Derivation of Risk-Based Investigation Levels
*Clandestine Drug Laboratory, Site Investigation Guidelines*

*Prepared for: Australian Crime Commission*

6 October 2009
Document History and Status

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Limitations

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It is prepared in accordance with the scope of work and for the purpose outlined in the Section 1 of this report.

The methodology adopted and sources of information used are outlined in this report. Environmental Risk Sciences has made no independent verification of this information beyond the agreed scope of works and assumes no responsibility for any inaccuracies or omissions. No indications were found that information contained in the report provided by the Australian Crime Commission was false.

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<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
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<tr>
<td>ACC</td>
<td>Australian Crime Commission</td>
</tr>
<tr>
<td>ADI</td>
<td>Acceptable Daily Intake</td>
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<tr>
<td>ANZECC</td>
<td>Australia and New Zealand Environment and Conservation Council</td>
</tr>
<tr>
<td>ASCC</td>
<td>Australian Safety and Compensation Council</td>
</tr>
<tr>
<td>AT</td>
<td>Averaging Time</td>
</tr>
<tr>
<td>BTEX</td>
<td>Benzene, toluene, ethylbenzene and total xylenes</td>
</tr>
<tr>
<td>BW</td>
<td>Body weight</td>
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<tr>
<td>CF</td>
<td>Unit conversion factor</td>
</tr>
<tr>
<td>CSMS</td>
<td>Contaminated Sites Monograph Series</td>
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<tr>
<td>ED</td>
<td>Exposure Duration</td>
</tr>
<tr>
<td>EF</td>
<td>Exposure Frequency</td>
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<tr>
<td>EIL</td>
<td>Ecologically-based Investigation Level</td>
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<tr>
<td>EPA</td>
<td>Environment Protection Authority</td>
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<tr>
<td>ET</td>
<td>Exposure Time</td>
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<tr>
<td>HIL</td>
<td>Health Investigation Level</td>
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<td>IL</td>
<td>Investigation Level</td>
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<tr>
<td>LOR</td>
<td>Limit of Reporting</td>
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<tr>
<td>NEPC</td>
<td>National Environment Protection Council</td>
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<tr>
<td>NEPM</td>
<td>National Environment Protection Measure</td>
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<tr>
<td>NHMRC</td>
<td>National Health and Medical Research Council</td>
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<tr>
<td>NSW DECC</td>
<td>New South Wales Department of Environment and Climate Change</td>
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<tr>
<td>RME</td>
<td>Reasonable maximum exposure</td>
</tr>
<tr>
<td>SA</td>
<td>Skin surface area available for contact</td>
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<tr>
<td>TC</td>
<td>Tolerable Concentration</td>
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<tr>
<td>TDI</td>
<td>Tolerable Daily Intake</td>
</tr>
<tr>
<td>TPH</td>
<td>Total petroleum hydrocarbons</td>
</tr>
<tr>
<td>TPHCWG</td>
<td>Total Petroleum Hydrocarbon Criteria Working Group</td>
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<tr>
<td>UCL</td>
<td>Upper Confidence Limit</td>
</tr>
<tr>
<td>USEPA</td>
<td>United States Environmental Protection Agency</td>
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<tr>
<td>VOC</td>
<td>Volatile Organic Compound</td>
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<tr>
<td>WHO</td>
<td>World Health Organization</td>
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<tr>
<td>L-PAC</td>
<td>L-phenylacetylcarbinol or 1-phenyl-1-hydroxy-2-propanone</td>
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<tr>
<td>MDA</td>
<td>3,4-methylenedioxyamphetamine</td>
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<tr>
<td>MDMA</td>
<td>3,4-methylenedioxyamphetamine, also known as “ecstacy”</td>
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<tr>
<td>MD-P-2-P</td>
<td>1-(3,4-methylenedioxyphenol)-2-propanone or piperonyl methyl ketone [PMK]</td>
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<tr>
<td>P-2-P</td>
<td>phenyl-2-propanone</td>
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Introduction

1.1 Background
The Australian Crime Commission (ACC) has commissioned Environmental Risk Sciences Pty Ltd (enRiskS) to derive appropriate investigation levels (ILs) to be used in the assessment and remediation of clandestine drug laboratories (clan labs).

Residual contamination associated with the manufacture of illicit drugs have the potential to be present as residues or dust on non-porous surfaces, absorbed by furnishings, walls and ducting etc in the vicinity of the clan labs, with other base chemicals and/or wastes (by-products and intermediates) having the potential to leak or be dumped into drains or directly to soil. These contaminants can in many instances persist within the building or in soil such that they have the potential to pose a risk to persons occupying the premises in the future or the environment.

ACC is developing guidelines for the assessment and remediation of sites where contamination has occurred as a result of the operation of a clan lab or other illicit drug process/laboratories. The guidelines are relevant to addressing adverse impacts the clan labs may have on the natural and built environment. They are to be applied after initial police operations have been completed and evidence and bulk chemicals/wastes have been removed from the premises.

The ILs derived in this report are intended to be included in the ACC guidelines.

1.2 Objectives
The overall objective of the work (as outlined the proposal dated 27 May 2009) is to derive appropriate investigation levels (ILs) that are relevant to the protection of human health and the environment to be used in the assessment and remediation of indoor and outdoor areas associated with clandestine drug laboratories (clan labs).

1.3 Methodology and Scope of Works
There are 2 key tasks associated with the derivation of ILs:

- Identification of key chemicals that warrant consideration in the derivation of ILs indoors and outdoors associated with the operation of clan labs in Australia; and
- Derivation of ILs that are based on the protection of human health and, where possible, the environment.

These tasks are outlined below:

Task 1 comprises a review of available information on common manufacturing methods in Australia, consideration of available data on the form, hazards and environmental fate of these compounds, available information on likely key by-products associated with the manufacturing methods and availability of toxicity studies and reviews. This review is presented to identify a key list of compounds that warrant the derivation of ILs associated with the remediation of former clan labs such that they are suitable for future use. It is not part of the scope of this assessment to undertake a detailed review and toxicological assessment of chemicals associated with former clan labs. Hence the review presented provides a summary of existing, published information.
Task 2 involves the derivation of ILs. With respect to the protection of human health the ILs will be derived using an approach consistent with the protocols/guidelines recommended by:

- National Environment Protection (Assessment of Site Contamination) Measure (NEPM), Schedule B(4), “Guideline on Health Risk Assessment Methodology” (1999a); and

Where required additional guidance will be obtained from relevant Australian and International guidance consistent with current industry best practice and relevant to the exposures that require consideration for clan labs.

With respect to the protection of the environment, ILs will not be derived, however currently available published screening level guidelines (threshold or benchmarks) relevant to the protection of the terrestrial or aquatic environment will be identified and presented.

In general, the ILs will be derived using an approach that is consistent with the derivation of NEPM HILs (1999b). The NEPM HILs only provide guidelines for a limited number of chemicals (non-volatile) in soil under a range of different land use scenarios. The ILs to be derived as part of these guidelines will follow an approach consistent with the NEPM HILs, for the same key land uses; however they will address the presence of key chemicals in all media significantly impacted by operations at a clan lab, namely soil (outdoors), air (indoors) and surface residues (indoors).

It is noted that a number of guidelines are available from the US that are associated with the assessment and remediation of former clan labs (refer to references). These guidelines focus on a limited number of compounds (mainly methamphetamine, VOCs as a group, lead and mercury). Many of the guidelines are based on analytical limits of detection rather than protection of human health or the environment and hence a more detailed review of clan lab methods in Australia and identified of key compounds associated with these methods has been undertaken.
2.1 General
This section provides information regarding the types of premises where clan labs are typically found, sources of contamination and the key exposures that require consideration in the derivation of ILs.

2.2 Exposure Scenarios and Premises
A large number of premises used as clan labs are residential type homes or properties. These may include single family residences in urban or rural areas, apartments, hotel/motel rooms and commercial buildings or storage areas.

The derivation of ILs requires consideration of a range of end-uses and as such will be derived to specifically address the following types of exposure scenarios and premises:

- Residential – includes single family homes in low-density residential (urban or rural) area or apartments with minimal access to soil. Guidelines have been developed for indoor and outdoor areas separately and hence they can be applied to other premises such as apartments or hotel/motels for the areas affected as required. Exposures in these homes/premises will be by residents of all ages (adults and children);
- Commercial – includes commercial buildings (of all types) that may be used for the purpose of commercial industrial purposes. Exposures are assessed for adult workers assuming potential exposure to environmental contamination from the former clan lab. Hence the approach adopted will be consistent with that adopted for the assessment of residential exposures rather than that which is applied as outlined by NOHSC (1995a,b) for occupational exposures. Other uses of commercially zoned premises such as childcare centres or schools (wherever children may remain on the site for any length of time) should be assessed as for a residential scenario;
- Environment – outdoor environment, particularly soil and water environments considered.

The assessment of recreational areas where no buildings are present can be assessed by considering the ILs for outdoor soil. The outdoor soil values derived under the residential scenario will be adopted for recreational exposures outdoors.

2.3 Sources and Hazards
From its initial establishment through its ultimate re-occupancy, a clan lab typically goes through four phases that vary with the nature of activities. The four phases may generally be described as

1. **Operational**: illicit drug manufacturing takes place;
2. **Discovery & Removal**: the lab is “busted” or “seized” (discovered by police) and bulk chemicals and equipment are removed.

During these phases, inhalation of airborne contaminants (such as methamphetamine, acidic and corrosive gases, and phosphine gas) and direct contact with primary sources and wastes are expected to represent the greatest hazard, along with physical hazards associated with the use and manufacture of chemicals that are flammable, reactive and potentially explosive. Removal of primary sources involves management of exposures through the use of appropriate personal protective equipment (PPE). Once the primary sources (reaction vessels, solvent in original containers and those in other containers, tanks of compressed gases) of contaminants have been physically removed, secondary sources may still remain in the premises. The focus of this report is on the presence of these secondary sources that require remediation prior to safe re-occupancy of the premises.
3. **Remediation & Verification**: samples are collected to characterise the distribution of contaminants within the premises, the contaminants are remediated, and samples are collected to verify that residual contaminant levels are below relevant clean-up levels; and

4. **Re-Occupancy**: a new group of residents or workers occupies the premises which housed the former clan lab.

Secondary sources that are the focus of remediation and validation works require prior to re-occupancy include:

- Solvent spills and "dumped" waste products that may be present indoors, or within the soil outside of the premises. Many liquid waste products are disposed of by pouring into indoor plumbing drains that then flow into the sewer or other sewage treatment systems (septic tanks). Some waste may be poured directly into soil outside the premises. Some contaminants in soil have the potential to leach to groundwater;
- Surface residues and dust deposited onto surfaces (porous and non-porous) during the operation of the clan lab. It is noted that deposition of chemicals can be widespread throughout a premises during the operation of the clan lab (refer to **Section 2.4**). Dust and surface residues are primarily associated with non-volatile compounds where key pathways of exposure include dermal absorption following skin contact with contaminated surfaces and ingestion following hand-to-mouth (and object-to-mouth) activities; and
- “soft” media (such as upholstered furniture, curtains, carpet and plasterboard) that have absorbed solvent vapours and volatile by-products during the operational phase of the clan lab. Re-release (or “off-gassing”) of volatile chemicals that have been absorbed into soft media appears to represent the primary inhalation hazard during remediation and validation activities. The time taken between discovery and source removal and re-occupancy varies but can be many months. The longer this time the greater the potential for off-gassing processes to have largely gone to completion reducing the potential significance of these inhalation exposures.

The purpose of the ILs presented in this report is to address these secondary sources such that remediation and validation can be undertaken to be protective of long-term (chronic) exposures by members of the public (residents and workers) and the environment.

### 2.4 Fate and Transport of Methamphetamine Indoors

The fate and transport of methamphetamine indoors has been studies more extensively than other products associated with the operation of clan labs. The available information is considered to be generally representative of the fate and transport of processes of key products and some by-products of the operation of clan labs.

A number of studies have been undertaken that look at levels of methamphetamine on indoor surfaces resulting from clandestine manufacture. The studies have looked into the concentrations present as well as the distance away from the main cook area. The studies include data collected from real clan labs during initial investigations by authorities as well as “controlled cooks” used to simulate exposures that may occur in a clan lab (Martyny et al 2004 (a, b, c) and 2005 (a, b), and OEHHA 2009a). From these studies the following has been noted:

- Methamphetamine is released as an aerosol during the production process and transported by air to locations distant from the site of synthesis. Hence surface residues associated with the clan lab are found throughout the premises not just in the room(s) used for manufacture;
- Residues may persist for a long period of time (many months at least) without any remediation;
- The primary mechanism of aerosol release appears to be “salting out” the free base form of methamphetamine. Some methods produce higher levels of methamphetamine than others, however
in all methods assessed, methamphetamine residues are found at the location of manufacture and throughout the premises;

- Methamphetamine residues have also been found to be present throughout a premise that has been used for smoking the drug. The greater the drug use, the higher the surface residues reported;
- Activity in a residence where methamphetamine has been manufactured can result in re-suspension of respirable fractions resulting in the potential for inhalation exposures;
- Washing of surfaces removes a significant portion of methamphetamine surface residues, in particular dislodgeable residues (which would be re-suspended with activity in the premises) are readily removed;
- The initial product of methamphetamine synthesis is the free base form of the drug, which is volatile. To prevent evaporative loss and facilitate storage and transport of the drug, methamphetamine base is converted to methamphetamine hydrochloride (a salt) using hydrogen chloride gas. Van Dyke et al. (2009) have suggested that “salting out” is a major mechanism for release of methamphetamine to the indoor environment. It is possible that both forms of methamphetamine – the hydrochloride salt and the free base – are released at this stage of synthesis. If this were the case, the base would not be expected to persist due to its volatility, but the salt would likely persist under most environmental conditions. However, if the hydrochloride salt comes into contact with moisture and the pH is greater than 4, the free base would be regenerated and the drug would once again have a tendency to volatilise. pH-dependent regeneration of the free base may be particularly important in understanding the success (or lack thereof) of using detergents to clean methamphetamine-contaminated surfaces, or using water-based latex paint to encapsulate the contamination, since cleaning detergents and latex paints are both alkaline.

The above comments are considered relevant to methamphetamine as well as other products and by-products produced by clan labs. The specific nature of each chemical will determine the potential for it to be of importance in surface residues, dust, indoor air or the outdoor environment.
### Identification and Toxicity of Key Chemicals

#### 3.1 General

This section provides a review of the methods and chemicals typically associated with the manufacture of illicit drugs in Australia, review of these methods and chemicals to identify key compounds for the derivation of ILs.

#### 3.2 Illicit Drug Manufacturing Methods

A large number of methods (well over 100 “recipes”) have been identified that involve the manufacture of amphetamine, methamphetamine, MDA (3,4-methylenedioxyamphetamine), MDMA (3,4-methylenedioxymethamphetamine, also known as “ecstasy”), MD-P-2-P (1-(3,4-methylenedioxyphe"nol)-2-propanone or piperonyl methyl ketone [PMK]), P-2-P (phenyl-2-propanone) and pseudoephedrine (as precursor to manufacture of other drugs).

Most methods associated with the manufacture of methamphetamine involve three basic steps: (1) the extraction of the precursor drug; (2) reduction of ephedrine or pseudoephedrine to methamphetamine; and (3) the “salting out” stage which extracts the methamphetamine (or other product) in a solid form. While many labs perform all stages of manufacture it is not uncommon for the various stages to be divided between labs. Therefore products, reagents and by-products may vary from site to site and include a wide range of compounds.

In Australia the following methods have been identified (following seizure of clan labs as per information from ACC) as the most comment methods that require consideration in the derivation of ILs:

- **MDMA from:**
  - MD-P-2-P, methylamine, aluminium and mercury (Shulgin Method);
  - MD-P-2-P, N-methylformamide and formic acid (Leuckart Method);
  - MD-P-2-P, methylamine and sodium cyano/borohydride; and
  - Safrole, hydrobromic acid and methylamine.

- **MD-P-2-P from sassafrass oil, potassium hydroxide, hydrogen peroxide and formic acid (Shulgin).**

- **Methamphetamine from:**
  - Pseudo/ephedrine, hypo/phosphorous acid and iodine;
  - Pseudo/ephedrine, phosphorous and iodine/hydroiodic acid;
  - Pseudo/ephedrine, lithium/sodium and ammonia (anhydrous);
  - P-2-P, methylamine, aluminium and mercury salt.

- **P-2-P from benzaldehyde, nitroethane and iron (or zinc);**

- **Pseudoephedrine from:**
  - pharmaceutical preparations;
  - benzaldehyde via L-PAC (L-phenylacetylcarbinol or 1-phenyl-1-hydroxy-2-propanone) and reductive animation by catalytic hydrogenation; and
  - benzaldehyde via L-PAC and reductive animation with sodium cyano/borohydride.

These methods have been reviewed in detail with respect to the potential base products, reactants, products and by-products. A list of these compounds associated with the above methods is presented in Appendix A.
3.3 Review and Identification of Key Chemicals

The range of compounds identified (Appendix A) have been reviewed further to identify key chemicals that require consideration in the assessment and potential remediation of former clan labs. The review undertaken has considered the following for each compound identified:

- The commonality of the compound is with respect to its potential use/presence from the range of methods considered. More common compounds have been considered of greater importance (unless there is another reason for inclusion such as toxicity). This review also considered discussion with chemists from the ACC regarding chemicals that may be of significance at former clan labs;
- Potential significance as identified from studies conducted at clan labs. It is noted that most of the studies undertaken on clan labs have not specifically addressed the presence of chemicals in environmental media (including indoors) following cessation of manufacturing. Most of the studies available address exposures during manufacturing, hazards during seizure of clan labs and analysis of products (including the identification of impurities). Hence the number of studies that address chemicals that may be of significance from a long-term environmental exposure perspective (as addressed in the report) are limited. The studies and reports used for this review include the following: Pal and Kirkbride (2009), USEPA (2008), CDTSC (2003), CDPH (2006), Gimeno et al (2005), JCLICA (October 1999, April 2006, January 2007, January 2008) and Shakula and Kulkarno (2000);
- Consideration of the toxicity of the chemicals. In particular the following was considered:
  - Availability of any data regarding the nature and potential toxicity of chemicals;
  - Acute toxicity and hazard associated with the chemicals. Some chemicals are acute hazards and toxicants and need to be handled with caution during the seizure of clan labs. Some of the chemicals identified as acute hazards have a low long term toxicity to human health or the environment and hence these have not been considered further in this assessment;
  - Potential for the sampling and analysis of the chemicals to be undertaken using commercially available methods consistent with those currently used in the assessment of contaminated sites in Australia. Chemicals that cannot be readily assessed using commercial available (and cost effective) methods have not been included (unless there is another reason for inclusion such as significant potential for chronic toxicity); and
  - Potential for high chronic toxicity to human and/or the environment (where information is available). The focus of the guidelines being developed is the assessment of long-term (chronic) exposures to chemicals that may remain in a former clan lab, hence the most significant issue that requires consideration is the potential for chronic effects to be of significance at low concentrations.

On the basis of the above review a range of chemicals have been identified that require consideration. These include the following:

- Methamphetamine and salts;
- MDMA and salts;
- Ephedrine and Pseudoephedrine and salts;
- Acids and bases – these include acetic acid, hydrochloric acid, sulphuric acid, formic acid and sodium hydroxide. The potential presence of these can be addressed through the assessment of pH;
- Solvents, which include the following:
  - Chlorinated solvents, namely chloroform and dichloromethane;
  - Petroleum hydrocarbon based solvents that include shellite (also known as Coleman Fuel or white spirits), mineral turpentine, methylated spirits, toluene and xylenes. These products are light petroleum products that can be characterised through the assessment of BTEX.
(benzene, toluene, ethylbenzene and total xylenes), naphthalene and total petroleum hydrocarbons (TPH).

- Ammonia;
- Iodine which may be associated with the use of hydriodic acid (also assessed on the basis of pH), hydrogen iodide and iodine as a reactant and by-product;
- Bromide which may be associated with the use of hydrobromic acid (also assessed on the basis of pH);
- Phosphorous which may be associated with the use of phosphorous and hypophosphorous acids (also assessed on the basis of pH) or the use of red phosphorous as a reactant;
- Methylamine;
- N-methylromamide;
- Nitroethane;
- Benzaldehyde;
- Phosphine;
- Safrole and isosafrole;
- Boron and compounds which may be associated with the use of sodium borohydride where borides and other boron compounds may remain;
- Mercury, principally mercuric chloride; and
- Lithium.

The key chemicals listed above are summarised in Table 1. It is noted that the potential presence of MDA, P-2-P, MD-P-2-P and some pharmaceutical ingredients (from pseudoephedrine preparations) may also be of significance at former clan labs, however there is little or no data regarding chronic toxicity of the compounds. In addition the analysis of these compounds requires more specialised methods not commonly used for contaminated sites. Hence they have not been included in the guidelines presented, however it is noted that should future analysis indicate that the presence of these compounds is of significance then further work may need to be undertaken to include these compounds in the guidelines.

### Table 1 Key Chemicals Indentified

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Category</th>
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<tbody>
<tr>
<td>Methamphetamine</td>
<td>Boron and compounds</td>
</tr>
<tr>
<td>MDMA</td>
<td>Mercury (inorganic)</td>
</tr>
<tr>
<td>Ephedrine and pseudoephedrine</td>
<td>Lithium</td>
</tr>
<tr>
<td>Ammonia</td>
<td>Benzaldehyde</td>
</tr>
<tr>
<td>Iodine</td>
<td>Phosphine</td>
</tr>
<tr>
<td>Bromide</td>
<td>Safrole and isosafrole</td>
</tr>
<tr>
<td>Phosphorous (acids) and red phosphorous</td>
<td>Chloroform</td>
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<td>N-Methylformamide</td>
<td>Dichloromethane</td>
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<td>Methylamine</td>
<td>pH</td>
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<td>Nitroethane</td>
<td>Petroleum Hydrocarbons:</td>
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<tr>
<td></td>
<td>Benzene</td>
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<td></td>
<td>Toluene</td>
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<tr>
<td></td>
<td>Ethylbezene</td>
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<td></td>
<td>Total Xylenes</td>
</tr>
<tr>
<td></td>
<td>Naphthalene</td>
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<tr>
<td></td>
<td>TPH (fractions C6-C9, C10-C14 and C15+)</td>
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</tbody>
</table>
### 3.3 Toxicity and Properties of Key Chemicals

The derivation of guidelines that are protective of human health and the environment requires the identification of key properties associated with the chemicals identified as well as quantitative toxicity data. The focus of this assessment is on long-term (chronic) exposures and hence short-term hazards and acute issues (that are of importance to law enforcement agencies during the initial discovery and seizure of a lab) have not been considered further. Quantitative data used to assess the toxicity of the chemical with respect to oral, inhalation and dermal exposures. The data presented are derived from published, peer-reviewed sources consistent with the approach outlines and presented in guidance from enHealth (2002) and NEPM (1999a).

For the purpose of deriving guidelines that can be used for the assessment of former clan labs, the nature of the key chemicals identified is important in identifying the relevance of deriving guidelines in various media. Specifically the following has been considered important:

#### Indoor Areas:

- **Surface Residues**: Chemicals that have the potential to remain on surfaces as a residue or dust are considered of importance. These are primarily non-volatile chemicals. Chemicals that are volatile are not expected to remain on non-porous surfaces for long enough to be of concern with respect to chronic exposures and hence a surface guideline has not been derived for these chemicals.

- **Indoor Air**: The focus of indoor air quality will be on the presence of volatile or gaseous compounds only. These compounds may have been absorbed into porous materials during the operation of the clan lab, after which they are expected to continue to off-gas to indoor air. The potential presence of chemicals in dust indoors is considered to be adequately addressed through the assessment of surface residues and outdoor soil (where inhalation of dust and volatiles derived from outdoor areas is also addressed).

#### Outdoor Areas:

- **Soil**: Soil guidelines have been derived for all chemicals identified that have the potential to remain in the soil environment for any significant period of time. In general most chemicals identified are considered of importance with respect to outdoor soils, except those that are essentially only likely to be present as gas phase compounds. For the chemicals considered in outdoor soil, criteria that are protective of human health will be derived.

- **Environment**: The potential environmental fate of chemicals has been considered, including the potential for bioaccumulation in aquatic species. Where available, existing guidelines relevant to the protection of the terrestrial or aquatic environments have been identified.

Appendix B presents a summary of the properties, fate and transport and toxicity of the key chemicals identified.

Table 2 presents a summary of the key aspects of the chemicals identified with respect to their relevance to the derivation of guidelines indoors and outdoors.
Table 2  Summary of Properties, Media of Concern and Availability of Guidelines and Toxicity Data for Key Chemicals

<table>
<thead>
<tr>
<th>Key Chemical</th>
<th>Properties*</th>
<th>Media of Concern</th>
<th>Toxicity Data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gas</td>
<td>Liquid</td>
<td>Solid</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>●</td>
<td>●</td>
<td>L</td>
</tr>
<tr>
<td>MDMA</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Ephedrine and pseudoephedrine</td>
<td>●</td>
<td>M</td>
<td>●</td>
</tr>
<tr>
<td>Ammonia</td>
<td>●</td>
<td>●</td>
<td>H</td>
</tr>
<tr>
<td>Iodine</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Bromide</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Phosphorus (acids) and red phosphorus</td>
<td>●</td>
<td>L</td>
<td>●</td>
</tr>
<tr>
<td>N-Methylformamide</td>
<td>●</td>
<td>M</td>
<td>●</td>
</tr>
<tr>
<td>Methylamine</td>
<td>●</td>
<td>●</td>
<td>G</td>
</tr>
<tr>
<td>Nitroethane</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Boron and compounds</td>
<td>●</td>
<td>L</td>
<td>●</td>
</tr>
<tr>
<td>Mercury (inorganic)</td>
<td>●</td>
<td>L</td>
<td>●</td>
</tr>
<tr>
<td>Lithium</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>●</td>
<td>●</td>
<td>H</td>
</tr>
<tr>
<td>Phosphine</td>
<td>●</td>
<td>G</td>
<td>H</td>
</tr>
<tr>
<td>Safrole and isosafrole</td>
<td>●</td>
<td>●</td>
<td>P</td>
</tr>
<tr>
<td>Chloroform</td>
<td>●</td>
<td>●</td>
<td>L</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>●</td>
<td>●</td>
<td>M-H</td>
</tr>
<tr>
<td>Benzene</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Toluene</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Ethylbenzene</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Xylenes</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Naphthalene</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>TPH C6-C9</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>TPH C10-C14</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>TPH C15+</td>
<td>●</td>
<td>●</td>
<td>M</td>
</tr>
<tr>
<td>Acids and bases (pH)</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
</tbody>
</table>

Notes:
* Properties relevant to the form of the compound expected to be present at former clan labs (included compounds, salts and various acids associated with the key chemicals)
** pH issues associated with the acids used in clan labs that are associated with the key chemicals identified
# Potential for environmental issues to be significant considered to be low based on the nature of the compound, however there may be some sites where environmental concerns may require additional consideration.
## Limited data which suggest ecotoxicity at high concentrations in soil, above those likely to be protective of human health.

Potential for degradation ranked as either L=Low, M= Moderate or H=High based on qualitative information on the chemical

Environmental Screening Guidelines are available for different media that include: S=soil and A=aquatic environments. Some guidelines are qualified: M=potential issues associated with methylmercury may need to be considered

Chronic toxicity data available from a number of sources (refer to Appendix B for details and quantitative values): W=WHO, N=NHMRC, U=USEPA, O=OEHHA, A=ATSDR, T=TPHCWG, T=TC EQ and D=derived from available information as noted below:

1 value for MDMA derived based on limited quantitative data and comparison of toxicity to methamphetamine. Confidence on value is considered low.
2 value derived on the basis of occupational data (TWA) and consideration of relevant modifying factors and uncertainty factors following guidance from the USEPA (2009).
Based on the summary presented in Table 2 (and Appendix B) ILs relevant to surface residues, indoor air and soil (outdoors) have been derived for only the key chemicals expected to be of significance in these media. Table 3 presents a summary of the key chemicals considered for surface residues, indoor air and soil.

**Table 3  Summary of Key Chemicals Relevant to Each Media of Concern**

<table>
<thead>
<tr>
<th>Surface Residues</th>
<th>Indoor Air</th>
<th>Outdoor Soil</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methamphetamine</td>
<td>Ammonia</td>
<td>Methamphetamine</td>
</tr>
<tr>
<td>MDMA</td>
<td>Iodine</td>
<td>MDMA</td>
</tr>
<tr>
<td>Ephedrine and pseudoephedrine</td>
<td>Bromide</td>
<td>Ephedrine and pseudoephedrine</td>
</tr>
<tr>
<td>Iodine</td>
<td>Methylamine</td>
<td>Ammonia</td>
</tr>
<tr>
<td>Bromide</td>
<td>Nitroethane</td>
<td>Iodine</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Benzaldehyde</td>
<td>Bromide</td>
</tr>
<tr>
<td>N-Methylformamide</td>
<td>Phosphine</td>
<td>Phosphorus</td>
</tr>
<tr>
<td>Boron and compounds</td>
<td>Safrole and isosafrole</td>
<td>N-Methylformamide</td>
</tr>
<tr>
<td>Mercury (inorganic)</td>
<td>Chloroform</td>
<td>Methylamine</td>
</tr>
<tr>
<td>Lithium</td>
<td>Dichloromethane</td>
<td>Nitroethane</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>Benzene</td>
<td>Boron and compounds</td>
</tr>
<tr>
<td>Safrole and isosafrole</td>
<td>Toluene</td>
<td>Mercury (inorganic)</td>
</tr>
<tr>
<td>TPH C15+</td>
<td>Ethylbenzene</td>
<td>Lithium</td>
</tr>
<tr>
<td>Acids and bases (pH)</td>
<td>Xylenes</td>
<td>Benzaldehyde</td>
</tr>
<tr>
<td></td>
<td>Naphthalene</td>
<td>Safrole and isosafrole</td>
</tr>
<tr>
<td></td>
<td>TPH C6-C9</td>
<td>Chloroform</td>
</tr>
<tr>
<td></td>
<td>TPH C10-C14</td>
<td>Dichloromethane</td>
</tr>
<tr>
<td></td>
<td>Benzene</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Toluene</td>
<td>Ethylbenzene</td>
</tr>
<tr>
<td></td>
<td>Xylenes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Naphthalene</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPH C6-C9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPH C10-C14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPH C15+</td>
<td>Acids and bases (pH)</td>
</tr>
</tbody>
</table>
3.4 Uncertainties

A number of uncertainties are inherent within the process adopted in this section for the selection of key chemicals and identification of chemical properties and toxicity relevant for the derivation of ILs. The key uncertainties include the following:

- There are very few studies or detailed investigations that have been completed on former clan labs where a comprehensive range of analytes has been assessed in surface residues or soil. Hence the identification of reactants, products, by-products and intermediates that may remain in residues and soil at level that require assessment and potential remediation following seizure of a clan lab has relied on limited information. It is expected that following the assessment of a number of former clan labs in Australia some of the key chemicals identified may not need to remain on the list, or some other chemicals may be present at higher concentrations than expected and may require the derivation of an IL.

- A number of products associated with a number of methods, namely MDA, P-2-P, MD-P-2-P and some pharmaceutical ingredients (from pseudoephedrine preparations) may also be of significance at former clan labs, however there is no data regarding chronic toxicity of these compounds and they are more difficult to analyse. These compounds may be included once additional studies have been undertaken.

- The environmental fate, transport and environmental effects of a number of the key products (particularly methamphetamine, MDMA, pseudo/ephedrine) is very limited. As more studies are undertaken and the fate and transport processes are better understood, these guidelines may need to be reviewed.

- The toxicity data available and relevant to the assessment of chronic environmental exposures to MDMA is limited. The level of confidence in the derived value (refer to Appendix B) is considered to be low and it should be considered guide only. The toxicity data should be further reviewed in detail, particularly if MDMA is found to be widely distributed and persistent in former clan labs.
Derivation of Investigation Levels

4.1 Approach

The derivation of ILs for the assessment of former clan labs has been undertaken on the basis of the following:

- ILs based on the protection of human health have been derived to address the most significant exposures expected indoors and outdoors associated with the key chemicals identified. This is based on the toxicity of the chemical (refer to Appendix B), the potential for exposure and the target risk level adopted. It is noted that for some key chemicals, existing ILs are available as part of national (NEPM) or state guidance. Where these existing ILs are available, these have been listed in preference to derived ILs. The derivation of health ILs is further discussed in this Section.

- ILs based on the protection of the environment have not been derived. Rather ILs have been identified based on existing trigger levels or benchmarks available for soil and water in published literature. The available guidelines are listed in Appendix B, with the most relevant values adopted as ILs.

4.2 Target Risk Levels

4.2.1 General

ILs have been derived for chemicals that are associated with non-threshold carcinogenic effects and threshold effects. Hence target risk levels relevant for both approaches are required to be established.

4.2.2 Threshold Effects

This relationship assumes that there is a level of exposure below which there is no (or no appreciable) risk of an adverse health effect. This is in contrast to the non-threshold relationship where there is an increased risk associated with any exposure. The WHO identifies threshold chemicals as those which are not suspected of exhibiting carcinogenic effects (non-carcinogens) or those which exhibit non-genotoxic carcinogenicity. Toxicity factors for these chemicals are referred to as an acceptable daily intake (ADI, by the WHO) or reference dose (RfD, by the USEPA) for oral exposures (in units of mg per kg body weight per day) and a tolerable concentration (TC, by WHO) or reference concentration (RfC, by USEPA) for inhalation exposures (in units of mg per cubic metre of air). The lower the ADI, RfD, TC or RfC, the more toxic the chemical and the lower the concentration above which there exists a potential for an adverse health effect.

The assessment of the potential for adverse effects to occur as a result a chronic exposure to the chemicals assessed has been undertaken by calculating a Hazard Index. This is simply the sum of the ratio of the exposure (as an intake or concentration) over all the relevant pathways to the relevant threshold toxicity value (adjusted for background). Background intakes relevant to each key chemical are noted in Appendix B.

For the purpose of deriving ILs a target $HI = 1$ has been adopted for each individual chemical. This means that the total chemical intake (or concentration) associated with the derived concentration (or surface residue limit) is equal to the threshold toxicity value (including background intakes). This approach is consistent with that used in the derivation of the existing HILs (NEPM, 1999b).

It is noted that the deviation of the ILs using this approach requires consideration of mixture effects in the application of the guidelines.
4.2.3 Non-Threshold Carcinogenic Effects

Non-threshold toxicity values assume that any amount of exposure to the chemical has the potential to result in an increased risk. These chemicals are typically carcinogens with their toxicity values referred to as cancer risk slope factors. The World Health Organisation (WHO) assigns slope factors (or unit risks) to chemicals identified as genotoxic carcinogens with other carcinogens identified evaluated on the basis of a threshold response relationship (refer above). A slope factor (or unit risk) is an upper bound estimate of the probability of a response (carcinogenic effects) occurring following the intake of a chemical over a lifetime via a specific exposure pathway (such as ingestion or inhalation). The calculation of a cancer risk implies that any exposure to these chemicals may result in an increased risk or probability of contracting cancer over a lifetime. Therefore the higher the slope factor (or unit risk), the higher the risk that may be associated with a given exposure.

Non-threshold carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential non-threshold carcinogen. The assessment of exposures to benzene and safrole/isosafrole (refer to Appendix B) is associated with non-threshold carcinogenic effects.

In identifying a target non-threshold risk for the purpose of deriving ILs the following is noted:

- An incremental lifetime cancer risk of $1 \times 10^{-6}$, means that in a population of 1 million people which has been exposed to the chemical for their lifetime one additional cancer is predicted over and above the background incidence of cancer in that population (1 million people). For the same population a cancer risk of $1 \times 10^{-4}$ implies that 100 additional cancers are predicted over and above the background incidence (for 1 million people).
- These values are extremely low when compared to the background incidence of cancer in our society. The background incidence for all cancers is in the order of 1 in 3 to 1 in 2 for an 85 year lifetime (AIHW, 2007). This means that for a population of 1,000,000 around 333,000 individuals are expected to contract cancer over an 85 year lifetime. An additional $1 \times 10^{-6}$, risk predicts 1 additional individual may develop cancer in that population over a lifetime.
- Specific Australian guidance related to the significance of cancer risk estimates is not currently available. Current US EPA policy states that: “Where the cumulative site risk to an individual based on reasonable maximum exposure for both current and future land use is less than $10^{-4}$,..action is generally not warranted unless there are adverse environmental impacts” (US EPA, 1991). If risks are found to be greater than the $10^{-4}$ probability, then the US EPA recommends that a preliminary remediation goal of $10^{-6}$ cancer risk be developed as the point of departure (ibid).
- A review of the origins of the $10^{-6}$ cancer risk number has been undertaken by Kelly (1991) and a review of the development of an Australian approach to the assessment of carcinogenic contaminants has been prepared for discussion by Fitzgerald (1993). Both these reviews indicate that the $10^{-6}$ was suggested by the United States Food and Drug Authority (USFDA) in 1961, as representing the de minimis legal risk. That is, the level of risk that can be identified, in a legal sense, as being representative of negligible or trivial risk. As the more recent US EPA policy (quoted above) indicates, the application of cancer risks has seen the acceptance of higher risk values with risks between $4 \times 10^{-3}$ and $1 \times 10^{-6}$ deemed acceptable.
- The application of cancer risk values in Australia and elsewhere is generally consistent with the US EPA policy. That is, the $10^{-6}$ risk value is commonly identified as the point of departure from negligible risk and the $10^{-4}$ risk value is commonly adopted as indicative of unacceptable risks (NSW DEC, 2005). The $10^{-6}$ risk value is sometimes used as the basis for defining ambient standards applicable to wide scale population exposure. For example, the NHMRC and the Agricultural and Resources Management Council of Australia and New Zealand (NHMRC/ARMCANZ 2004) have used the $10^{-6}$ value for the derivation of the Australian drinking water guidelines for genotoxic carcinogens. The
WHO and Health Canada, on the other hand, have used the $10^{-5}$ risk as the basis for the derivation of the WHO drinking water guidelines (WHO 2008) and the Dutch use the $10^{-4}$ lifetime cancer risk as the basis for the derivation of human Intervention Values for soil and groundwater for genotoxic carcinogens.

- Health Canada (2004) have undertaken a review of what is considered an acceptable risk and noted that given the conservatism (safety) margin associated with the derivation of cancer slope factors and unit risks, and the negligible impact of a 1-in-100,000 incremental risk level for contaminated site exposures, a cancer risk level of 1-in-100,000 ($1 \times 10^{-5}$) is recommended for the purposes of assessing and managing sites contaminated with carcinogenic substances.

- It is understood that a goal of $10^{-5}$ is generally accepted by Victorian EPA accredited auditors as indicating conditions that might warrant specific management or remedial action.

### Adopted Risk Targets

Based on the above discussion it is considered that the following reflects current practice in Australia:

- Incremental risks below $1 \times 10^{-6}$ would be considered to be effectively zero;
- Incremental risks between $1 \times 10^{-6}$ and $1 \times 10^{-5}$ would be considered acceptable;
- Risks greater than $1 \times 10^{-4}$ would be considered unacceptable and warrant some form of action or management to reduce the risk.

