

HIL Scenario	Interim Soil Vapour HIL# (mg/m <sup>3</sup> )
Residential A	2
Residential B	2
Recreational C	40
Commercial D	8

# 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

- ATSDR 1997, *Toxicological Profile for Tetrachloroethylene*, available from: <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=265&tid=48>.
- Cavalleri, A, Gobba, F, Paltrinieri, M, Fantuzzi, G, Righi, E & Aggazzotti, G 1994, 'Perchloroethylene exposure can induce colour vision loss', *Neurosci. Lett.* vol. 179, pp. 162–166, [http://dx.doi.org/10.1016/0304-3940\(94\)90959-8](http://dx.doi.org/10.1016/0304-3940(94)90959-8).
- DEC 2003, *Ambient Air Quality Research Project (1996-2001), Internal working paper no. 4, Ambient concentrations of heavy metals in NSW*, Department of Environment and Conservation (NSW).
- Echeverria, D, White, RF & Sampaio, C 1995, 'A behavioral evaluation of PCE exposure in patients and dry cleaners: A possible relationship between clinical and preclinical effects', *J. Occup. Environ. Med.* Vol. 37, pp. 667–680.
- Health Canada 1993, *Tetrachloroethylene, Priority Substances List, Assessment Report*.
- Hawas, O, Hawker, D, Chan, A, Cohen, D, Christensen, E, Golding, G & Vowles, P 2001, 'Characterisation and Identification of Sources of Volatile Organic Compounds in an

- Industrial Area in Brisbane', *16th Int. Clean Air Conf.*, Christchurch, New Zealand, 18–22 August 2002, pp. 307–313.
- IARC 1995, *Dry cleaning, some chlorinated solvents and other industrial compounds, Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*, vol. 63, International Agency for Research on Cancer, Lyon, France.
- Mutti, A, Alinovi, R, Bergamaschi, E, Biagini, C, Cavazzini, S, Franchini, I, Lauwerys, RR, Bernard, AM, Roels, H, Gelpi, E, Rosello, J, Ramis, I, Price, RG, Taylor, SA, de Broe, M, Nuyts, GD, Stolte, H, Fels, LM & Herbort, C 1992, 'Nephropathies and exposure to perchloroethylene in dry-cleaners', *Lancet*, vol. 330, pp. 189–193, [http://dx.doi.org/10.1016/0140-6736\(92\)90463-D](http://dx.doi.org/10.1016/0140-6736(92)90463-D).
- NEPC 1999, *Schedule B (7a), Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council, Australia.
- NICNAS 2001, *Tetrachloroethylene – Priority Existing Chemical Assessment Report No. 15*, National Industrial Chemicals Notification and Assessment Scheme.
- NHMRC 1999, *Toxicity assessment for carcinogenic soil contaminants*, National Health and Medical Research Council, Australia.
- NHMRC 2011, *National water quality management strategy, Australian drinking water guidelines*, National Health and Medical Research Council, Australia.
- RAIS 2010, *Risk Assessment Information System*, website and database maintained by the Oak Ridge Operations Office, available from: <http://rais.ornl.gov/>.
- RIVM 2001, *Re-evaluation of human-toxicological Maximum Permissible Risk levels*. National Institute of Public Health and the Environment, Bilthoven, Netherlands, available from: <http://www.rivm.nl/bibliotheek/rapporten/711701025.html>.
- Seeber, A 1989, 'Neurobehavioral toxicity of long-term exposure to tetrachloroethylene', *Neurotoxicol. Teratol.*, Vol. 11, pp. 579–583. [http://dx.doi.org/10.1016/0892-0362\(89\)90041-X](http://dx.doi.org/10.1016/0892-0362(89)90041-X).
- US EPA (IRIS 2012), data and information available from the *Integrated Risk Information System*, an online database, available from <http://www.epa.gov/iris/>.
- US EPA 1995, *Technical Guidance Manual, Assessing Dermal Exposure from Soil*, US EPA Region 3, December 1995, available from: <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.
- US EPA 2012, *Toxicological Review of Tetrachloroethylene (Perchloroethylene)*, in support of Summary Information on the Integrated Risk Information System (IRIS), February 2012, EPA/635/R-08/011F.
- WHO 2000, *Air Quality Guidelines for Europe, 2<sup>nd</sup> edn*, WHO European Publication Series No. 91, WHO Regional Office for Europe, Copenhagen.
- WHO 2006, *Concise International Chemicals Assessment Document (CICAD) 68, Tetrachloroethene*, World Health Organization, 2006, available from <http://www.inchem.org/documents/cicads/cicads/cicad68.htm#1.0>.
- WHO 2010. WHO Guidelines for Indoor Air Quality, Selected Pollutants. WHO Regional Office for Europe.
- WHO 2011, *Guidelines for drinking-water quality, 4<sup>th</sup> edn*, World Health Organization, Geneva, available from [http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/en/index.html](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 its toxicity to humans are available and should be consulted for more detailed information (ATSDR 1996; WHO 2011). The following provides a summary of the key aspects of DCE that is relevant to the derivation of interim HILs.

DCE is a colourless, volatile and flammable liquid with a characteristic sharp, harsh odour. It is one of two isomers of 1,2-DCE, the second being *trans*-1,2-DCE. *cis*-1,2-DCE is considered to be more toxic than *trans*-1,2-DCE and hence the HILs derived for the *cis*-isomer are adequately protective of exposures associated with the *trans*-isomer.

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

#### 4.4.1 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.

#### 4.4.2 Dermal absorption

Insufficient data is available on the dermal absorption of DCE from soil. Given the volatility of the compound, dermal absorption is expected to be low though, 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).

### 4.4.3 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 the text of 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 an 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.

### 4.4.4 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 for Australian cities. *Cis*-1,2-DCE has been detected in VOC sampling from Perth (WA DEP 2000), with average concentrations of 0.2 ppb (0.8 µg/m<sup>3</sup>) and a maximum reported concentration of 2.1 ppb (8.3 µg/m<sup>3</sup>). These values were comparable to average concentrations reported in air in the USA 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%).

It is noted that other sources found indoors (from a wide range of common products) are likely to be present and may contribute more significantly to background exposures. These sources need to be addressed on a site-specific basis.

## 4.5 Identification of Toxicity Reference Values

### 4.5.1 Classification

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

US EPA (2010) has classified 1,2-DCE as 'inadequate information to assess the carcinogenic potential'.

### 4.5.2 Review of Available Values/Information

There is no adequate data available to assess the carcinogenicity of DCE. Review of available genotoxicity studies by WHO (2011) 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 US EPA (2010) suggested that, overall, data for 1,2-DCE (both isomers) is not positive for genotoxicity and mutagenicity. The positive results (considered by RIVM) are considered inconsistent by 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
<b>Australian</b>		
ADWG (NHMRC 2011)	TDI = 0.017 mg/kg/day for <i>trans</i> -isomer	The Australian Drinking Water Guidelines (NHMRC 2011) have derived a drinking water guideline of 0.06 mg/L for 1,2-DCE (both isomers) following guidance from WHO (refer below).
<b>International</b>		
WHO	TDI = 0.017	WHO (2011) has derived a guideline of 0.05 mg/L

Source	Value	Basis/Comments
(2011)	mg/kg/day for <i>trans</i> -isomer	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 <i>trans</i> -1,2-DCE in drinking water, and an uncertainty factor of 1000. This guideline is relevant to the sum of both <i>cis</i> - and <i>trans</i> - isomers, however this is due to WHO adopting a conservative approach where there is no data available for the derivation of a <i>cis</i> - isomer value.
RIVM (2001)	TDI = 0.006 mg/kg/day TC = 0.03 mg/m <sup>3</sup>	A TDI of 0.006 mg/kg/day has been established for <i>cis</i> -1,2-DCE based on a NOAEL of 32 mg/kg/day from a 90-day oral rat study (using the <i>cis</i> - isomer), and an uncertainty factor of 5000. Inhalation tolerable concentrations (TC) were derived for <i>cis</i> -1,2-DCE using route extrapolation from the oral study, resulting in a TC of 0.03 mg/m <sup>3</sup>
ATSDR (1996)	No chronic MRLs derived	
US EPA (2010)	RfD = 0.002 mg/kg/day for <i>cis</i> -isomer	RfD derived on the basis of a BMDL <sub>10</sub> of 5.1 mg/kg/day associated with increased kidney weight in male rats and a 3000-fold uncertainty factor (includes 3-fold factor for database deficiencies). No inhalation RfC was derived for the <i>cis</i> -isomer. For the <i>trans</i> -isomer an oral RfD of 0.02 mg/kg/day was derived and no inhalation RfC was derived.

For the assessment of inhalation exposures (relevant to the derivation of soil vapour Interim HILs), there are no specific TRVs derived from inhalation studies associated with *cis*-1,2-DCE. An inhalation value can be derived from route extrapolation from an oral value (as undertaken by RIVM). In relation to the available oral TRVs, the most recent evaluation conducted by US EPA is considered the most appropriate. From this oral TRV, an inhalation TRV of 0.007 mg/m<sup>3</sup> can be derived (for a 70 kg adult where 20 m<sup>3</sup> of air is inhaled each day).