On this basis a Target Risk value of $1 \times 10^{-5}$ has been adopted for the derivation of ILs. It is noted that neither NEPM (1999 a, b) or enHealth (2002) provide guidance on an acceptable risk level for non-threshold carcinogenic effects.

### 4.3 Exposure Settings, Receptors and Pathways

With respect to the protection of human health, ILs have been derived for indoor areas (surface residues and indoor air) and outdoor soil. The media of concern relevant to each of the key chemicals identified are listed in Table 3. For these media of concern ILs have then been derived for the exposure scenarios considered, namely residential and commercial/industrial. The exposure settings considered have been selected (also adopting the terminology) to be consistent with the exposure settings adopted in the existing HILS (NEPM 1999b).

Table 4 provides a summary of the exposure settings considered in the derivation of the ILs as well as the key receptors and pathways considered:

The assessment presented has addressed potential worst-case exposure to the key chemicals based on a Reasonable Maximum Exposure (RME) scenario. This is estimated by using intake variables that define the highest exposure that is reasonably likely to occur in the exposures scenarios assessed. The RME is likely to provide a conservative or overestimate of total exposure and therefore health risk. Hence the derived ILs are expected to be conservative.

The methodology adopted for the derivation of ILs, exposure (intake) assumptions adopted and calculations undertaken are presented in the following:

- **Appendix C** – derivation of surface residue ILs;
- **Appendix D** – derivation of indoor air ILs;
- **Appendix E** – derivation of outdoor soil ILs.
# Table 4  Summary of Exposure Settings, Receptors and Pathways – Derivation of ILs

<table>
<thead>
<tr>
<th>Exposure Setting and Media of Concern</th>
<th>Exposure Pathways</th>
<th>Key (significant) Receptor</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Residential A</strong> – low density residential homes and childcare centres (indoor values can be applied to other exposure scenarios such as apartments/hotels etc)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surface Residues</td>
<td>Dermal contact by hands</td>
<td>Most significant exposures occur by young children who spend more time in contact with residues during crawling and floor play. Also have greatest exposures during mouthing of hands and objects. The potential for exposure decreases with age. Hence ILs for residential surface residues have been based on exposures by young children.</td>
</tr>
<tr>
<td></td>
<td>Dermal contact by rest of body</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ingestion of residues from mouthing hands</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ingestion of residues by mouthing objects</td>
<td></td>
</tr>
<tr>
<td>Indoor Air</td>
<td>Inhalation of volatile chemicals derived from off-gassing from porous materials</td>
<td>Exposures that may occur by residents of all ages</td>
</tr>
<tr>
<td>Outdoor Soil</td>
<td>Incidental ingestion of soil</td>
<td>Exposures by young children most significant for the assessment of threshold effects. Exposures by residents of all ages (over a lifetime) relevant for non-threshold carcinogenic effects.</td>
</tr>
<tr>
<td></td>
<td>Dermal Contact with soil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inhalation of dust and volatiles from chemicals in outdoor soil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(exposures outdoors with 75% assumed to be present indoors)</td>
<td></td>
</tr>
<tr>
<td><strong>Residential E</strong> – Exposures to soil in outdoor areas (note indoor values presented for Residential A can be used if a building is present)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outdoor Soil</td>
<td>Incidental ingestion of soil</td>
<td>Exposures by young children most significant for the assessment of threshold effects. Exposures by residents of all ages (over a lifetime) relevant for non-threshold carcinogenic effects.</td>
</tr>
<tr>
<td></td>
<td>Dermal Contact with soil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inhalation of dust and volatiles from chemicals in outdoor soil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(exposures outdoors with 75% assumed to be present indoors)</td>
<td></td>
</tr>
<tr>
<td><strong>Commercial/Industrial F</strong> – exposures by adult workers in commercial or industrial areas. Note that scenarios that include children, such as childcare centres and schools need to use Residential A ILs.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surface Residues</td>
<td>Dermal contact by hands</td>
<td>Only adult workers indoors considered.</td>
</tr>
<tr>
<td></td>
<td>Dermal contact by rest of body</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ingestion of residues from incidentally placing hands in mouth</td>
<td></td>
</tr>
<tr>
<td>Indoor Air</td>
<td>Inhalation of volatile chemicals derived from off-gassing from porous materials</td>
<td>Exposures that may occur by workers indoors</td>
</tr>
<tr>
<td>Outdoor Soil</td>
<td>Incidental ingestion of soil</td>
<td>Only adult workers indoors considered.</td>
</tr>
<tr>
<td></td>
<td>Dermal Contact with soil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inhalation of dust and volatiles from chemicals in outdoor soil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(exposures outdoors with 75% assumed to be present indoors)</td>
<td></td>
</tr>
</tbody>
</table>
4.4 Derived Investigation Levels

Table 5 presents a summary of the derived ILs for the exposure settings outlined above as well as the available screening level guidelines identified that are relevant to the protection of the terrestrial and aquatic environments (where relevant).

The following is noted with respect to the application of the ILs presented:

- Where available existing national (NEPM) or state ILs have been listed in preference to derived ILs. If the NEPM guidelines are revised or expanded to include key chemicals also addressed in this report, the revised NEPM values should be adopted as ILs. Derived ILs for these chemicals are presented in the relevant appendices and can be utilised where relevant, however in most cases the existing national or state guidelines should be adopted;
- As with the NEPM HILs, these values should be used where there has been adequate characterisation of a site (i.e. sufficient and appropriate sampling). The arithmetic mean can be compared to the values given in Table 5. The relevance of localised elevated values should be considered and should not be obscured by consideration only of the arithmetic mean of the results. The results must also meet the following criteria:
  - the standard deviation of the results must be less than 50% of the ILs; and
  - no single value exceeds 250% of the relevant IL;
- The ILs derived are intended to be used for the purpose of comparison with data collected from former clan lab sites. In particular the indoor air ILs are based on a total air concentration that may be measured (that may be from all sources, ambient and the former clan lab). If no air samples are collected then the application of the indoor air ILs to modelled or estimated concentrations needs to be carefully considered;
- ILs derived for soil are for outdoor areas only. If volatile chemicals are present in soil directly beneath an existing or proposed building then the potential for vapour intrusion (VI) needs to be addressed separately. This is because VI issues vary depending on the nature and depth of contamination, site (soil) characteristics and building characteristics;
- The ILs presented have not considered agricultural areas where crops may be produced (including home-grown fruit and vegetables), poultry kept (for production of eggs) or livestock. Where relevant, these issues need to be addressed in a site-specific assessment;
- The ILs presented have been rounded to 1 or 2 significant figures considered relevant for the level of uncertainty inherent in the approach adopted;
- The ILs presented should not be rigidly interpreted. Consideration of the land-use and contaminant distribution is important in interpreting the results for each site;
- Potential issues associated with the presence of mixtures (for example BTEX mixtures or methamphetamine/MDMA mixtures) should be considered in the application of the ILs at any one site; and
- The ILs presented are intended to be used for the purpose of investigation. Exceedance of the ILs does not imply that health effects will occur, rather that a more detailed, site-specific assessment may be required.
### Table 5 Summary of Investigation Levels (ILs) - Assessment of Former Clan Lab Sites

<table>
<thead>
<tr>
<th>Key Chemical</th>
<th>Residential (A)</th>
<th>Recreational (E)</th>
<th>Commercial/Industrial (F)</th>
<th>Environmental #</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Indoor Criteria</td>
<td>Outdoor</td>
<td>Indoor Criteria</td>
<td>Outdoors</td>
</tr>
<tr>
<td></td>
<td>Surface (µg/100cm²)</td>
<td>Air (mg/m³) Soil (mg/kg)</td>
<td>Surface (µg/100cm²)</td>
<td>Air (mg/m³) Soil (mg/kg)</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>0.5 b 5 5 10 b 45 x x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MDMA</td>
<td>7 b 60 60 130 b 600 x x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudo/Ephedrine</td>
<td>600 b 6000 6000 10000 b 50000 x x-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ammonia</td>
<td>a 0.1 1800 1800 a 0.3 10000 x 0.9AFM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iodine</td>
<td>20 0.0008 2 2 450 0.003 6 4^{I} x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bromide</td>
<td>2000 0.0008 2 2 50000 0.003 4 10^{I} x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.07 b 0.6 2 b 7 x</td>
<td></td>
<td></td>
<td>AFM^{I}</td>
</tr>
<tr>
<td>N-Methylformamide</td>
<td>10 b 120 270 b 1200 x x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methyamine</td>
<td>a 0.004 70 70 a 0.01 600 x x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitroethane</td>
<td>a 0.4 4400 4400 a 1 20000 x x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boron and compounds</td>
<td>1800 b 3000 (N) 6000 (N) 40000 b 150000 (N) 0.5^{II} 0.37^{AF}, 5.1^{AM}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mercury (inorganic)</td>
<td>35 b 15 (N) 30 (N) 800 b 75 (N) 1 (NE) 0.0006^{AF}, 0.004^{AM}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lithium</td>
<td>46 b 230 230 1000 b 5700 2^{II} 0.014^{I}</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Benzaldehyde</td>
<td>1500 0.4 6300 6300 35000 1 35000 0.6^{II} 0.01^{I}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phosphine</td>
<td>a 0.0004 c c a 0.001 c x x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Safrole and isosafrole</td>
<td>16 0.0002 1 1 16 0.001 6 0.4^{I} x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chloroform</td>
<td>a 0.1 240 240 a 0.4 1400 1.2^{II}, 170^{I} 0.37^{AFM}</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Dichloromethane</td>
<td>a 1 120 120 a 4 3300 4^{II}, 4^{I}^{AF}</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Benzene</td>
<td>a 0.0095 (A) 1 (S) 1 (S) a 0.0095 (A) 1 (S) 210^{I} 0.95^{II}, 0.5^{AM}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toluene</td>
<td>a 0.4 (A) 130 (S) 130 (S) a 0.4 (A) 130 (S) 1.4 (S) 0.1^{AF}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethylbenzene</td>
<td>a 26 50 (S) 50 (S) a 80 50 (S) 3.1 (S) 0.08^{II}, 0.005^{AM}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Xylenes</td>
<td>a 0.9 (A) 25 (S) 25 (S) a 0.9 (A) 25 (S) 14 (S) 0.2 to 0.35^{I}^{AF}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Naphthalene</td>
<td>a 0.003 150 150 a 0.01 400 56^{II} 0.016^{II}, 0.05^{AFM}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TPH</td>
<td>C6-C9 (aliphatic)^{I} 0.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C10-C14 0.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C15+ 0.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>6.5-8.5 b 6.5-8.5 6.5-8.5 b 6.5-8.5 x</td>
<td></td>
<td></td>
<td>(6 to 9) A^{**}</td>
</tr>
</tbody>
</table>
Notes for Table 5 (refer to Environmental Risk Sciences [2009] report for full detail on derivation):

a No surface residue IL has been derived for these key chemicals as they are considered volatile and would not be present as surface residues (or dust) for sufficient time to be of concern.

b No indoor air IL has been derived for these key chemicals. Only volatile chemicals (or gases) have been considered as they may continue to off-gas from porous surfaces over time.

c No soil IL has been derived for phosphine gas as it is not expected to be present in outdoor soil for sufficient time to be of concern.

d No Tier 1 or screening level guidelines are available for these chemicals from peer reviewed sources that are relevant to the protection of the terrestrial or aquatic environments.

x IL derived for TPH fractions C6-C9 are for the aliphatic fraction. This is calculated based on the total TPH C6-C9 reported minus total BTEX (the major contributors to aromatics).

A Monitoring Investigation Levels are available and presented for benzene, toluene and xylenes in air as per NEPM (2004), converted (at STP) from ppmv to mg/m^3. Values relevant to chronic exposures (annual averages) have been presented for use in preference to derived ILs. Derived ILs are noted in the calculations presented in Appendices C to E of the Environmental Risk Sciences (2009) report and can be referenced where relevant. ILs adopted should reflect any changes to NEPM guidelines.

N Health Investigation Levels are available from NEPM (1999b) for these chemicals in soil and are presented in this table, relevant to the land-use. Derived ILs are noted in the calculations presented in Appendices C to ED of the Environmental Risk Sciences (2009) report and can be referenced where relevant. ILs adopted should reflect any changes to NEPM guidelines.

NE Ecological Investigation Level available for mercury from NEPM (1999b) presented in this table. ILs adopted should reflect any changes to NEPM guidelines.

S No NEPM values are currently available, however ILs presented in NSW EPA Service Station Guidelines (1994) are commonly adopted as ILs in Australia. These are presented in the table. Exceptions are in Queensland where state specific values should be considered. Derived ILs are noted in the calculations presented in Appendices C to E of the Environmental Risk Sciences (2009) report and can be referenced where relevant. ILs adopted should reflect changes to state guidelines and/or release of relevant NEPM guidelines.

# Environmental Screening Guidelines available from a ranges of peer-reviewed sources. Qualifiers noted in table refer to source of guidelines available as follows:

A = available from ANZECC/ARMCANZ (2000) for:

F=freshwater

M=marine water

*Guideline for ammonia based on pH of 8, guideline must be adjusted for other pH levels

** Range of guidelines available for phosphorous and pH in water for a range of ecosystems in different areas of Australia, refer to guidance document for relevant values

U = available from USEPA Region 5 or 6

R = Guideline available from RIVM (2001)

O = Guideline available from OECD (2005)

4.5 Uncertainties and Considerations

As the ILs are relevant to any residential, commercial or recreational area, the methods and assumptions adopted are considered to be sufficiently conservative to ensure all uses are adequately addressed. There are, however a number of key areas where uncertainty is considered high. The ILs derived and presented in this report are considered to be a starting point and hence a number of the uncertainties require further consideration in the ongoing review and development of these guidelines. The key uncertainties/areas that require further consideration include the following:

- The identification of key chemicals that require consideration in the derivation of ILs is based on limited information. None of the available data is specifically relevant to the nature of contamination that remains on surface, in indoor air and outdoor soil at a former clan lab following seizure and removal of materials, wastes and products. It is recommended that some former clan labs be characterised on the basis of a wide range of chemicals (based on the relevant method). This data, along with data collected from other clan labs during investigation (and remediation) should be reviewed with the aim of better defining the key chemicals that require ILs. This may involve the inclusion or removal of some key chemicals addressed in this report.

- Some of the key chemicals identified are also associated with staining, such as iodine. It is likely that remediation methods adopted to address aesthetic issues associated with these contaminants may also adequately address human health (or environmental where relevant) issues. This needs to be confirmed once data is available from a number of former clan labs where these issues are present.

- The toxicological values adopted for MDMA have been considered to be associated with a low level of confidence. In addition effects associated with exposures by the general public to environmental levels of other key products such as L-PAC, P-2-P, MDP-2-P and MDA is not currently available. These chemicals may require further review and assessment should initial testing on former clan labs indicate that these may be present at significant concentrations prior to remediation.

- It is expected that initial sampling of some sites may identify other media of concern, such as indoor air associated with vapour intrusion issues, groundwater or surface water impacts. These need to be addressed on a site-specific basis as they have not been specifically addressed in the ILs derived and presented in this report.

- The potential for remediation methods to achieve levels below the ILs established needs to be considered and reviewed. Where the ILs cannot be met, then a site-specific risk assessment may be required.
References

US Clan Lab Guidelines:


Other References:


Journal of the Clandestine Laboratory Investigating Chemists Association (JCLICA), Volume 9, Number 4, October 1999.

Journal of the Clandestine Laboratory Investigating Chemists Association (JCLICA), Volume 16, Number 2, April 2006.

Journal of the Clandestine Laboratory Investigating Chemists Association (JCLICA), Volume 17, Number 1, January 2007.

Journal of the Clandestine Laboratory Investigating Chemists Association (JCLICA), Volume 18, Number 1, January 2008


OECD, 2005. Screening Information Data Set (SIDs), Benzaldehyde.


Appendix A – Summary of Chemicals Associated with Former Clan Labs
This appendix presents a summary of the potential compounds that may be derived from base products, reactants, products and by-products from former clan labs in Australia. In Australia the following methods have been identified (following seizure of clan labs as per information from ACC) as the most common methods that require consideration in the derivation of ILs:

- **MDMA from:**
  - MD-P-2-P, methylamine, aluminium and mercury (Shulgin Method);
  - MD-P-2-P, N-methylformamide and formic acid (Leuckart Method);
  - MD-P-2-P, methylamine and sodium cyano/borohydride; and
  - Safrole, hydrobromic acid and methylamine.

- **MD-P-2-P from sassafrass oil, potassium hydroxide, hydrogen peroxide and formic acid (Shulgin).**

- **Methamphetamine from:**
  - Pseudo/ephedrine, hypo/phosphorous acid and iodine;
  - Pseudo/ephedrine, phosphorous and iodine/hydroiodic acid;
  - Pseudo/ephedrine, lithium/sodium and ammonia (anhydrous);
  - P-2-P, methylamine, aluminium and mercury salt.

- **P-2-P from benzaldehyde, nitroethane and iron (or zinc);**

- **Pseudoephedrine from:**
  - pharmaceutical preparations;
  - benzaldehyde via L-PAC (L-phenylacetylcarbinol or 1-phenyl-1-hydroxy-2-propanone) and reductive animation by catalytic hydrogenation; and
  - benzaldehyde via L-PAC and reductive animation with sodium cyano/borohydride.
<table>
<thead>
<tr>
<th>Derivation of Risk-Based Investigation Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clandestine Drug Laboratory, Site Investigation Guidelines</td>
</tr>
<tr>
<td>Ref: ACC/09/R001-A</td>
</tr>
</tbody>
</table>

### Amphetamines and products, including intermediates

<table>
<thead>
<tr>
<th>Product/By-product</th>
<th>Amphetamine and salts</th>
<th>√</th>
<th>√</th>
<th>√</th>
</tr>
</thead>
<tbody>
<tr>
<td>Product/Reactant</td>
<td>Methamphetamine and salts</td>
<td>√</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Product/Reactant/By-product</td>
<td>MDMA and salts</td>
<td>√</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>By-product</td>
<td>N-acetylamphetamine and salts</td>
<td>√</td>
<td></td>
<td></td>
</tr>
<tr>
<td>By-product</td>
<td>N,N-diacetylamphetamine and salts</td>
<td>√</td>
<td></td>
<td></td>
</tr>
<tr>
<td>By-product</td>
<td>3,4-dimethoxymethamphetamine and salts</td>
<td>√</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>By-product</td>
<td>P-2-P</td>
<td>√</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>By-product</td>
<td>1-(3,4-methylenedioxyphenyl)-2-bromopropane</td>
<td>√</td>
<td></td>
<td></td>
</tr>
<tr>
<td>By-product</td>
<td>1-(3,4-methylenedioxyphenyl)propane</td>
<td>√</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>By-product</td>
<td>1-(3,4-methylenedioxyphenyl)-2-iminopropane</td>
<td>√</td>
<td></td>
<td></td>
</tr>
<tr>
<td>By-product</td>
<td>L-PAC = L-phenylacetylcarbinol</td>
<td>√</td>
<td>√</td>
<td></td>
</tr>
</tbody>
</table>

### Ephedrine and Pseudoephedrine and products/by-products

| Product/Reactant | Ephedrine and salts | √ | √ | √ |
| Product/Reactant | Pseudoephedrine and salts | √ | √ | √ |
| Product/Reactant | L-PAC = L-phenylacetylcarbinol | √ | √ |
| By-product       | Pseudoephedrine pharmaceutical ingredients | √ | √ | √ |
### Derivation of Risk-Based Investigation Levels

**Clandestine Drug Laboratory, Site Investigation Guidelines**

Ref: ACC/09/R001-A

<table>
<thead>
<tr>
<th>MDMA from:</th>
<th>Meth from:</th>
<th>Pseudoephedrine from:</th>
</tr>
</thead>
<tbody>
<tr>
<td>MD-P-2-P, methamphetamine, aluminum and mercury</td>
<td>MD-P-2-P, sodium pyrophosphate and sodium carbonate</td>
<td>MD-P-2-P, benzyl alcohol, sodium bicarbonate, iron (or zinc)</td>
</tr>
<tr>
<td>MD-P-2-P, N-methylformamide and formic acid</td>
<td>MD-P-2-P, sodium pyrophosphate and sodium carbonate</td>
<td>MD-P-2-P, benzyl alcohol, sodium bicarbonate, iron (or zinc)</td>
</tr>
<tr>
<td>Saffrole, hydrobromic acid and miltamylne</td>
<td>MD-P-2-P, sodium pyrophosphate and sodium carbonate</td>
<td>MD-P-2-P, benzyl alcohol, sodium bicarbonate, iron (or zinc)</td>
</tr>
</tbody>
</table>

#### By-products from Pseudoephedrine extraction

<table>
<thead>
<tr>
<th>By-product</th>
<th>By-product</th>
<th>By-product</th>
</tr>
</thead>
<tbody>
<tr>
<td>acelaminophen</td>
<td>4-aminophenol</td>
<td>brompheniramine</td>
</tr>
</tbody>
</table>
| 2-amino-2-
|aminopyridine | doxylamine | 1,2-dihydroxybenzene |
| N-cyclohexyl-N-methyl-2-
|aminophenyl)methylamine | desloratadine | 1,2-dihydroxy-3-isopropyl-benzene |
| dextromethorphan | dextromethorphan | 1,2-dihydroxy-4-isopropyl-benzene |
| diphenhydramine | diphenhydramine | 2-[(2-dimethylaminomethyl)-
| amino]pyridine | doxylamine | p-methylphenol |
| fexofenadine | loratadine | pyrilamine |
| guaiphenesin | loratadine | tripolidine |
| 2-hydroxyphenylglyceryl ether | loratadine | tripolidine |
| 2-(a-methylbenzyl]pyridine | loratadine | tripolidine |
| 1-(methylphenyl)-1-(2-pyridyl)-3-
|pyrrolidino-propane | Loratadine | tripolidine |
| p-methylphenol | Loratadine | tripolidine |
| pyrilamine | Loratadine | tripolidine |
| tripolidine | Loratadine | tripolidine |
### Base Products and Reactants

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</tr>
<tr>
<td>hydrochloric acid</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>sulfuric acid</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>sodium hydroxide (caustic soda, lye)</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>acetone</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>diethyl ether</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>isopropyl alcohol</td>
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</tr>
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<td>methanol</td>
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</tr>
<tr>
<td>methylated spirits</td>
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<td>shellite</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
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<tr>
<td>mineral turpentine</td>
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<td>xylene</td>
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<td>dichloromethane</td>
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<tr>
<td>ammonia</td>
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</tr>
<tr>
<td>bromine</td>
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</tr>
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</tr>
<tr>
<td>glucose/sucrose</td>
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<tr>
<td>hydriodic acid (liquid) or hydrogen iodide (gas)</td>
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<tr>
<td>hydrobromic acid</td>
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<tr>
<td>hydrogen gas</td>
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<tr>
<td>hydrogen peroxide</td>
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<td>hypophosphorous acid</td>
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</tr>
<tr>
<td>iodine (also as by-product)</td>
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</tr>
<tr>
<td>methylamine</td>
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</tr>
<tr>
<td>N-methylformamide</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>nitroethane</td>
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</tr>
<tr>
<td>phosphorus (red phosphorus)</td>
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</tr>
<tr>
<td>phosphorous acid</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
<tr>
<td>sodium cyano/borohydride</td>
<td>√ √ √ √ √ √ √ √ √ √</td>
</tr>
</tbody>
</table>

### MDMA from:
- MD-P-2-P, methylamine, aluminium and mercury
- MD-P-2-P, N-methylformamide and formic acid
- MD-P-2-P, methylamine, sodium cyanide and methylene
- Saffrole, hydrobromic acid and methylamine
- Pseudoephedrine, nitroethane, iron (or zinc)
- Pseudoephedrine, N-methylformamide and formic acid
- Pseudoephedrine, methylamine, aluminium and mercury salt
- Pharmaceutical preparations

### Meth from:
- MD-P-2-P, potassium hydroxide, hydrogen peroxide, formic acid (Shulgin)
- Pseudoephedrine, hypophosphorous acid and iodine
- Pseudoephedrine, lithium/sodium and ammonia (anhydrous)
- Pharmaceutical preparations benzaldehyde via L-PAC and reductive amination by catalytic hydrogenation
- Benzaldehyde via L-PAC and reductive amination with sodium cyano/borohydride

### Pseudoephedrine from:
- MD-P-2-P, potassium hydroxide, nitrogen, iron (or zinc)
- Pharmaceutical preparations

---

Derivation of Risk-Based Investigation Levels
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<table>
<thead>
<tr>
<th></th>
<th>MDMA from:</th>
<th>Meth from:</th>
<th>Pseudoephedrine from:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MD-P-2-P, methylene, aluminum and mercury</td>
<td>MD-P-2-P, N-methylformamide and formaldehyde</td>
<td>MD-P-2-P, methylene, potassium hydroxide, formaldehyde, and aluminum</td>
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<tr>
<td></td>
<td>MD-P-2-P, methylene, sodium and acetic acid</td>
<td>MD-P-2-P, methylene, sodium and acetic acid</td>
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</tr>
<tr>
<td></td>
<td>Sulfate, hydrobromic acid, and methylene</td>
<td>MD-P-2-P, methylene, sodium and acetic acid</td>
<td>MD-P-2-P, methylene, lithium, sodium, and ammonium</td>
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<td>MD-P-2-P, methylene, aluminum and mercury</td>
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<td>Meth from:</td>
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<td>MD-P-2-P, methylene, sodium and acetic acid</td>
<td>MD-P-2-P, methylene, sodium and acetic acid</td>
<td>MD-P-2-P, methylene, sodium and acetic acid</td>
</tr>
<tr>
<td></td>
<td>Sulfate, hydrobromic acid, and methylene</td>
<td>MD-P-2-P, methylene, sodium and acetic acid</td>
<td>MD-P-2-P, methylene, lithium, sodium, and ammonium</td>
</tr>
</tbody>
</table>

### Metals

| Reactants | aluminium | √ | | | | |
| Reactants | iron | | | | | |
| Reactants | lithium | | | | | |
| Reactant/By-product | mercury salts (mercuric chloride) | | | | | |
| Reactants | metal catalyst (nickel, palladium, platinum) | | | | | |
| Reactants | sodium (metal) | | | | | |
| Reactants | zinc | | | | | |
| By-product | aluminium cation | | | | | |
| By-product | barium cation | | | | | |
| By-product | iron cation | | | | | |
| By-product | lithium cation | | | | | |
| By-product | nickel cation | | | | | |
| By-product | palladium cation | | | | | |
| By-product | platinum cation | | | | | |
| By-product | sodium cation | | | | | |
| By-product | zinc cation | | | | | |

### Other Reactants and By-products

| Reactant/By-product | piperonal | | | | | |
| By-product | pyruvic acid | | | | | |
| By-product | acetaldehyde | | | | | |
| By-product | benzaldehyde (also a reactant) | | | | | |
| By-product | benzoic acid and salts | | | | | |
| By-product | benzyl alcohol | | | | | |
| By-product | 1,3-benzodioxole | | | | | |
| By-product | 1-benzyl-3-methylnaphthalene | | | | | |
| By-product | 1-(1,4-cyclohexadienyl)-2-methylaminopropane (CMP) and salts | | | | | |
| By-product | 1-dimethylamino-2-(p-methoxybenzylamino)-ethane | | | | | |
| By-product | 1-dimethylamino-2-(p-methoxyhydroxethylidene)-methyaminopropane (CMP) and salts | | | | | |
| By-product | 1,3-dimethyl-2-phenylpentane | | | | | |
| By-product | glutaraldehyde | | | | | |
| By-product | 1-methoxy-1-(3,4-methylenedioxyphenyl)-2-methylaminopropane and salts | | | | | |
| By-product | 1-methyaminol-1-(3,4-methylenedioxyphenyl)-propane and salts | | | | | |
| By-product | 1-(3,4-methylenedioxy-phenyl)-2-methylaminopropane | | | | | |
| By-product | di-(1-(3,4-methylenedioxy-phenyl)isoamylamine | | | | | |

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| By-product/Reactant | di-[1-(3,4-methylenedioxy-phenyl)-isopropyl]methylamine | √ | √ | √ | √ |
| By-product | N-methyl-piperonilamine and salts | ✓ | ✓ |
| By-product/Reactant | 1-phenyl-2-nitropropane | ✓ |
| By-product/Reactant | 1-phenyl-1,2-propanediol | ✓ | ✓ |
| By-product | phosphine | ✓ | ✓ |
| By-product | piperonyl alcohol | ✓ | ✓ |
| By-product | piperonyl chloride | ✓ | ✓ |
| By-product | pyridine | ✓ |
| By-product/Reactant | isosafrole | ✓ | ✓ | ✓ | ✓ | ✓ |
| By-product/Reactant | safrole | ✓ | ✓ | ✓ | ✓ |
| Reactant | sassafras oil (safrole) and associated containants | ✓ | ✓ |
| By-product/Reactant | methylammonium cation | ✓ | ✓ |
| By-product | acetate anion | ✓ | ✓ |
| By-product | borate anion | ✓ | ✓ |
| By-product | bromide anion | ✓ | ✓ |
| By-product | chloride anion | ✓ | ✓ |
| By-product | cyanide anion | ✓ | ✓ |
| By-product | formate anion | ✓ | ✓ |
| By-product | hypophosphite anion | ✓ | ✓ |
| By-product | iodide anion | ✓ | ✓ |
| By-product | nitrate anion | ✓ | ✓ |
| By-product | phosphate anion | ✓ | ✓ |
| By-product | phosphate anion | ✓ | ✓ |
| By-product | pyruvate anion | ✓ | ✓ |
| By-product | sulfate anion | ✓ | ✓ |

Note: Green highlighted row = key chemical identified for derivation of IL based on how common, significance and potential presence at labs following seizure and toxicity.
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B1 Introduction
This appendix presents a summary of the key aspects associated with the chemicals identified for the purpose of establishing guidelines relevant to the assessment and remediation of former clan labs. The summary provided is based on available information from published sources and databases and include information on:

- Common uses for the chemical, nature, appearance, key chemical/physical parameters and odour/taste (where relevant).
- Health effects, including the potential carcinogenic and genotoxicity of the chemical and potential for dermal absorption relevant to the assessment of exposure.
- Fate and transport in the environment including the potential to be present indoors on surfaces or in air as a volatile, fate in the atmosphere, soil and water environment. The potential for degradation and bioaccumulation is also presented.
- Quantitative data used to assess the toxicity of the chemical with respect to oral, inhalation and dermal exposures. The data presented are derived from published, peer-reviewed sources consistent with the approach outlines and presented in guidance from enHealth (2002) and NEPM (1999a), unless noted otherwise. The following is noted with respect to the identification of toxicity data:
  - Toxicity data have been selected to be representative of long-term (chronic) exposures by all members of the public (including sensitive subgroups);
  - Where quantitative data is not available for inhalation exposures, available oral data have been assumed to be relevant for the assessment of inhalation exposures. The oral data has been converted from an intake in mg/kg/day to an air concentration in mg/m³ on the basis of a 70 kg body weight and daily inhalation rate of 20 m³/day. This conversion is consistent with guidance from USEPA (2009a); and
  - Where quantitative data is not available for dermal exposures, available oral data has been assumed to be relevant for the assessment of dermal intakes.
- Environmental data available from published peer-reviewed sources that are relevant to the screening level assessment of potential environmental issues. These data have been presented for aquatic and terrestrial environments (where available).

The scope of the assessment presented in this report does not include a detailed review of toxicological effects, and derivation of quantitative toxicological data associated with the chemicals identified. It is noted that the assessment of potential health effects associated with MDMA requires some interpretation of available data for the purpose of identifying a relevant quantitative dose-response value for the purpose of deriving investigation guidelines. The review presented is not a complete literature review or summary of all available studies. The approach adopted is presented in this appendix.
B2 Methamphetamine

B2.1 General
Clandestine laboratories produce two chemical forms of methamphetamine, the free base ("methamphetamine base") and the hydrochloride salt ("methamphetamine hydrochloride"). The free base, which is the initial product of a clandestine synthesis, is a liquid at room temperature. The hydrochloride salt is produced from the free base by bubbling hydrogen chloride gas through a solution of the free base.

There are two isomeric forms of methamphetamine, called \(d\)-methamphetamine and \(l\)-methamphetamine. The \(d\)-isomer is a potent central nervous system (CNS) stimulant, and virtually all of the methamphetamine produced by clandestine laboratories is the \(d\)-isomer. The other isomer, the \(l\)-isomer has little CNS activity and is used in pharmaceutical preparations for the temporary relief of nasal congestion.

Appearance: Methamphetamine hydrochloride is usually found as a yellow or white crystalline powder. However, "street" grade methamphetamine may be found in a variety of colours depending on how it was manufactured and what impurities are present in the final product. Methamphetamine hydrochloride may also be found as "ice," a large, usually clear crystal of high purity. Methamphetamine base is a yellow to brown liquid which is soluble in organic solvents.

Chemical/Physical Properties: d-Methamphetamine (CASRN: 537-46-2) has the following properties (HSDB):

- Molecular formula: \(\text{C}_{10}\text{H}_{15}\text{N}\) (\(\text{C}_{10}\text{H}_{16}\text{ClN}\) methamphetamine hydrochloride)
- Molecular weight: 149.24 (185.69 methamphetamine hydrochloride)
- Log Kow: 2.07
- Solubility: 0.5 g/ml water

Odour and Taste: Methamphetamine hydrochloride is odourless, however it has a bitter taste. Methamphetamine base has a sharp biting odour resembling geranium leaves. No odour threshold is available.

B2.2 Exposure, Fate and Transport

Indoor Surfaces: Methamphetamine base is volatile and would not be expected to persist on indoor surfaces.

Methamphetamine hydrochloride is a salt which is not volatile and may persist on surfaces and hence a number of US agencies have developed clean-up standards for methamphetamine contamination on indoor surfaces. Studies have shown that methamphetamine residues may persist for at least several months particularly in the absence of human activity or remediation. However, if the hydrochloride salt comes into contact with moisture and the pH is greater than 4, the free base would be regenerated and the drug would once again have a tendency to volatilise. pH-dependent regeneration of the free base may be particularly important in understanding the success (or lack thereof) of using detergents to clean methamphetamine-contaminated surfaces, or using water-based latex paint to encapsulate the contamination, since cleaning detergents and latex paints are both alkaline.

The size distribution of airborne methamphetamine particles identified during and immediately following a controlled cook is consistent with a condensation aerosol and it has been proposed that methamphetamine is initially released as a vapour during the "salting out" process. This occurs when methamphetamine base is precipitated out of solution by bubbling hydrogen chloride gas through it to produce methamphetamine hydrochloride. Once released, the vapour condenses into very small particles that stay suspended in air and
are able to migrate to all portions of a residence. This is consistent with wipe sample results from controlled cooks where residues are found throughout a premises, away from the synthesis area.

From the perspective of a potential hazard to human health, once these small particles are inhaled, they will penetrate into the deep pulmonary portion of the lung and be absorbed quickly into the bloodstream. While this is important in the days immediately following the manufacturing of the drug, with respect to long-term exposures following seizure and initial removal of drug manufacturing equipment, the potential for re-suspension of aerosols is important. If surfaces are cleaned, the potential for re-suspension, and hence inhalation exposures, is limited.

**Environment:** Limited data is available on the fate and transport of methamphetamine in the air, soil or water environment. Limited, steady degradation of methamphetamine was identified in non-sterile and sterile soil conditions in studies undertaken by Pal and Kirkbride (2009), where degradation rates of 0.0006 to 0.0023 per day were derived. Methamphetamine was considered to be persistent in soil when compared with MDMA and pseudoephedrine over a one year incubation period. Koc values derived for methamphetamine for the soil types assessed range from approximately 100 to 600 ml/g. Methamphetamine is expected to be less mobile in soil compared with pseudoephedrine based on the desorption study undertaken Pal and Kirkbride (2009).

**B2.3 Ecological Effects and Guidelines**

Limited information is available on the ecotoxicity of methamphetamine. Methamphetamine was included as one of the chemicals evaluated with respect to fate and transport and ecotoxicity in soil by Pal and Kirkbride (2009). The EC50 (defined as a 50% reduction in dehydrogenase activity in spiked soil relative to unspiked soil) for the soil materials tested with methamphetamine where close to or greater than 1 g/kg. While the study was limited to 3 soil types it suggested that the potential for disruption of normal soil microbial function is only likely to occur at high concentrations. These are likely to be higher than the guidelines that are derived to be protective of human health issues.

**B2.4 Health Effects**

**General:** Methamphetamine hydrochloride can be inhaled (smoked), snorted, injected, or ingested. The route of exposure primarily affects the rate of absorption and onset of effects, with injection and inhalation producing the most rapid onset, and ingestion resulting in delayed onset. In general, once methamphetamine is absorbed, the biological effects are the same regardless of the route of exposure. Amphetamines are concentrated in the kidney, lungs, cerebrospinal fluid and brain. They are highly lipid soluble and readily cross the blood-brain barrier. Under normal conditions, about 30% of amphetamine is excreted unchanged in the urine but this excretion is highly variable and is dependent on urinary pH. The biologic half-life of orally administered methamphetamine is approximately four to five hours.

Effects of methamphetamine exposure vary widely and depend on a number of individual factors with adults and children showing different responses to the drug. Repeated exposure may lead to the development of tolerance to one of more of the effects, and symptoms resulting from withdrawal different from those associated with methamphetamine exposure.

**Acute Effects:** Methamphetamine is irritating to skin, eyes, mucous membranes, and the upper respiratory tract. Eye contact may cause pupil dilation and retraction of the upper lid. Acute intoxication can cause dizziness, headache, dry mouth, a metallic taste, anorexia, insomnia, tremor, rash, chest pain, difficulty breathing, fainting, blurred vision, dilated pupils, impotence, bluish skin colour, lung congestion, convulsions, and coma. Overdose may cause exaggeration of reflexes, rapid breathing, confusion, panic states, aggressiveness, hallucinations, brain oxygen loss, elevated body temperature, skeletal muscle wasting, fatigue, depression, acute paranoia, and a schizophrenic-like state. Other effects include nausea, vomiting, diarrhoea, cramps, irregular heartbeat, high or low blood pressure, and circulatory collapse.
Chronic Effects: Long-term exposure may cause severe skin conditions, insomnia, irritability, poor concentration, hyperactivity, personality changes, weight loss, teeth grinding and tooth loss, ulcers of the lips and tongue, physical and psychological dependence, anxiety, fear, compulsive behaviour, delirium, disorientation, hallucinations, or a psychotic schizophrenic-like condition with possible self-injury. Prolonged use results in tolerance and psychological and physical dependence. Withdrawal can occur from abrupt cessation of high doses resulting in mental depression, fatigue, vomiting, and nausea.

Carcinogenicity: No human data available. The available studies on animals show no evidence of carcinogenic activity for d- or l-amphetamines (NTP, 2005).

Genotoxicity: The available studies show negative results in mutagenic testing (NTP, 2005).

Susceptible Populations: There are some differences in d- and l- pharmacokinetic parameters in adults and children (NTP, 2005). At the equivalent doses, the half life is shorter but systemic bioavailability is greater in children compared with adults (even when body weight differences are accounted for).