#### 4.5.3 Recommendation

In relation to *cis*-1,2-DCE, only soil vapour Interim HILs have been derived. Hence only the inhalation pathway has been quantified in the development of these HILs. On the basis of the discussion above, the following inhalation toxicity reference values (TRVs) have been adopted for *cis*-1,2-DCE:

##### **Recommendation for *cis*-1,2-DCE**

Inhalation TRV (TRV<sub>I</sub>) = 0.007 mg/m<sup>3</sup> (US EPA 2010)

Background intakes from other sources (as % of TRV):

Bli = 0% for inhalation

#### 4.6 Calculated Interim HILs

On the basis of the above, the following interim soil vapour HILs have been derived for DCE (refer to Appendix B for equations used to calculate the HILs and Appendix C for calculations):

HIL Scenario	Interim Soil Vapour HIL# (mg/m <sup>3</sup> )
Residential A	0.08
Residential B	0.08
Recreational C	2
Commercial D	0.3

# 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 0.1 for HILs A, B and D and an outdoor attenuation factor of 0.05 for HIL C.

#### 4.7 References

- ATSDR 1996, *Toxicological Profile for 1,2-Dichloroethene*, available on website 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, Australia.
- NHMRC 2011, *National water quality management strategy, Australian drinking water guidelines*, National Health and Medical Research Council, Australia.
- RIVM 2001, *Re-evaluation of human-toxicological Maximum Permissible Risk levels*, National Institute of Public Health and the Environment, Bilthoven, Netherlands, available from: <http://www.rivm.nl/bibliotheek/rapporten/711701025.html>.
- US EPA (IRIS 2012), data and information available from the *Integrated Risk Information System*, an online database, available from <http://www.epa.gov/iris/>.
- US EPA 1995, *Technical Guidance Manual, Assessing Dermal Exposure from Soil*, US EPA Region 3, December 1995, available from: <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.
- US EPA 2010, *Toxicological Review of cis-1,2-Dichloroethylene and trans-1,2-Dichloroethylene*, in support of Summary Information on the Integrated Risk Information System (IRIS), September 2010, EPA/635/R-09/006F.
- WA DEP 2000, *Volatile Organic Compounds Monitoring in Perth, Baseline Air Toxics Project*, Western Australian Department of Environmental Protection.
- WHO 2011, *Guidelines for drinking-water quality, 4<sup>th</sup> edn*, World Health Organization, Geneva, available from [http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/en/index.html](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 its 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 is 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 polyvinyl chloride (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, but 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

#### 5.4.1 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 chemicals 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.

#### **5.4.2 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 though, 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).

#### **5.4.3 Plant Uptake**

No data is 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 the text of 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 an 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.

#### **5.4.4 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<sup>3</sup>). 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 2011). 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**

#### **5.5.1 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 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.

## 5.5.2 Review of Available Values/Information

Exposure to vinyl chloride via inhalation has been associated with increases 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 and ATSDR 2006).

Vinyl chloride has been identified as genotoxic and mutagenic (WHO 1999, ATSDR 2006 and 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 WHO (2000; 2011). 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 an 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
<b>Australian</b>		
ADWG (NHMRC 2011)	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 associated with a no-effect level of 0.13 mg/kg/day from lifetime studies in rats, and 1000-fold uncertainty factor.
OCS (2012)	No evaluation available	

<b>International</b>		
WHO DWG (2011)	SF = 1.15 (mg/kg/day) <sup>-1</sup> (for exposures from birth) SF = 0.7 (mg/kg/day) <sup>-1</sup> (for exposures as adults)	WHO (2011, last review in 2004) 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 <sup>-6</sup> (µg/m <sup>3</sup> ) <sup>-1</sup>	Inhalation unit risk 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) <sup>-1</sup>	Slope factor 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 WHO and US EPA and does not include any consideration of early life sensitivity.
RIVM (2001)	SF = 0.17 (mg/kg/day) <sup>-1</sup> UR = 2.8x10 <sup>-5</sup> (µg/m <sup>3</sup> ) <sup>-1</sup>	Slope factor 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 unit risk 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 <sup>3</sup> 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.

US EPA (IRIS 2012)	SF = 1.5 (mg/kg/day) <sup>-1</sup> (for exposures over lifetime) SF = 0.75 (mg/kg/day) <sup>-1</sup> (for exposures as adult) UR = 8.8x10 <sup>-6</sup> (µg/m <sup>3</sup> ) <sup>-1</sup> for exposures over lifetime) UR = 4.4x10 <sup>-6</sup> (µg/m <sup>3</sup> ) <sup>-1</sup> for exposures as adult)	Slope factor (last reviewed in 2000) 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 unit risk 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 <sup>3</sup> based on route-extrapolation from the oral value.
--------------------	---	---

Both WHO and US EPA 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, of the non-threshold reference values available, the inhalation values presented by US EPA are the most relevant and current (and adequately address early lifetime exposures) and suitable for the derivation of soil vapour Interim HILs.

### 5.5.3 Recommendation

In relation to vinyl chloride, only soil vapour Interim HILs have been derived. Hence only the inhalation pathway has been quantified in the development of these HILs. On the basis of the discussion above, the following inhalation toxicity reference values (TRVs) have been adopted for vinyl chloride:

<p><b><u>Recommendation for Vinyl Chloride (quantitative inhalation toxicity values)</u></b>  Carcinogenic end points most sensitive and evaluated on the basis of:  Inhalation TRV = 0.0088 (mg/m<sup>3</sup>)<sup>-1</sup> (US EPA (IRIS 2012)) for inhalation exposures from birth (HIL A, B and C)  Inhalation TRV = 0.0044 (mg/m<sup>3</sup>)<sup>-1</sup> (US EPA (IRIS 2012)) for inhalation exposures as adults (HIL D)</p>
---

### 5.6 Calculated Interim HILs

On the basis of the above, the following interim soil vapour HILs have been derived for vinyl chloride (refer to Appendix B for equations used to calculate the HILs and Appendix C for calculations):

HIL Scenario	Interim Soil Vapour HIL# (mg/m <sup>3</sup> )
Residential A	0.03
Residential B	0.03

Recreational C	0.5
Commercial D	0.1

# 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 0.1 for HILs A, B and D and an outdoor attenuation factor of 0.005 for HIL C.

## 5.7 References

- ATSDR 2006, *Toxicological Profile for Vinyl Chloride*, available from: <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=282&tid=51>.
- DEC 2003, *Ambient Air Quality Research Project (1996-2001), Internal working paper no. 4, Ambient concentrations of heavy metals in NSW*, Department of Environment and conservation (NSW).
- Health Canada 1992, *Vinyl Chloride, Guidelines for Canadian Drinking Water Quality, Supporting Documentation*.
- IARC 2008, *International Agency for Research on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans: 1,3-Butadiene, Ethylene Oxide and Vinyl Halides (Vinyl Fluoride, Vinyl Chloride and Vinyl Bromide)*, World Health Organization, International Agency for Research on Cancer, Lyon, France.
- NEPC 1999, *Schedule B (7a), Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council, Australia.
- NHMRC 2011, *National water quality management strategy, Australian drinking water guidelines*, National Health and Medical Research Council, Australia.
- NJ DEP 2005, *Field Sampling Procedures Manual, Chapter 9 – Soil Gas Surveys*, New Jersey Department of Environmental Protection.
- RIVM 2001, *Re-evaluation of human-toxicological Maximum Permissible Risk levels*, National Institute of Public Health and the Environment, Bilthoven, Netherlands, available from: <http://www.rivm.nl/bibliotheek/rapporten/711701025.html>.
- Scheutz, C 2002, 'Attenuation of Methane and Trace Organics in Landfill Soil Covers', PhD Thesis, September 2002, also updated paper 'Biodegradation of Trace Gases in Simulated Landfill Soil Cover Systems', *Journal of Air & Waste Management Association*, July 2005, vol. 55(7), pp. 878–885.
- US EPA (IRIS 2012), data and information available from the *Integrated Risk Information System*, an online database, available from <http://www.epa.gov/iris/>.
- US EPA 1995, *Technical Guidance Manual, Assessing Dermal Exposure from Soil*, US EPA Region 3, December 1995, available from <http://www.epa.gov/reg3hwmd/risk/human/info/solabsg2.htm>.
- US EPA 2000, *Toxicological Review of Vinyl Chloride*, in support of Summary Information on the Integrated Risk Information System (IRIS), May 2000.
- WA DEP 2000, *Volatile Organic Compounds Monitoring in Perth, Baseline Air Toxics Project*, Western Australian Department of Environmental Protection.

- WHO 1999, *Environmental Health Criteria 215, Vinyl Chloride*, International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organisation, World Health Organization, Geneva.
- WHO 2000, *Air Quality Guidelines for Europe, 2<sup>nd</sup> edn*, World Health Organization, Geneva.
- WHO 2004, *Vinyl Chloride in Drinking-water, Background document for development of WHO Guidelines for Drinking Water Quality*, World Health Organization, available from [http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/en/index.html](http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html)
- WHO 2011, *Guidelines for drinking-water quality, 4<sup>th</sup> edition*, World Health Organization, Geneva available from [http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/en/index.html](http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html).