Dermal Absorption: In the course of an in vitro absorption study of methamphetamine hydrochloride across human skin it was suspected that the hydrochloride salt may be unstable at neutral to alkaline pH. The studies undertaken confirmed that skin pH is a critical factor affecting the rate and magnitude of dermal absorption. Dermal absorption of methamphetamine has been assessed as 57% (OEHHA, 2009a).

B2.5 Quantitative Toxicity Data

A number of studies are available regarding the toxicity of methamphetamine. The most recent and comprehensive review by OEHHA (2009b) provided a peer-reviewed threshold dose for the quantification of adverse health effects associated with exposures by all members of the public. The review undertaken considered any effect induced by methamphetamine as an adverse effect and potentially a critical effect for the purpose of deriving a threshold value.

The OEHHA (2009b) review identified a sub-chronic RfD of 0.3 μg/kg-day. The toxicity endpoint for the RfD was appetite suppression and reduction in body weight gain which were identified as well characterised, centrally mediated indicators of methamphetamine’s pharmacological activity. The primary study was a three-dose, placebo-controlled, double blind investigation of weight gain during pregnancy involving a total of 84 women. The mean duration of dosing was 15-17 weeks, although one quarter of the women in the three methamphetamine dose groups received the drug for 20-21 weeks. An aggregate uncertainty factor of 300 was used in combination with a Lowest Observed Adverse Effect Level (LOAEL) of 5 mg/day (0.08 mg/kg-day) to calculate the RfD (0.3 μg/kg-day). The RfD was based on the most sensitive indicator of toxicity is consistent with the methodology developed by U.S. EPA, as any other manifestations of methamphetamine toxicity would occur at higher doses.

The sub-chronic RfD is adopted by OEHHA as relevant for the assessment of potential exposures to methamphetamine residues in a residence (or other premises) which will decrease over time and where peak exposures occur during early childhood (12 to 18 months). It was considered (by OEHHA) unnecessary to include any additional factors to extrapolate from sub-chronic to chronic exposures.

It should be noted that additive toxic effects have been observed in studies in rats with respect to exposures to both methamphetamine and MDMA (Clemens et al, 2005).

No background intakes of methamphetamine are expected for the general population. Hence background intakes have been assumed to be essentially zero.
B3 MDMA

B3.1 General

3,4-Methylenedioxymethamphetamine (MDMA) also known as “ecstasy”, a derivative of methamphetamine (METH) both of which are substituted amphetamines with potent central nervous stimulant effects. Acutely, METH and MDMA have numerous peripheral and central effects, leading to psychomotor activation, euphoria, decreased appetite, and hyperthermia. Because of their strong euphoric properties, METH and MDMA have a high abuse liability, and chronic use of either one can lead to psychotic and violent behaviours.

MDMA belongs to the family of phenethylamine drugs and shares a chemical similarity to the stimulant amphetamine and the hallucinogen mescaline. Also within the phenethylamine group, and chemically similar, are 4-bromo-2,5-dimethoxyamphetamine [DOB], 2,5-dimethoxy-4-methylamphetamine [DOM], 3,4-methylenedioxymethylamphetamine [MDEA], 3,4-methylenedioxyamphetamine [MDA], mescaline, paramethoxyamphetamine [PMA] and trimethoxyamphetamine [TMA] (ACC 2003).

Other than its manufacture and use as an illicit drug, MDMA does not have any other uses (HSDB, 2009). Hence a great deal of the data and studies on MDMA are focused on key effects associated with its use as an illicit drug rather than addressing potential health effects associated with occupational or environmental exposures (by all members of the general public). This limits the available data for the purpose of quantifying potential toxicity effects relevant to environmental exposures. The following provides a summary of the available information on MDMA. It is outside the scope of this assessment to undertake a detailed toxicological review and assessment of the available studies hence the assessment undertaken has a number of limitations. It is expected that the toxicity data adopted for the assessment of MDMA would be reviewed and revised as new data and information (that is relevant) is available.

Appearance: MDMA is listed in HSDB as an oil, however it is produced (as hydrochloride salt) in clan labs as a powder, pressed into pills or as a geltab.

Chemical/Physical Properties: MDMA (CASRN: 42542-10-9) has the following properties (HSDB):

- Molecular formula: \( \text{C}_{11}\text{H}_{15}\text{NO}_2 \)
- Molecular weight: 193.24
- Log Kow: 2.28
- Solubility in water: 5400 mg/L at 25°C
- Vapour pressure: 0.0016 mmHg at 25°C
- Henry’s Law constant: 2.75x10⁻⁹ atm.m³/mol at 25°C

Odour and Taste: MDMA has a musty smell with a searing bitter taste. MDMA as a powder has a distinctive liquorice scent (NZ NDP, 2004).

B3.2 Exposure, Fate and Transport

Indoor Surfaces: MDMA hydrochloride is a salt, similar to methamphetamine salt, which is not volatile and may persist on surfaces for a significant period of time. Hence the derivation of a standard for indoor surface is considered relevant.

Environment: Limited data is available on the fate and transport of MDMA in the air, soil or water environment. Degradation of MDMA was identified in non-sterile soil conditions in studies undertaken by Pal and Kirkbride (2009), where degradation rates of 0.005 to 0.019 per day were derived. Degradation rates were consistently lower for studies undertaken in sterile soils. MDMA was considered to be less persistent in soil when compared with methamphetamine and other compounds over a one year incubation period. Koc values derived for MDMA for the soil types assessed range from approximately 100 to 600 ml/g. MDMA is
expected to be less mobile in soil compared with pseudoephedrine based on the desorption study undertaken Pal and Kirkbride (2009).

B3.3 Ecological Effects and Guidelines

Limited information is available on the ecotoxicity of MDMA. MDMA was included as one of the chemicals evaluated with respect to fate and transport and ecotoxicity in soil by Pal and Kirkbride (2009). The EC50 (defined as a 50% reduction in dehydrogenase activity in spiked soil relative to unspiked soil) for the soil materials tested with MDMA where close to or greater than 1 g/kg. While the study was limited to 3 soil types it suggested that the potential for disruption of normal soil microbial function is only likely to occur at high concentrations. These are likely to be higher than the guidelines that are derived to be protective of human health issues.

B3.4 Health Effects

The following has been summarised from WHO (2001), Green et al (2003), Maxwell (2006) and Quinton and Yamamoto (2006).

**General:** Common adverse physical effects of MDMA include agitation, anxiety, tachycardia and hypertension; more serious adverse reactions include hyperthermia, rhabdomyolysis, disseminated intravascular coagulation, renal failure, cardiac complications, intracranial haemorrhage, and hepatotoxicity. Another term used to describe these adverse effects is “serotonin syndrome,” which is characterised by enhanced physical activity, sweating, lack of coordination, mental confusion, trismus, jaw clenching, agitation, hyperreflexia, hyperthermia, shivering, rhabdomyolysis, metabolic acidosis, myoclonus, tremor, and nystagmus.

Long-term neurotoxic effects of MDMA, particularly in the serotonergic system, are not fully known however animal studies suggest the potential for significant long-term harm associated with neurotoxicity (WHO 2001). Studies have suggested use of MDMA affects depression, other mood disorders, impulsiveness or hostility, psychotic symptoms, anxiety and panic disorders, and other psychopathologic disturbances. The selective impairments of neuropsychological performance associated with regular ecstasy use have been found not to be reversed by prolonged abstinence, which is consistent with evidence that ecstasy has potent and selective neurotoxic effects on brain serotonergic systems in humans (Maxwell, 2006).

MDMA has a high affinity for serotonin receptors and transport sites in the brain. Serotonin-producing neurones in the brain regulate aggression, mood, sexual activity, sleep and sensation to pain. Serotonin is also important in memory and temperature regulation. Initially MDMA emphasises extracellular levels of serotonin, however ultimately levels are decreased. MDMA exposure also leads to increased levels of dopamine.

The neurochemical and degenerative effects of MDMA on animals were found to be dose-related (WHO 2001), however the mechanisms involve in producing these effects are poorly understood (Quinton and Yamamoto 2006). Most of the animal research has concentrated on the neurotoxicity of MDMA. Of main concern, and considered to be the most sensitive end-point, are the long term effects of MDMA on the serotonergic system. It is these effects that have been most widely studied with limited quantitative data available.

MDMA (like all amphetamines) is well absorbed from the gastrointestinal tract where it is rapidly distributed through the body, including the brain.

**Carcinogenicity:** No human or animal data are available for MDMA. With respect to amphetamines in general, available studies on animals show no evidence of carcinogenic activity (NTP, 2005).
**Genotoxicity:** No data are available for MDMA, however for amphetamines in general, the available studies show negative results in mutagenic testing (NTP, 2005).

**Dermal Absorption:** No specific data is available on dermal absorption of MDMA, hence the value adopted for the purpose of assessing methamphetamine, 57% has been adopted for the assessment of MDMA.

### B3.5 Quantitative Toxicity Data

No detailed review and quantitative assessment of MDMA is available. The WHO (2001) assessment presented a range of data available, but did not identify quantitative values protective of chronic exposures by the general public. The neurotoxicity of MDMA in humans has not been well established; therefore, based on human data, a level of exposure at which MDMA becomes neurotoxic cannot be determined. However, the phenomenon of neurotoxicity has been investigated extensively in animals, and appears to be dose-related. Van Aerts (1998) provides a general summary of the dose response relationship associated with MDMA in humans based on animal studies, as follows:

<table>
<thead>
<tr>
<th>Dose (human) (mg/kg/day)</th>
<th>Effect</th>
<th>Neurotoxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.4</td>
<td>No observable effect</td>
<td>No</td>
</tr>
<tr>
<td>1.0</td>
<td>Some changes of serotonin</td>
<td>No, probably neuromodulatory effects, neuronal markers, among amongst which up-regulation of which increases in serotonin serotonin system components</td>
</tr>
<tr>
<td>2.0</td>
<td>More long term changes in serotonin neuronal markers</td>
<td>Disputed. - Either neuromodulatory effects, predominantly down-regulation of serotonin system components, or, as some claim, the observed reductions reflect nerve terminal degeneration</td>
</tr>
<tr>
<td>4.0</td>
<td>Disappearance of serotonergic fibres and swollen and fragmented axons</td>
<td>Yes, nerve terminal degeneration</td>
</tr>
<tr>
<td>10.0</td>
<td>Disappearance of serotonergic fibres and swollen and fragmented axons</td>
<td>Yes, axonal degeneration</td>
</tr>
</tbody>
</table>

from: van Aerts (1998)

A large number of studies have been undertaken on MDMA with respect to neurotoxicity and behavioural endpoints, as summarised in literature reviews completed by Baggott and Jerome (2001), Jerome and Baggott (2003) and Jerome (2004, 2005, 2007 and 2008). Few of the studies have been undertaken at low doses with the aim of identifying a threshold (NOAEL or LOAEL) that can then be used to establish a toxicity value for use in the assessment of environmental exposures by the general public. A comprehensive review of these studies has not been undertaken in this report (not part of the scope of this assessment), however the following provides a summary of the key reviews available.

- A few reviews have been undertaken where threshold values have been identified and discussed. These reviews are not comprehensive reviews of all available studies and effects; however they do provide an indication of a potential threshold that may be relevant to neurotoxicity effects from MDMA exposure.
- Gaylor and Skinner (1990) conducted a limited review of MDMA with respect to neurochemical, neurohistological and behavioural endpoints. The review identified either NOAEL or LOAEL’s from the available studies and considered the application of adopted default uncertainty factors of 100 (where a NOAEL was available) and 1000 (where only a LOAEL was available). Different uncertainty factors may be relevant depending on the studies undertaken (not reviewed). This review identified threshold values for these endpoints that ranged from 0.005 to 0.05 mg/kg/day.
- Van Aerts (1998) identified studies where a LOAEL or NOAEL has been established and converted these to human equivalent doses. A four month oral study on squirrel monkeys and a one to two week study in rats identified a NOAEL that ranged from 0.4 to 1.0 mg/kg. Other observations from available studies are summarised in the dose-response table presented above.
Van Aerts et al (2000) conducted a limited review of MDMA and MBDA (N-methyl-1-(1,3-benzodioxol-5-y1)-2-aminobutane) where neurotoxicity (including serotonin neuronal markers were also assessed) was the key endpoint. Based on this review a NOAEL of 0.34 to 0.5 mg/kg was identified based on single dose or short duration multiple dose studies. A NOAEL of 0.4 mg/kg was estimated to be a reasonable estimate for short duration exposures. If an uncertainty factor of 100 to 1000 (assumed) were applied to the NOAEL the threshold value for this endpoint would be in the order of 0.004 to 0.0004 mg/kg/day respectively.

A study by Armstrong and Noguchi (2004) found that, with respect to neurotoxic effects in rats, methamphetamine was more toxic than MDMA. While this is a limited study it suggests that the use of the lowest threshold value which is similar to the RfD for methamphetamine may be overly conservative. On this basis a threshold value of 0.004 to 0.005 mg/kg/day is considered reasonable. For the purpose of this assessment the threshold value of 0.004 mg/kg/day has been adopted.

The level of confidence in this value is considered to be low and it should be considered guide only. The toxicity data should be further reviewed in detail, particularly if MDMA is found to be widely distributed and persistent in former clan labs.

It should be noted that additive toxic effects have been observed in studies in rats with respect to exposures to both methamphetamine and MDMA (Clemens et al, 2005) suggesting a similar mechanism for neurotoxicity effects.

Background intakes of MDMA are expected to be associated with illicit use of the drug. Hence with respect to the assessment of environmental exposures no background intake of MDMA has been considered.
B4 Ephedrine and Pseudoephedrine
The following is summarised from OEHHA (2003c), CANTOX (2000), IPCS (2000) and HSDB (2009).

B4.1 General
Ephedrine and pseudoephedrine are precursors in the synthesis of methamphetamine. In addition both are ephedrine alkaloids which also include N-methylephedrine, N-methylpseudoephedrine, norpseudoephedrine and norephedrine (phenylpropanolamine). Ephedrine alkaloids can be prepared synthetically or derived from Ephedra species (plant genus with only a few Ephedra species containing ephedrine alkaloids). In general, all the ephedrine alkaloids show significant differences between diastereomers (e.g., ephedrine and pseudoephedrine) with regard to pharmacokinetic and pharmacodynamic effects. All have effects on the cardiovascular and respiratory system, but not to the same degree.

Appearance: Ephedrine appears as a waxy solid or as white to colourless granules, powder, or crystals. Pseudoephedrine is a white powder. The powders may become yellow upon standing. Both are non-volatile solids at room temperature.

For pharmaceutical uses, ephedrine and pseudoephedrine are often marketed as white, red, or blue tablets. In clan methamphetamine labs, the tablets may be ground up and partially dissolved in water or alcohol with the sludge filtered with makeshift devices such as coffee filters.

Chemical/Physical Properties: Ephedrine (CASRN: 299-42-3) and pseudoephedrine (CASRN: 90-82-4) have the following properties (HSDB):

- Molecular formula: C₁₀H₁₅NO (ephedrine and pseudoephedrine)
- Molecular weight: 165.23 (ephedrine and pseudoephedrine)
- Log Kow: 1.13 (ephedrine), 0.89 (pseudoephedrine)
- Koc: 73 L/kg (pseudoephedrine)
- Solubility: 63,600 mg/l water at 30°C (ephedrine), 106,000 mg/L at 25°C (pseudoephedrine)
- Vapour pressure: 0.00083 mmHg at 25°C (ephedrine and pseudoephedrine)
- Henry’s Law: 8.65x10⁻¹¹ atm.m³/mol at 25°C (ephedrine and pseudoephedrine)

Odour and Taste: Ephedrine and pseudoephedrine are essentially odourless or have a slight aromatic (or musty) odour. They have a bitter taste.

B4.2 Exposure, Fate and Transport
Indoor Surfaces: Ephedrine and pseudoephedrine are likely to be preset as non-volatile salts which are relatively stable under normal indoor environmental conditions and hence may persist in surface residues indoors.

Air: If present as a vapour (not expected from former clan labs), ephedrine and pseudoephedrine will be rapidly degraded with a half-life of approximately 4 hours. Particulate ephedrine will undergo gradual decomposition with exposure to light. Pseudoephedrine is not expected to be susceptible to direct photolysis by sunlight.

Soil: Ephedrine will primarily exist in the cation form which more strongly adsorb to soil containing organic carbon and clay. Volatilisation from soil is not significant. It is considered readily biodegradable.

Pseudoephedrine is expected to be highly mobile based on a Koc of 73 L/kg (HSDB, 2009), also noted as potentially significant by Pal and Kirkbride (2009), however it is likely that pseudoephedrine will exist in the environment in the cation form which more strongly adsorb to soil containing organic carbon and clay. Degradation of pseudoephedrine was identified in non-sterile soil conditions in studies undertaken by Pal and Kirkbride (2009), where degradation rates of 0.01 to 0.09 per day was derived. Degradation rates were
significantly less for studies undertaken in sterile soils. Pseudoephedrine was considered to be less persistent in soil when compared with methamphetamine and other compounds over a one year incubation period. Koc derived for pseudoephedrine for the soil types assessed range from approximately 50 to 250 ml/g.

**Water:** Ephedrine and pseudoephedrine cations may adsorb to suspended particulates and sediments. Volatilisation from surface water is not significant.

**Bioaccumulation:** An estimated BCF of 0.3 for ephedrine and 3 for pseudoephedrine suggests the potential for bioaccumulation in aquatic species in low.

### B4.3 Ecological Effects and Guidelines

Limited information is available on the ecotoxicity of ephedrine and pseudoephedrine. Some information is available on these compounds with respect to pharmaceuticals (in general) in the environment, from many sources. In general, pharmaceuticals have the potential for ubiquitous direct release into the environment worldwide (where humans live or visit). Other possible sources include disposal of unwanted illicit drugs and synthesis by-products into domestic sewage systems by clandestine drug operations; disposal of raw products and intermediates (e.g., ephedrine) via toilets is not uncommon in illegal laboratories.

Although most pharmaceuticals are designed to target specific metabolic pathways in humans and domestic animals, they can have numerous often unknown effects on metabolic systems of non-target organisms, especially invertebrates. Although many non-target organisms share certain receptors with humans, effects on non-target organisms are usually unknown.

A predicted no effect concentration (PNEC) based on aquatic toxicity has been estimated for ephedrine (based on data from ephedra herbal products). The estimated PNEC for ephedrine is 0.0198 mg/L (DEFRA, 2004). No data is available for pseudoephedrine.

Ephedrine and pseudoephedrine were included as chemicals evaluated with respect to fate and transport and ecotoxicity in soil by Pal and Kirkbride (2009). The EC50 (defined as a 50% reduction in dehydrogenase activity in spiked soil relative to unspiked soil) for the soil materials tested with pseudo/ephedrine where close to or greater than 1 g/kg. While the study was limited to 3 soil types it suggested that the potential for disruption of normal soil microbial function is only likely to occur at high concentrations. These are likely to be higher than the guidelines that are derived to be protective of human health issues.

### B4.4 Health Effects

**General:** In general, the pharmacokinetics of pseudoephedrine is similar to ephedrine absorption of oral ephedrine. After oral administration, ephedrine is rapidly and completely absorbed from the gastrointestinal tract with adsorption complete within 2 to 2.5 hours. The major route of elimination for ephedrine in humans is urinary excretion. Up to 95% of an oral dose may be excreted in the urine within 24 hours. Ephedrine is rapidly and extensively distributed throughout the body, with distribution to the liver, lungs, kidneys, spleen, and brain. Ephedrine is highly lipophilic and crosses the blood-brain barrier. Ephedrine is also excreted in breast milk. Excretion patterns may be much more rapid in children. Ephedrine has a plasma half-life ranging from 3 to 6 hours depending on urinary pH.

Ephedrine is a CNS stimulant that may produce nervousness, anxiety, apprehension, fear, tension, agitation, excitement, restlessness, weakness, irritability, talkativeness, or insomnia. Large doses of ephedrine may result in dizziness, light-headedness, tremor, hyperactive reflexes, hypertension (high blood pressure), and vertigo. Large (routes other than oral) doses of ephedrine may cause confusion, delirium, hallucinations, or euphoria. In addition, paranoid psychoses and visual auditory hallucinations may occur at extremely high doses. Ephedrine may also cause the following: throbbing headache; respiratory difficulty; fever or a feeling
of warmth; paleness; dryness of the nose and throat; chest pain; sweating; mild abdominal discomfort; vomiting; palpitation; tachycardia (rapid heartbeat); potentially fatal arrhythmias (alteration in heartbeat), including ventricular fibrillation; acute urinary retention or difficulty in urination; hypertension (high blood pressure), which may result in intracranial haemorrhage; nausea; and loss of appetite. Ephedrine has been known to cause allergic sensitisation. Ephedrine hydrochloride may have a local anaesthetic effect on exposed skin, may cause skin, eye, mucous membrane, upper respiratory tract, and digestive tract irritation.

Symptoms of toxicity associated with pseudoephedrine include convulsions, hallucinations, irregular or slow heartbeat, shortness of breath, trouble breathing, an increase in blood pressure, nervousness, restlessness, excitement, trouble sleeping, difficult or painful urination, dizziness, light-headedness, drowsiness, fast or pounding heartbeat, increased sweating, nausea, vomiting, trembling, unusual paleness, or weakness. Pseudoephedrine hydrochloride exposure may cause eye, skin, and digestive tract irritation, as well as neurological and CNS effects.

**Main Risks and Target Organs:** Cardiovascular: heart and arterial vessels, CNS stimulation. Chronic use can lead to tolerance with dependence.

**Chronic Effects:** Tolerance to ephedrine may develop with prolonged or excessive use. Prolonged or repeated exposure to pseudoephedrine hydrochloride can cause hallucinations, CNS stimulation, psychic abnormalities such as anxiety, depression and excitability, and possibly coma.

**Carcinogenicity:** The available studies on animals show no evidence of carcinogenic activity at doses higher than those associated with other effects (such as decreased body weight), (CANTOX 2000).

**Genotoxicity:** The available studies show negative results in mutagenic testing (CANTOX, 2000).

**Dermal Absorption:** No data is available on the dermal absorption of ephedrine or pseudoephedrine. On this basis it has been conservatively assumed that 100% is absorbed through the skin.

### B4.5 Quantitative Toxicity Data

CANTOX (2000) completed a detailed review of ephedra and ephedrine alkaloids. The non-clinical toxicology of ephedrine and ephedra was reviewed to assess its consistency with data obtained from clinical studies. The chemical characteristics of ephedra are dependent upon its chemical composition. Since the dominant ephedrine alkaloid isomer of most *Ephedra* species is ephedrine, the characteristics of ephedrine was identified as a good indicator of the expected chemistry, pharmacology, and toxicology. As with any mixture, the characteristics of only one, albeit major, component cannot define all of the characteristics of ephedra (and ephedrine alkaloids). The studies evaluated addressed the acute, subchronic and chronic safety, carcinogenicity, reproductive toxicity and mutagenicity of ephedrine. Most clinical and non-clinical studies have been undertaken on ephedrine. With regard to pseudoephedrine, since its effects are similar to ephedrine but are somewhat weaker with respect to the hypertensive effects and stimulation of the central nervous system, the assessment of toxicity using ephedrine as a surrogate is expected to provide a conservative evaluation.

Based on the review undertaken by CANTOX (2000) a dose-response assessment relevant for the general population was undertaken. The assessment resulted in the derivation of a Tolerable Upper Intake Level or Upper Limit (UL). The term Tolerable Upper Intake Limit (UL) is defined as the maximum level of total chronic daily intake of a substance judged unlikely to pose a risk of adverse health effects to the most sensitive members of the healthy population developed by applying uncertainty factors.

The UL derived is 90 mg/day based on a NOAEL of 90 mg/day from a clinical study and an uncertainty factor of 1. This is relevant to the total of all ephedrine alkaloids. Based on a 70 kg adult, the UL can be converted to a TDI of 1.3 mg/kg/day.
No background intakes of ephedrine/pseudoephedrine are available for the general population. Intakes may occur through the use of pharmaceutical preparations. To address potential exposures by the general public to residual ephedrine and pseudoephedrine levels at former clan labs it has been assumed that up to 50% of the UL (and TDI) may be ingested via pharmaceutical preparations. This is expected to be conservative as common use of ephedrine and pseudoephedrine in cold preparations is declining.
**B5 Chloroform**
The following is summarised from ATSDR (1997), WHO (2004) and HSDB (2009).

**B5.1 General**
Chloroform (also known as trichloromethane, methenyl chloride, methane trichloride, methyl trichloride and formyl trichloride) is both a synthetic and naturally occurring compound, with anthropogenic sources responsible for most of the chloroform in the environment. Chloroform is mainly used in the production of other materials, principally fluorocarbons, used in the synthesis of tetrafluoroethylene and polytetrafluoroethylene, and as a refrigerant and propellant. Chloroform is also widely employed as an organic solvent in industry and in analytical laboratories. It has also been used as an ingredient of pharmaceuticals, drugs, cosmetics, grain fumigants, dyes and pesticides.

**Appearance:** Chloroform is a volatile colourless liquid.

**Chemical/Physical Properties:** Chloroform (CASRN: 67-66-3) has the following properties (HSDB and RAIS):

- Molecular formula: $\text{CHCl}_3$
- Molecular weight: 119.38
- Log Kow: 1.97
- Koc: 35.04 L/kg
- Soluble: water and carbon disulfide, miscible with alcohol, ether, benzene, carbon tetrachloride, fixed and volatile oils
- Solubility in water: 7950 water at 25°C
- Vapour pressure: 197 mmHg at 20°C
- Henry's Law: 0.00387 atm.m$^3$/mol at 25°C
- Air diffusion coefficient: 0.104 cm$^2$/s
- Water diffusion coefficient: $1 \times 10^{-5}$ cm$^2$/s

**Odour and Taste:** Chloroform has a pleasant, non-irritating odour and slightly sweet taste. The odour threshold is listed as 85 ppm (421 mg/m$^3$).

**B5.2 Exposure, Fate and Transport**

**Indoor Surfaces:** Nearly all chloroform released to the environment will ultimately be present in the atmosphere due to its volatility. If spilled onto a nonporous surface, chloroform is not expected to remain and hence surface residues from non-porous materials are not considered to be of concern. Porous materials such as concrete, furnishings and wood may absorb chloroform with the potential for off-gassing over time, however eventually all of the chemical will dissipate.

**Air:** In the atmosphere, chloroform may be transported long distances before degrading via photochemical reactions with free radicals such as hydroxyl (which form low levels of phosgene and hydrogen chloride). Half-lives vary from 55 to 20 days.

**Soil:** Following releases to soil, most chloroform is expected to evaporate rapidly due to its high volatility and low soil adsorption. Most of the remaining chloroform will travel through the soil because of its low adsorption onto soils with leaching of chloroform to groundwater considered to be a significant pathway.

**Water:** Because of its volatility, evaporation is considered to be the main process for the removal of chloroform from aquatic systems. Chloroform is not expected to adsorb significantly to sediment or suspended organic matter in surface water.

**Biodegradation:** Chloroform is generally considered persistent in water and soils with a low potential for degradation. Under correct conditions, chloroform may undergo anaerobic biodegradation. Concentrations of
chloroform in soil or water above a certain threshold levels results in toxic conditions which inhibits bacteria, methane-fermenting bacteria under anaerobic conditions.

**Bioaccumulation:** The potential for bioaccumulation in aquatic biota is considered to be low.

### B5.3 Ecological Effects and Guidelines

Chloroform is toxic to the embryo-larval stages of some amphibian and fish species. It is less toxic to fish and *Daphnia magna*. There is little difference in sensitivity between freshwater and marine fish. Chloroform is of low toxicity to algae and other microorganisms (WHO, 1994). No terrestrial data is available for chloroform hence available soil guidelines are based on equilibrium partitioning from water guidelines.

With respect to the protection of the long term environment the following screening level guidelines/benchmarks are available from ANZECC/ARMCANZ (2000) for chlorinated methanes:

- Low reliability trigger value of 0.37 mg/L for chloroform in fresh and marine water which should only be used as an indicative interim working level.

Other screening level guidelines/benchmarks available for chloroform from USEPA, Canada and Dutch peer reviewed sources include the following for soil:

- Dutch (RIVM, 2001) Serious Risk Concentration (SRC) in soil based on ecotoxicity endpoints = 170 mg/kg
- USEPA Region V (2003) Ecological Screening Level (ESL) in soil = 1.2 mg/kg.

### B5.4 Health Effects

**General:** Human exposure to chloroform can occur orally, dermally, or by inhalation. Chloroform is the principal trihalomethane generated as by-products during the chlorination of drinking water. The primary sources of chloroform in the environment are chlorinated drinking water and wastewater, pulp and paper mills, and chemical and pharmaceutical manufacturing plants. The general population is exposed to chloroform mainly in food, drinking-water and indoor air. Most of the chloroform released to the environment eventually enters the atmosphere, while much smaller amounts enter groundwater as a result of filtration through the soil.

Chloroform is rapidly absorbed through the lungs and the gastrointestinal tract, and to some extent through the skin. In humans, the respiratory absorption of chloroform ranges from 49 to 77% and absorption from the gastrointestinal tract approximates 100%, with peak blood levels being reached within 1 hour.

Following its absorption, chloroform is distributed to all organs. The distribution of chloroform in the body does not differ qualitatively between the various routes of exposure. A number of studies have shown that chloroform distributes to fat tissue. It is lipid soluble, readily passes through cell membranes, reaching relatively high concentrations in nervous tissue. Chloroform concentrations in tissues are dose-related and occur in the following order: adipose > brain > liver > kidney > blood. Chloroform passes through the placenta and has been detected in fresh cow’s milk and foetal blood at levels equal to or greater than that in maternal blood.

**Main Risks and Target Organs:** Target organs for chloroform toxicity are the liver, kidneys, central nervous system and developmental toxicity.

**Acute Effects:** In humans, acute exposures to chloroform have been associated with fainting, vomiting dizziness, salivation, fatigue, headache, respiratory depression and coma. Cardiac arrhythmia, bradycardia and cardiac arrest resulting death have been reported following use of chloroform as an anaesthetic. Other effects noted following acute exposures include liver and kidney damage.
**Chronic Effects:** Limited information is available regarding possible adverse effects associated with chronic exposure to chloroform, however in the available studies liver and kidney toxicity have been reported.

**Carcinogenicity and Genotoxicity:** Although chloroform has produced tumours in mice and in male rats in several bioassays, the currently available data provide convincing support for the view that the mechanism of tumour induction does not depend on direct DNA damage and that chloroform can be considered to be a non-genotoxic carcinogen.

Chloroform has been classified as a "probable" human carcinogen (Category B2) by the USEPA and IARC has classified chloroform in Group 2B (possibly carcinogenic to humans) based on carcinogenicity in animals.

Review of chloroform by the USEPA (2001) indicates that it is considered likely to be carcinogenic to humans by all routes of exposure under high-dose conditions that lead to cytotoxicity and regenerative hyperplasia. Chloroform is not likely to be carcinogenic to humans by any routes of exposure at doses that do not cause cytotoxicity and cell regeneration. Hence the USEPA has concluded that the threshold effects value established is also protective against increased risk of cancer. In addition the weight of evidence associated with genotoxicity data supports the conclusion that chloroform is not strongly mutagenic and that genotoxicity is not likely to be the predominant mode of action. Similarly a review of the mode of action by the WHO (2004) has identified that mechanism for induction of tumours is consistent with a non-linear (or threshold) dose-response relationship for the induction of tumours.

**Dermal Absorption:** The USEPA (2004) guidance on dermal exposure assessment suggests that for volatile chemicals dermal absorption can be effectively considered negligible as the chemical is not expected to remain on the skin long enough to be absorbed. Prior to this recommendation the dermal absorption of chloroform was taken to be 0.05% for VOC based on experimental data (USEPA 1995). On this basis a value of 0.05% has been adopted for chloroform.

**B5.5 Quantitative Toxicity Data**

The following are available from the WHO (2004):

- **TDI = 0.015 mg/kg/day** based on PBPK model and BMD value based on liver effects in dogs, with consideration of an uncertainty factor of 25 (10 for intraspecies toxicokinetics and toxicodynamics differences and 2.5 for interspecies toxicodynamics differences)
- **TC = 0.14 mg/m³** based on the same study and approach presented for the derivation of the TDI (relevant given the use of the PBPK model to address route specific issues).

The TDI and TC values derived by the WHO (2004, an update of previous assessments provided in 1994 and 2000) have considered these issues as well as the route of exposure and are considered the most relevant threshold values to adopt. The TDI is essentially equivalent to the oral RfD derived using a similar approach by the USEPA. The values adopted are considered protective of all health effects (non-neoplastic and neoplastic) associated with exposure to chloroform, including those of sensitive groups such as children.

Based on available data on intakes that may be derived from urban air, food and drinking water (WHO, 2004), background intakes have been estimated to be approximately 50% of the available TDI and TC, of which 50% is assumed to be derived from air.
B6  Dichloromethane
The following is summarised from ATSDR (2000), WHO (1996) and HSDB (2009).

B6.1 General
Dichloromethane (also commonly known as methylene chloride as well as methane dichloride, methylene dichloride or DCM) is a synthetic compound, which is not known to occur naturally in the environment. DCM is primarily used as a solvent, especially for grease, plastics and various paint-binding agents. Among its uses are: as a component of paint and varnish strippers, and adhesive formulations; solvent in aerosol formulations; extractant in food and pharmaceutical industries; process solvent in cellulose ester production and fibre and film forming; process solvent in polycarbonate production; blowing agent in flexible polyurethane foams; the extraction of fats and paraffins; plastics processing, and metal and textile treatment; a vapour degreasing solvent in metal-working industries. The main use in consumer products is in paint strippers, where DCM is the main constituent (70-75%).

Appearance: Dichloromethane is a volatile colourless liquid.

Chemical/Physical Properties: Dichloromethane (CASRN: 75-09-2) has the following properties (HSDB and RAIS):

- Molecular formula: CH₂Cl₂
- Molecular weight: 84.93
- Log Kow: 1.25
- Koc: 23.74 L/kg
- Soluble: water and carbon tetrachloride, miscible with alcohol, ether, dimethylformamide, ethanol and ethyl ether
- Solubility in water: 13200 at 20°C
- Vapour pressure: 435 mmHg at 20°C
- Henry's Law: 0.00219 atm.m³/mol at 25°C
- Air diffusion coefficient: 0.101 cm²/s
- Water diffusion coefficient: 1.17x10⁻⁵ cm²/s

Odour: Chloroform has a penetrating ether-like odour. The odour threshold is listed as 540 to 2160 mg/m³.

B6.2 Exposure, Fate and Transport

Indoor Surfaces: Nearly all DCM released to the environment will ultimately be present in the atmosphere due to its volatility. If spilled onto a non-porous surface, DCM is not expected to remain and hence surface residues from non-porous materials are not considered to be of concern. Porous materials such as concrete, furnishings and wood may absorb DCM with the potential for off-gassing over time, however eventually all of the chemical will dissipate.

Air: In the atmosphere, DCM will degrade by reaction with photochemically produced hydroxyl radicals with a half-life of approximately six months. Transport can occur to regions far removed from the emission source. DCM is expected to have no significant impact on stratospheric ozone depletion nor will it contribute significantly to photochemical smog formation.

Soil: Following releases to soil, most DCM is expected to evaporate rapidly due to its high volatility and low soil adsorption. Most of the remaining DCM will travel through the soil because of its low adsorption onto soils with leaching of DCM to groundwater considered to be a significant pathway.

Water: Because of its volatility, evaporation is considered to be the main process for the removal of DCM from aquatic systems. DCM is not expected to adsorb significantly to sediment or suspended organic matter in surface water.
**Biodegradation:** DCM undergoes slow hydrolysis in water and hence it is not considered to be a significant degradation process in water. Both aerobic and anaerobic biodegradation may be important for DCM in water. Degradation of DCM in soils was found to occur with the rate of degradation dependant on the soil type, concentration and redox state of the soil with degradation observed under both aerobic and anaerobic conditions. Biodegradation of DCM appears to be accelerated by the presence of elevated levels of organic carbon.

**Bioaccumulation:** The potential for bioaccumulation in aquatic biota is considered to be low.

**B6.3 Ecological Effects and Guidelines**

In the aquatic environment, fish and amphibian embryos have been shown to be the most sensitive to DCM, with effects on hatching identified from 5.5 mg/L; adult aquatic organisms are relatively insensitive even after prolonged exposure. There is no evidence to suggest that DCM and/or its metabolites bioaccumulate in the environment.

Localised contamination of soil will not significantly disperse despite the mobility of DCM in groundwater and soils. However biological degradation processes have been identified capable of mineralising DCM in a few days. From the limited information on soil organisms, it may be assumed that contamination of soil has only a local and transient effect (WHO, 1996). Hence the potential for long term environmental effects associated with the improper disposal of DCM at former clan lab sites is considered low, particularly for the terrestrial environment. If significant quantities are identified in groundwater, with the potential for discharge to a receiving environment the environmental issues may need to be addressed.

With respect to the protection of the long term environment the following screening level guidelines/benchmarks are available from ANZECC/ARMCANZ (2000) for chlorinated methanes:

- Low reliability trigger value of 4 mg/L for DCM in fresh and marine water which should only be used as an indicative interim working level.

Other screening level guidelines/benchmarks available for DCM from USEPA, Canada and Dutch peer reviewed sources include the following for soil:

- Dutch (RIVM, 2001) Serious Risk Concentration (SRC) in soil based on ecotoxicity endpoints = 3.9 mg/kg
- USEPA Region V (2003) Ecological Screening Level (ESL) in soil = 4 mg/kg.

**B6.4 Health Effects**

**General:** Human exposure to DCM occurs principally through inhalation. However exposure may also occur via oral and dermal routes, particularly during occupational or consumer use of products containing DCM. Humans and animals readily absorb DCM from the lungs and the gastrointestinal tract into systemic circulation. The compound is also absorbed to some extent through intact skin. Following absorption, DCM concentrations rapidly increase in the blood to reach equilibrium levels that depend primarily on exposure concentrations. A fairly uniform distribution to heart, liver, and brain is reported with increased concentrations also reported in adipose tissue. DCM is quite rapidly excreted, mostly via the lungs in the exhaled air. It can cross the blood-brain barrier and be transferred across the placenta, and small amounts can be excreted in urine or in milk.

**Main Risks and Target Organs:** Target organs for DCM toxicity are the cardiovascular system and central nervous system.
**Acute Effects:** Acute toxicity of DCM is considered to be low. Effects associated with acute exposures (at high doses) are primarily associated with the CNS and the liver.

**Chronic Effects:** Effects associated with chronic exposure to DCM are associated with the CNS, and blood carboxyhaemoglobin, cardiovascular system and liver.

**Carcinogenicity and Genotoxicity:** DCM has been classified as a "probable" human carcinogen (Category B2) by the USEPA and IARC has classified DCM in Group 2B (possibly carcinogenic to humans) based on carcinogenicity in animals. Review of genotoxicity data available suggests that there is no conclusive evidence that DCM is genotoxic. In addition the carcinogenic potency of DCM in humans is expected to be low. On this basis the adoption of a threshold dose-response assessment for the assessment of DCM exposures is considered appropriate.

**Dermal Absorption:** The USEPA (2004) guidance on dermal exposure assessment suggests that for volatile chemicals dermal absorption can be effectively considered negligible as the chemical is not expected to remain on the skin long enough to be absorbed. Prior to this recommendation the dermal absorption of volatile organic compounds was taken to be 0.05% based on experimental data (USEPA 1995). On this basis a value of 0.05% has been adopted for DCM.

**B6.5 Quantitative Toxicity Data**

The following are available for DCM:

- **TDI** = 0.0012 mg/kg/day (NHMRC, 2004) based on a lowest effect level from a 2 year drinking water study and application of a 5000 fold safety factor.
- **TC** = 1 mg/m³ (ATSDR 1996) based liver effects from an inhalation specific study.

Based on available data on intakes that may be derived from urban air (no data is available for food and drinking water) (Hawas 2001 and WA DEP 2000), background intakes have been estimated to be approximately 20% of the available TDI and TC.
B7  Ammonia

The following is summarised from OEHHA (2003d), ATSDR (2004), WHO (1986) and HSDB (2009).

B7.1 General

Anhydrous ammonia is used with an alkali metal (typically lithium or sodium) in the “Nazi Method” to convert ephedrine to methamphetamine. Ammonia is a naturally occurring compound that is an intermediate in the global nitrogen cycle. It is essential for many biological processes and is a central compound in all living things. Because of its role in natural processes and cycles, ammonia is found at low concentrations in most environmental media.

Appearance: Ammonia is a colourless gas at room temperature and easily dissolves in water to form ammonium hydroxide. Ammonium hydroxide is a weak base that is partly ionised in water, the amount of ionisation is dependent on pH and at most environmentally relevant pH the ammonia is largely ionised. The ammonium ions are not gaseous and have no odour. In clan labs, anhydrous ammonia is often stored in insulated coolers, small propane fuel cylinders, and propane tanks.

Chemical/Physical Properties: Ammonia (CASRN: 7664-41-7) have the following properties (HSDB and RAIS):

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula</td>
<td>NH₃</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>17.03</td>
</tr>
<tr>
<td>Log Kow</td>
<td>0.23 to 1.155</td>
</tr>
<tr>
<td>Koc</td>
<td>363 L/kg</td>
</tr>
<tr>
<td>Solubility</td>
<td>482000 water at 20°C</td>
</tr>
<tr>
<td>Vapour pressure</td>
<td>7510 mmHg at 25°C</td>
</tr>
<tr>
<td>Henry’s Law</td>
<td>1.6x10⁻⁵ atm.m³/mol at 25°C</td>
</tr>
<tr>
<td>Air diffusion coefficient*</td>
<td>0.28 cm²/s</td>
</tr>
<tr>
<td>Water diffusion coefft*</td>
<td>1.9x10⁻⁶ cm²/s</td>
</tr>
</tbody>
</table>

* Diffusion coefficients as referenced and presented by Wu et al (2003)

Odour and Taste: Ammonia has a strong pungent suffocating odour. The odour threshold is listed as 18 to 38 mg/m³.

B7.2 Exposure, Fate and Transport

Indoor Surfaces: Contamination of indoor surfaces by anhydrous ammonia is not a concern. If spilled onto a nonporous surface, liquefied anhydrous ammonia will not remain, because it is a gas at room temperature. Porous materials such as concrete and wood may absorb ammonia and delay its rate of evaporation, but eventually all of the chemical will dissipate.

General: In considering the environmental fate of ammonia it is necessary to note that ammonia is very important in nature and in nature’s biological cycles. In addition an important consideration that affects the transport and partitioning of ammonia in the environment is that ammonia is a base and as such the physical and chemical properties are pH dependant.

Air: Most of the ammonia entering the atmosphere will be transported back to the earth by both wet and dry deposition predominantly as ammonium sulphate and ammonium nitrate. Wet deposition includes rainfall, snow, hail, fog, and dew, while dry deposition mainly concerns gaseous ammonia. The half life of ammonia in air is expected to be a few days.

Soil: Ammonia ion in soil is very mobile in soil due to the conversion to nitrate by nitrification. Nitrate is lost rapidly (within a few days or weeks) in warm, moist soils due to uptake by plant root systems. Ammonia does
not leach readily through soil however nitrate derived from ammonia may leach. Ammonia is a key compound in the nitrogen cycle and as such is continually recycled in the environment.

**Water:** Ammonia can be removed from water via volatilisation to the atmosphere (pH dependant), transformation into other nitrogen species and/or adsorption onto sediments and other organic materials. Transformation in water is primarily through the process of nitrification. The term 'ammonia' refers to two chemical species of ammonia that are in equilibrium in water: the un-ionised ammonia, $\text{NH}_3$, and the ionised ammonium ion, $\text{NH}_4^+$. The proportion of the two chemical forms in water varies with the physico-chemical properties of the water, particularly pH and temperature.

**Bioaccumulation:** As ammonia is naturally occurring and a key intermediate in the nitrogen cycle, the potential for bioaccumulation is not considered relevant.

### B7.3 Ecological Effects and Guidelines

Ammonia in the form of a liquid, concentrated solution, or at a high vapour concentration, is acutely toxic and will destroy most living organisms. However, ammonia is so readily diluted and degraded in the environment that accidental spillages or emissions will not persist and will be widely dispersed. Nevertheless, persistently high concentrations of ammonia may occur in the atmosphere where there is intensive use or production of animal manure in farming. High concentrations may also occur in water in certain elevated, isolated lakes, and where sewage is discharged WHO (1990). While acute issues associated with the potential improper disposal of ammonia are high, as ammonia is a non-persistent and non-cumulative toxicant the potential for long-term effects at former clan lab sites are considered to be low, particularly for the terrestrial environment. If significant quantities are present in groundwater, with the potential for discharge to a receiving environment the environmental issues may need to be addressed.

The toxicity of ammonia can depend on pH, temperature and ionic composition of water, particularly at the point of discharge. The toxicity of ammonia is primarily attributed to the un-ionised $\text{NH}_3$. In general, more un-ionised ammonia exists at higher pH and hence overall toxicity is greater, although the toxicity of the un-ionised form is less at higher pH. However, data also indicate that at lower pH, less un-ionised $\text{NH}_3$ is needed to produce its toxic effects because the ammonium ion is responsible for some of the toxicity. At sufficiently low pH, the relative amount of ammonium ion increases and it dominates toxicity. Overall, the effect of pH on toxicity of ammonia is largely explained by a combined toxicity of the un-ionised ammonia and ammonium ion, with un-ionised ammonia contributing mostly to toxicity at high pH and ammonium ion being more important at lower pH (ANZECC/ARMCANZ, 2000).

With respect to the protection of the long term environment the following screening level guidelines/benchmarks are available from ANZECC/ARMCANZ (2000) for ammonia:

- Freshwater high reliability trigger value of 0.9 mg/L (95% protection level) at pH 8;
- Marine water high reliability trigger value of 0.91 mg/L (95% protection level) at pH 8.

As noted above the toxicity of ammonia is pH dependant. Hence the above guideline trigger values are given as total ammonia (ionised plus un-ionised ammonia) concentrations. The guideline trigger values were calculated by converting all data, reported at different pH values, to total ammonia at a common pH value of 8 before applying any statistical distribution derivation method. Guideline trigger values at other pH conditions need to be assessed as outlined by ANZECC/ARMCANZ (2000).

No ecological screening level guidelines are available for ammonia in soil.
B7.4 Health Effects

General: Ammonia dissolves in moisture in the air and on tissue or mucous membranes to form ammonium hydroxide, a strong base. Hence the main health effects of ammonia are related to corrosive nature of the compound. Ammonium is produced in the intestines by bacteria and is efficiently absorbed from the gastrointestinal system. It is noted that ammonia has a toxic effect on healthy humans only if the intake becomes higher than the capacity to detoxify. Studies indicate that ammonia is absorbed by the mucous membranes and intestinal tract following inhalation and oral routes of exposure. Although ammonia rapidly enters the eye, systemic absorption is not considered to be significant.

Most inhaled ammonia is held in the upper respiratory tract then excreted in exhaled air. Both endogenously and exogenously produced ammonia is readily absorbed by the intestinal tract before entering the hepatic portal vein. In the liver, ammonium ions are extensively metabolised to urea and glutamine. Consequently, levels of ammonia that reach the circulatory system are low. Hepatic insufficiency can affect ammonium ion metabolism. Metabolised ammonia is primarily excreted by the body through urea.

Main Risks and Target Organs: Respiratory system, skin and eyes.

Acute Effects: Ammonia and ammonia solutions are irritating and corrosive and may and can cause burns to the skin, eyes, mouth and lungs. Acute oral exposure rapidly results in pain, excessive salivation and burns to the mouth, throat and oesophagus. Acute inhalation may cause upper respiratory tract irritation. Substantial exposures can result in burns as well as airway obstruction, respiratory disease and bronchiolar and alveolar oedema. Ammonia and ammonia solutions are corrosive via direct contact with tissues and splashes to the eye may result in serious injury.

Chronic Effects: Effects associated with chronic oral exposure to ammonia have not been identified in humans, however data from animals suggest osteoporosis, occurring secondary to chronic metabolic acidosis and the key endpoints. Chronic inhalation exposure has been associated with increase cough, phlegm, wheeze and asthma. Limited data is available, however it is unlikely that exposure to environmental levels of ammonia would result in reproductive or developmental toxicity. Data from animal studies suggest that foetal toxicity or embryo toxicity may occur by secondary to maternal toxicity after very high exposures.

Carcinogenicity and Genotoxicity: The limited data available are inconclusive with respect to carcinogenicity and genotoxicity of ammonia. Ammonia has not been classified by IARC or USEPA.

Dermal Absorption: No data is available on the dermal absorption of ammonia or ammonium. As ammonia is a gas it is not expected to be significantly absorbed by the skin, however it has been conservatively assumed that 1% may be absorbed through the skin.

B7.5 Quantitative Toxicity Data

Limited quantitative data is available, however the USEPA has derived an inhalation reference concentration (RfC) of 0.1 mg/m³.

Based on available data on intakes that may be derived from urban air (WHO, 1986) and drinking water (NHMRC, 2004), background intakes have been estimated to be approximately 20% of the available RfC (of which 50% is derived from air).
B8 Iodine
The following is summarised from OEHHA (2003e), ATSDR (2004b), IPCS (1990), WHO (2009a) and HSDB (2009).

B8.1 General
Iodine is primarily used in anti-infective agents. It is also used in the manufacture of a wide range of organic and inorganic chemicals and pharmaceuticals. In clan labs iodine is combined with red phosphorous to make hydroiodic acid, an essential ingredient in the manufacture of methamphetamine from ephedrine.

Appearance: Laboratory grade elemental iodine (I₂) consists of heavy greyish-black to purple crystals that have a metallic lustre. Laboratory grade iodine also may appear as a brown powder. At room temperature iodine crystals readily volatilise to a violet gas.

Chemical/Physical Properties: Iodine (CASRN: 7553-56-2) has the following properties (HSDB and RAIS):

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula:</td>
<td>I₂</td>
</tr>
<tr>
<td>Molecular weight:</td>
<td>253.81</td>
</tr>
<tr>
<td>Log Kow:</td>
<td>2.49</td>
</tr>
<tr>
<td>Koc:</td>
<td>NA</td>
</tr>
<tr>
<td>Solubility:</td>
<td>chloroform, carbon tetrachloride and carbon disulfide to form a purple solution. Also soluble in water, cyclohexane, methanol, alcohol, ether, benzene, hexane, butanol, ethylacetate and toluene</td>
</tr>
<tr>
<td>Solubility in water:</td>
<td>330 mg/L at 25°C</td>
</tr>
<tr>
<td>Vapour pressure:</td>
<td>0.3 mmHg at 25°C</td>
</tr>
<tr>
<td>Henry’s Law:</td>
<td>NA</td>
</tr>
</tbody>
</table>

No data is available regarding diffusion coefficients, Henry’s Law or Koc for iodine, hence values available for methyl iodide have been used for the purpose of modelling volatilisation. The values available are: air diffusion coefficient = 0.039 cm²/s, water diffusion coefficient = 1.04x10⁻⁵ cm²/s, Koc = 14 L/kg.

Odour and Taste: Iodine gas (vapour) has a highly characteristic, sharp, irritating odour. Iodine has a sharp, acrid, metallic taste. The odour threshold is listed as 8 mg/m³.

B8.2 Exposure, Fate and Transport

Indoor Surfaces: Long-term contamination of indoor surfaces is not expected. Elemental iodine readily volatilises at room temperature. If present on indoor surfaces, elemental iodine will be removed through volatilisation where inhalation exposure could occur. Iodine compounds however may remain on surfaces long enough to require consideration with respect to long-term exposure. Iodine has the potential to stain surfaces and hence visual issues may also need to be addressed in the remediation of iodine on surfaces in any premises.

General: Iodine is found in the earth’s crust, much of it inaccessible, except for a small portion that is liberated through weathering into the oceans. Iodine is largely found in the ocean where it is transferred to air, surface soil and sediments etc.

Air: The gaseous and particulate forms of iodine in the atmosphere are deposited onto ocean or land surfaces through wet and dry deposition. In air, iodine vapour will be hydrolysed by water vapour to iodate (IO₃⁻) and iodide (I⁻) ions, which have a relatively low order of toxicity. At ordinary pressures and temperature, methyl iodide and iodine have high vapour pressures and will exist predominately in a free gaseous form in air. Both iodine and methyl iodide undergo photochemical reactions to form iodine radicals (IC), which can then go on to form a number of other iodine species through a complex series of reaction pathways.
Soil: In soil, iodine will be oxidised to iodate (IO$_3^-$) and reduced to iodide (I$^-$) ions, which have a relatively low order of toxicity and are essential micronutrients in the diet. Evaporation of iodine from the land surface to the atmosphere is only about 1% of the flux of iodine from the atmosphere to the land surface and iodine is cycled back to the ocean through groundwater and river effluent. The low flux of iodine from land surfaces to the atmosphere is due to the retention of iodine within surface soils, especially in soils rich in organic matter and iron/aluminum oxides.

Water: In aqueous solution, iodine is oxidised to iodate (IO$_3^-$) and reduced to iodide (I$^-$) ions. Iodine in drinking water normally contributes a small proportion of total daily iodine intake; the majority of ingested iodine comes from food. Therefore, unless a source of drinking water is highly contaminated with iodine, ingestion of iodine from drinking water is unlikely to cause adverse effects. Contamination of groundwater by iodine is unlikely given the rapid conversion of iodine (I$_2$) to iodide (I$^-$) in the presence of organic materials. Iodide has a relatively low order of toxicity and is an essential constituent in the human diet.

Bioaccumulation: Iodine can bioaccumulate to varying extents in aquatic organisms where the higher levels of bioaccumulation are usually associated with the relatively high levels of iodine in seawater.

B8.3 Ecological Effects and Guidelines
Aqueous iodine (I$_2$(aq)) acts as a biotic killing agent. It is a strong oxidant and therefore damaging to biological systems. Aqueous I$_2$ is most stable at lower pH and as solution pH increases I equilibrium shifts toward the non-bactericidal I$^-$ species. Unlike mammals, plants do not require I and they can be adversely affected by low (micromolar) concentrations. The degree of phytotoxicity is dependent on the form of I that exists in the soil solution. Typically, I$^-$ is more phytotoxic than IO$_3^-$ which may be due to the greater ability of plant roots to absorb the reduced form. Once in the plant, I$^-$ may oxidise to I$_2$.

No screening level guidelines are available for iodine or iodide in Australia. Screening level guidelines/benchmarks as listed by RAIS (2009) from USEPA, Canada and Dutch peer reviewed sources include the following for iodide soil:

- USEPA (Region VI and ORNL) Benchmark for protection of plants = 4 mg/kg

B8.4 Health Effects
General: Exposure to iodine may occur via oral, dermal or inhalation. When ingested iodine appears to be inactivated by combination with gastrointestinal contents. Absorption is poor due to rapid conversion to iodide. Iodine is absorbed from the lungs and converted to iodide in the body. Only small quantities of iodine are absorbed through the intact skin; however it can be absorbed through wounds and abrasions.

Iodine reaches the blood stream mainly in form of iodide, and it is incorporated into the thyroglobulin form in the thyroid gland. Iodine is excreted mainly in the urine. Iodides diffuse across the placental barrier and into breast milk. Infant death from respiratory distress secondary to goitre (enlargement of the thyroid) has been reported as a result of mothers taking medications containing iodides. Compared to adults, children have a greater ratio of lung surface area to body weight and may be more susceptible to inhalation effects associated with exposure to iodine.

Main Risks and Target Organs: Target organs are mucous membranes of pharynx, larynx and oesophagus for the concentrated iodine, and thyroid for the diluted form as a systemic effect.

Acute Effects: Concentrated iodine is corrosive. Main risks in acute exposure to high iodine concentrations are largely due to the highly corrosive effect of iodine on the entire gastrointestinal tract and resultant shock. Iodine vapour causes eye, skin, nose and throat irritation, coughing, wheezing, and laryngitis. Exposure to high concentrations may result in airway spasm, chest tightness, breathing difficulty, severe inflammation, and
fluid accumulation in the voice box, upper airways, and lungs. Some people develop allergic hypersensitivity to iodine vapour.

**Chronic Effects:** Effects of long-term exposure to iodine vapour in humans are not known. Iodide is an essential micronutrient in the diet. It is required in small amounts for normal function of the thyroid gland. In laboratory animals, long-term inhalation of iodine vapours disrupts thyroid function and reduces the ability of the lungs to take up oxygen. A number of health effects can be attributed to both deficiency and excess of iodine in the diet. While inadequate intake of iodine leads to hypothyroidism and compensatory increase in the size of the thyroid gland (goitre), excessive intake of iodine can be associated with both hyperthyroidism and hypothyroidism.

**Carcinogenicity and Genotoxicity:** Increased incidence of thyroid cancer has been observed both in association with endemic hypothyroidism and with increased dietary iodine intake in endemic goitre areas. However studies of populations in which iodine intakes are sufficient have not found significant associations between iodine intake and thyroid cancer. A number of studies have shown that iodine or potassium iodide do not cause mutagenic effects. Iodine has not been classified with respect to carcinogenicity by IARC or USEPA.

**Susceptible Populations:** Individuals susceptible to iodine-induced hypo-thyroidism include foetuses and newborn infants, the elderly, persons who have underlying thyroid disease and patients who have received treatment with anti-thyroid medications.

**Dermal Absorption:** Based on available data presented in ATSDR (2004) dermal absorption of iodine may range from 0.1% to 14%. While the WHO (2009a) suggests a value of <1% may be relevant, a value of 14% has been conservatively assumed.

**B8.5 Quantitative Toxicity Data**

The ATSDR (2004) and WHO (2009a) has derived an oral chronic TDI of 0.01 mg/kg/day based on hypothyroidism in children. The TDI derived satisfies the requirements of both protecting human health from excessive iodine exposure and ensuring that iodine exposure does not conflict with essential dietary intake levels.

The only available inhalation guideline is available from the Texas Commission on Environmental Quality (TCEQ, 2009) where an interim chronic effects screening level (ESL) of 0.001 mg/m³, based on the protection of health effects is available.

As a wide range of background intake estimates are available, it has been assumed that background intakes of iodine may contribute up to 60% of the available TDI (and ESL) based on estimates from the US population by WHO (2009a), of which 50% is assumed to be from air.
B9 Bromide

B9.1 General

The following is summarised for the bromide ion (Br-) and bromine (Br) by IPCS (1992), HSDB (2009) and Flury and Papritz (1993).

In clan labs hydrobromic acid, the aqueous acid of hydrogen bromide is used as a reactant in the manufacture of methamphetamine. Apart from issues associated with pH, the presence of bromide from the use and disposal of this acid provides an indicator of the presence of these contaminants. Bromide is present in seawater with seafoods generally reporting high levels of bromide. Bromide compounds have been used for a range of purposes including sedatives. Ozone is a powerful oxidant and can convert naturally occurring bromide to bromine in the environment hence most of the information available relates to the more prevalent bromide ion (Br-).

Appearance: Bromine is a dark reddish-brown, volatile, diatomic liquid with a suffocating odour at room temperature. It is the only liquid non-metallic element. Because the vapour pressure is so high, the dark red vapours are immediately detectable when a container is opened.

Odour and Taste: Bromine has a suffocating, bleach-like, penetrating odour. The odour threshold is listed as 0.05 to 3.5 ppm.

B9.2 Exposure, Fate and Transport

Indoor Surfaces: If contamination occurs, bromide would be expected to persist to some extent.

Air: Bromine (Br₂) will photolyse rapidly (about 1 minute) to form Br atoms, which then react with ozone to ultimately form aerosol and/or particulate bromine. No information on the atmospheric half-life and lifetime of bromine was found in the readily-available literature. Information is available on methyl bromide (commonly used as a fumigant) which also releases bromide ions to the environment. Parameters relevant to methyl bromide (HSDB 2009 and USDA 2009) have been used for the purpose of volatilisation modelling.

Soil and Water: Bromides are considered ideal tracers in soil as they do not adsorb to negatively charged constituents in the solid phase. Therefore they have the potential to rapidly migrate through soil, reportedly faster than water in soil. An important source of bromide and bromine in soil are bromide containing pesticides such as 1,2-dibromomethane, methyl bromide (bromomethane) and 1,2-dibromo-3-chloropropane which degrade to form bromide in soil and subsequently groundwater (via leaching).

B9.3 Ecological Effects and Guidelines

Bromide has a low acute toxicity to mammals and aquatic organisms (Flury abd Papritz, 1993). However reproduction and growth of some species are affected at concentrations exceeding 2 to 10 mg/L. Compared to the knowledge about the aquatic environment, ecotoxicological data on soil organisms are scarce. The Br- concentrations occurring naturally in soils are usually too small to cause problems in agricultural crop production. However, with the use of brominated pesticides that readily hydrolyse to inorganic Br-, increased concentrations of this substance may occur in soils and may harm crop plants. Bromide is easily taken up by plants. The symptoms of bromide toxicity are similar to those of excess of salts, namely necrosis of the tips and edges of leaves, chlorosis, and shrivelling of leaves of carnation, wheat, and citrus. Other symptoms are poor germination and slower growth (Flury abd Papritz, 1993).

No screening level guidelines are available for bromide or bromine in Australia. Screening level guidelines/benchmarks as listed by RAIS (2009) from USEPA, Canada and Dutch peer reviewed sources include the following for bromine soil:
• USEPA (Region VI and ORNL) Benchmark for protection of plants = 10 mg/kg

B9.4 Health Effects

**General:** Bromine occurs naturally in the earth's crust as a non-metallic element. Like other halogens, it is very reactive and principally found in the form of inorganic bromides (Na, K, NH4, Ca and Mg) and as a component secondary to chlorine in minerals and biological systems (humans and animals). Bromine is slightly soluble in water, producing hydrogen bromide. Hydrogen bromide is a corrosive colourless gas with a pungent odour that is extremely soluble in water. In the presence of sunlight and humid air or hot water, it forms hydrobromic acid. Although less toxic than bromine, it has all the irritant qualities of bromine.

Bromine vapours can be inhaled where effects to the respiratory tract may occur. Due to its reactivity, bromine does not persist as an element in living tissue and quickly forms bromide. Bromide is deposited to the tissues, displacing other halogens. There is no data on the metabolism of inhaled bromine or bromide.

Acute toxicity effects associated with exposure to bromide include gastrointestinal and central nervous system effects. Chronic studies show effects in the central nervous system, endocrine and reproductive systems.

**Main Risks and Target Organs:** Target organs are central nervous system, endocrine and reproductive systems.

**Carcinogenicity and Genotoxicity:** While limited data is available, no data are available that suggest bromine is carcinogenic or mutagenic. Bromine has not been classified by IARC or USEPA.

**Dermal Absorption:** No dermal absorption data is available for bromide or bromine hence a conservative default value of 1% has been assumed as for metals (USEPA 1995) in the absence of other data.

B9.5 Quantitative Toxicity Data

Studies have been undertaken on bromide and bromine with the lowest tentative **ADI of 0.1 mg/kg/day** identified on the basis of endocrine effects associated with sodium bromide (Flury and Papritz, 1993).

The Texas Commission on Environmental Quality (TCEQ, 2009) has derived an interim chronic effects screening level (**ESL**) of **0.0007 mg/m³** for bromine based on the protection of health effects.

Bromide concentrations are reported to be low in the natural environment however bromine occurs naturally in food products. As current data is limited it has been assumed that background intakes of bromide and bromine comprise **10%** of the ADI and ESL.
B10  Phosphorus
The following is summarised from OEHHA (2003f), Environment Canada (2005), HSDB (2009).

B10.1 General
Phosphorus is present in clan labs through the use of hypophosphorous acid, phosphorous acid and red phosphorous as a reactant in the manufacture of methamphetamine. With respect to hypophosphorous acid ($\text{H}_3\text{PO}_2$) and phosphorous acid ($\text{H}_3\text{PO}_3$), apart from issues associated with pH, the presence of phosphorus from these acids provides an indicator of the presence of these contaminants. The summary presented here addresses phosphorus (derived from these acids) and the solid red phosphorus as used in clan labs. The most common use for phosphorus compounds is in fertilisers.

**Appearance:** Hypophosphorous acid is a clear to yellow liquid. Phosphorous acid is a yellow to clear crystal that is soluble. Red phosphorus is a dark reddish powder or crystal that is largely insoluble in water.

**Chemical/Physical Properties:** Phosphorus (red) and acids are not considered volatile. There are a range of properties that define phosphorus compounds, which have not been summarised here.

**Odour and Taste:** Phosphorus (elemental) has an acrid, garlic like odour. Red phosphorus has no odour.

B10.2 Exposure, Fate and Transport

**Indoor Surfaces:** If contamination occurs, red phosphorus would be expected to persist. On indoor surfaces it would be found as a solid where spilled. Skin contact and ingestion resulting from hand-to-mouth activity could occur. Hence the presence of red phosphorus in surface residues is of potential importance. Red phosphorus will slowly degrade to highly toxic phosphine gas ($\text{PH}_3$) and phosphorus acids in the environment.

**Soil and Water:** Red phosphorus will slowly degrade to phosphine ($\text{PH}_3$) and phosphorus acids in the environment. Phosphine is very reactive and usually undergoes rapid oxidation. The final products, phosphates, are harmless. Phosphorus ($\text{P}$) is an important plant nutrient that limits productivity in many agricultural systems, and in natural aquatic and terrestrial ecosystems. The soluble fraction is readily ‘bioavailable’. When phosphorus is added to the soil in recycled water, most of the soluble fraction becomes adsorbed onto soil particles, is retained in the surface soils and is sparingly available to plants. Transport of phosphorus through soil is often limited because of strong sorption to the soil matrix, uptake of dissolved phosphorus by plants and micro-organisms and reaction with soil minerals. Leaching can occur where phosphorus concentrations are elevated. In water and wastewater, phosphorus will adsorb to sediments and sewage sludge.

In freshwater systems, phosphorus occurs in three forms: (i) inorganic phosphorus, (ii) particles of organic phosphorus, and (iii) dissolved organic phosphorus. Aquatic algae and plants use an inorganic form of phosphorus for their nutrition. In most lakes and rivers, phosphorus is the primary nutrient that limits the growth of algae and plants. In some systems, the nutrient form of phosphorus is taken up very quickly and so is difficult to measure accurately.

Excessive phosphorus in a freshwater system increases plant and algal growth. This can lead to: changes in number and type of plants and animals; increases in animal growth and size; increases in turbidity; more organic matter falling to the bottom of the system in the form of dead plants and animals; and losses of oxygen in the water. When there is no oxygen at the bottom of a freshwater system, phosphorus that previously had been locked in the sediment can be released back into the water. This is called internal loading and exacerbates the problem of excessively high productivity.
B10.3 Ecological Effects and Guidelines

Key environmental issues associated with the presence of phosphorus is the eutrophication of soil and surface water and potential toxic effects on plants (sensitive species).

Phosphorus can be toxic, but toxicity occurs rarely in nature and is generally not a concern. Of more concern are the indirect effects of phosphorus. All algae and plants require phosphorus to grow. Elevated phosphorus levels, however, can increase a freshwater system’s productivity and result in large amounts of organic matter falling to the bottom. Bacteria and other organisms decompose this matter and in the process use a lot of oxygen. In very productive freshwater systems, the oxygen levels can be in such short supply that fish kills occur. A type of algae, called cyanobacteria, grows particularly well in high levels of phosphorus. Cyanobacterial blooms can cause a range of water quality problems, including summer fish kills, bad odours, and tainted drinking water. Some cyanobacteria produce toxins that can kill livestock and wildlife (EC, 2005).

The principal force driving significant changes in soil chemistry is soil pH. Phosphorus interacts strongly with iron, calcium and aluminium in the soil at different pH levels. In acid soils (pH <7 in a CaCl₂ extract), phosphorus and iron combine in a form that makes both unavailable for plant uptake. This interaction is the key to understanding potential iron deficiency (which may be a form of phosphorus toxicity) in plants.

Various factors, such as flow, light, turbidity, temperature, nitrogen levels, zooplankton grazing, etc., can limit plant and algae growth. Thus, it is not possible to identify absolute total phosphorus concentrations for aquatic environments that will prevent plant and algae blooms.

ANZECC/ARMCANZ (2000) have outlined a range of trigger values for total phosphorus in upland rivers, lowland rivers, freshwater lakes and reservoirs, wetlands, estuaries and marine environments relevant to south-eastern Australia, tropical Australia, south-western Australia and central Australia. In additional guidelines are also available for total phosphorus in irrigation water and phosphates in aquiculture water. These guidelines should be utilised, where relevant, for the screening of water quality data with respect to the protection of the environment.

No guidelines are available for phosphorus in soil. Ontario has established a sediment guideline for total phosphorus of 600 mg/kg.

B10.4 Health Effects

General: Pure red phosphorus does not usually represent a significant health hazard. It is essentially non-volatile, insoluble in water, and poorly absorbed into the body. Red phosphorus does react with water vapour and oxygen in air to form extremely toxic phosphine gas, phosphorus oxyacids, white phosphorus, and phosphoric acid. Red phosphorus may also be contaminated with white phosphorus (White-P) and/or yellow phosphorus (Yellow-P, a form of White P that contains impurities), which are more toxic.

Health effects associated with exposure to red phosphorus include respiratory effects associate with inhalation of dusts, skin irritation, eye irritation. More significant effects are noted for white phosphorus which may be present as a contaminant in red phosphorus. Health effects associated with phosphorus include liver and kidney damage, skin and eye effects (including burning), gastrointestinal effects, anaemia and cardiovascular effects. Long term ingestion of red phosphorus contaminated with white phosphorus may result in jaw bone degeneration.

Main Risks and Target Organs: Target organs are respiratory, skin, liver and kidney.

Carcinogenicity and Genotoxicity: No data is available with respect to carcinogenicity and genotoxicity. The USEPA has classified white phosphorus, Group D – not classifiable as to human carcinogenicity.
Dermal Absorption: No dermal absorption data is available for phosphorus hence a conservative default value of 10% has been assumed in the absence of other data.

B10.5 Quantitative Toxicity Data

No data is available for red phosphorus, hence available data on phosphorus (as white or yellow phosphorus) has been used as a surrogate for the presence of phosphorus from the use of acids and red phosphorus. It is noted that red phosphorus is noted to be significantly less toxic than white or yellow phosphorus, however as red phosphorus can be contaminated with white or yellow phosphorus and no toxicity data is available for red phosphorus date from the more toxic phosphorus forms is assumed to be representative. This is expected to result in the derivation of be conservative guidelines.

The following data are available for phosphorus as a total from red phosphorus and phosphorus from acid residues:

- The USEPA (2009b) has derived an oral RfD of 0.00002 mg/kg/day based on a reproductive study with white phosphorus;
- The Texas Commission on Environmental Quality (TCEQ, 2009) has derived an interim chronic effects screening level (ESL) of 0.0001 mg/m³ for yellow phosphorus based on the protection of health effects. A value (less conservative than for yellow phosphorus) is also available for phosphorous acid as a mist which is more relevant for exposures during use or manufacture rather than environmental exposures.

Phosphates are found in foods as naturally occurring components of biological molecules and as food additives in the form of various phosphate salts. Phosphorus is an essential nutrient in the diet and hence health effects can occur if phosphorus intakes are insufficient. As no specific data is available, it has been assumed that background intakes of phosphorus may contribute 50% of the RfD and ESL adopted.
B11 N-Methylformamide

The following is summarised from HSDB (2009).

B11.1 General

N-Methylformamide (NMF) is used as an intermediate in the synthesis of pesticides; extraction solvent for aromatic hydrocarbons. It is also present in cigarette smoke. In clan labs NMF is used in the manufacture of MDMA.

Appearance: NMF is a colourless viscous liquid that decomposes on heating or burning to produce nitrogen oxides. Reacts with strong oxidants and attacks some forms of rubber and plastics.

Chemical/Physical Properties: NMF (CASRN: 123-39-7) has the following properties (HSDB):

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula</td>
<td>C₂H₅NO</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>59.07</td>
</tr>
<tr>
<td>Log Kow</td>
<td>-0.97</td>
</tr>
<tr>
<td>Koc</td>
<td>7 L/kg</td>
</tr>
<tr>
<td>Soluble in water</td>
<td>water, alcohols, benzene and acetone</td>
</tr>
<tr>
<td>Solubility in water</td>
<td>1000000 mg/L</td>
</tr>
<tr>
<td>Vapour pressure</td>
<td>0.253 mmHg at 25°C</td>
</tr>
<tr>
<td>Henry's Law</td>
<td>2.0X10⁻¹ atm-m³/mole</td>
</tr>
</tbody>
</table>

B11.2 Exposure, Fate and Transport

Indoor Surfaces: NMF, if spilled indoors, may remain on surfaces for some time and degradation may occur under some conditions. Therefore its presence in surface residues should be considered.

Air: If released to air, NMF will exist solely as a vapour in the ambient atmosphere. Vapour-phase NMF will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 57 hours.

Soil: If released to soil, NMF is expected to have very high mobility based upon an estimated Koc. Volatilisation from dry or moist soil surfaces is not expected to be an important fate process based upon the estimated Henry's Law constant and vapour pressure. NMF has been shown to biodegrade by microorganisms obtained through soil enrichment.

Water: If released into water, NMF is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. NMF may biodegrade in the aquatic environment. Volatilisation from water surfaces is not expected to be an important fate process based upon this compound's estimated Henry's Law constant.

Bioaccumulation: An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low.

B11.3 Ecological Effects and Guidelines

Review of the available data on NMF (USEPA, 2007) indicates that the chemical is likely to be of low concern with respect to acute toxicity to algae, invertebrates or fish. As NMF is expected to degrade in the soil and water environment, is volatile and does not bioaccumulate in aquatic species the potential for chronic ecotoxicity effects are considered low. No long-term ecological effects have been identified and no guidelines are available.
B11.4 Health Effects

**General:** NMF is a metabolite of dimethylformamide (DMF), an industrial solvent. The major potential of NMF is its ability to cause liver damage. NMF is an eye and respiratory irritant. NMF is also teratogenic and embryotoxic. Based on limited data NMF appears to be more toxic than its methyl homologue, dimethylformamide, or its N-desmethyl analogue, formamide. However no quantitative toxicity data is available for NMF and hence data available for DMF is considered representative. This may need to be revised if more a more detailed assessment of NMF studies is undertaken or more data becomes available.

**Main Risks and Target Organs:** Target organs are liver and developmental toxicity.

**Carcinogenicity and Genotoxicity:** No studies are available for NMF. IARC has reviewed the supporting chemical, DMF, for carcinogenicity and categorised it as Group 3, not classifiable as to its carcinogenicity to humans. This is based on inadequate evidence in humans and evidence from animal studies (inhalation) showing no increase in tumour incidence above controls. With respect to DMF, available studies indicate that it is not genotoxic.

**Dermal Absorption:** Dermal absorption of NMF and DMF is expected to be high for both the vapour and liquid. Available data on DMF suggests dermal absorptions of 26% to 62%. A conservative dermal absorption value of 60% has been assumed for NMF (CERI, 2007).

B11.5 Quantitative Toxicity Data

As no quantitative data is available for NMF, data from DMF has been adopted as a surrogate. Chronic inhalation guidelines are available from the WHO (2001b) of 0.1 mg/m³, Health Canada (2001) of 0.1 mg/m³, the UESPA (2009b) of 0.03 mg/m³ and OEHHA (2009c) of 0.08 mg/m³. While these values could be reviewed in detail, it is noted that the toxicity of NMF may be higher than for DMF and hence the lower value available from the USEPA, an RfC of 0.03 mg/m³ has been selected as a surrogate for the assessment of inhalation exposures to NMF.

Background intakes of NMF are assumed to be negligible for the general population. It is noted that background intakes may be more significant for smokers as NMF is found in cigarette smoke.
B12 Methylamine
The following is summarised from HSDB (2009).

B12.1 General
Methylamine is an aliphatic amine that is used in organic synthesis and tanning. It is also used as an intermediate in the synthesis of pharmaceuticals, pesticides, solvents and other compounds. In clan labs methylamine is used in the manufacture of MDMA, methamphetamine and pseudo/ephedrine (a precursor).

Appearance: Methylamine is a colourless, flammable gas or compressed liquefied gas. It is also frequently encountered in solution with water and is also soluble in alcohol and ether.

Chemical/Physical Properties: Methylamine (CASRN: 74-89-5) has the following properties (HSDB):

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula</td>
<td>CH₃N</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>31.06</td>
</tr>
<tr>
<td>Log Kow</td>
<td>-0.57</td>
</tr>
<tr>
<td>Koc</td>
<td>389 to 449 L/kg</td>
</tr>
<tr>
<td>Soluble</td>
<td>water and solvents (including ethanol, benzene and acetone)</td>
</tr>
<tr>
<td>Solubility in water</td>
<td>1250000 mg/L at 25°C</td>
</tr>
<tr>
<td>Vapour pressure</td>
<td>2650 mmHg at 25°C</td>
</tr>
<tr>
<td>Henry’s Law</td>
<td>1.11x10⁻⁹ atm-m³/mole at 25°C</td>
</tr>
</tbody>
</table>

It is noted that chemical properties required for the quantification of volatilisation are not available for methylamine, values available for dimethylamine (USEPA 2004) have been adopted. The values adopted are the air diffusion coefficient (0.12 cm²/s) and water diffusion coefficient (1.3x10⁻⁵ cm²/s).

Odour and Taste: Methylamine has a strong fish/ammonia type of odour. The odour threshold is listed as 0.021 ppm (recognition) which becomes strong from 20 to 100 ppm.

B12.2 Exposure, Fate and Transport

Indoor Surfaces: Methylamine is a gas at room temperature and pressure and hence if an aqueous solution spilled indoors it is expected to volatilise readily from surfaces. Hence long-term issues that may be associated with methylamine on surfaces are not considered to be significant. As a gas it may be absorbed by porous materials such as furnishings, walls and floors with the potential for off-gassing over time.

Air: If released to air, methylamine will exist solely as a gas in the ambient atmosphere. Gas-phase methylamine will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 18 hours. Gas phase methylamine is degraded in the atmosphere by a reaction with ozone, with a half life of 540 days. It is not susceptible to direct photolysis by sunlight.

Soil: If released to soil, methylamine is expected to have moderate mobility based upon an estimated Koc. It is expected to exist almost entirely in the cation form in the environment where volatilisation is not significant from the cation form. However volatilisation from dry soils is expected to be significant.