## 6 Shortened forms

<b>ADAF</b>	age-dependent adjustment factor
<b>ADI</b>	acceptable daily intake
<b>ADWG</b>	Australian Drinking Water Guidelines
<b>AI</b>	adequate intake
<b>ANZECC</b>	Australia and New Zealand Environment and Conservation Council
<b>APVMA</b>	Australian Pesticides and Veterinary Medicines Authority
<b>ATDS</b>	Australian Total Diet Survey
<b>ATSDR</b>	Agency for Toxic Substances and Disease Registry
<b>BA</b>	bioavailability
<b>BI</b>	background intake
<b>BMD</b>	benchmark dose
<b>BMDL</b>	Benchmark dose lower confidence limit
<b>CCME</b>	Canadian Council of Ministers of the Environment
<b>CICAD</b>	Concise International Chemicals Assessment Document
<b>CNS</b>	central nervous system
<b>DAF</b>	dermal absorption factor
<b>DCE</b>	dichloroethene
<b>DW</b>	dry weight
<b>EA</b>	Environment Agency (England and Wales)
<b>EHC</b>	Environmental Health Criteria
<b>EPA</b>	Environment Protection Authority
<b>FSANZ</b>	Food Standards Australia and New Zealand
<b>GAF</b>	gastrointestinal absorption factor
<b>GV</b>	guideline value
<b>HCB</b>	hexachlorobenzene
<b>HEC</b>	human equivalent concentration
<b>HED</b>	human equivalent dose
<b>HIARC</b>	Hazard Identification Assessment Review Committee
<b>HIL</b>	health investigation level
<b>HSDB</b>	Hazardous Substances Data Bank



<b>HSL</b>	health screening level
<b>IARC</b>	International Agency for Research on Cancer
<b>IEUBK</b>	Integrated exposure uptake biokinetic model
<b>IRIS</b>	Integrated Risk Information System
<b>JECFA</b>	Joint FAO/WHO Expert Committee on Food Additives
<b>JMPR</b>	WHO/FAO Joint Meeting on Pesticide Residues
<b>LOAEL</b>	lowest observed adverse effect level
<b>LOEL</b>	lowest observed effect level
<b>MF</b>	modifying factor
<b>MoA</b>	mode (or mechanism) of action
<b>MoE</b>	margin of exposure
<b>MPC</b>	maximum permissible concentration
<b>MRL</b>	maximum residue limit
<b>MRL</b>	minimal risk level
<b>NDI</b>	negligible daily intake
<b>NEPC</b>	National Environment Protection Council
<b>NEPM</b>	National Environment Protection Measure
<b>NHMRC</b>	National Health and Medical Research Council
<b>NOAEL</b>	no observable adverse effect level
<b>NOEL</b>	no observable effect level
<b>NSW DECC</b>	New South Wales Department of Environment and Climate Change
<b>OCS</b>	Office of Chemical Safety
<b>PBDE</b>	polybrominated diphenyl ether
<b>PCB</b>	polychlorinated biphenyl
<b>PCE</b>	perchloroethene (tetrachloroethene)
<b>PNEC</b>	predicted no-effect concentration
<b>POP</b>	persistent organic pollutant
<b>PTDI</b>	provisional tolerable daily intake
<b>PTMI</b>	provisional tolerable monthly intake
<b>PTWI</b>	provisional tolerable weekly intake
<b>PVC</b>	polyvinyl chloride

<b>RAIS</b>	Risk Assessment Information System
<b>RDI</b>	recommended daily intake
<b>REL</b>	reference exposure level
<b>RfC</b>	reference concentration
<b>RfD</b>	reference dose
<b>RME</b>	reasonable maximum exposure
<b>SF</b>	slope factor
<b>TC</b>	tolerable concentration
<b>TCA</b>	trichlorethane
<b>TCE</b>	trichlorethene
<b>TD</b>	tumorigenic dose
<b>TDI</b>	tolerable daily intake
<b>TRV</b>	toxicity reference value
<b>UF</b>	uncertainty factor
<b>UL</b>	upper limit
<b>UR</b>	unit risk
<b>US EPA</b>	United States Environmental Protection Agency
<b>VOC</b>	volatile organic compound
<b>WHO</b>	World Health Organization
<b>WHO DWG</b>	World Health Organization Drinking Water Guidelines