Water: If released into water, methylamine is expected to adsorb to suspended solids and sediment based upon the estimated Koc. It is expected to exist almost entirely in the cation form in the environment where volatilisation from water is not significant from the cation form.

Degradation: In both soil and water, methylamine is expected to degrade.
Bioaccumulation: An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low.

B12.3 Ecological Effects and Guidelines

Few data or studies are available with respect to the potential effects of methylamine on the terrestrial or aquatic environment. Methylamine may be harmful to wildlife in high concentrations (UK, 2009). When released to the air it will degrade relatively quickly through reaction with hydroxyl radicals (days). As a volatile organic compound (VOC), methylamine can be involved in reactions with other air pollutants that can form ground-level ozone, which can damage to crops and materials. High concentrations (not considered likely at former clan labs) would need to be present for these effects to be significant. As it is expected to degrade in the soil and water environment, is a gas at room temperature and pressure and does not bioaccumulate in aquatic species the potential for chronic ecotoxicity effects are considered low. No long-term ecological effects have been identified and no guidelines are available.

B12.4 Health Effects

General: Key health effects associated with exposure (particularly acute exposures) to methylamine, as with other aliphatic amines, include irritation to the eye, skin and respiratory system. Other effects identified include cardiovascular and liver effects. Aliphatic amines have been identified in breast milk.

Main Risks and Target Organs: Target organs are skin and respiratory system.

Carcinogenicity and Genotoxicity: There is no evidence methyamine is associated with carcinogenic or genotoxic effects.

Dermal Absorption: No data is available on dermal absorption of methylamine or other aliphatic amines. The USEPA (2004) guidance on dermal exposure assessment suggests that for volatile chemicals dermal absorption can be effectively considered negligible as the chemical is not expected to remain on the skin long enough to be absorbed. Prior to this recommendation the dermal absorption of volatile organic compounds was taken to be 0.05% to 3% (depending on the vapour pressure) based on experimental data (USEPA 1995). On this basis a value of 3% has been adopted for methylamine.

B12.5 Quantitative Toxicity Data

No quantitative public health data is available on methylamine. A dose-response relationship for aliphatic amines is presented in HSDB however no specific data is available for methylamine. It is noted that design criteria for the purpose of modeling air emissions from sources in NSW, Victoria and SA area available that provides 3-minute or 1 hour average criteria for methylamine and dimethylamine. These are typically based on an odour threshold, however the VicEPA (2001) has established design ground level concentrations of 0.33 ppm (as 1 hour average) for both methylamine and dimethylamine, noted to be based on toxicity. These are relevant to the assessment of modeled air emissions and not specifically relevant to ambient/indoor air concentrations. No basis for the derivation is provided and hence a more robust value is required for the assessment of chronic exposures by the general public.

The only available guideline for methylamine is the occupational TWA available from Safe Work Australia (2009) of 10ppm or 13 mg/m³. The value is derived by OSHA based on data from an occupational study (duration unknown) where no irritation effects were observed at 10ppm. Following guidance presented in USEPA (2009) the occupational guideline can be converted to a public health value by consideration of an exposure adjustment factor of 4.2 (based on converting from an 8 hour exposure to 24 hours and adjusting from a 5 day week to a 7 day week) and an uncertainty factor. Based on the limited information available it is considered relevant to apply an uncertainty factor of 1000 (10 for intraspecies variability [address sensitive}
members of the population, 10 for the duration of the study [unknown] and 10 for database deficiencies). Applying these to the available TWA a **TC of 0.003 mg/m$^3$** is derived.

It is noted that the derived TC is similar to the RfC available for triethylamine of 0.007 mg/m$^3$ (available from USEPA, 2009b).

Background intakes of methylvamine are not known. Given most significant exposures are expected to occur in an occupational environment, potential background intakes by the general public are considered to be negligible.
**B13 Nitroethane**  
The following is summarised from HSDB (2009).

**B13.1 General**

Nitroethane is a solvent used for cellulose esters, vinyl and other resins and waxes. It is also used as a nail polish remover, propellant for military use and an intermediate in pharmaceutical and pesticide manufacture. In clan labs nitroethane is used in the manufacture of P-2-P.

**Appearance:** Nitroethane is an oily colourless liquid.

**Chemical/Physical Properties:** Nitroethane (CASRN: 79-24-3) has the following properties (HSDB):

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula</td>
<td>C₂H₅NO₂</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>75.07</td>
</tr>
<tr>
<td>Log Kow</td>
<td>0.18</td>
</tr>
<tr>
<td>Koc</td>
<td>30 L/kg</td>
</tr>
<tr>
<td>Soluble</td>
<td>water, acetone, chloroform, dilute alkali</td>
</tr>
<tr>
<td>Solubility in water</td>
<td>48000 mg/L</td>
</tr>
<tr>
<td>Vapour pressure</td>
<td>20.8 mmHg at 25°C</td>
</tr>
<tr>
<td>Henry's Law</td>
<td>4.76x10⁻⁵ atm-m³/mole at 25°C</td>
</tr>
</tbody>
</table>

It is noted that chemical properties required for the quantification of volatilisation are not available for nitroethane, values available for nitromethane have been adopted.

**Odour and Taste:** Nitroethane has a mild, fruity, somewhat disagreeable chloroform-like odour. The odour threshold is listed as 163 ppm.

**B13.2 Exposure, Fate and Transport**

**Indoor Surfaces:** Nitroethane is volatile and hence if spilled indoors it is expected to volatilise from surfaces. Hence long-term issues that may be associated with nitroethane on surfaces are not considered to be significant.

**Air:** If released to air, nitroethane will exist solely as a vapour in the ambient atmosphere. Vapour-phase nitroethane will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 110 days.

**Soil:** If released to soil, nitroethane is expected to have very high mobility based upon an estimated Koc of 30 L/kg. Volatilisation from dry or moist soil surfaces is expected to be an important fate process based upon the estimated Henry's Law constant and vapour pressure. Nitroethane may degrade in aerobic conditions, however the data is conflicting.

**Water:** If released into water, nitroethane is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Nitroethane may biodegrade under aerobic conditions, but not under anaerobic conditions. Volatilisation from water surfaces is expected to be an important fate process based upon this compound's estimated Henry's Law constant.

**Bioaccumulation:** An estimated BCF of 1 suggests the potential for bioconcentration in aquatic organisms is low.
B13.3 Ecological Effects and Guidelines

Few data or studies are available with respect to the potential effects of nitroethane on the terrestrial or aquatic environment. Acute issues associated with hazardous properties of nitroethane (flammable, explosive and reactive) may be of importance for acute environmental exposures. As it is expected to degrade in the soil and water environment, is volatile and does not bioaccumulate in aquatic species the potential for chronic ecotoxicity effects are considered low.

Nitroethane was included as one of the chemicals evaluated with respect to ecotoxicity in soil by Pal and Kirkbride (2009). The EC50 (defined as a 50% reduction in dehydrogenase activity in spiked soil relative to unspiked soil) for the soil materials tested with nitroethane ranged from 0.6 to 1.6 g/kg.

No long-term ecological effects have been identified and no guidelines are available.

B13.4 Health Effects

**General:** Methemoglobinemia results from accidental oral exposure to nitroethane. Because methemoglobinemia may be delayed, individuals who ingest nitroethane should be monitored closely for at least 24 hours after ingestion. If inhaled nitroethane can cause respiratory effects including irritation and difficulty breathing. Nitroethane is considered irritating to skin and eyes. Other effects include CNS depression, liver and kidney effects.

**Main Risks and Target Organs:** Target organs are respiratory and central nervous system.

**Carcinogenicity and Genotoxicity:** There is no evidence nitroethane is associated with carcinogenic or genotoxic effects.

**Dermal Absorption:** No dermal absorption data is available for nitroethane. The USEPA (2004) guidance on dermal exposure assessment suggests that for volatile chemicals dermal absorption can be effectively considered negligible as the chemical is not expected to remain on the skin long enough to be absorbed. Prior to this recommendation the dermal absorption of volatile organic compounds was taken to be 0.05% to 3% (depending on the vapour pressure) based on experimental data (USEPA 1995). On this basis a value of 3% has been adopted for nitroethane.

B13.5 Quantitative Toxicity Data

The only available inhalation guideline is available from the Texas Commission on Environmental Quality (TCEQ, 2009) where an interim chronic effects screening level (ESL) of \(0.31 \text{ mg/m}^3\), based on the protection of health effects is available.

Background intakes of nitroethane are not known. Given most significant exposures are expected to occur in an occupational environment, potential background intakes by the general public are considered to be negligible.
B14  Boron and compounds


B14.1  General

In clan labs the use of sodium borohydride (CASRN: 16940-66-2) as a reagent in the synthesis of MDMA and pseudo/ephedrine may result in the presence of inorganic boron compounds. The presence of boron ions an inorganic boron compounds provides an indication of the former use and presence of sodium borohydride.

Boron (B) (CASRN: 7440-42-8) is the 51st most common element present in compounds in the earth's crust, and is found at an average concentration of 8 mg/kg. Boron is generally found in nature bound to oxygen, and is never found as the free element. The most common boron containing ores are alkali and alkaline earth borates, including borax, kernite, colemanite, and ulexite, and as borosilicate minerals. Boron containing mineral are concentrated in arid regions. Borates are found in ocean water, sedimentary rocks, coal, shale, and soils. Once a tetra-, di-, meta-, ortho-, or pyroborate salt dissolves in a buffered solution, one borate cannot be distinguished on chemical or toxicological grounds, from any one of the others.

Appearance: Elemental boron may be present as filaments, powder, whisker or single crystals. It is insoluble in water.

B14.2 Exposure, Fate and Transport

Indoor Surfaces: As inorganic boron compounds are not volatile they are expected to remain in surface indoors. Hence the potential for boron to be present in surface dust and residues within a former clan lab needs to be considered.

Air: Inorganic boron compounds (with the exception of hydroboranes) are expected to be non-volatile and will exist solely in the particulate phase in the ambient atmosphere. Particulate-phase boron compounds will be removed from the atmosphere by wet and dry deposition.

Soil: The chemistry of boron is dominated by its tendency to form stable bonds with electronegative atoms, especially oxygen. Reduced boron compounds (halides, hydrides, alkyls and aryls) tend to oxidise and hydrolyse readily, and would be expected to be converted into various boron-oxide compounds in the environment. The extent of boron adsorption depends on the chemical composition of the soil, pH, salinity, organic matter content, iron and aluminium oxide content, iron- and aluminium-hydroxy content, and clay content. The adsorption of boron to soils can be variable and range from being fully reversible to irreversible. The adsorption of boron is expected to be most significant for soils that contain high concentrations of amorphous aluminium and iron oxides and hydroxides. Inorganic boron compounds are non-volatile and would not be expected to volatilise from moist or dry soil surfaces. No biotransformation processes have been reported for boron compounds.

Water: In aqueous solution, boron is normally present as boric acid and borate ions, with the dominant form of inorganic boron as undissociated boric acid in natural aqueous systems. In aqueous solution, boric acid acts as an electron acceptor, accepting hydroxide from water to form \((\text{B(OH)}_4)^-\) ion. In concentrated solutions (>0.1 M boric acid) polymeric species are formed. Boron compounds in water may be absorbed by soils and sediments. The extent of boron adsorption depends on the pH of the water where the greatest adsorption is observed at pH 7.5-9.0. Since the adsorption of boron is expected to be most significant for soils that contain high concentrations of amorphous aluminium and iron oxides and hydroxides, sediments with these characteristics may also strongly adsorb boron compounds.

Bioaccumulation: Boron does not accumulate in aquatic species; however uptake by plants can occur. Boron but does not magnify through the food-chain.
B14.3 Ecological Effects and Guidelines

Boron is a naturally occurring element found combined with other elements (primarily oxygen) throughout the environment. It is found in the Earth’s crust, with the majority of readily available forms occurring in the ocean. It is estimated that more boron is released into the environment by natural weathering than from anthropogenic sources. Boron is present in surface water and groundwater and is readily adsorbed on the surfaces of soil particles. Boron is present in food, beverages, and drinking-water (WHO, 1998).

Boron is an essential micronutrient for cyanobacteria and diatoms. Toxicity to aquatic organisms, including vertebrates, invertebrates, and plants can vary depending on the organism’s life stage and environment. Early stages are more sensitive to boron than later ones, and the use of reconstituted water shows higher toxicity in lower boron concentrations than natural waters. In mammals, excessive consumption can adversely affect growth, reproduction or survival (WHO, 1998).

Boron is an essential micronutrient for higher plants, with interspecies differences in the levels required for optimum growth. In plants, there is only a narrow margin between boron deficiency and excess boron uptake leading to toxicity (WHO, 1998).

With respect to the protection of the environment the following screening level guidelines/benchmarks are available from ANZECC/ARMCANZ (2000) for boron:

- Freshwater high reliability trigger value of 0.37 mg/L (95% protection level); and
- No guideline has been established for marine water; however it is recommended that the background concentration in seawater of 5.1 mg/L be used as a low reliability guideline.

Other screening level guidelines/benchmarks available for boron as listed by RAIS (2009) from USEPA, Canada and Dutch peer reviewed sources include the following for soil:

- USEPA (Region VI and ORNL) Benchmark for protection of plants = 0.5 mg/kg
- USEPA (ORNL) Benchmark for protection of soil microbes = 20 mg/kg

B14.4 Health Effects

General: Acute exposure is associated with short-term irritant effects on the upper respiratory tract, nasopharynx, and eye. Health effects associated with acute ingestion of boron include liver, kidney, central nervous system, gastrointestinal effects, skin lesions and respiratory effects. Animal studies show ingestion of boron is associated with developmental and reproductive toxicity, which are considered the most sensitive targets for boron toxicity. Other effects identified following ingestion include liver effects and haematological alterations. The primary health effects associated with dermal exposure are irritation of the eyes and reversible skin changes.

Boron compounds are absorbed from the respiratory and gastrointestinal tracts. Dermal absorption of boron across intact skin is negligible.

Borates are essentially complete absorbed by the body following ingestion. Once in the body boron is distributed passive diffusion throughout the body fluids. In contrast to soft tissues and blood, bone shows selective uptake of boron and significantly longer retention times. Boron is poorly metabolised in the body and has a relatively short half-life in the body (plasma half-life is report to be 21 hours).

Main Risks and Target Organs: Developmental, reproductive and respiratory systems.
Carcinogenicity and Genotoxicity: Based on the lack of human data and the limited animal data, boron has not been classified by IARC or USEPA. No mutagenic activity has been indentified in available studies on boric acid or borax (WHO, 2009b).

Dermal Absorption: Limited data is available on the dermal absorption of boron, however is expected to be negligible for intact skin. The default absorption of 1% for metals (USEPA, 1995) is therefore assumed. This is consistent with that used in the derivation of the current soil HIL (Mangas, 1998).

B14.5 Quantitative Toxicity Data

The following data is available for boron:

- **TDI of 0.2 mg/kg/day** proposed by the WHO (2009b). The value is consistent with that adopted (as rounded) from the previous review by the WHO (2008), used in the derivation of the current soil HIL (Mangas, 1998) and the oral RfD available from the USEPA (2009b).

- An interim air ESL is available from the TCEQ (2009) for boron of 0.005 mg/m³ based on health effects. It is also noted that TCEQ also has an interim ESL of 0.0001 mg/m³ for sodium borohydride. This value is considered relevant where sodium borohydride (powder) is present in significant quantities for dust levels to be of concern. With respect to the assessment of former clan labs where remediation is expected to occur the value available for boron is considered appropriate.

Based on available data associated with intakes of boron from food and water (Mangas 1998 and 2009b) background intakes have been estimated to be 20% to 60% of the TDI. A value of 60% has been used in this assessment.
B15 Mercury (inorganic)

B15.1 General
Mercury is a heavy metal which exists in three oxidation states: 0 (elemental), +1 (mercurous) and +2 (mercuric). As well as the common mercurous and mercuric inorganic salts, mercury can also bind covalently to at least one carbon atom. Thus the most commonly encountered exposures associated with mercury are with elemental mercury, inorganic mercuric compounds and methyl mercury. In clan labs the most common form of mercury is mercuric chloride used as a reagent in methamphetamine synthesis. Hence this summary addresses the key aspects of inorganic mercuric chloride.

Appearance: Mercuric chloride is an odourless white solid, crystals, granules or powder).

Chemical/Physical Properties: Mercuric chloride (CASRN: 7487-94-7) has the following properties (HSDB, ATSDR and RAIS):

- Molecular formula: HgCl₂
- Molecular weight: 271.52
- Log Kow: 0.22
- Koc: NA
- Solubility: 73100 in water at 20°C
- Vapour pressure: NA
- Henry’s Law constant: 7.09x10⁻¹⁰ atm.m³/mol at 25°C

B15.2 Exposure, Fate and Transport

Indoor Surfaces: As inorganic mercury is not volatile and is persistent it is expected to remain in surface should it be spilled. Hence the potential for inorganic mercury to be present in surface dust and residues within a former clan lab needs to be considered.

Air: Mercury is released into the atmosphere from anthropogenic emissions as either vapour (elemental or oxidised mercury) or as particles (oxidised compounds). Natural emissions are mainly in elemental mercury form. Mercury may reside in the atmosphere for about one year, allowing global circulation systems to transport mercury emissions from source of emission to anywhere on earth before transformation and deposition take place. Mercury is transferred from the atmosphere to the earth’s surface via wet or dry deposition.

Soil: The majority of mercury in surface soil is in the form of oxidised mercury complexes/compounds; however, a small fraction is methyl mercury and elemental mercury. Mercury complexes deposited in soils can be transformed back into gaseous mercury by light and humic substances and re-enter the atmosphere. Studies have consistently shown that plant uptake is negligible and consequently, animals foraging on plants accumulate little mercury. In addition to direct deposition, mercury can also reach water from soil run-off, although the amount partitioning to run-off is expected to be small since mercury binds to soil; run-off is probably in the form of suspended sediments.

Water: Once in water, mercury can enter the food chain, settle into sediment, or volatilise back into the atmosphere. Entrance into the food chain begins with bacteria in water which can take up mercury in its inorganic form and metabolise it to methyl mercury. The methyl mercury-containing bacteria may be consumed by the next level in the food chain, or they may excrete the methyl mercury into the water where it can absorb to plankton, which are also consumed by the next level in the food chain. Even small environmental concentrations of mercury in water can readily accumulate to potentially harmful concentrations.
in fish and fish-eating people. Fish species higher in the food chain have much higher mercury concentrations than species that are lower on the food chain.

**Bioaccumulation:** On the basis of the potential for long-range transport, persistence in water, soil and sediment, bioaccumulation, toxicity and ecotoxicity, mercury is considered persistent and is addressed in the 1998 UN-ECE Convention on Long-Range Transboundary Air Pollution on Heavy Metals (UN-ECE, 1998). The United Nations Environment Programme (UNEP) Governing Council concluded, at its 22nd session in February 2003, after considering the key findings of the Global Mercury Assessment report, that there is sufficient evidence of significant global adverse impacts from mercury to warrant further international action to reduce the risks to humans and wildlife from the release of mercury to the environment. The UN Governing Council decided that national, regional and global actions should be initiated as soon as possible and urged all countries to adopt goals and take actions, as appropriate, to identify populations at risk and to reduce human-generated releases. While mercury is not listed as one of the 12 chemicals listed in the Stockholm Convention on Persistent Organic Pollutants (POPs), it chemical meets criteria listed (annex D) in the convention for consideration as persistent and bioaccumulative.

**B15.3 Ecological Effects and Guidelines**

Current literature indicates that mercury (Hg) in the environment, including groundwater, exhibits complex behaviour that affects both its mobility and potential toxicity. Mercury has a low solubility in water; however, it also has the potential to form multiple species in the environment, which can lead to increased total mercury concentrations in aqueous systems. The relative toxicity of mercury is also dependent on the form in which it occurs, which, in groundwater, is dependent on: biogeochemical processes; partitioning between solids, groundwater, and vapour; and complexation with dissolved organic and inorganic ligands. Redox, pH conditions, and groundwater composition are, consequently, all important components of determining the likely form, and therefore, potential fate of mercury in the environment.

There are a number of pathways by which mercury can enter the terrestrial, fresh or marine water environments. Once in the soil or water system, complexation and transformation processes will occur. Once entering a water body, mercury can remain in the water column (further partitioned as dissolved or attached to suspended material), be lost from the water body through drainage water, undergo transformation to more volatile elemental mercury or dimethyl mercury and re-volatilise into the atmosphere, settle into the sediment or be taken up by aquatic biota.

Methylation is a key step in the entrance of mercury into the food chain. The biotransformation of inorganic mercury species to methylated organic species in water bodies can occur in the sediment and the water column. Methylmercury is very bioavailable and accumulates in fish through the aquatic food web; nearly 100% of the mercury found in fish muscle tissue is methylated.

Bioaccumulation of mercury has been shown to occur in aquatic plants, invertebrates, insects, scavengers, fish and mammals. Benthic organisms are particularly susceptible to bioaccumulation of mercury (especially methyl mercury) due to their close ties to the geochemistry of the sediments that they live on and in. Uptake occurs primarily via dissolved-phase mercury in interstitial pore waters, with the mass of mercury bound in the sediment serving as a source. Studies have shown that bioaccumulation of mercury in invertebrate benthic organisms is relatively low in comparison to higher trophic level organisms such as mussels, shrimp, crabs and fish. This is due in part to the ability of the different organisms to eliminate mercury from their systems following initial uptake.

Methylmercury is preferentially bioaccumulated in organisms, although bioaccumulation of mercury in the inorganic has also been documented. Biomagnification of mercury through the food chain as methyl mercury has been demonstrated in high trophic-level piscivorous fish and can be especially significant in marine mammals that feed on these fish, although this varies widely between species.
The bioavailability of mercury to an organism is dependent both on the physical and chemical nature of the impacted media (e.g., sediment) and the ecological habits (e.g., feeding) and physiological characteristics of the organism (e.g., physiological aspects that promote bioaccumulation). The same geochemical factors that govern the fate and transport of mercury in the environment affect bioavailability to organisms. Characteristics of soil, sediment or water that promote the bioavailability of mercury in organisms include high concentrations of methyl mercury, low concentration of organic carbon (both dissolved and particulate) available for binding, low capacity to form charged inorganic complexes, and moderate redox conditions.

Bioavailability for birds and mammals is highly variable, depending in part on feeding habits. Primary uptake in earthworms plays an important role in bioaccumulation of mercury in terrestrial food chains. Absorption of methyl mercury and HgCl₂ in the gastro-intestinal tract of birds and mammals is greater than for inorganic forms. Bioaccumulation is generally higher in predators in comparison to herbivores.

The toxicity of mercury is dependent on the form of mercury present, geochemical factors such as temperature, salinity and pH, the sensitivity of individual species at different growth stages and the tolerance of individual organism. Toxicological effects of mercury on aquatic organisms can include neurological damage, reproductive impairment, growth inhibition, developmental abnormalities, and altered behavioural responses. Reproductive endpoints have generally been shown to be more susceptible to mercury toxicity than growth or survival endpoints. Methyl mercury has been shown to be significantly more toxic to aquatic life than inorganic forms of mercury. Toxicity has also been shown to increase with increasing temperature, decreasing oxygen content and reduced salinity in marine environments. The complexity of mercury toxicity in the environment with respect to these and other factors often necessitates the collection of site-specific data to accurately assess biological effects, rather than reliance on chemical data and generic screening levels or model results.

With respect to the protection of the environment the following screening level guidelines/benchmarks are available from ANZECC/ARMCANZ (2000):

- Interim urban Ecological Investigation Level (EIL) of 1 mg/kg in soil is available from NEPM (1999b);
- Interim sediment quality value of 1 mg/kg for total mercury;
- Freshwater high reliability trigger value of 0.0006 mg/L (95% protection) for inorganic mercury;
- For slightly to moderately disturbed systems a trigger level for inorganic mercury in fresh water of 0.00006 mg/L is recommended (99% protection);
- Marine water high reliability trigger value of 0.0004 mg/L (95% protection) for inorganic mercury; and
- For slightly to moderately disturbed systems a trigger level for inorganic mercury in marine water of 0.0001 mg/L is recommended (99% protection). This value is the same as that recommended by Canada to protect consumers of fish.

No Trigger Value is provided for methyl mercury in ANZECC/ARMCANZ (2000). Consideration of the potential for methyl mercury to be present in environmental media should be noted and addressed in a site-specific assessment where required. Relevant guidelines for methyl mercury should be obtained where required.

**B15.4 Health Effects**

**General:** Current literature indicates that mercury in the environment, including groundwater, exhibits complex behaviour that affects both its mobility and potential toxicity. Mercury has a low solubility in water; however, it also has the potential to form multiple species in the environment, which can lead to increased total mercury concentrations in aqueous systems. The relative toxicity of mercury is also dependent on the form in which it occurs, which, in groundwater, is dependent on: biogeochemical processes; partitioning between solids, groundwater, and vapour; and complexation with dissolved organic and inorganic ligands.
Redox, pH conditions, and groundwater composition are, consequently, all important components of determining the likely form, and therefore, potential fate of mercury in the environment.

Limited data is available concerning the absorption of inhaled mercury compounds; however it is expected to be determined by the size and solubility of the particles. Absorption of ingested inorganic mercury has been estimated to be approximately 5 to 10% with absorption be children greater than for adults. Absorption via the dermal route is considered low and not significant, however indirect evidence of dermal absorption is provided by case studies following dermal application of ointments that contained inorganic mercury salts.

Inorganic mercury compounds are rapidly distributed to all tissues following absorption. The fraction that crosses the blood-brain and foetal barriers is less than for elemental mercury due to poor lipid solubility. The major site of systemic deposition of inorganic mercury is the kidney. Most inorganic mercury is excreted in the urine or faeces.

Main Risks and Target Organs:  Kidney and central nervous system.

Acute Effects:  Acute exposure to high concentrations of ingestion of inorganic mercury has been associated with gastrointestinal damage, cardiovascular damage, acute renal failure and shock.

Chronic Effects:  The kidney is the critical organ associated with chronic exposure to inorganic mercury compounds. The mechanism for the end toxic effect on the kidney, namely autoimmune glomerulonephritis, is the same for inorganic mercury compounds and elemental mercury and results in a condition sometimes known as nephrotic syndrome.

There is some evidence that inorganic mercury may cause neurological effects, particularly associated with studies of mercuric chloride. Reproductive and developmental effects have been observed in rats given mercuric chloride.

Carcinogenicity and Genotoxicity:  IARC have considered inorganic mercury compounds not classifiable as to human carcinogenicity. The USEPA has classified mercuric chloride as a possible human carcinogen (Class C) based on increased incidence of squamous cell papillomas of the forestomach and marginally increased incidence of thyroid follicular cell adenomas and carcinomas from a long term oral studies in rats. Mercuric chloride has produced some evidence of an action on the chromosomes, and mixed results associated with mutagenic activity has been reported. The USEPA evaluation of mercuric chloride indicate that a linear low-dose extrapolation is not appropriate as kidney tumour seen in mice occurred at doses that were also nephrotoxic.

Dermal Absorption:  No data is available on the dermal absorption of mercury, hence the default absorption of 1% for metals (USEPA, 1995) is therefore assumed.

B15.5 Quantitative Toxicity Data

The following data is available for inorganic mercury:

- **TDI of 0.00071 mg/kg/day** available from the WHO (2008) for total mercury. Key studies associated with inorganic mercury toxicity primarily relate to mercuric chloride.
- **Inhalation guideline value (GV) of 0.001 mg/m³** from the WHO (2000) for inorganic mercury in air.

Based on available data on intakes that may be derived from urban air, food, dental amalgams and drinking water (NHMRC 1999), background intakes have been estimated to be approximately 25% of the available TDI and GV.
**B16 Lithium**
The following is summarised from OEHHA (2003g) and HSDB (2009).

**B16.1 General**
Lithium (Li) (CASRN: 7439-93-2) is commonly used in rechargeable batteries and pharmaceutical products. Lithium metal in the form of ribbon, wire, rod, ingot, granules, powder, or shot can be purchased from chemical supply houses. In clan labs lithium is used as a catalyst and is a by-product of the synthesis of methamphetamine from pseudo/ephedrine.

**Appearance:** Lithium is a soft, silvery-white metal that becomes yellowish upon exposure to moist air. It is soluble in liquid ammonia, producing a blue solution.

**Odour:** Lithium is odourless.

**B16.2 Exposure, Fate and Transport**

**General:** Lithium is widely distributed in nature; trace amounts are found in many minerals, in most rocks and soils, and in many natural waters.

**Indoor Surfaces:** Metallic lithium will react with nitrogen, oxygen, and water vapour in air. Consequently, the lithium surface becomes coated with a mixture of lithium hydroxide (LiOH), lithium carbonate (Li₂CO₃), and lithium nitride (Li₃N). Lithium hydroxide represents a potentially significant hazard because it is extremely corrosive. Physically, lithium used for synthesis of methamphetamine is often in the form of small pieces of foil. In most cases, the amount of LiOH that forms on the surface of these pieces will be small. The potential for lithium (and lithium salts) to be present on indoor surfaces, mainly as a dust, needs to be considered.

**Air:** If released to air, lithium compounds should exist in the particulate phase in the ambient atmosphere since the ionic nature of lithium compounds makes them essentially non-volatile. Particulate-phase lithium may be physically removed from the air by wet and dry deposition.

**Soil:** Lithium compounds are not expected to adsorb strongly to soils and sediments. Lithium ion would not be expected to undergo oxidation-reduction reactions under environmental conditions, and would exist in its +1 oxidation state either in compounds or as dissolved ions. The ionic nature of lithium compounds makes them essentially non-volatile; hence lithium compounds would not be volatilised from moist or dry soil surfaces.

**Water:** In water, adsorption to suspended solids and sediments is not expected to be important fate processes for lithium compounds. Lithium ions may undergo precipitation, sorption, or ligand exchange reactions in the environment. Due to the ionic nature of most lithium compounds, volatilisation from water surfaces will not occur.

**Bioaccumulation:** Bioconcentration is not expected to be an important fate process due to the ionic nature of lithium compounds.

**B16.3 Ecological Effects and Guidelines**
Lithium is found naturally in the aquatic and terrestrial environments but at low concentrations. Lithium is found primarily in the ionic form in water. A review of the aquatic toxicity of lithium indicates that for most natural waters the presence of sodium is sufficient to prevent lithium toxicity (Aral and Vecchio-Sadus, 2008).

In soil lithium can be taken up by plants, although it appears not to be required for growth and development. Uptake is affected by soil acidity with greater uptake observed in acidic soils (similar to that observed for heavier metals). At high levels, lithium can be toxic to plants, however this is dependent on the species.
present. The presence or addition of calcium can prevent toxicity and uptake of lithium (Aral and Vecchio-Sadus, 2008).

A search of ecological screening benchmarks (RAIS, 2009) from USEPA, Canada and Dutch peer reviewed sources has identified the following guidelines for lithium:

- USEPA (Region VI and ORNL) Benchmark for protection of plants = 2 mg/kg
- USEPA (ORNL) Benchmark for protection of soil microbes = 10 mg/kg
- USEPA (Region VI and Tier II) Benchmark for surface water (chronic) = 0.014 mg/L

B16.4 Health Effects

**General:** Dermal contact and inhalation are not likely to be significant routes of exposure to this material. In general, metals are poorly absorbed across the skin, so it is likely that contact with metallic lithium will not result in appreciable systemic absorption. Ingestion of small pieces of lithium foil, particularly by children, appears to be the most likely route of exposure. Little information on the toxicity of elemental lithium is available. However, lithium salts such as lithium carbonate and lithium citrate are used in medicine to treat manic-depressive illness (bipolar disorder), and information on the toxicity of ingested lithium salts is available. These effects have been assumed representative of potential health effects associated with exposure to lithium. Ingested in excessive amounts, lithium primarily affects the gastrointestinal (GI) tract, the central nervous system (CNS), and the kidneys.

Acute GI effects include abdominal pain, nausea, vomiting, and diarrhoea. Nervous system effects include tremors, loss of muscle coordination, muscle rigidity, and exaggerated reflexes. Sedation, mental confusion, agitation, seizures, and coma may occur at high doses. Symptoms associated with kidney toxicity include an initial increase in urine output (polyuria), subsequent elevation in blood non-protein nitrogen, and finally, diminished urine output. Inhalation of finely divided lithium particles may result in serious injury to the nasal passages, upper airways, and lungs due to formation of lithium hydroxide (LiOH), a strong base that is highly corrosive.

Chronic lithium therapy may result in kidney toxicity. Chronic lithium intoxication may lead to temporary blurred vision and blindness; unusual sensitivity to light (photophobia) has also been reported. Reduced heart rate (bradycardia) and significant drop in blood pressure (hypotension) may develop as a result of severe, usually chronic exposure to lithium. Chronic exposure to lithium may be aggravated by dehydration, concurrent illness, or interactions with other drugs. In such cases, symptoms of neurological toxicity may develop after exposure to relatively small amounts of lithium. Dehydration is a common finding in persons with chronic lithium toxicity

**Main Risks and Target Organs:** Target organs are gastrointestinal (GI) tract, the central nervous system (CNS), developmental and the kidneys.

**Carcinogenicity and Genotoxicity:** No information is available on carcinogenic effects of lithium compounds. However based on the known mechanisms of action for lithium carcinogenic effects are considered unlikely (Aral et al 2008). Limited data is available with respect to the mutagenic potential of lithium compounds. The available studies do not suggest lithium is mutagenic.

**Dermal Absorption:** Dermal uptake of lithium is not likely to be a significant route of exposure. Absorption of solid lithium across the skin is poor, although contact with finely divided lithium or lithium powder might present a hazard because these forms may react with skin moisture to form corrosive LiOH. To provide a conservative assessment of potential intakes of lithium the default absorption of 1% for metals (USEPA, 1995) is therefore assumed relevant for dermal absorption of lithium.
B16.5 Quantitative Toxicity Data

Limited quantitative data is available for lithium and lithium salts. The only value currently available is a USEPA a provisional chronic oral RfD of 0.002 mg/kg/day (USEPA 2009b). The basis for the provisional value is not available.

Background intakes of lithium are not known. Given most significant exposures are expected to occur in an occupational environment or from pharmaceutical preparations, potential background intakes by the general public are considered to be negligible.
B17 Benzaldehyde
The following is summarised from OECD (2005), CIR (2006) and HSDB (2009).

B17.1 General
Benzaldehyde was formerly used as an active ingredient in pesticides. It is also used as an intermediate in the manufacture of aromatic alcohols, solvents, odourants and flavouring chemicals particularly artificial cherry and almond flavours. It is also a starting material in pharmaceuticals. In clan labs benzaldehyde is a reactant and by-product of the manufacture of methamphetamine, P-2-P and pseudo/ephedrine.

Appearance: Benzaldehyde is a colourless to yellowish strongly refracting volatile oil.

Chemical/Physical Properties: Benzaldehyde (CASRN: 100-52-7) has the following properties (HSDB and RAIS):

- Molecular formula: C₇H₆O
- Molecular weight: 106.2
- Log Kow: 1.48
- Koc: 33 L/kg
- Solubility: 6950 mg/L in water at 25°C
- Vapour pressure: 0.127 mm Hg at 25°C
- Henry's Law constant: 2.6x10⁻⁵ atm.m³/mol at 25°C
- Air diffusion coefficient: 0.0744 cm²/s
- Water diffusion coefficient: 9.46x10⁻⁶ cm²/s

Odour: Benzaldehyde has a characteristic odour similar to bitter almond. The odour threshold is listed as 0.042 ppm.

B17.2 Exposure, Fate and Transport

Indoor Surfaces: Benzaldehyde is volatile and hence if spilled indoors it is expected to volatilise from surfaces. Hence long-term issues that may be associated with benzaldehyde on surfaces are not likely to be significant. However as benzaldehyde is an oily chemical, under some conditions there is the potential for an oily residue of benaldehyde to remain on surfaces. As there is limited data available regarding the long-term presence of benzaldehyde on surfaces, a surface residue IL may need to be considered in the initial guidelines. The potential for absorption by porous materials and subsequent off-gassing to indoor air should be considered.

Air: If released to air, benzaldehyde will exist solely as a vapour in the ambient atmosphere. Vapour-phase benzaldehyde will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals with a half-life estimated to be 30 hours. Small quantities of benzaldehyde have been detected in atmospheric aerosol particulates which can be physically removed from air via dry and wet deposition.

Soil: If released into soil, benzaldehyde is expected to have very high mobility based upon the estimated Koc. Volatilisation from dry and moist soil surfaces is expected to be an important fate process based upon the vapour pressure and Henry's Law constant. A number of biological screening studies have demonstrated that benzaldehyde is readily biodegradable.

Water: In water, benzaldehyde is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process with volatilisation half-lives estimated to be between 1.5 and 14 days.

Bioaccumulation: An estimated BCF of 2.7 suggests the potential for bioconcentration in aquatic organisms is low.
B17.3 Ecological Effects and Guidelines

Ecotoxicological data indicate that benzaldehyde is acutely toxic to fish, harmful to daphnia and slightly toxic to algae (OECD, 2005). Little data is available for benzaldehyde, however the following have been identified for benzaldehyde based on the available information (OECD, 2005):

- Predicted No Effect Level = 0.0107 mg/L for aquatic organisms
- Predicted No Effect Level = 0.6 mg/kg for soil organisms

Benzaldehyde was included as one of the chemicals evaluated with respect to ecotoxicity in soil by Pal and Kirkbride (2009). The EC50 (defined as a 50% reduction in dehydrogenase activity in spiked soil relative to unspiked soil) for the soil materials tested with benzaldehyde ranged from 1.7 to 3.3 g/kg.

B17.4 Health Effects

General: Exposure to benzaldehyde has been associated with skin effects (dermatitis), CNS effects and irritation to the eye and mucous membranes of the respiratory system. Because benzaldehyde rapidly metabolises to benzoic acid in the skin, the available dermal irritation and sensitisation data demonstrating no adverse reactions to benzoic acid were considered supportive of the safety of benzaldehyde for use in consumer products.

Benzaldehyde is absorbed through skin and by the lungs, distributes to all well-perfused organs, but does not accumulate in any specific tissue type. After being metabolised to benzoic acid, conjugates are formed with glycine or glucuronic acid, and excreted in the urine.

In general, benzaldehyde is considered to have a low acute toxicity.

Main Risks and Target Organs: Target organs are gastrointestinal (GI) tract, the central nervous system (CNS), developmental and the kidneys.

Carcinogenicity and Genotoxicity: Benzaldehyde was evaluated by the National Toxicology Program, which found no evidence of carcinogenicity in rats, and some evidence of carcinogenicity in mice. Overall, at the concentrations used in cosmetics, benzaldehyde was not considered a carcinogenic risk to humans. The available studies do not suggest benzaldehyde is mutagenic.

Dermal Absorption: Dermal uptake of benzaldehyde is expected to occur (particularly where present in consumer products), however as it is a volatile compound, absorption from environmental exposures is expected to be low. The USEPA (2004) guidance on dermal exposure assessment suggests that for volatile chemicals dermal absorption can be effectively considered negligible as the chemical is not expected to remain on the skin long enough to be absorbed. Prior to this recommendation the dermal absorption of volatile organic compounds was taken to be 0.05% to 3% (depending on the vapour pressure) based on experimental data (USEPA 1995). On this basis a value of 3% has been adopted for benzaldehyde.
B17.5 Quantitative Toxicity Data

Limited quantitative data is available for benzaldehyde. The WHO (2001c) presents an ADI of 0.5 mg/kg/day based on benzoic acid equivalents for consideration in food products. The USEPA (2009b) provides an RfD of 0.1 mg/kg/day based on gastrointestinal and kidney effects associated with benzaldehyde. For the assessment of potential environmental exposures to benzaldehyde, the USEPA RfD of 0.1 mg/kg/day has been used.

An odour based guideline is available from the TCEQ (2009), however no toxicity based inhalation guideline is available.

Background intakes of benzaldehyde may occur through its use in food and consumer products. The assessment undertaken by OECD (2005) identified that background intakes of benzaldehyde were 100 times lower than the WHO ADI. These are considered essentially negligible.
**B18 Phosphine**
The following is summarised from OEHHA (2003h), IPCS (1997) and HSDB (2009).

**B18.1 General**
Phosphine is extremely rare in nature. Aluminium and zinc phosphide is used as rodenticides, fumigants and pesticides release phosphine gas when exposed to moisture. In clan labs phosphine is a by-product generated during the synthesis of hydriodic acid from iodine and red phosphorus during the manufacture of methamphetamine. It is produced when red phosphorus contacts caustics and/or acids, especially in the presence of a metal. It is a significant hazard during the manufacture of methamphetamine with the death of a number of cooks because of inadvertent exposure to phosphine.

**Appearance:** Phosphine is a colourless gas at room temperature.

**Chemical/Physical Properties:** Phosphine (CASRN: 7803-51-2) has the following properties (HSDB and RAIS):

- Molecular formula: PH₃
- Molecular weight: 34
- Log Kow: NA
- Koc: NA
- Soluble: Water, alcohol, ether and coprous chloride solution
- Solubility in water: 397 mg/L at 25°C
- Vapour pressure: 29300 mm Hg at 25°C
- Henry’s Law constant: 0.0244 atm.m³/mol at 25°C

**Odour:** Phosphine is often reported as having a disagreeable, garlic-like odour; like the odour of decaying fish. Pure grade phosphine is essentially odourless. The odour threshold is listed as 0.03 to 0.5 ppm, odour recognition of 2ppm.

**B18.2 Exposure, Fate and Transport**

**Indoor Surfaces:** Since phosphine is a gas at room temperature, residues of phosphine will not accumulate on indoor surfaces. Hence long-term issues that may be associated with phosphine on surfaces are not considered to be significant. The potential for absorption by porous materials during the cooking process (where high concentrations may be present in indoor air) and subsequent off-gassing to indoor air should be considered.

**Air:** In the atmosphere, phosphine exists solely as a gas. In the troposphere, phosphine reacts primarily with hydroxyl radicals; eventual products are water and phosphorus oxyacids with a half-life and lifetime of phosphine calculated to be 0.7 and 1 day, respectively. Phosphine is not expected to be susceptible to direct photolysis by sunlight. Phosphine would also be expected to react with water vapour in the air. Phosphine is removed from air by soil and oxidised to orthophosphate.

**Soil:** Phosphine is a gas at room temperature, and any residue of phosphine present in a solid or liquid waste would quickly evaporate under most circumstances. However studies have shown that phosphine may bind to soil. Laboratory studies do suggest that phosphine present below the soil surface is quickly adsorbed and degraded. Phosphine interaction with soil is soil-type dependent. It is noted that large amounts of buried phosphorus-containing waste may represent a significant source of soil and groundwater contamination.

**Water:** Due to its volatility, any release of phosphine to surface water would quickly evaporate to the surrounding air. Waste generated by clandestine laboratories may produce small amounts of phosphine, but the small amount potentially released to soil would not be sufficient to cause contamination of groundwater.
However, large amounts of buried phosphorus-containing waste may represent a significant source of soil and groundwater contamination.

Biodegradation, hydrolysis and bioconcentration are not expected to be important fate processes when compared to volatilisation.

B18.3 Ecological Effects and Guidelines

Ecological effects or potential risks to non-targeted organisms resulting from indoor use of phosphine as a fumigation gas is considered to be very minimal. Phosphine would be highly toxic to small mammals and birds that might remain in indoor sites (e.g., warehouses) during fumigation. However no long-term ecological effects have been identified (USEPA, 1999). No guidelines are available for a screening level assessment of ecological effects associated with phosphine.

B18.4 Health Effects

General: Inhalation of phosphine gas can result in adverse effects on the central nervous system (CNS), gastrointestinal (GI) tract, lungs, and heart. CNS effects include fatigue, headache, restlessness, irritability, drowsiness, tremors, dizziness, double vision, and impaired gait. GI symptoms may include nausea, vomiting, abdominal pain, and diarrhoea. Effects on the lungs include chest tightness, cough, and shortness of breath. Severe (acute) exposure may lead to accumulation of fluid in the lungs, but the onset of this effect may be delayed by seventy-two hours or more. Other symptoms may include a marked decrease in blood pressure, rapid and/or irregular heartbeat, and cardiac arrest. There are no biological indicators for exposure to phosphine. Therefore, analysis of hair, nails, urine, blood, or exhaled air will not provide evidence of exposure to phosphine. Acute toxicity of phosphine is considered to be high.

Phosphine is rapidly absorbed and distributed throughout the body and is acutely toxic. The onset of symptoms is rapid following phosphine inhalation or the ingestion/inhalation of metal phosphides, which release phosphine on contact with moisture or stomach acid. Dermal absorption of phosphine or phosphides is not considered a significant route of exposure.

Symptoms of chronic exposure may include anaemia, bronchitis, gastrointestinal disorders, speech and motor disturbances, weakness, weight loss, toothache, swelling of the jaw, mandibular necrosis, and spontaneous fractures. Some chronic effects can be confused with symptoms of acute poisoning.

The majority of absorbed phosphine is excreted in exhaled air; minor amounts are metabolised and excreted in urine as hypophosphite and phosphate.

Children exposed to phosphine will have the same symptoms of toxicity as adults.

Main Risks and Target Organs: Target organs are central nervous system (CNS), gastrointestinal (GI) tract, lungs, and heart.

Carcinogenicity and Genotoxicity: Phosphine is clastogenic and genotoxic in vitro but is not considered to be mutagenic in vivo and has not been associated with carcinogenic effects (HPA, 2007).

Dermal Absorption: The skin is not a common route of absorption of phosphine and phosphides. As phosphine is essentially a gas the default dermal absorption value of 0.05% (for highly volatile compounds) from the USEPA (1995) has been adopted.
B18.5 Quantitative Toxicity Data

The USEPA (2009b) has derived the following for phosphine:

- **RfD of 0.003 mg/kg/day** based on body weight effects;
- **RfC of 0.0003 mg/m³**. The OEHHA (2009c) has derived a chronic REL of 0.0008 mg/m³ and TCEQ (2009) has an interim chronic ESL of 0.00042 mg/m³ (based on health effects) both of which are similar to similar to the RfC. The **RfC of 0.0003 mg/m³** has been used in this assessment.

Background intakes of phosphine are expected to be significant in occupational environments. Data on exposures by the general public are limited, but expected to be low. Hence no background intakes of phosphine by the general public have been considered.
B19 Safrole and Isosafrole

The following is summarised from WHO (1974) and HSDB (2009).

B19.1 General

Safrole (1,2-methylenedioxy-4-allylbenzene) is the principal constituent of oil of sassafras and a minor constituent of many other essential oils. The related substance, isosafrole (1,2-methylene-dioxy-4-propenylbenzene), also occurs as a minor constituent of many essential oils with a distribution similar to that of safrole. Both were formerly used as pesticides. Currently, both are used in perfumery, the manufacture of soaps and as a constituent in essential oils. In clan labs safrole and isosafrole are reactants and by-products of the manufacture of MDMA. Limited information is available on isosafrole and hence both safrole and isosafrole are assessed as a group with data primarily derived for safrole used for the purpose of this assessment.

Appearance: Safrole is a colourless or pale yellow oil or crystals. Isosafrole is a colourless liquid.

Chemical/Physical Properties: Safrole (CASRN: 94-59-7) and isosafrole (CASRN: 120-58-1) has the following properties (HSDB and RAIS):

- Molecular formula: C10H10O2
- Molecular weight: 162.18
- Log Kow: 3.45 (safrole) and 3.37 (isosafrole)
- Koc: 300
- Soluble: Safrole is soluble in water and alcohol, slightly soluble in propylene glycol, miscible in chloroform and ether. Isosafrole is soluble in organic solvents and partially soluble in water.
- Solubility in water: 121 (safrole) and 144 (isosafrole) mg/L at 25°C
- Vapour pressure: 0.0706 (safrole) and 0.0245 (isosafrole) mm Hg at 25°C
- Henry’s Law constant: 9.07x10^{-6} (safrole) and 4.08x10^{-6} (isosafrole) atm·m³/mol at 25°C

As safrole and isosafrole are moderately volatile compounds if present in dry soil, volatilisation has been considered. No air and water diffusion parameters are available for either chemical; hence the default values relevant to pesticides have been used. These parameters vary little for pesticides and hence the default values are considered relevant for these chemicals that have been used in pesticides in the past. These default values are 0.05 cm²/s air diffusion coefficient and 5x10^{-6} cm²/s water diffusion coefficient (Jury, 1983).

Odour: Safrole has a distinctive sassafras odour. Isosafrole has a fragrant anise odour.

B19.2 Exposure, Fate and Transport

Indoor Surfaces: Safrole and isosafrole may be present as a residue indoors and hence consideration should be given to the potential exposure to these compounds on indoor surfaces.

Air: In the atmosphere, safrole and isosafrole will exist solely as a vapour. The vapour-phase will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals with a half-life estimated to be approximately 5 hours. The half-life for the vapour-phase reaction of safrole with ozone has been estimated to be 23 days and for isosafrole 4 (cis-) and 2 (trans-) hours. Safrole and isosafrole are not expected to be susceptible to direct photolysis by sunlight.

Soil: If released to soil, safrole/isosafrole is expected to have moderate mobility based the estimated Koc. Volatilisation from moist soil surfaces is expected to be an important fate process (based on the estimated Henry’s Law constant) but safrole is not expected to volatilise from dry soil surfaces based upon its vapour pressure.
**Water:** If released into water, safrole/isosafrole may adsorb to suspended solids and sediment based upon the estimated $K_{oc}$. Volatilisation from water surfaces is expected to be an important fate process based upon this compound's estimated Henry's Law constant. USEPA (1985) lists safrole and isosafrole as moderately volatile compounds if present in dry soil and slightly volatile from aqueous solutions.

**Bioconcentration:** An estimated BCF of 90 (safrole) and 80 (isosafrole) suggests the potential for bioconcentration in aquatic organisms is moderate.

**B19.3 Ecological Effects and Guidelines**

Little data is available on effects of safrole or isosafrole in the aquatic or terrestrial environment. A search of ecological screening benchmarks (RAIS, 2009) from USEPA, Canada and Dutch peer reviewed sources has identified the following guidelines:

- USEPA (Region V) Ecological Soil Screening Benchmark for safrole = 0.404 mg/kg
- USEPA (Region V) Ecological Soil Screening Benchmark for isosafrole = 9.94 mg/kg

These guidelines are understood to be based on phytotoxic effects primarily associated with the use of these chemicals in pesticides.

Safrole was included as one of the chemicals evaluated with respect to ecotoxicity in soil by Pal and Kirkbride (2009). The EC50 (defined as a 50% reduction in dehydrogenase activity in spiked soil relative to unspiked soil) for the soil materials tested with safrole where greater than 1 g/kg (approximately 8 g/kg).

No other guidelines are available that are associated with potential ecological effects.

**B19.4 Health Effects**

**General:** Acute exposure to safrole can result in irritation to the skin, gastrointestinal effects (such as nausea and vomiting) and central nervous system (CNS) effects (such as headache, dizziness, drowsiness, convulsions and sometimes unconsciousness). Chronic exposures are associated with liver, kidney and reproductive effects. It is also considered to be a carcinogen.

Following ingestion safrole is rapidly absorbed, metabolised and excreted. Safrole can cross the placenta and be excreted in breast milk.

**Carcinogenic Potential:** IARC note that safrole and isosafrole are carcinogenic in mice and rats; they produce liver tumours following their oral administration. Safrole also produced liver and lung tumours in male infant mice following its subcutaneous injection. On this basis safrole is classified by IARC a Group 2B (possibly carcinogenic) and isosafrole a Group 3 (not classifiable based on lack of data) carcinogen. The USEPA has classified both safrole and isosafrole as a Group B2 carcinogen (probably carcinogenic to humans). The available data suggests that there is the potential for safrole to be considered genotoxic.

**Main Risks and Target Organs:** Target organs are central nervous system (CNS), gastrointestinal (GI) tract, kidney and reproductive system.

**Dermal Absorption:** No data is available for dermal absorption of safrole/isosafrole. As these may be considered semivolatile compounds a default of 10% (USEPA 1995) has been adopted.
B19.5 Quantitative Toxicity Data

As safrole is considered to be a genotoxic carcinogen it is relevant to assess potential carcinogenic effects on the basis of the non-threshold approach. Limited data is available regarding a dose-response relationship for safrole, however OEHHA (2009d) list the following non-threshold values for safrole:

- Oral slope factor = 0.22 (mg/kg/day)$^{-1}$
- Inhalation unit risk = $6.3 \times 10^{-5} (\mu g/m^3)^{-1}$

No data is available for isosafrole and hence the data available for safrole is considered representative for both compounds.
B20 Hydrocarbon Based Solvents

B20.1 General

A number of hydrocarbon based solvents have been identified that may be used clan labs. The products used are those which are commercially available and include mineral turpentine, shellite (also known as Coleman Fuel and or white gas/spirits), methylated spirits (ethanol solution), methanol, toluene and xylenes. In general the products used are typically light petroleum products, typically with a low benzene content. The assessment of the potential presence of these light petroleum hydrocarbon products in a former clan can be addressed by the assessment of the key hydrocarbons, benzene, toluene, ethylbenzene and total xylenes (known as BTEX), naphthalene and total petroleum hydrocarbons (TPH).

TPH is a term used to describe a wide group of chemicals typically derived from petroleum products. TPH is the most common reporting acronym used by environmental laboratories in Australia to describe the measurable amount of petroleum based hydrocarbons in relevant media such as soil and water. TPH data is used extensively as the basis for the assessment of contamination from petroleum products. There are several hundred individual hydrocarbon chemicals defined as petroleum based.

Because TPH is a complex mixture with variable composition depending on sources and time, a generic assessment of the toxicity of TPH is difficult. The assessment of risks associated with exposure to constituents of TPH involves a process of calculating intake of the various fractions distinguishing between volatile fractions and semi volatile fractions (as necessary to enable evaluation of relevant exposure pathways). As toxicity values are not generally available for TPH fractions, the risk assessment typically utilises surrogates or reference chemicals (ie. a single chemical) to provide an estimate of the potential toxicity of specific groups of TPH fractions.

The TPHCWG (1997-1999) details an approach that utilises both individual indicator compounds (for specific compounds such as benzene and others such as TEX and naphthalene) and data for whole products or well-defined mixtures that are representative of TPH fractions.

The evaluation of TPH has focused on the fraction groups typically provided by analytical methods adopted in Australia (C6-C9, C10-C14, C15-C28 and C29-C36). With respect to the toxicity of these factors, data from sources such as the TPHCWG is split into aromatic and aliphatic fractions to enable further assessment of properties and toxicities that are associated with these groupings. The analysis of TPH in Australia does not routinely provide aromatic and aliphatic fractions and therefore for the purpose of this assessment some assumptions are required. These assumptions are:

- The assessment of BTEX provides adequate quantification of aromatic C6-C9 fractions. Hence the quantification of C10-C28 fractions is based on remaining aliphatics;
- Benzene is the only genotoxic carcinogen likely to be present in the light petroleum products used in clan labs. Benzene is quantified and assessed separately. However other carcinogens that are associated with heavier products such as oils have not been addressed separately;
- The assessment of higher fractions has been undertaken assuming they are 100% aromatic. Aromatic fractions have a higher toxicity than aliphatics and hence the approach adopted is considered conservative. It is noted that for volatile fractions, aliphatics are typically more volatile, however they more rapidly reach a saturated vapour phase concentration and hence the potential for volatile aliphatics to be present at concentrations that are higher than aromatics is low.
- The assessment of volatilisation has addressed volatile indicator chemicals and TPH fractions only, namely BTEX, naphthalene and TPH C6-C14.
B20.2 Environmental Fate and Transport and Ecotoxicity

The following points associated with the environmental fate and transport and ecotoxicity of petroleum hydrocarbons particularly with respect to light petroleum products expected to be used as solvents in clan labs have been summarised from TPHCWG (199-19997), CCME (2000 and 2008) and CONCAWE (1992 and 1995):

- Petroleum products released into the environment undergo weathering processes with time. These processes include volatilisation, leaching (migration to aqueous phase) through solution and entrainment, chemical oxidation and microbial degradation.
- The rate of weathering is highly dependent on environmental conditions and the physico-chemical properties of the hydrocarbons present.
- Volatilisation is important for lightweight and volatile hydrocarbon fractions. In the atmosphere these volatile hydrocarbons will be photodegraded by reaction with hydroxyl radicals in the atmosphere.
- Surface water runoff may lead to surface water contamination.
- BTEX compounds, tend to be the most water-soluble fractions, and tend to migrate more readily than other TPH fractions.
- Leaching of petroleum hydrocarbons in soil can be a significant pathway.
- Biodegradation processes can be complex.
- Toxicity of petroleum hydrocarbons in the aquatic and terrestrial environments depends on the individual hydrocarbons that are present. The concentrations of these will vary over time and in different media due to weathering and transport processes.
- The bulk of the available literature on gasoline relates to the environmental impact of monoaromatic (BTEX) and diaromatic (naphthalene, methyl-naphthalenes) constituents.
- In general, lighter hydrocarbons products exhibit some short-term toxicity to freshwater and marine organisms, especially under closed vessel or flow-through exposure conditions in the laboratory. The components which result in aquatic toxicity are also highly volatile and can be readily biodegraded by microorganisms.
- Petroleum hydrocarbons a readily metabolised by vertebrates, modified into a more readily excitable form and do not accumulate in tissues. In addition petroleum hydrocarbons are not readily absorbed into or accumulated in plants. Hence consumption of plants or other animals does not tend to be a significant issue with respect to exposure by wildlife and livestock populations.

Screening Level Guidelines

The following guidelines are available for the propose of conducting a screening level review of ecotoxicity issues associated with the presence of petroleum hydrocarbons in soil and water:

- Benzene – the following is available from ANZECC/ARMCANZ (2000) and CCME (2000):
  - Trigger Value for fresh water (95% protection level) = 0.95 mg/L;
  - Trigger Value for marine water (95% protection level) = 0.5 mg/L;
  - Soil Protective Benchmark based on soil invertebrate and plant endpoints = 210 mg/kg
- Toluene - the following is available from ANZECC/ARMCANZ (2000) and CCME (2000):
  - Low reliability Trigger Value for fresh and marine water = 0.18 mg/L;
  - Soil Protective Benchmark based on soil invertebrate and plant endpoints = 5 to 126 mg/kg; and
  - Threshold concentration in soil = 1.4 mg/kg (NSW EPA 1994).
- Ethylbenzene - the following is available from ANZECC/ARMCANZ (2000) and CCME (2000):
  - Low reliability Trigger Value for fresh water = 0.08 mg/L;
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Low reliability Trigger Value for marine water = 0.005 mg/L;
Soil Protective Benchmark based on soil invertebrate and plant endpoints = 9 to 155 mg/kg;
Threshold concentration in soil = 3.1 mg/kg (NSW EPA 1994).

Xylenes - the following is available from ANZECC/ARMCANZ (2000) and CCME (2000):
- Trigger value for fresh and marine water (95% protection level) for m- and p-xylene = 0.2 mg/L;
- Trigger value for fresh and marine water (95% protection level) for o-xylene = 0.35 mg/L;
- Soil Protective Benchmark based on soil invertebrate and plant endpoints = 9 to 97 mg/kg;
- Threshold concentration in soil = 14 mg/kg (NSW EPA 1994).

Naphthalene - the following is available from ANZECC/ARMCANZ (2000) and CCME (2000):
- Trigger value for fresh water (95% protection level) = 0.016 mg/L;
- Trigger value for marine water (95% protection level) = 0.05 mg/L; and
- Soil Protective Benchmark based on soil invertebrate and plant endpoints = 56 to 108 mg/kg.

TPH - the following is available from CCME (2000):
- No water quality guidelines are available for TPH, based on aquatic toxicity;
- Soil quality guideline based on soil invertebrate and plant endpoints (agricultural [A], residential [R], parkland [P] and commercial/industrial [C/I]):
  - TPH C₆-C₁₀ = 130 mg/kg (A,R,P) and 330 mg/kg (C/I)
  - TPH C₁₀-C₁₆ = 450 mg/kg (A,R,P) and 760 mg/kg (C/I)
  - TPH C₁₆-C₃₄ = 400 mg/kg (A,R,P) and 1700 mg/kg (C/I)
  - TPH C₃₄+ = 2800 mg/kg (A,R,P) and 3300 mg/kg (C/I)
- Soil quality guideline based on protection of aquatic species in surface water body 10m from source:
  - TPH C₆-C₁₀ = 230 mg/kg
  - TPH C₁₀-C₁₆ = 150 mg/kg

B20.3 Quantitative Toxicity Data

General
A number of summaries are available from key sources such as the WHO, ASTDR and USEPA that provide detail on the key health effects associated with petroleum hydrocarbons. The reader is referred to these sources for this information. The following provides a summary of the quantitative data used for the purpose of deriving guidelines:

Benzene
The following data are available for the quantitative assessment of potential health effects associated with exposure to benzene. It is noted that both non-threshold and threshold effects have been considered as threshold effects associated with BTEX mixture (ATSDR, 2004c) may require further consideration in the application of the guidelines.

- Non-threshold slope factor = 0.035 (mg/kg/day)⁻¹ available from the WHO (2008);
- Non-threshold Inhalation Unit Risk = 6x10⁻⁶ (μg/m³)⁻¹ available from the WHO (2000);
- Chronic oral RfD = 0.004 mg/kg/day available from the USEPA (2009b);
- Chronic inhalation RfC = 0.03 mg/m³ available from the USEPA (2009b);
- Dermal absorption = 0.05% (default value for VOCs such as benzene, USEPA 1995);
- Background intakes (relevant for the assessment of threshold effects) taken to be 10% based on reported concentrations of benzene in Australian urban air (DEC 2004);
- Physical/chemical parameters used to assess volatilisation available from RAIS (2009) database.
**Toluene**
The following data are available for the quantitative assessment of potential health effects associated with exposure to toluene:

- Chronic oral RfD = 0.08 mg/kg/day available from the USEPA (2009b);
- Chronic inhalation GV = 5 mg/m³ available from the USEPA (2009b);
- Dermal absorption = 3% (default value for VOCs less volatile than benzene, USEPA 1995);
- Background intakes (relevant for the assessment of threshold effects) taken to be 10% based on reported concentrations of toluene in Australian urban air (DEC 2004);
- Physical/chemical parameters used to assess volatilisation available from RAIS (2009) database.

**Ethylbenzene**
The following data are available for the quantitative assessment of potential health effects associated with exposure to ethylbenzene:

- Chronic oral TDI = 0.097 mg/kg/day available from the WHO (2008) and NHMRC (2004);
- Chronic inhalation GV = 22 mg/m³ available from the WHO (2000);
- Dermal absorption = 3% (default value for VOCs less volatile than benzene, USEPA 1995);
- Background intakes (relevant for the assessment of threshold effects) are essentially negligible based on reported concentrations of ethylbenzene in Australian urban air (DEC 2004) and the available TDI and GV;
- Physical/chemical parameters used to assess volatilisation available from RAIS (2009) database.

**Xylenes**
The following data are available for the quantitative assessment of potential health effects associated with exposure to total xylenes:

- Chronic oral TDI = 0.179 mg/kg/day available from the WHO (2008) and NHMRC (2004);
- Chronic inhalation GV = 0.87 mg/m³ available from the WHO (2000);
- Dermal absorption = 3% (default value for VOCs less volatile than benzene, USEPA 1995);
- Background intakes (relevant for the assessment of threshold effects) are essentially negligible based on reported concentrations of xylenes in Australian urban air (DEC 2004) and the available TDI and GV;
- Physical/chemical parameters used to assess volatilisation available from RAIS (2009) database.

**Naphthalene**
While naphthalene has been shown to be carcinogenic in animals, review of the available data by the UK (2003) and the EU (2003) indicate that the tumours observed following inhalation exposure did not arise by a direct genotoxic mechanism. On this basis, use of a non-threshold approach was not considered appropriate in the quantification of risk associated with naphthalene. The following data are available for the quantitative assessment of potential health effects associated with exposure to naphthalene:

- Chronic oral RfD = 0.02 mg/kg/day available from the USEPA (2009b);
- Chronic inhalation RfC = 0.003 mg/m³ available from the USEPA (2009b);
- Dermal absorption = 13% for higher fractions based on data for polycyclic aromatic hydrocarbons (PAHs);
- Background intakes (relevant for the assessment of threshold effects) estimated to be 10% based on reported concentrations of naphthalene in Australian urban air (DEH 1999, Berko 1999) and the available threshold values;
Physical/chemical parameters used to assess volatilisation available from RAIS (2009) database.

**Total Petroleum Hydrocarbons (TPH)**

As BTEX is quantified it is assumed that the remaining TPH C₆-C₉ is aliphatic. Higher fractions have been assumed to be 100% aromatic. Only TPH fractions C₆-C₁₄ are considered volatile.

The following data are available for the quantitative assessment of potential health effects associated with exposure to TPH:

- **Aliphatic C₆-C₉ fractions:**
  - Chronic oral RfD = 5 mg/kg/day available from the TPHCWG (1999);
  - Chronic inhalation RfC = 0.7 mg/m³ based on the assumption that n-hexane is a surrogate with the most current toxicity data available from the USEPA (200b) considered appropriate. No additional weighting of the toxicity value (as presented by the TPHCWG) has been undertaken.

- **C₁₀-C₁₄ fractions - assumed 100% aromatic:**
  - Chronic oral RfD = 0.03 mg/kg/day available from the TPHCWG (1999);
  - Chronic inhalation RfC = 0.2 mg/m³ available from the TPHCWG (1999).

- **C₁₅+ fractions - assumed 100% aromatic:**
  - Chronic oral RfD = 0.03 mg/kg/day available from the TPHCWG (1999);
  - Chronic inhalation RfC = NA as fractions not volatile. Inhalation of dust assessed on the basis of the oral RfD.

- Dermal absorption = assumed to be 3% for TPH C₆-C₉ and 13% for higher fractions based on data for polycyclic aromatic hydrocarbons (PAHs), likely to significantly contribute to aromatic TPH C₁₀+ fractions;

- Background intakes (relevant for the assessment of threshold effects) are taken to be 20% based on the likely common use and exposure to TPH in household products.
B21 pH

pH provides an indicator of the presence of an acid or base. With respect to the range of pH that are considered acceptable for long-term exposures by residents, workers or the environment, guidelines available in ANZECC/ARMCANZ (2000) and the NHMRC (2004) provide the following:

- pH between 6 and 9 (depending on the ecosystem) will generally be protective of ecotoxicity effects;
- pH < 6.5 may be corrosive;
- pH > 8.5 may cause aesthetic issues such as taste
- extreme pH values (<4 and >11) may adversely affect health

On the basis of the above a pH range of 6.5 to 8.5 in soil or surface residues is considered adequately protective of long-term effects associated with all land-use scenarios considered.
B22 References


OECD, 2005. Screening Information Data Set (SIDs), Benzaldehyde.


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Appendix C – Methodology and Derivation of ILs for Indoor Surface Residues
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C1 Introduction

This appendix presents the methodology and basis for assumptions adopted in the derivation of ILs for indoor surface residues. The quantification of ILs involves consideration of exposure and relevant toxicity data. The approach to quantification of dose-response and the toxicity data used in this assessment are presented in Appendix B. The following presents the approach and assumptions adopted for the quantification of exposure to surface residues and the methodology adopted for the derivation of the ILs.

The quantification of exposure to residues indoors has considered the following pathways as significant:

- Dermal absorption by hands and body following contact with hard and soft surfaces (relevant to adults and children);
- Ingestion of residues from hands (relevant to adults and children); and
- Ingestion of residues by children during the mouthing of objects that have been in contact with the contaminated surface.

The available information relating to the potential intake of contaminants derived from hard surfaces (such as floor boards, walls, tiles etc) and soft surfaces (such as carpet and furnishings) suggests some differences, however these differences are only in the order of 2 fold and not considered to be sufficiently great to warrant a separate assessment of these surfaces. Hence the approach adopted has considered exposure on all surfaces, hard and soft, in the building, namely flows, window sills, doors, benches, carpets and other soft furnishings that may be affected by residues. This means that the derived ILs can be applied to any building where different mixes of hard and soft flooring may be present.

Exposure to a contaminant that may be present as a surface residue is dependent on a number of variables (Cohen Hubal et al, 2000) including the contaminant concentration on the surface on which individual may make contact, the contact rate of the individual with the surface, the transfer efficiency from the surface to the skin or objects (and subsequently to the mouth), contaminant uptake rates and activity patterns. Hence the potential for exposure and intake of a contaminant from surface residues can vary significantly. The approach adopted in deriving ILs has focused on calculating a reasonable maximum exposure (RME) that will be protective of most significant exposures inside a building.

With respect to the scenarios for which ILs have been derived the following have been considered as the most sensitive groups relevant for the derivation of ILs:

- **Residential** – Assessed on the basis of potential exposure by young children aged 6 months to 2 years. Exposures by children in this age group were identified (OEHHA, 2009) as the most sensitive group based on the length of time spent indoors, time and proportion of the body surface area spent in contact with the floor or other surfaces and a greater likelihood of placing hands and objects in the mouth. These activities result in a higher intake of contaminants that may be present in surface residues compared with older children and adults, in a residential setting. On this basis ILs for indoor surfaces relevant for residential land use have been derived on the basis of the protection of exposures by young children aged 6 months to 2 years.

- **Commercial** – Assessed on the basis of potential exposures by adult workers in a workplace. It is assumed that the application of ILs for commercial/industrial use will be relevant for these uses only. Any more sensitive uses that may be allowable under a particular zoning, such as childcare, schools or hotel/motels need to be considered as residential scenarios. Hence the commercial ILs have been derived on the basis of the protection of exposures by adults in the workplace only.

Recreational use has not included consideration of indoor exposures, hence no surface residue ILs have been derived for this use.
The overall approach to the quantification of exposures indoors to surface residues is derived from studies and models established to assess exposures to pesticides. The most recent and detailed model is presented in the USEPA Model SHEDS (Stochastic Human Exposure and Dose Simulation Model for Multimedia, Multipathway Chemicals) (2007) as used in the OEHHA (2009) derivation of a cleanup standard for surface methamphetamine contamination and USEPA (2005) SHEDS-Wood Assessment. It is noted that the SHEDS model allows for complex, small interval-based assessment of activities and exposures. For the purpose of the derivation of ILs, the equations presented in SHEDS (2007) and USEPA (2005) have been used with more conservative general activity and exposure parameters to ensure the approach is transparent and adequately conservative.

The total intake that may occur as a result of contact with residues on soft or hard surfaces inside any building (by adults and children, where relevant) has been calculated using the following equation:

\[ \text{Intake}_{SR} = D_H + D_B + O_H + O_O \]  
\[(\text{Equation C1})\]

Where:
- \(D_H\) = Intake of contaminant via dermal contact with surface by hands, refer to Section C2 (mg/kg/day)
- \(D_B\) = Intake of contaminant via dermal contact with surface by rest of body, refer to Section C2 (mg/kg/day)
- \(O_H\) = Intake of contaminant via ingestion of residues from hands, refer to Section C3 (mg/kg/day)
- \(O_O\) = Intake of contaminant via ingestion of residues from mouthing of objects, refer to Section C4 (mg/kg/day)

The above approach does not take into account losses that occurring through the washing of hands or bathing and hence is considered conservative. The following sections presents further detail on the calculation of dermal and ingestion intakes.

**C2 Dermal Intake**

Dermal exposure (by hands and the rest of the body) to residual surface contamination has been estimated as follows (chronic exposures based on transfer efficiency equation presented in USEPA 2007):

\[ D_X = \frac{C_S \times SA_X \times CR_X \times FTSS \times ABSd \times Bd \times EF \times ED}{BW \times AT} \]  
\[(\text{Equation C2})\]

Where:
- \(C_S\) = Dislodgeable surface residue level on contaminated surface [hard or soft surface] (mg/cm\(^2\)) – this is the residue level measured during site investigations
- \(SA_X\) = Exposed skin surface area for part of body X (either hands [H] or rest of body [B]) (cm\(^2\))
- \(CR_X\) = Surface contact rate for part of body X (either hands [H] or rest of body [B]) (# per day)
- \(FTSS\) = Fraction transferred from surface to skin (unitless)
- \(ABSd\) = Dermal absorption fraction (unitless)
- \(Bd\) = Dermal bioavailability (unitless)
- \(EF\) = Exposure frequency (days/year)
- \(ED\) = Exposure duration (years)
- \(BW\) = Body weight (kg)
- \(AT\) = Averaging time (days)
Skin Surface Area (SA)
The skin surface area of each group (adult or child) has been obtained from body part specific data presented in the USEPA Exposure Factors Handbook (1997). The following values have been adopted for the assessment presented:

- **Residential** - Young children aged 6 months to 2 years:
  - Hands – 301 cm² based on 5.68% total body surface area relevant for children aged 1 to 2 years. The total body surface area (5300 cm²) has been taken as the 10% value for children aged 2 to 3 years as no data is available for children under 2 years. The 10% value for children aged 3 to 4 years is approximately the same as the mean value for the younger age group of 2 to 3 years, hence the approach adopted is considered reasonable.
  - Unclothed rest of body – 3122 cm² based on approach presented above for the head, arms, legs and feet (58.9% body surface area).

- **Commercial** - Adult Workers:
  - Hands – 1024 cm² based on the average of the 90th percentile male and female surface area for hands (USEPA 1997).
  - Unclothed rest of body – 1350 cm² based on the remainder of the lower arms potentially touching surfaces (lower arms). Calculated from the average 90th percentile male and female for adult (USEPA 1997).

Surface Contact Rate (CR)
The surface contact rate is one of the most variable parameters as it depends on the nature of the activities being undertaken. The SHEDS model (USEPA, 2007) has utilised probability distributions associated with a range of activities that result in exposure to different surfaces and rooms in a residential home as well as statistical data based on diary events from microactivity studies. It is considered overly complex to utilise such an approach for the derivation of ILs, hence a point (single value) has been selected from the available distributions presented in the SHEDS guidance. It is noted that the guidance provides exposure distributions for young children only and hence values adopted for adults are based on the available information and professional judgement. For the assessment of potential exposures by different groups, the following is noted:

- **Residential** - Young children aged 6 months to 2 years:
  - Hands: the contact rate is based on the fraction of the total hand skin surface area contacting surface residues. The value adopted is 0.74 (mean as per USEPA 2005) over each 20 minute contact event. It is assumed that up to 20 (20 minute) contact events with the hands occur each day resulting in a contact rate of 14.8 per day. It is expected that there may be more frequent events where a smaller fraction of the hands is in contact with surfaces event that may occur, however on average over the day, the approach adopted is considered adequately representative of activities and exposures that may occur every day. It should also be noted that the approach adopted has not considered any removal processes as a result of washing or dry brushing of hands. Hence the approach is expected to be conservative. The total time assumed in contact with surfaces (by hands) with residues is 400 minutes each day.
  - Body (non hands): the contact rate is based on the fraction of unclothened body skin surface area (non-hands) contacting surface residues of 0.158 (mean as per USEPA 2005) over a 20 minute contact event. It is assumed that up to 20 (20 minute) contact events occur each day resulting in a total contact rate of 3.16 per day. As with the contact rate for hands, this approach is considered representative of activities and exposures that may occur every day. It should also be noted that the approach adopted has not considered any removal processes as a result of dry brushing or bathing. Hence the approach is expected to be conservative.
The total time assumed in contact with surfaces (by the body) with residues is 400 minutes each day.

- Based on the above assumptions total time in contact with surfaces (hands plus body [together or separately]) may be in the range of 400 to 800 minutes or 6.7 to 13.3 hours. Data on child microactivity patterns presented in the USEPA Child-Specific Exposure Factors Handbook (2002) indicates that the hours spent each day by a child aged 1 to 2 years eating, playing, watching TV, reading and relaxing when contact with hard and soft surface may take place is approximately 8.7 hours. This is consistent with the range of contact time assessed.

- **Commercial** – Adult Workers:
  - Hands: the contact rate is based on the fraction of the total hand skin surface area contacting surface residues. The value adopted is 0.74 (mean as per USEPA 2005) over each 20 minute contact event, the same as adopted for children which is conservatively assumed relevant for adults as well. It is assumed that up to 8 (20 minute) contact events with the hands occur each day resulting in a contact rate of 5.9 per day. It should also be noted that the approach adopted has not considered any removal processes as a result of washing or dry brushing of hands. Hence the approach is expected to be conservative. The total time assumed in contact with surfaces (by hands) with residues is 160 minutes each day.
  
  - Body (non hands): the contact rate is based on the fraction of unclothed body skin surface area (non-hands) contacting surface residues of 0.158 (mean as per USEPA 2005) over a 20 minute contact event, the same as for young children. It is assumed that up to 8 (20 minute) contact events occur each day resulting in a total contact rate of 1.3 per day. It should also be noted that the approach adopted has not considered any removal processes as a result of dry brushing or bathing. Hence the approach is expected to be conservative. The total time assumed in contact with surfaces (by the body) with residues is 160 minutes each day.

- Based on the above assumptions total time in contact with surfaces (hands plus body [together or separately]) may be in the range of 160 to 320 minutes or 2.7 to 5.3 hours. This comprises 33% to 66% of the workday where some part of the hands and forearms are in contact with a contaminated surface.

**Fraction Transferred from Surface to Skin (FTSS)**

Residue-to-skin transfer efficiency is likely to be dependent on the chemical properties of the contaminating substance and (if applicable) the carrier (e.g. solvent) in which the chemical is present. Nevertheless, the transfer efficiencies reported by Camann et al. (2000) for chlorpyrifos, pyrethrin I and piperonyl butoxide – three chemically distinct substances – typically varied over a range of 2-fold or less, which is not considered to be significant. The nature of the surface had a much greater effect on transfer efficiency. According to the authors, transfers from vinyl flooring were 2- to 10-fold greater than from plush carpets where the mean values ranged from 0.01 [1%] for carpet to 0.04 [4%] for vinyl. The USEPA (2007) adopted a distribution of transfer efficiencies from a number of studies where the mean transfer efficiency was 0.07 (or 7%). The data was reviewed by OEHHA (2009) and the mean value of 7% adopted for the derivation of a cleanup level. The value of 7% has also been adopted in this assessment for the transfer efficiency for all compounds assessed for surfaces within a residential or commercial building.

**Dermal Absorption (ABSd)**

Where available, compound specific values have been adopted as noted in Appendix B for each key chemical considered (per day).

**Dermal Bioavailability (Bd)**

Assumed to be 100% for all compounds assessed.
Body Weight (BW)
The body weight of each group (adult or child) has been obtained from data presented in the USEPA Exposure Factors Handbook (1997). The statistical analysis of body weights in the US for different age groups in the population as well as adults (males and females) is considered to be representative of expected body weights in Australia. Hence data available from this source has been used for the derivation of ILs. The following values have been adopted for the age groups considered:

- **Residential** - Young children aged 6 months to 2 years: 9.84 kg based on the average male and female 50% body weight for male and females aged 1 year;
- **Commercial** – Adult Workers: 70 kg based on the default value used by the USEPA considered relevant for the Australian population.

Exposure Frequency and Duration (EF, ED)
The derivation of ILs has assumed:

- **Residential** exposures will occur on 365 days per year for the age groups assessed, which is 18 months for young children (peak exposures); and
- **Commercial/industrial** worker exposures will occur on 240 days per year for 30 years (enHealth 2002).

Averaging Time (AT)
The averaging time adopted for the assessment of exposure depends on whether the assessment is undertaken on the basis of a non-threshold or threshold approach.

For a threshold approach the potential for adverse effects to occur is only relevant when exposed, hence the averaging time is equal to the exposure duration (converted to days).

For a non-threshold approach any exposure has the potential to result in adverse (carcinogenic) effects to occur at some point during a lifetime. Hence the averaging time for non-threshold effects is equal to a lifetime, taken to be 70 years (converted to days).

C3 Oral Intake – Hand to Mouth
Incidental ingestion as a result of placing hands in the mouth following exposure to surface residues has been estimated as follows (chronic exposures based on the equation presented in USEPA (2007):

\[
O_H = \frac{DL_H \cdot HF \cdot (1 - (1 - MRE)(FQH \cdot T)) \cdot ABSo \cdot Bo \cdot EF \cdot ED}{BW \cdot AT}
\]

...(Equation B3)

Where:
- \(DL_H\) = Dermal loading on one hand, based on Equation C2 (mg/day)
- \(HF\) = Fraction of one hand in the mouth (unitless)
- \(MRE\) = Mouthing removal efficiency from skin (unitless)
- \(FQH\) = Frequency of hand to mouth behaviour (events/hour)
- \(T\) = Time spent mouthing hands or objects (hours)
- \(ABSo\) = Oral absorption fraction (unitless)
- \(Bo\) = Oral bioavailability (unitless)
Fraction of Hand in Mouth (HF)
Residential: For young children (6 months to 2 years) the fraction of one hand that enters the mouth during mouthing has been taken to be 0.165 [or 16.5%], the 75th percentile from the distribution presented by USEPA (2005).

Commercial: For adult workers the fraction of each hand that is placed in the mouth has been taken to be 0.088 [or 8.3%], the 25th percentile from the distribution presented by USEPA (2005), assumed relevant for adult workers who are not expected to place as much of their hands in their mouth.

Mouthing Removal Efficiency (MRE)
Residential: For young children (6 months to 2 years) the efficiency of residue removal from the skin during mouthing of the hand has been taken to be 0.849 [84.9%], the 75th percentile from the distribution presented by USEPA (2005).

Commercial: For adult workers the efficiency of residue removal from the skin during mouthing of the hand has been taken to be 0.72 [72%], the 25th percentile from the distribution presented by USEPA (2005), assumed relevant for adults.

Frequency of Hand to Mouth Behaviour (FQH)
Residential: For young children (6 months to 2 years), 10 mouthing (of hands) events per hour has been considered based on the mean value presented by OEHHA (2009) which is consistent with the 75th percentile from the distribution presented by USEPA (2005).

Commercial: For adult workers 1.27 mouthing (of hands) events per hour has been considered based on the 25th percentile from the distribution presented by USEPA (2005), assumed relevant for adults.

Time Spent Mouthing (T)
Residential: For young children (6 months to 2 years) it has been assumed that mouthing of hands occurs for 6 hours of the day. The value adopted is higher than the value considered by OEHHA (2009) of 4 hours.

Commercial: For adult workers it has been assumed that mouthing of hands occurs for 1 hour per day in total (1/8th of the work day).

Oral Absorption (ABSo)
Assume to be 100% for all compounds assessed (per day). This is likely to be conservative.

Oral Bioavailability (Bo)
Assumed to be 100% for all compounds assessed.

C4 Oral Intake – Objects to Mouth
Potential ingestion of contaminants via the mouthing of objects that may have come into contact with surface residues is relevant for the assessment of exposures by very young children only. A study into micro activity patterns of children by Freeman et al. (2001) showed that the observed activity rate for object to mouth behaviour as essentially 0 for children 5 years and over. Rates in children aged 3 and 4 years were significantly lower than observed in other studies for infants and children aged 2 years. Exposures by adults are essentially negligible.

Incidental ingestion as a result of placing an object that may have also been in contact with a surface where a residue is present in the mouth has been estimated as follows (chronic exposures based on the equation presented in USEPA (2007):
Derivation of Risk-Based Investigation Levels

Concentration Ratio (OR)
The ratio of the concentration on an object compared with that on the surface has been taken to be 0.2, the maximum value considered by OEHHA (2009).

Area of Object in Mouth (OA)
The area of an object that may be put into the mouth has been taken to be 35 cm² each day when indoors noted as a default by USEPA (2007) and higher than the maximum considered in the distribution adopted by OEHHA (2009).

Transfer Efficiency from Object to Mouth (TFOB)
The efficiency of residue removal from an object by the mouth has been taken to be 0.5, the maximum from the distribution presented by OEHHA (2009).

Frequency of Object to Mouth Behaviour (FQM)
The frequency of events where objects are placed in the mouth is 15 per hour based on the maximum value presented by OEHHA (2009).

Time Spent Mouthing (T)
For young children (6 months to 2 years) it has been assumed that mouthing of hands occurs for 6 hours of the day. The value adopted is higher than the value considered by OEHHA (2009) of 4 hours.

C5 Approach to Derivation of Surface Residue IL
Calculated ILs for residential (young children) and commercial (adults) are attached to this appendix. The following shows the equations used in the derivation of the ILs for threshold and non-threshold carcinogenic chemicals where a surface residue IL is considered relevant.

Equations C2 to C4 are used to derive an intake (by young children or adults) via the relevant pathways assessed, as presented in Equation C1. For the derivation of ILs, the equations have been rearranged to enable the calculation of the dislodgable surface residue concentration. This is the mass of the chemical, per unit surface area, that can be removed from the surface and transferred to hands or objects. It is the concentration that is measured and reported using a surface wipe sampling method. Hence the value of Cs is the IL to be derived for residential and commercial users of the site.

On this basis these equations (intakes over each pathway, expressed in mg/kg/day) have been combined and rearranged in the following way:

\[ D_H = C_s \cdot I F_{DH} \]
\[ D_B = C_s \cdot I F_{DB} \]
\[ O_H = C_s \cdot I F_{OH} \]
Derivation of Risk-Based Investigation Levels

Clandestine Drug Laboratory, Site Investigation Guidelines
Ref: ACC/09/R001-A

\[ O_o = C_s \cdot IFOO \]

\( I_{DH} = \text{intake factor for dermal absorption from hands (cm}^2/\text{kg/day)} \) from Equation C2:

\[ I_{DH} = \frac{SA_H \cdot CR_H \cdot FTSS \cdot ABSd \cdot Bd \cdot EF \cdot ED}{BW \cdot AT} \]

\( I_{DB} = \text{intake factor for dermal absorption from rest of the body (cm}^2/\text{kg/day)} \) from Equation C2:

\[ I_{DB} = \frac{SA_B \cdot CR_B \cdot FTSS \cdot ABSd \cdot Bd \cdot EF \cdot ED}{BW \cdot AT} \]

\( I_{OH} = \text{intake factor for ingestion of residues from the mouthing of hands (cm}^2/\text{kg/day)} \) from Equation C3:

\[ I_{OH} = \frac{SH_H \cdot CR_H \cdot FTSS \cdot HF \cdot (1 - (1 - MRE)^{(TFQH+T)}) \cdot ABSo \cdot Bo \cdot EF \cdot ED}{BW \cdot AT} \]

\( I_{OO} = \text{intake factor for ingestion of residues from the mouthing of objects (cm}^2/\text{kg/day)} \) from Equation C4:

\[ I_{OO} = \frac{OR \cdot OA \cdot (1 - (1 - TFOB)^{(TFQMT)}) \cdot ABSo \cdot Bo \cdot EF \cdot ED}{BW \cdot AT} \]

Equation C1 then becomes:

\[ \text{Intake}_{SR} = C_s \cdot (I_{DH} + I_{DB} + I_{OH} + I_{OO}) ....(\text{Equation C5}) \]

or

\[ \text{Intake}_{SR} = C_s \cdot I_{\text{total}} \] where \( I_{\text{total}} \) is the total intake factor from all pathways (relevant)

Calculation of Surface Residue IL for Threshold Chemicals

The calculation of a threshold HI compares the calculated intake with the threshold dose-response (toxicity value) identified in Appendix B for each threshold key chemical, accounting for background intakes (B) where relevant. This is presented in the following equation:

\[ H_I = \frac{\text{Intake}_{SR}}{TDI - B} \] ....(Equation C6)

Where the TDI is the threshold toxicity value (or tolerable daily intake) relevant for the chemical (expressed in mg/kg/day). The value may be derived from different sources and hence may be termed an ADI, RfD or MRL etc.

The HI adopted in the derivation of ILs is a value of 1 (as noted in the main report). This is the target HI identified and used in this assessment.

Combining Equations C5 and C6 and rearranging for \( C_s \), the IL for surface residues, the following equation is used for deriving the IL for surface residues for threshold chemicals:
Calculation of Surface Residue IL for Non-Threshold Chemicals

The calculation of a non-threshold risk involves the multiplication of the calculated intake with the slope factor identified in Appendix B for oral and dermal (same as oral) slope factor (SF, expressed in \((\text{mg/kg/day})^{-1}\)) for each key chemical where non-threshold carcinogenic effects are of most significance. The approach adopted does not require consideration of background intakes as the target risk level is an incremental risk target, above background. This is presented in the following equation:

\[
\text{Risk} = \text{Intake}_{SR} \times (\text{Slope Factor})
\]

...(Equation C8)

The Risk level adopted in the derivation of ILs is a value of \(1 \times 10^{-5}\) (as noted in the main report). This is the target risk identified and used in this assessment for each key chemical where this approach is relevant.

Combining Equations C5 and C8 and rearranging for \(C_s\), the IL for surface residues, the following equation is used for deriving the IL for surface residues for non-threshold chemicals:

\[
IL_{surface} = C_s = \frac{T_{arg}et \text{ Risk}}{IF_{DH} + IF_{DB} + IF_{OH} + IF_{OO}} \times SF
\]

...(Equation C9)

Presentation of Surface Residue ILs

The attached tables present a summary of the assumptions presented in this Appendix, along with the calculations associated with the derivation of ILs for surface residues. It is noted that the IL is presented as \(\text{mg/cm}^2\) as well as \(\mu\text{g}/100\text{cm}^2\). The presentation of the IL in units of \(\mu\text{g}/100\text{cm}^2\) is relevant so that a direct comparison of the IL with measured surface residue concentrations (levels) from surface wipe sampling methods (typically calculated and presented as \(\mu\text{g}/100\text{cm}^2\)).

It is noted that the calculations attached show a level of accuracy no considered to be appropriate for the ILs to be used on any site. This is due to the level of uncertainty inherent in the assumptions adopted for the quantification of exposure, the toxicity values adopted and the level of uncertainty expected in the sampling and analysis of surface residues. Hence the ILs for surface residues presented in the main report have been rounded to no more than 2 significant figures.

C6 Review of ILs Calculated

The approach adopted for the derivation of an IL for surface residues has used equations and point values considered to provide a conservative estimate of intake vial dermal and ingestion pathways. The approach is a point value, simplistic application of a more complex exposure model (which considers exposure distributions and microactivity patterns based on diary entries), SHEDS (USEPA, 2007). The conservative nature of the approach adopted can be illustrated by comparison of the IL derived for methamphetamine with that derived by OEHHA (2009) using the more complex SHEDS model. The comparison is considered to be appropriate as both the OEHHA guideline and the IL derived in this assessment have adopted the same threshold RfD derived by OEHHA (2009b) and are derived for the most sensitive residential receptor, young children aged 6 months to 2 years. Dermal absorption values adopted are also equivalent. The following is noted with respect to the IL and calculations presented by OEHHA (2009a):
IL derived in this assessment for methamphetamine surface residues = 0.5 µg/100cm²;

OEHHA (2009) guideline for methamphetamine surface residues = 1.5 µg/100cm². Based on this value the 95th to 99th percentile intakes of methamphetamine (from the output distribution generated) are essentially equal to the adopted RfD. Hence the guideline value adopted by OEHHA is a conservative value. This is 3 times higher (less conservative) than the IL derived.

Intake of methamphetamine calculated by OEHHA is noted to be dominated by dermal absorption from the rest of the body (approximately 80% of total intake). Review of the intakes calculated in the derivation of the ILs suggests dermal absorption contributes approximately 65% of the total intake. The difference in contribution is expected to be associated with the adoption of more conservative point values in the ILs for intake parameters for the ingestion pathways.

The approach adopted by OEHHA identified that the model is most sensitive (direct relationship) to the skin surface area (particularly for the body) and fraction transferred from surface to skin (FTSS). This is consistent with that observed in the approach adopted in this assessment.

On the basis of the above review, the approach adopted for the derivation of ILs for surface residues is considered to be appropriate and adequately conservative.

C7 References


Attachment – Calculations
### Table C1  Summary of Exposure Assumptions –ILs Surface Residues – Residential Use

<table>
<thead>
<tr>
<th>Assessment of Exposure to Surface Residues</th>
<th>Abbrev.</th>
<th>Units</th>
<th>Parameter</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hands - Surface Area Exposed to residue</td>
<td>SAH</td>
<td>cm$^2$</td>
<td>301</td>
<td>Area of hands for child aged 1-2 years (USEPA 1997)</td>
</tr>
<tr>
<td>Hands - Contact Rate</td>
<td>CRH</td>
<td>per day</td>
<td>14.8</td>
<td>Assuming contact rate of 0.74 over 20 minutes for hands (mean from USEPA [2005]) with up to 20 events each day where residue remains on skin</td>
</tr>
<tr>
<td>Body (no hands) - Surface Area Exposed to residue</td>
<td>SAB</td>
<td>cm$^2$</td>
<td>3122</td>
<td>Area of unclothed body (head, arms, legs and feet) for child aged 1-2 years (USEPA 1997)</td>
</tr>
<tr>
<td>Body (no hands) - Contact Rate</td>
<td>CRB</td>
<td>per day</td>
<td>3.16</td>
<td>Assuming contact rate of 0.158 over 20 minutes for body (mean from USEPA [2005]) with up to 20 events each day where residue remains on skin</td>
</tr>
<tr>
<td>Fraction transferred surface to skin</td>
<td>FTSS</td>
<td>-</td>
<td>0.07</td>
<td>OEHHA 2009 for all surfaces (hard and soft)</td>
</tr>
<tr>
<td>Fraction of hand in mouth</td>
<td>HF</td>
<td>-</td>
<td>0.165</td>
<td>75th percentile from USEPA (2005)</td>
</tr>
<tr>
<td>Mouthing removal efficiency</td>
<td>MRE</td>
<td>-</td>
<td>0.849</td>
<td>75th percentile from USEPA (2005)</td>
</tr>
<tr>
<td>Frequency of hand-mouth behaviour</td>
<td>FQH</td>
<td>#/hour</td>
<td>10</td>
<td>Mean value OEHHA (2009) and 75% from USEPA (2005)</td>
</tr>
<tr>
<td>Time for mouthing hands and objects</td>
<td>T</td>
<td>hours</td>
<td>6</td>
<td>Assumed amount of time in contact with surfaces and objects (Note: 4 hours assumed in OEHHA [2009])</td>
</tr>
<tr>
<td>Concentration ratio object to surface</td>
<td>OR</td>
<td>-</td>
<td>0.2</td>
<td>Maximum from OEHHA (2009)</td>
</tr>
<tr>
<td>Area of object in mouth</td>
<td>OA</td>
<td>cm$^2$ each day</td>
<td>35</td>
<td>Default from USEPA (2007)</td>
</tr>
<tr>
<td>Transfer fraction from object to mouth per event</td>
<td>TFOB</td>
<td>-</td>
<td>0.5</td>
<td>Default from USEPA (2007)</td>
</tr>
<tr>
<td>Frequency of mouthing (objects) events</td>
<td>FQM</td>
<td>#/hour</td>
<td>15</td>
<td>Upper mean value from OEHHA (2009)</td>
</tr>
<tr>
<td>Oral absorption fraction</td>
<td>ABSo</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Oral bioavailability</td>
<td>Bo</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Dermal bioavailability</td>
<td>Bd</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Exposure Frequency</td>
<td>EF</td>
<td>days/year</td>
<td>365</td>
<td>Assume at home indoors every day of the year</td>
</tr>
<tr>
<td>Exposure Duration</td>
<td>ED</td>
<td>years</td>
<td>1.5</td>
<td>Assume peak exposure occurs from 6 months to 2 years</td>
</tr>
<tr>
<td>Averaging Time (non-carcinogenic)</td>
<td>AT$_{NC}$</td>
<td>days</td>
<td>547.5</td>
<td>Based on threshold (non carcinogenic) effects</td>
</tr>
<tr>
<td>Averaging Time (carcinogenic)</td>
<td>AT$_C$</td>
<td>days</td>
<td>25550</td>
<td>Based on 70 year lifetime for carcinogenic (non-threshold) effects</td>
</tr>
<tr>
<td>Body Weight</td>
<td>BW</td>
<td>kg</td>
<td>9.84</td>
<td>Average male and female body weight for 1 year old (USEPA, 1997)</td>
</tr>
</tbody>
</table>
## Table C2  Derivation of ILs Surface Residues – Threshold Effects - Residential Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd) (-)</th>
<th>Tolerable Daily Intake (TDI) (mg/kg/day)</th>
<th>Background Intake (B) (mg/kg/day)</th>
<th>Total intake factor (IF) (cm²/kg/day)</th>
<th>Target HI</th>
<th>Derived Residue IL - Surface Threshold (mg/cm²)</th>
<th>Calculated Intakes from Derived ILs (mg/kg/day)</th>
<th>Total Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methamphetamine</td>
<td>0.57</td>
<td>0.0003</td>
<td>0</td>
<td>61.4</td>
<td>1</td>
<td>4.9E-06</td>
<td>8.8E-05</td>
<td>3.0E-04</td>
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<tr>
<td>MDMA</td>
<td>0.57</td>
<td>0.004</td>
<td>0</td>
<td>61.4</td>
<td>1</td>
<td>6.5E-05</td>
<td>1.2E-03</td>
<td>4.0E-03</td>
</tr>
<tr>
<td>Pseudo/Ephedrine</td>
<td>1</td>
<td>1.3</td>
<td>0.65</td>
<td>105.2</td>
<td>1</td>
<td>6.2E-03</td>
<td>2.0E-01</td>
<td>6.5E-01</td>
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<tr>
<td>Iodine</td>
<td>0.14</td>
<td>0.01</td>
<td>0.006</td>
<td>17.6</td>
<td>1</td>
<td>2.3E-04</td>
<td>1.0E-03</td>
<td>4.0E-03</td>
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<td>Bromide</td>
<td>0.01</td>
<td>0.1</td>
<td>0.01</td>
<td>4.3</td>
<td>1</td>
<td>2.1E-02</td>
<td>6.6E-03</td>
<td>9.0E-02</td>
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<td>Phosphorus</td>
<td>0.1</td>
<td>0.00002</td>
<td>0.0001</td>
<td>13.5</td>
<td>1</td>
<td>7.4E-07</td>
<td>2.3E-06</td>
<td>1.0E-05</td>
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<tr>
<td>N-Methylformamide</td>
<td>0.6</td>
<td>0.0086</td>
<td>0</td>
<td>64.4</td>
<td>1</td>
<td>1.3E-04</td>
<td>2.5E-03</td>
<td>8.6E-03</td>
</tr>
<tr>
<td>Boron</td>
<td>0.01</td>
<td>0.2</td>
<td>0.12</td>
<td>4.3</td>
<td>1</td>
<td>1.8E-02</td>
<td>5.8E-03</td>
<td>8.0E-02</td>
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<tr>
<td>Mercury</td>
<td>0.01</td>
<td>0.002</td>
<td>0.0005</td>
<td>4.3</td>
<td>1</td>
<td>3.5E-04</td>
<td>1.1E-04</td>
<td>1.5E-03</td>
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<tr>
<td>Lithium</td>
<td>0.01</td>
<td>0.002</td>
<td>0</td>
<td>4.3</td>
<td>1</td>
<td>4.6E-04</td>
<td>1.5E-04</td>
<td>2.0E-03</td>
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<tr>
<td>Benzaldehyde</td>
<td>0.03</td>
<td>0.1</td>
<td>0</td>
<td>6.4</td>
<td>1</td>
<td>1.6E-02</td>
<td>1.5E-02</td>
<td>1.0E-01</td>
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<tr>
<td>TPH C15+</td>
<td>0.13</td>
<td>0.03</td>
<td>0.006</td>
<td>16.6</td>
<td>1</td>
<td>1.4E-03</td>
<td>6.0E-03</td>
<td>2.4E-02</td>
</tr>
</tbody>
</table>

## Table C3  Derivation of ILs Surface Residues – Non-Threshold Effects - Residential Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd) (-)</th>
<th>Non-Threshold Slope Factor (SF) (mg/kg/day)</th>
<th>Total intake factor (IF) (cm²/kg/day)</th>
<th>Target Risk</th>
<th>Derived Surface Residue IL - Non-Threshold (mg/cm²)</th>
<th>Calculated Intakes from Derived ILs (mg/kg/day)</th>
<th>Total Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Safrole and Isosafrole</td>
<td>0.1</td>
<td>0.22</td>
<td>0.290</td>
<td>1E-05</td>
<td>1.6E-04</td>
<td>11.1E-05</td>
<td>4.5E-05</td>
</tr>
</tbody>
</table>
### Table C4  Summary of Exposure Assumptions –ILs Surface Residues – Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Assessment of Exposure to Indoor Surface Residues</th>
<th>Abbrev.</th>
<th>Units</th>
<th>Parameter</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hands - Surface Area Exposed to residue</td>
<td>SAH</td>
<td>cm²</td>
<td>1024</td>
<td>Average 90th percentile male/female surface area for hands (USEPA 1997)</td>
</tr>
<tr>
<td>Hands - Contact Rate</td>
<td>CRH</td>
<td>per day</td>
<td>5.9</td>
<td>Assuming contact rate of 0.74 over 20 minutes for hands (mean from USEPA [2005]) with up to 8 events each day where residue remains on skin</td>
</tr>
<tr>
<td>Body (no hands) - Surface Area Exposed to residue</td>
<td>SAB</td>
<td>cm²</td>
<td>1350</td>
<td>Area of unclothed body potentially touching surfaces (lower arms) average 90th percentile male/female for adult (USEPA 1997)</td>
</tr>
<tr>
<td>Body (no hands) - Contact Rate</td>
<td>CRB</td>
<td>per day</td>
<td>1.3</td>
<td>Assuming contact rate of 0.158 over 20 minutes for body (mean from USEPA [2005]) with up to 8 events each day where residue remains on skin</td>
</tr>
<tr>
<td>Fraction transferred surface to skin</td>
<td>FTSS</td>
<td>-</td>
<td>0.07</td>
<td>OEHHA 2009 for all surfaces (hard and soft)</td>
</tr>
<tr>
<td>Fraction transferred from hand to mouth</td>
<td>FTSM</td>
<td>-</td>
<td>0.5</td>
<td>50% efficiency based on pesticide studies</td>
</tr>
<tr>
<td>Fraction of hand in mouth</td>
<td>HF</td>
<td>-</td>
<td>0.083</td>
<td>25th percentile from USEPA (2005) for young children assumed relevant for adults</td>
</tr>
<tr>
<td>Mouthing removal efficiency</td>
<td>MRE</td>
<td>-</td>
<td>0.72</td>
<td>25th percentile from USEPA (2005) for young children assumed relevant for adults</td>
</tr>
<tr>
<td>Frequency of hand-mouth behaviour</td>
<td>FQH</td>
<td>#/hour</td>
<td>1.27</td>
<td>25th percentile from USEPA (2005) for young children assumed relevant for adults</td>
</tr>
<tr>
<td>Time for mouthing hands</td>
<td>T</td>
<td>hours</td>
<td>1</td>
<td>Assumed for adults</td>
</tr>
<tr>
<td>Oral absorption fraction</td>
<td>ABS0</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Oral bioavailability</td>
<td>Bo</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Dermal bioavailability</td>
<td>Bd</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Exposure Frequency</td>
<td>EF</td>
<td>days/year</td>
<td>240</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Exposure Duration</td>
<td>ED</td>
<td>years</td>
<td>30</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Averaging Time (non-carcinogenic)</td>
<td>AT&lt;sub&gt;NC&lt;/sub&gt;</td>
<td>days</td>
<td>10950</td>
<td>Based on threshold (non-carcinogenic) effects</td>
</tr>
<tr>
<td>Averaging Time (carcinogenic)</td>
<td>AT&lt;sub&gt;C&lt;/sub&gt;</td>
<td>days</td>
<td>25550</td>
<td>Based on 70 year lifetime for carcinogenic (non-threshold) effects</td>
</tr>
<tr>
<td>Body Weight</td>
<td>BW</td>
<td>kg</td>
<td>70</td>
<td>Default for adults from USEPA considered relevant for Australian population</td>
</tr>
</tbody>
</table>
### Table C5  Derivation of ILs Surface Residues – Threshold Effects - Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd) (-)</th>
<th>Tolerable Daily Intake (TDI) (mg/kg/day)</th>
<th>Background Intake (B) (mg/kg/day)</th>
<th>Total intake factor (IF) (cm²/kg/day)</th>
<th>Target HI</th>
<th>Derived Surface Residue IL - Threshold (µg/100 cm²)</th>
<th>Calculated Intakes from IL (mg/kg/day)</th>
<th>Dermal intake via hands (DH)</th>
<th>Dermal intake via rest of body (DB)</th>
<th>Oral intake from hands (OH)</th>
<th>Total Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methamphetamine</td>
<td>0.57</td>
<td>0.0003</td>
<td>0</td>
<td>3.043</td>
<td>1</td>
<td>9.9E-05</td>
<td>2.2E-04</td>
<td>6.3E-05</td>
<td>1.3E-05</td>
<td>3.0E-04</td>
<td></td>
</tr>
<tr>
<td>MDMA</td>
<td>0.57</td>
<td>0.004</td>
<td>0</td>
<td>3.043</td>
<td>1</td>
<td>1.3E-03</td>
<td>131</td>
<td>3.0E-03</td>
<td>8.4E-04</td>
<td>1.7E-04</td>
<td></td>
</tr>
<tr>
<td>Pseudo/Ephedrine</td>
<td>1</td>
<td>1.3</td>
<td>0.65</td>
<td>5.239</td>
<td>1</td>
<td>1.2E-01</td>
<td>12408</td>
<td>4.9E-01</td>
<td>1.4E-01</td>
<td>1.6E-02</td>
<td>6.5E-01</td>
</tr>
<tr>
<td>Iodine</td>
<td>0.14</td>
<td>0.01</td>
<td>0.006</td>
<td>0.847</td>
<td>1</td>
<td>4.7E-03</td>
<td>472</td>
<td>2.6E-03</td>
<td>7.4E-04</td>
<td>6.3E-04</td>
<td>4.0E-03</td>
</tr>
<tr>
<td>Bromide</td>
<td>0.01</td>
<td>0.1</td>
<td>0.1</td>
<td>0.184</td>
<td>1</td>
<td>4.9E-01</td>
<td>49027</td>
<td>2.0E-02</td>
<td>5.5E-03</td>
<td>6.5E-02</td>
<td>9.0E-02</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.1</td>
<td>0.000002</td>
<td>0.0001</td>
<td>0.643</td>
<td>1</td>
<td>1.6E-05</td>
<td>1.55</td>
<td>6.2E-06</td>
<td>1.7E-06</td>
<td>2.1E-06</td>
<td>1.0E-05</td>
</tr>
<tr>
<td>N-Methylformamide</td>
<td>0.6</td>
<td>0.0086</td>
<td>0</td>
<td>3.196</td>
<td>1</td>
<td>2.7E-03</td>
<td>268</td>
<td>6.4E-03</td>
<td>1.8E-03</td>
<td>3.6E-04</td>
<td>8.6E-03</td>
</tr>
<tr>
<td>Boron</td>
<td>0.01</td>
<td>0.2</td>
<td>0.12</td>
<td>0.184</td>
<td>1</td>
<td>4.4E-01</td>
<td>43580</td>
<td>1.7E-02</td>
<td>4.9E-03</td>
<td>5.8E-02</td>
<td>8.0E-02</td>
</tr>
<tr>
<td>Mercury</td>
<td>0.01</td>
<td>0.002</td>
<td>0.005</td>
<td>0.184</td>
<td>1</td>
<td>8.2E-03</td>
<td>817</td>
<td>3.3E-04</td>
<td>9.2E-05</td>
<td>1.1E-03</td>
<td>1.5E-03</td>
</tr>
<tr>
<td>Lithium</td>
<td>0.01</td>
<td>0.002</td>
<td>0.005</td>
<td>0.184</td>
<td>1</td>
<td>1.1E-02</td>
<td>1089</td>
<td>4.3E-04</td>
<td>1.2E-04</td>
<td>1.4E-03</td>
<td>2.0E-03</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>0.03</td>
<td>0.1</td>
<td>0</td>
<td>0.286</td>
<td>1</td>
<td>3.5E-01</td>
<td>35003</td>
<td>4.2E-02</td>
<td>1.2E-02</td>
<td>4.6E-02</td>
<td>1.0E-01</td>
</tr>
<tr>
<td>TPH C15+</td>
<td>0.13</td>
<td>0.03</td>
<td>0.006</td>
<td>0.796</td>
<td>1</td>
<td>3.0E-02</td>
<td>3014</td>
<td>1.6E-02</td>
<td>4.4E-03</td>
<td>4.0E-03</td>
<td>2.4E-02</td>
</tr>
</tbody>
</table>

### Table C6  Derivation of ILs Surface Residues – Non-Threshold Effects - Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd) (-)</th>
<th>Non-Threshold Slope Factor (SF) (mg/kg/day)</th>
<th>Total intake factor (IF) (cm²/kg/day)</th>
<th>Target Risk</th>
<th>Derived Surface Residue IL - Non-Threshold (µg/100 cm²)</th>
<th>Calculated Intakes from IL (mg/kg/day)</th>
<th>Dermal intake via hands (DH)</th>
<th>Dermal intake via rest of body (DB)</th>
<th>Oral intake from hands (OH)</th>
<th>Total Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Safrole and Isosafrole</td>
<td>0.1</td>
<td>0.22</td>
<td>0.276</td>
<td>1E-05</td>
<td>16.5</td>
<td>2.8E-05</td>
<td>7.9E-06</td>
<td>9.4E-06</td>
<td>4.5E-05</td>
<td></td>
</tr>
</tbody>
</table>
Appendix D – Methodology and Derivation of ILs for Indoor Air
# Table of Contents

D1  Introduction.................................................................................................................. D-1
D2  Approach to Derivation of Indoor Air ILs..................................................................... D-2
D3  Derivation of Indoor Air ILs....................................................................................... D-3
D4  References................................................................................................................... D-4
D1 Introduction
This appendix presents the methodology and basis for assumptions adopted in the derivation of ILs for indoor air. The quantification of ILs involves consideration of exposure and relevant toxicity data. The approach to quantification of dose-response and the toxicity data used in this assessment for key chemicals for which indoor air has been identified as important are presented in Appendix B. The following presents the approach and assumptions adopted for the quantification of exposure to indoor air and the methodology adopted in the derivation of the ILs.

The quantification of exposure to indoor air has considered inhalation exposures the most significant pathways for exposure. In addition the ILs for indoor air have focused on key chemicals that may have been absorbed by porous surfaces (such as soft furnishings, curtains and plasterboard) during the manufacturing phase of clan labs (where high concentrations are present indoors). The chemicals may continue to off-gas from these materials for some time following seizure and removal of manufacturing equipment and products. As this is the key mechanism addressed for indoor air in this assessment the focus is on volatile chemicals (and gasses) only. There are no significant pathways of concern that relate to particulate phase contaminants. Some chemicals (non-volatile) may be present in surface residues where activity may result in some particulate phase being released to indoor air. However during remediation more readily dislodgable residues are expected to be removed, and if the ILs derived for surface residues are met, particulate phase issues are not considered to be of significance.

With respect to the scenarios for which ILs have been derived for indoor air the following have been considered as the most sensitive groups relevant for the derivation of ILs:

- **Residential** – Residents are the most sensitive group assessed. However for the derivation of ILs for chemicals assessed on the basis of a threshold dose-response approach, young children aged 0 to 5 years are the most sensitive and the ILs have been derived on the basis of exposure assumptions relevant to this group. For the derivation of ILs for chemicals assessed on the basis of a non-threshold carcinogenic approach, exposures over a lifetime (as a child and adult) are considered relevant. Hence the non-threshold ILs have been derived on the basis of lifetime exposures by residents.

- **Commercial** – Assessed on the basis of potential exposures by adult workers in a workplace. It is assumed that the application of ILs for commercial/industrial use will be relevant for these uses only. Any more sensitive uses that may be allowable under a particular zoning, such as childcare, schools or hotel/motels need to be considered as residential scenarios. Hence the commercial ILs have been derived on the basis of the protection of exposures by adults in the workplace only.

- The approach adopted for the derivation of ILs for residents and adults assumes no change in exposure concentration over time. This is considered to be a conservative approach as it is expected that the rate of off-gassing to indoor air will decline over time, particularly for chemicals with shorter atmospheric half-lives.

Recreational use has not included consideration of indoor exposures, hence no indoor air ILs have been derived for this use.
D2  Approach to Derivation of Indoor Air ILs

The overall approach to the quantification of exposures to indoor air has followed inhalation guidance provided by USEPA (2009). This approach recognises the approach and uncertainties associated with the derivation of inhalation toxicity values and requires the consideration of an exposure concentration (EC) rather than an intake. Therefore the approach adopted does not require receptor and age specific inputs on body weight and inhalation rates, rather it requires consideration of exposure time, frequency and duration as noted in the following equation:

\[ EC_{\text{indoors}} = \frac{Ca \cdot ET \cdot ABSi \cdot Bi \cdot EF \cdot ED}{AT} \quad (\text{mg/m}^3) \quad \ldots (\text{Equation D1}) \]

where:
- \( Ca \) = Concentration of chemical in air (mg/m\(^3\)), this is the indoor air IL derived in this assessment.
- \( ET \) = Exposure time (hours/day)
- \( ABSi \) = Absorption fraction or rate associated with inhalation exposures (unitless)
- \( Bi \) = Bioavailability associated within inhalation exposures (unitless)
- \( EF \) = Exposure frequency (days/year)
- \( ED \) = Exposure duration (years)
- \( AT \) = Averaging time (in hours)

**Exposure Time (ET)**

For the purpose of deriving indoor air ILs, the following exposure times have been adopted:

- **Residential** – All residents are assumed to be indoors for up to 20 hours each day (as per enHealth 2002 and NEPC 1999).
- **Commercial** - Adult workers are assumed to be indoors for up to 10 hours each day. This allows for longer work days which may occur in different industries.

**Absorption via Inhalation (ABSi)**

Assumed to be 100% for all compounds assessed.

**Bioavailability via Inhalation (Bi)**

Assumed to be 100% for all compounds assessed.

**Exposure Frequency and Duration (EF, ED)**

The derivation of ILs has assumed:

- **Residential** exposures will occur on 365 days per year for 5 years as a young child aged 9-5 years, 10 years as an older child aged 5 to 15 years and 55 years as an adult, where the lifetime exposure for residents is assumed to be 70 years;
- **Commercial/industrial** worker exposures will occur on 240 days per year for 30 years (enHealth 2002).

**Averaging Time (AT)**

The averaging time adopted for the assessment of exposure depends on whether the assessment is undertaken on the basis of a non-threshold or threshold approach.

For a threshold approach the potential for adverse effects to occur is only relevant when exposed, hence the averaging time is equal to the exposure duration (converted to hours).

For a non-threshold approach any exposure has the potential to result in adverse (carcinogenic) effects to occur at some point during a lifetime. Hence the averaging time for non-threshold effects is equal to a lifetime, taken to be 70 years (converted to hours).
D3 Derivation of Indoor Air ILs

Calculated ILs for residential and commercial areas are attached to this appendix. The following shows the equations used in the derivation of the ILs for threshold and non-threshold carcinogenic chemicals where an indoor air IL is considered relevant.

Calculation of Indoor Air IL for Threshold Chemicals

The calculation of a threshold HI compares the calculated intake with the threshold dose-response (toxicity value) identified in Appendix B for each threshold key chemical, accounting for background intakes (B) where relevant. This is presented in the following equation:

\[ HI = \frac{EC_{\text{indoors}}}{TC - B} \]  

...(Equation D2)

Where the TC is the threshold toxicity value (or tolerable daily intake) relevant for inhalation exposures to the chemical (expressed in mg/m³). The value may be derived from different sources and hence may be termed a MRL, RfC or guideline value.

It is noted that the background intakes adopted for the derivation of ILs are based on potential intakes via food, water and soil. As the ILs are to be used for comparison against measured indoor air concentrations, background (urban/ambient) air concentrations will be measured along with any contribution from off-gassing indoors. Hence the background intakes adopted for the derivation of the ILs for indoor air differs from those adopted for the derivation of ILs for other media (surface residues and soil). In particular, the background intake for most volatile/gaseous chemicals assessed has been taken to be negligible as inhalation (from all sources) is expected to be the most significant exposure. The only chemicals where some background intake has been assumed are chloroform and ammonia (common by-products of disinfection and exposure can occur while bathing) and iodine (where other intakes may occur).

The HI adopted in the derivation of ILs is a value of 1 (as noted in the main report). This is the target HI identified and used in this assessment.

Combining Equations D1 and D2 and rearranging for \( C_a \), the IL for surface residues, the following equation is used for deriving the IL for surface residues for threshold chemicals:

\[ IL_{\text{indoor air}} = C_a = \frac{T_{\text{target HI}} \cdot (C - B) \cdot AT_{\text{threshold}}}{ET \cdot ABS_i \cdot B_i \cdot EF \cdot ED} \]  

\((\text{mg/m}^3)\)  

...(Equation D3)

Calculation of Surface Residue IL for Non-Threshold Chemicals

The calculation of a non-threshold risk involves the multiplication of the calculated intake with the inhalation unit risk identified in Appendix B (UR, expressed in (mg/m³)⁻¹) for each key chemical where non-threshold carcinogenic effects are of most significance. The approach adopted does not require consideration of background intakes as the target risk level is an incremental risk target, above background. This is presented in the following equation:

\[ \text{Risk} = EC_{\text{indoors}} \cdot (\text{Unit Risk}) \]  

...(Equation D4)

The Risk level adopted in the derivation of ILs is a value of \( 1 \times 10^{-5} \) (as noted in the main report). This is the target risk identified and used in this assessment for each key chemical where this approach is relevant.
Combining Equations D1 and D4 and rearranging for $C_a$, the IL for indoor air, the following equation is used for deriving the IL for non-threshold chemicals for residents and commercial/industrial workers:

$$IL_{\text{indoor air}} = C_a = \frac{T \text{arget Risk} \cdot AT_{\text{non-threshold}}}{UR \cdot (ET \cdot ABS_i \cdot B_i \cdot EF \cdot \sum ED)}$$

**(mg/m³)** ...(Equation D5)

**Presentation of Indoor Air ILs**

The attached tables present a summary of the assumptions presented in this Appendix, along with the calculations associated with the derivation of ILs for indoor air. It is noted that the calculations attached show a level of accuracy considered to be appropriate for the ILs to be used on any site. This is due to the level of uncertainty inherent in the assumptions adopted for the quantification of exposure, the toxicity values adopted and the level of uncertainty expected in the sampling and analysis of indoor air. Hence the ILs for indoor air presented in the main report have been rounded to no more than 2 significant figures.

**D4 References**


http://enhealth.nphp.gov.au/council/pubs/ecpub.htm and


### Table D1  Summary of Exposure Assumptions – ILs Indoor Air – Residential Use

<table>
<thead>
<tr>
<th>Assessment of Exposure to Indoor Air</th>
<th>Abbrev.</th>
<th>Units</th>
<th>Parameter</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time Spent Indoors</td>
<td>ETI</td>
<td>hours</td>
<td>20</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Inhalation absorption fraction</td>
<td>ABSi</td>
<td>per day</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Inhalation bioavailability</td>
<td>Bi</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Exposure Frequency</td>
<td>EF</td>
<td>days/year</td>
<td>365</td>
<td>Assume at home indoors every day of the year</td>
</tr>
<tr>
<td>Exposure Duration</td>
<td>ETI</td>
<td>years</td>
<td>5</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>- Young children (0-5 years)</td>
<td>EDYC</td>
<td>years</td>
<td>10</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>- Older children (5-15 years)</td>
<td>EDOC</td>
<td>years</td>
<td>55</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>- Adults</td>
<td>EDₐ</td>
<td>years</td>
<td>55</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>Averaging Time (non-carcinogenic)</td>
<td>ATₐNC</td>
<td>hours</td>
<td>ED<em>365</em>24</td>
<td>Calculated based on ED for each relevant age group</td>
</tr>
<tr>
<td>Averaging Time (carcinogenic)</td>
<td>ATₐC</td>
<td>hours</td>
<td>613200</td>
<td>Based on lifetime of 70 years (converted to hours)</td>
</tr>
</tbody>
</table>

### Table D2  Derivation of ILs Indoor Air - Residential Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Tolerable Concentration in Air (mg/m³) (TC)</th>
<th>Background Intake (B)</th>
<th>Target HI</th>
<th>Derived Indoor Air IL - Threshold (mg/m³)</th>
<th>Non-Threshold Unit Risk (mg/m³)^-1</th>
<th>Non-Threshold Target Risk</th>
<th>Derived Indoor Air IL - Non-Threshold (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroform</td>
<td>0.14</td>
<td>25%</td>
<td>1</td>
<td>0.13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dichloromethane</td>
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<td>0</td>
<td>1</td>
<td>1.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ammonia</td>
<td>0.1</td>
<td>10%</td>
<td>1</td>
<td>0.11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iodine</td>
<td>0.001</td>
<td>30%</td>
<td>1</td>
<td>0.00084</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bromide</td>
<td>0.0007</td>
<td>0</td>
<td>1</td>
<td>0.00084</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methylamine</td>
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<td>0</td>
<td>1</td>
<td>0.0036</td>
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<td></td>
</tr>
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<td>Nitroethane</td>
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<td>0</td>
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<td>Ethylbenzene</td>
<td>22</td>
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<td>1</td>
<td>26</td>
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<tr>
<td>Xylenes (total)</td>
<td>0.57</td>
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<td>1</td>
<td>1.0</td>
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<td>Naphthalene</td>
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Table D3  Summary of Exposure Assumptions – ILs Indoor Air – Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Assessment of Exposure to Indoor Air</th>
<th>Abbrev.</th>
<th>Units</th>
<th>Parameter</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time Spent Indoors</td>
<td>ET _i</td>
<td>hours</td>
<td>10</td>
<td>Assumed relevant for commercial workers indoors</td>
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<tr>
<td>Inhalation absorption fraction</td>
<td>ABS _i</td>
<td>per day</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
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<tr>
<td>Inhalation bioavailability</td>
<td>Bi</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
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<tr>
<td>Exposure Frequency</td>
<td>EF</td>
<td>days/year</td>
<td>240</td>
<td>Assume at home indoors every day of the year</td>
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<tr>
<td>Exposure Duration - Adults</td>
<td>ED _A</td>
<td>years</td>
<td>30</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
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<tr>
<td>Averaging Time (non-carcinogenic)</td>
<td>AT _NC</td>
<td>hours</td>
<td>ED<em>365</em>24</td>
<td>Calculated based on ED for each relevant age group</td>
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<tr>
<td>Averaging Time (carcinogenic)</td>
<td>AT _C</td>
<td>hours</td>
<td>613200</td>
<td>Based on lifetime of 70 years (converted to hours)</td>
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Table D4  Derivation of ILs Indoor Air - Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Tolerable Concentration in Air (mg/m³) (TC)</th>
<th>Background Intake (B)</th>
<th>Target HI</th>
<th>Derived Indoor Air IL - Threshold (mg/m³)</th>
<th>Non-Threshold Unit Risk (mg/m³)(^{-1})</th>
<th>Non-Threshold Effects</th>
</tr>
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<tr>
<td>Chloroform</td>
<td>0.14</td>
<td>25%</td>
<td>1</td>
<td>0.38</td>
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<td>Dichloromethane</td>
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<td>0</td>
<td>1</td>
<td>3.7</td>
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<tr>
<td>Ammonia</td>
<td>0.1</td>
<td>10%</td>
<td>1</td>
<td>0.33</td>
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<tr>
<td>Iodine</td>
<td>0.001</td>
<td>30%</td>
<td>1</td>
<td>0.003</td>
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<td>Bromide</td>
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<td>0</td>
<td>1</td>
<td>0.003</td>
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<td>Methylene</td>
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<td>0</td>
<td>1</td>
<td>0.11</td>
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<td></td>
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<tr>
<td>Nitroethane</td>
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<td>0</td>
<td>1</td>
<td>1.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benzaldehyde</td>
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<td>0</td>
<td>1</td>
<td>1.3</td>
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<td></td>
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<tr>
<td>Phosphine</td>
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<td>0</td>
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<td>0.0011</td>
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<tr>
<td>Safrole and Isosafrole</td>
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<td></td>
<td></td>
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<td>0.063</td>
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<td>Benzene</td>
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<td>0.11</td>
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<td>Toluene</td>
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<td>18</td>
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</tr>
<tr>
<td>Ethylbenzene</td>
<td>22</td>
<td>0</td>
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<td>80</td>
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<td>Xylenes (total)</td>
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<td></td>
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<tr>
<td>Naphthalene</td>
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<td>0</td>
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<td>0.011</td>
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<td>TPH C6-C9</td>
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<td>TPH C10-C14</td>
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<td>0.73</td>
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</tbody>
</table>
Appendix E – Methodology and Derivation of ILs for Outdoor Soil
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E1 Introduction
This appendix presents the methodology and basis for assumptions adopted in the derivation of ILs for outdoor soil. The quantification of ILs involves consideration of exposure and relevant toxicity data. The approach to quantification of dose-response and the toxicity data used in this assessment are presented in Appendix B. The following presents the approach and assumptions adopted for the quantification of exposure to outdoor soil and the methodology adopted for the derivation of the ILs.

The quantification of exposure to outdoor soil has considered the following pathways as significant:

- Dermal absorption of key chemicals during contact with soil (or dust indoors that may be derived from outdoor soil);
- Ingestion of soil (or dust indoors that may be derived from outdoor soil);
- Inhalation of volatile chemicals that may be derived from contamination remaining on outdoor soil; and
- Inhalation of key chemicals that may be present in particulates (dust) generated from outdoor soil (where surface cover is poor).

The ILs derived for soil are relevant for outdoor areas of a former clan lab. The operation of a former clan lab may have resulted in the “dumping” of base and waste products outdoors. It is noted that should ILs be required to be used on a site where the existing building is to be demolished and there is the potential for a new building to be constructed in another location, including above contaminated soil, then the IL may require revision (for volatile key chemicals) to address the potential for vapour intrusion. This would need to be undertaken on a site-specific basis.

With respect to the scenarios for which ILs have been derived the following have been considered as the most sensitive groups:

- Residential – Residents are the most sensitive group assessed. However for the derivation of ILs for chemicals assessed on the basis of a threshold dose-response approach, young children aged 0 to 5 years are the most sensitive and the ILs have been derived on the basis of exposure assumptions relevant to this group. For the derivation of ILs for chemicals assessed on the basis of a non-threshold carcinogenic approach, exposures over a lifetime (as a child and adult) are considered relevant. Hence the non-threshold ILs have been derived on the basis of lifetime exposures by residents.
- Commercial – Assessed on the basis of potential exposures by adult workers in a workplace. It is assumed that the application of ILs for commercial/industrial use will be relevant for these uses only. Any more sensitive uses that may be allowable under a particular zoning, such as childcare, schools or hotel/motels need to be considered as residential scenarios. Hence the commercial ILs have been derived on the basis of the protection of exposures by adults in the workplace only.
- The approach adopted for the derivation of ILs for residents and adults assumes no change in soil concentration over time. This is considered to be a conservative approach as it is expected that the concentration in soil will deplete/ degrade over time.

As the assessment of residential exposure has considered exposure to contamination in soil outdoors only, the ILs for recreational exposures have been assumed to be equal to those derived for residential areas. Recreational use may involve exposures over a lifetime (as per enHealth 2002). In addition exposures in a recreational area that is in close proximity to a residential area may result in exposures that are similar to that assumed on a residential property. Hence the ILs derived for residential outdoor soil have been considered representative and conservative for use as ILs for recreational exposures.
The following sections present further detail on the calculation of dermal and ingestion intakes and exposure concentrations relevant for inhalation exposures of volatiles and dust.

E2 Dermal Intake

Dermal exposure to surface soil contamination has been estimated as follows:

\[
\text{Daily Chemical Intake} = \frac{C_s \times \text{SA}s \times \text{AF} \times \text{FD} \times \text{ABSd} \times B_d \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad \text{(mg/kg/day)}
\]  

where:
- \(C_s\) = Concentration in soil (mg/kg), which is the IL to be derived for outdoor soil.
- \(\text{SA}s\) = Surface area of body exposed to soil each day (cm\(^2\)/day)
- \(\text{ABSd}\) = Dermal absorption fraction (unitless)
- \(\text{AF}\) = Adherence factor, amount of soil that adheres to the skin per unit area which depends on soil properties and area of body (mg/cm\(^2\) per event)
- \(\text{FD}\) = Fraction of day exposed, or dirty (unitless)
- \(\text{CF}\) = Conversion factor of \(1\times10^{-6}\) to convert mg to kg
- \(B_d\) = Dermal bioavailability (unitless)
- \(\text{EF}\) = Exposure frequency (days/year)
- \(\text{ED}\) = Exposure duration (years)
- \(\text{BW}\) = Body weight (kg)
- \(\text{AT}\) = Averaging time (days)

Skin Surface Area (SA)

The skin surface area of each group (adult or child) has been obtained from body part specific data presented in the USEPA Exposure Factors Handbook (1997). The following values have been adopted for the assessment presented:

- **Residential:**
  - Young children aged 0-5 years – 2625 cm\(^2\) based on 30% total body surface area relevant for contact with soil (and dust) as per enHealth (2003);
  - Older children aged 5-15 years - 4700 cm\(^2\) based on 28% total body surface area relevant for contact with soil (and dust) as per enHealth (2003); and
  - Adults - 4700 cm\(^2\) based on 24% total body surface area relevant for contact with soil (and dust) as per enHealth (2003).

- **Commercial:**
  - Adults workers - 4700 cm\(^2\) based on 24% total body surface area relevant for contact with soil (and dust) as per enHealth (2003).

Adherence Factor

This is the mass of soil that adheres to each cm\(^2\) area of skin. It varies depending on the activity undertaken and the area of the body. Activity and body part specific adherence factors are available (USEPA, 1997). However for the purpose of deriving ILs that can be used on a range of sites and a range of activities, a more general, conservative value of 0.5 mg/cm\(^2\) (enHealth, 2002) has been adopted for all receptors considered.

Dermal Absorption (ABSd)

Where available, compound specific values have been adopted as noted in Appendix B for each key chemical considered (per day).


**Fraction of Day Exposed**

The dermal absorption factors used for the key chemicals considered are based on dermal absorption over a 24 hour period of exposure. While USEPA (2004) guidance recommends adjusting the exposure frequency (EF) to address less than 24 hour exposures rather than the ABSd, the approach adopted in this assessment has to be more transparent by using an additional factor that specifically addresses the fraction of the day when the skin is considered to remain dirty. With respect to the receptor groups considered the following has been considered:

- **Residential**:
  - Children (aged to 15 years) = 1, assumed that children may not wash (properly) at the end of each day and soil may remain on the skin for the full 24 hours; and
  - Adults = 0.5, assumed that an adult may remain dirty for up to 12 hours each day when they are in contact with outdoor soil.

- **Commercial** – Adult workers = 0.5, assumed that a worker may remain dirty for up to 12 hours each day when they are in contact with outdoor soil.

**Dermal Bioavailability (Bd)**

Assumed to be 100% for all compounds assessed.

**Body Weight (BW)**

The body weight of each group (adult or child) has been obtained from data presented in the USEPA Exposure Factors Handbook (1997). The statistical analysis of body weights in the US for different age groups in the population as well as adults (males and females) is considered to be representative of expected body weights in Australia. Hence data available from this source has been used for the derivation of ILs. The following values have been adopted for the age groups considered:

- **Residential**:
  - Young children aged 0-5 years = 13.2 kg based on a child aged 2½ years (enHealth 2002);
  - Older children aged 5-15 years = 35.6 kg based on the mean body weight for male and female children aged 10-11 years (USEPA 1997); and
  - Adults = 70 kg based on the default adopted by the USEPA, considered representative of the Australian population.

- **Commercial** – Adult Workers: 70 kg based on the default value used by the USEPA considered relevant for the Australian population.

**Exposure Frequency and Duration (EF, ED)**

The derivation of ILs has assumed:

- **Residential** exposures will occur on 365 days per year for the age groups assessed. The exposure duration considered is 5 years for young children aged 0-5 years, 10 years for children 5-15 years and 55 years for adults;

- **Commercial/industrial** worker exposures will occur on 240 days per year for 30 years (enHealth 2002).

**Averaging Time (AT)**

The averaging time adopted for the assessment of exposure depends on whether the assessment is undertaken on the basis of a non-threshold or threshold approach.

For a threshold approach the potential for adverse effects to occur is only relevant when exposed, hence the averaging time is equal to the exposure duration (converted to days).
For a non-threshold approach any exposure has the potential to result in adverse (carcinogenic) effects to occur at some point during a lifetime. Hence the averaging time for non-threshold effects is equal to a lifetime, taken to be 70 years (converted to days).

### E3 Oral Intake

Incidental ingestion of soil (or dust) as a result of exposure to contaminated soil in outdoor accessible areas has been estimated as follows:

\[
\text{Daily Chemical Intake}_{\text{Is}} = \frac{C_s \cdot \text{IRs} \cdot \text{Bo} \cdot \text{ABSo} \cdot \text{CF} \cdot \text{EF} \cdot \text{ED}}{\text{BW} \cdot \text{AT}} \quad \text{(mg/kg/day)} \quad \text{(Equation E2)}
\]

where:
- \(C_s\) = Concentration in soil (mg/kg), which is the IL to be derived for outdoor soil.
- \(\text{IRs}\) = Ingestion rate of soil (mg/day)
- \(\text{ABSo}\) = Oral absorption fraction (unitless)
- \(\text{Bo}\) = Oral bioavailability (unitless)
- \(\text{CF}\) = Conversion factor of \(1 \times 10^{-6}\) to convert mg to kg
- \(\text{EF}\) = Exposure frequency (days/year), refer to Section E2
- \(\text{ED}\) = Exposure duration (years), refer to Section E2
- \(\text{BW}\) = Body weight (kg), refer to Section E2
- \(\text{AT}\) = Averaging time (days, refer to Section E2)

#### Soil Ingestion Rate (IRs)

The mass of soil that may be incidentally ingested from a residential or commercial area has been assumed to be equal to the default values recommended by enHealth (2002) and NEPC (1999). The application of these values assumes that 100% of the soil ingested each day is absorbed by the body and is derived from contaminated soil or dust indoors that is derived from the contaminated soil outdoors. The ingestion rates adopted are as follows:

- **Residential:**
  - Young children aged 0 to 5 years = 100 mg/day;
  - Older children aged 5 to 15 years = 50 mg/day; and
  - Adults = 25 mg/day.

- **Commercial:** Adult Workers = 25 mg/day.

#### Oral Absorption (ABSo)

Assumed to be 100% for all compounds assessed.

#### Oral Bioavailability (Bo)

Assumed to be 100% for all compounds assessed.

### E4 Inhalation Exposures

The overall approach to the quantification of inhalation exposures (volatiles and dust) outdoors has followed inhalation guidance provided by USEPA (2009). This approach recognises the approach and uncertainties associated with the derivation of inhalation toxicity values and requires the consideration of an exposure concentration (EC) rather than an intake. Therefore the approach adopted does not require receptor and age specific inputs on body weight and inhalation rates, rather it requires consideration of exposure time, frequency and duration. With respect to the assessment of particulates the following approach has assumed that 100% of the inhaled dust is small enough to penetrate deep into the lungs where chemical absorption can occur (also assumed to be 100%). Hence the approach is considered overly conservative for the assessment.
of exposures to particulates that may be present in air. The following equation has been used in this
assessment:

\[ EC = \frac{C_a \cdot ET \cdot ABS_i \cdot B_i \cdot EF \cdot ED}{AT} \]  

\( \text{(mg/m}^3\text{)} \)  

...(Equation E3)

where:

- \( C_a \) = Concentration of chemical in air (mg/m\textsuperscript{3}) – the volatile and/or dust concentration calculated as:

\[ C_a = C_s \cdot \left(1 + \frac{1}{VF \cdot PEF}\right) \]  

\( \text{(mg/m}^3\text{)} \)  

...(Equation E4)

- \( C_s \) = Concentration in soil (mg/kg), which is the IL to be derived for outdoor soil;

- \( VF \) = Volatilisation Factor, the ratio of the soil concentration \( C_s \) to the vapour concentration \( C_v \). This is calculated for each volatile key chemical as outlined below;

- \( PEF \) = Particulate Emission Factor (PEF), the ratio of the soil concentration \( C_s \) to the dust concentration \( C_d \). This is calculated as outlined below;

- \( ET \) = Exposure time (hours/day), this has been calculated as noted below;

- \( ABS_i \) = Absorption fraction or rate associated with inhalation exposures (unitless)

- \( B_i \) = Bioavailability associated within inhalation exposures (unitless)

- \( EF \) = Exposure frequency (days/year), refer to Section E2

- \( ED \) = Exposure duration (years), refer to Section E2

- \( AT \) = Averaging time (in hours), refer to Section E2

**Exposure Time (ET)**

For the purpose of deriving ILs for outdoor soil, inhalation exposures have been assumed to occur outdoors
(where the contamination is present) as well as indoors assuming that outdoor air concentrations move into
the building. The following approach assumptions have been adopted for exposure times:

- **Residential:**
  - All residents are assumed to be outdoors for up to 4 hours each day (as per enHealth 2002
    and NEPC 1999) where 100% of the calculated vapour or dust concentration is inhaled; and
  - All residents are assumed to be indoors for up to 20 hours each day (as per enHealth 2002
    and NEPC 1999) where vapour or dust concentrations are 75% of the outdoor air
    concentrations (allowing for some dispersion from outdoors to indoors).

- **Commercial:**
  - All workers are assumed to be outdoors for up to 2 hours each day where 100% of the calculated
    vapour or dust concentration is inhaled; and
  - All workers are assumed to be indoors for up to 8 hours each day where vapour or dust
    concentrations are 75% of the outdoor air concentrations (allowing for some dispersion from
    outdoors to indoors).

**Absorption via Inhalation (ABS\textsubscript{i})**

Assumed to be 100% for all compounds assessed.

**Bioavailability via Inhalation (B\textsubscript{i})**

Assumed to be 100% for all compounds assessed.

**Particulate Emission Factor (PEF)**

The ratio of soil concentration to dust/particulate concentration has been calculated assuming that dust
loading/levels in air on the site (residential or commercial) is equivalent to 50 µg respirable dust/m\textsuperscript{3} air (as per enHealth 2002), where 100% is derived from the contaminated soil. This results in a PEF of 5×10\textsuperscript{7} [m\textsuperscript{3}/kg or
mg/kg soil per mg/m\textsuperscript{3} dust]. It is noted that this dust loading is based on reasonable upper levels of dust in
urban air. The dust level in urban air is derived from a range of sources such as combustion emissions,
industrial emissions and dust generated from a number of sources. Hence the adoption of this value is considered conservative.

**Volatilisation Factor**

The volatilisation factor has been calculated for each volatile key chemical identified using equations presented in USEPA (2002) for vapour phase concentrations in air above surface soil impacts outdoors.

\[
VF = \frac{Q/C_V \cdot (3.14 \cdot D_A \cdot T)^{0.5} \cdot 1 \times 10^{-4}}{2 \cdot \rho_b \cdot D_A} \quad \text{(m}^3/\text{kg})
\]  
\text{...(Equation E5)}

Where:

\[
D_A = \left(\frac{(\rho_a^{2.3} \cdot D_i \cdot H' + \rho_w^{2.3} \cdot D_w)}{\rho_b \cdot k_d + \theta_w + \theta_a \cdot H'}\right)^{\frac{1}{2}} \quad \text{(cm}^2/\text{s})
\]  
\text{...(Equation E6)}

\[
Q/C = \text{Inverse of the ratio of the geometric mean air concentration to the volatilisation flux at centre of a square source (g/m}^2/\text{s per kg/m}^3), \text{calculated based on the approach outlines in USEPA (1996) guidance to be 150 (g/m}^2/\text{s per kg/m}^3) \text{based on an average outdoor area that may be contaminated of 10m x 10m and a climate similar to the south-east coast of Australia.}
\]

\[
T = \text{Exposure interval (s), where a value of 2.2x10}^9 \text{ s has been used for the assessment of residential exposures (based on 70 year lifetime) and 9.5x10}^8 \text{ has been used for commercial/industrial exposures (based on a 30 year lifetime), consistent with USEPA (2002 guidance);}
\]

\[
\rho_b = \text{dry soil bulk density (g/cm}^3) = 1.6 \text{ g/cm}^3 \text{ assumed for outdoor fill/loam materials;}
\]

\[
\theta_a = \text{air-filled soil porosity (L/L or cm}^3/cm^3) = n-\theta_w, \text{ calculated to be 0.32;}
\]

\[
n = \text{total soil porosity (L/L or cm}^3/cm^3) = 1-(\rho_b / \rho), \text{ calculated to be 0.4;}
\]

\[
\theta_w = \text{water-filled soil porosity (L/L or cm}^3/cm^3) = MC \times \rho_b, \text{ calculated to be 0.08}
\]

\[
\rho = \text{soil particle density (g/cm}^3) = 2.65 \text{ g/cm}^3
\]

\[
MC = \text{soil moisture content (g/g) = 0.05, or 5% assumed relevant to drier fill/loam outdoors;}
\]

\[
D_A = \text{diffusivity in air (cm}^2/\text{s), chemical-specific;}
\]

\[
H' = \text{dimensionless Henry's law constant (unitless), chemical-specific;}
\]

\[
D_W = \text{diffusivity in water (cm}^2/\text{s), chemical-specific}
\]

\[
K_a = \text{soil-water partition coefficient (cm}^3/\text{g) for organics: } = K_{oc} \times f_{oc}
\]

\[
K_{oc} = \text{soil organic carbon partition coefficient (cm}^3/g), \text{chemical-specific;}
\]

\[
f_{oc} = \text{fraction organic carbon in soil (g/g) = 0.001 (0.1%), considered representative (conservative) for most surface soil in Australia.}
\]

For the volatile chemicals identified and assessed in this report, the following presents the VF calculated for threshold and non-threshold assessments (residential and commercial where relevant):
Table E1  Calculated Volatilisation Factors

<table>
<thead>
<tr>
<th>Volatile Key Chemicals</th>
<th>Henry’s Law Constant, $H$ (unitless)</th>
<th>Air Diffusion Coeff, $D_A$ (cm$^2$/s)</th>
<th>Water Diffusion Coeff, $D_W$ (cm$^2$/s)</th>
<th>Koc (cm$^3$/g)</th>
<th>Apparent Diffusivity (cm$^2$/s)</th>
<th>Volatilisation Factor Residential (m$^3$/kg)</th>
<th>Volatilisation Factor Commercial (m$^3$/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroform</td>
<td>0.15</td>
<td>0.104</td>
<td>1.0E-05</td>
<td>35.04</td>
<td>1.2E-2</td>
<td>3.6E+3</td>
<td>2.3E+3</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>0.090</td>
<td>0.28</td>
<td>1.9E-05</td>
<td>363</td>
<td>8.5E-3</td>
<td>4.2E+3</td>
<td>2.8E+3</td>
</tr>
<tr>
<td>Ammonia</td>
<td>0.00066</td>
<td>0.28</td>
<td>1.0E-05</td>
<td>14</td>
<td>6.8E-3</td>
<td>4.7E+3</td>
<td>3.1E+3</td>
</tr>
<tr>
<td>Iodine</td>
<td>0.216</td>
<td>0.039</td>
<td>1.0E-05</td>
<td>22</td>
<td>2.8E-2</td>
<td>2.3E+3</td>
<td>1.5E+3</td>
</tr>
<tr>
<td>Bromide</td>
<td>0.30094</td>
<td>0.144</td>
<td>1.4E-05</td>
<td>389</td>
<td>1.1E-5</td>
<td>1.2E+5</td>
<td>7.8E+4</td>
</tr>
<tr>
<td>Methylamine</td>
<td>0.00046</td>
<td>0.12*</td>
<td>1.3E-05*</td>
<td>30</td>
<td>2.5E-4</td>
<td>2.5E+4</td>
<td>1.6E+4</td>
</tr>
<tr>
<td>Nitroethane</td>
<td>0.0020</td>
<td>0.119**</td>
<td>1.4E-05**</td>
<td>363</td>
<td>8.2E-5</td>
<td>4.3E+4</td>
<td>2.8E+4</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>0.0011</td>
<td>0.074</td>
<td>9.2E-06</td>
<td>33</td>
<td>4.6E-6</td>
<td>1.8E+5</td>
<td>1.2E+5</td>
</tr>
<tr>
<td>Saffrole and Isosafrole</td>
<td>0.00037</td>
<td>0.05</td>
<td>5.0E-06</td>
<td>50</td>
<td>1.1E-2</td>
<td>3.7E+3</td>
<td>2.4E+3</td>
</tr>
<tr>
<td>Benzene</td>
<td>0.228</td>
<td>0.086</td>
<td>9.8E-06</td>
<td>58.9</td>
<td>4.9E-3</td>
<td>5.6E+3</td>
<td>3.7E+3</td>
</tr>
<tr>
<td>Toluene</td>
<td>0.27</td>
<td>0.078</td>
<td>9.2E-06</td>
<td>268</td>
<td>4.4E-3</td>
<td>5.9E+3</td>
<td>3.9E+3</td>
</tr>
<tr>
<td>Ethylbenzene</td>
<td>0.323</td>
<td>0.075</td>
<td>7.8E-06</td>
<td>363</td>
<td>3.5E-3</td>
<td>6.6E+3</td>
<td>4.3E+3</td>
</tr>
<tr>
<td>Xylenes (total)</td>
<td>0.301</td>
<td>0.07</td>
<td>7.8E-06</td>
<td>407</td>
<td>5.3E-5</td>
<td>5.3E+4</td>
<td>3.5E+4</td>
</tr>
<tr>
<td>Naphthalene</td>
<td>0.0198</td>
<td>0.059</td>
<td>7.5E-06</td>
<td>1837</td>
<td>2.2E-2</td>
<td>2.6E+3</td>
<td>1.7E+3</td>
</tr>
<tr>
<td>TPH C6-C9</td>
<td>0.067</td>
<td>0.048</td>
<td>7.7E-06</td>
<td>2510</td>
<td>2.2E-4</td>
<td>2.6E+4</td>
<td>1.7E+4</td>
</tr>
</tbody>
</table>

Refer to Appendix B for chemical-specific parameters for most key chemicals
* Values for nitromethane (RAIS)
** Values for dimethylamine (USEPA 2004)

E5  Approach to Derivation of Outdoor Soil ILs

Calculated ILs for residential and commercial receptors are attached to this appendix. The following shows the equations used in the derivation of the ILs for threshold and non-threshold carcinogenic chemicals for outdoor soil.

For the derivation of ILs, Equations E1 to C5 have been rearranged to enable the calculation of the soil concentration $C_s$. This has been calculated as follows:

**Dermal and Oral Intakes**

\[ \text{Intake}_D = C_s \times IF_D \]  
(based on Equation E1) ...(Equation E7)

Where $IF_D = \text{intake factor for dermal absorption from soil (kg/kg/day)}$ from Equation E1:

\[ IF_D = \frac{SAs \times AF \times FD \times ABSd \times B_d \times CF \times EF \times ED}{BW \times AT} \]

For threshold effects:

\[ AF \times ABSd \times B_d \times EF \times CF \times \sum SA_x \times ED_x \times FC_x / BW_x \]

For non-threshold effects:  
\[ IF_D = \frac{\sum SA_x \times ED_x \times FC_x}{AT} \]

\[ \text{Intake}_O = C_s \times IF_O \]  
(based on Equation E2) ...(Equation E8)
Where $IF_O$ = intake factor for oral ingestion from soil (kg/kg/day) from Equation E2:

For threshold effects:  
$$IF_O = \frac{IRs \cdot Bo \cdot ABSo \cdot CF \cdot EF \cdot ED}{BW \cdot AT}$$

For non-threshold effects:  
$$IF_O = \frac{Bo \cdot ABSo \cdot CF \cdot EF \cdot \sum (IR_x \cdot ED_x) / BW_x}{AT}$$

**Inhalation Exposure Concentration**

$$EC = C_s \cdot IF_A$$  
(based on Equation E3)  
...(Equation E9)

Where $IF_A$ = intake factor for inhalation exposures that may be derived from soil outdoors (kg/m³) from Equations E3 and E4:

For Threshold effects:  
$$IF_A = \left[\frac{1}{VF} + \frac{1}{PEF}\right] \cdot \frac{ET \cdot ABS_i \cdot B_i \cdot EF \cdot ED}{AT}$$

For Threshold effects:  
$$IF_A = \left[\frac{1}{VF} + \frac{1}{PEF}\right] \cdot \frac{ET \cdot ABS_i \cdot B_i \cdot EF \cdot \sum ED_x}{AT}$$

**Calculation of Soil IL for Threshold Chemicals**

The calculation of a threshold HI compares the calculated intake (for oral and dermal exposures) and the exposure concentration (for inhalation exposures) with the threshold dose-response (toxicity value) identified in Appendix B for each threshold key chemical, accounting for background intakes (B) where relevant. This is presented in the following equation:

$$HI = \frac{(Intake_D + Intake_O)}{TDI - B} + \frac{EC}{TC - B}$$  
...(Equation E10)

Where the TDI (oral/dermal) and TC are the threshold toxicity values relevant for the chemical (expressed in mg/kg/day for the TDI and mg/m³ for the TC).

The HI adopted in the derivation of ILs is a value of 1 (as noted in the main report). This is the target HI identified and used in this assessment and is based on exposures overall exposures pathways relevant for outdoor soil.

Combining Equations C7 to E10 and rearranging for $C_s$, the IL for outdoor soil, the following equation is used for deriving the IL for threshold chemicals:
Derivation of Risk-Based Investigation Levels

Calculation of Surface Residue IL for Non-Threshold Chemicals
The calculation of a non-threshold risk involves the multiplication of the calculated intake with the slope factor (SF) (for oral and dermal exposures) and a unit risk (UR) (for inhalation exposures) identified in Appendix B for each key chemical where non-threshold carcinogenic effects are of most significance. The approach adopted does not require consideration of background intakes as the target risk level is an incremental risk target, above background. This is presented in the following equation:

\[ \text{Risk} = (\text{Intake}_D + \text{Intake}_O) \times SF \times EC \times UR \]  
...(Equation E12)

The Risk level adopted in the derivation of ILs is a value of \(1 \times 10^{-5}\) (as noted in the main report) relevant for the total risk over all pathways of exposure. This is the target risk identified and used in this assessment for each key chemical where this approach is relevant.

Combining Equations C7, C8, C9 and C11 and rearranging for \(C_s\), the IL for soil, the following equation is used for deriving the IL for non-threshold chemicals:

\[ \text{IL}_{\text{soil}} = C_s = \frac{\text{Target Risk}}{(\text{IF}_D + \text{IF}_O) \times SF + \text{IF}_A \times UR} \]  
...(Equation E13)

Presentation of Soil ILs
The attached tables present a summary of the assumptions presented in this Appendix, along with the calculations associated with the derivation of ILs for outdoor soil. It is noted that the calculations attached show a level of accuracy no considered to be appropriate for the ILs to be used on any site. This is due to the level of uncertainty inherent in the assumptions adopted for the quantification of exposure, the toxicity values adopted and the level of uncertainty expected in the sampling and analysis of soil. Hence the ILs for soil presented in the main report have been rounded to no more than 2 significant figures.

The attached tables present calculated ILs for all key chemicals. However, it is noted that where relevant currently available ILs (from NEPM or NSW EPA) have been referenced in the main report for some key chemicals. These reflect current ILs available for these chemicals and would need to be updated as ILs were revised.
E6 References


Attachment – Calculations
Table E1 Summary of Exposure Assumptions – ILs Outdoor Soil – Residential Use

<table>
<thead>
<tr>
<th>Assessment of Exposure to Soil Outdoors</th>
<th>Abbrev.</th>
<th>units</th>
<th>Parameter</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil Ingestion Rate - Young children (0-5 years)</td>
<td>IRY</td>
<td>mg/day</td>
<td>100</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>- Older children (5-15 years)</td>
<td>IRO</td>
<td>mg/day</td>
<td>50</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>- Adults</td>
<td>IRA</td>
<td>mg/day</td>
<td>25</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Surface Area of Skin - Young children (0-5 years)</td>
<td>SAYC</td>
<td>cm²/day</td>
<td>2625</td>
<td>Assume 30% of body surface area gets dirty as per enHealth (2003)</td>
</tr>
<tr>
<td>- Older children (5-15 years)</td>
<td>SAOC</td>
<td>cm²/day</td>
<td>4300</td>
<td>Assume 28% of body surface area gets dirty as per enHealth (2003)</td>
</tr>
<tr>
<td>- Adults</td>
<td>SAA</td>
<td>cm²/day</td>
<td>4700</td>
<td>Assume 24% of body surface area gets dirty as per enHealth (2003)</td>
</tr>
<tr>
<td>Soil to Skin Adherance Factor</td>
<td>AF</td>
<td>-</td>
<td>0.5</td>
<td>Default as per enHealth (2002)</td>
</tr>
<tr>
<td>Fraction of Day (where soil remains on skin) - children</td>
<td>FC</td>
<td>-</td>
<td>1</td>
<td>Assume children’s skin remains dirty for 24 hours (before washing)</td>
</tr>
<tr>
<td>Fraction of Day (where soil remains on skin) - adults</td>
<td>FA</td>
<td>-</td>
<td>0.5</td>
<td>Assume adults wash after 12 hours</td>
</tr>
<tr>
<td>Time Spent Outdoors</td>
<td>ET₀</td>
<td>hours</td>
<td>4</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Time Spent Indoors</td>
<td>ETᵢ</td>
<td>hours</td>
<td>20</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Particulate Emission Factor</th>
<th>PEF_res</th>
<th>(m³/kg)</th>
<th>5.00E+07</th>
<th>Default assumed based on 50 µg/m³ respirable dust (enHealth 2002) where 100% is derived from the site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio of air concentrations indoors to outdoors</td>
<td>Cfi</td>
<td>-</td>
<td>0.75</td>
<td>Assume concentrations of volatiles and dusts indoors are 75% of those outdoors (outdoor sources) as per NEPM (1999)</td>
</tr>
<tr>
<td>Bioavailability - Oral</td>
<td>Bo</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>- Dermal</td>
<td>Bd</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>- Inhalation</td>
<td>Bi</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Absorption Fraction - Oral</td>
<td>ABSo</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>- Inhalation</td>
<td>ABSi</td>
<td>-</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Body weight - Young children (0-5 years)</td>
<td>BWYC</td>
<td>kg</td>
<td>13.2</td>
<td>Based on child aged 2.5 years as per enHealth (2002)</td>
</tr>
<tr>
<td>- Older children (5-15 years)</td>
<td>BWOC</td>
<td>kg</td>
<td>35.6</td>
<td>Mean body weight for boys and girls aged 5 to 15 years (USEPA 1997)</td>
</tr>
<tr>
<td>- Adults</td>
<td>BWA</td>
<td>kg</td>
<td>70</td>
<td>Default for adults from USEPA considered relevant for Australian population</td>
</tr>
<tr>
<td>Exposure Frequency</td>
<td>EF</td>
<td>days/year</td>
<td>365</td>
<td>Assume at home indoors every day of the year</td>
</tr>
<tr>
<td>Exposure Duration - Young children (0-5 years)</td>
<td>EDYC</td>
<td>years</td>
<td>5</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>- Older children (5-15 years)</td>
<td>ED_OC</td>
<td>years</td>
<td>10</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>- Adults</td>
<td>ED_A</td>
<td>years</td>
<td>55</td>
<td>Relevant to age group assessed</td>
</tr>
<tr>
<td>Averaging Time (noncarcinogenic)</td>
<td>AT_NC</td>
<td>hours</td>
<td>ED¹365*24</td>
<td>Calculated based on ED for each relevant age group</td>
</tr>
<tr>
<td></td>
<td></td>
<td>days</td>
<td>ED¹365</td>
<td></td>
</tr>
<tr>
<td>Averaging Time (carcinogenic)</td>
<td>AT_C</td>
<td>hours</td>
<td>613200</td>
<td>Based on lifetime of 70 years (converted to hours)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>days</td>
<td>25550</td>
<td></td>
</tr>
</tbody>
</table>
### Table E2 Derivation of ILs Outdoor Soil – Threshold Effects - Residential Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd) (unitless)</th>
<th>Tolerable Daily Intake (TDI) (mg/kg/day)</th>
<th>Tolerable Concentration in Air (TC) (mg/m³)</th>
<th>Background Intake (B) (% of TDI or TC)</th>
<th>Target HI</th>
<th>Derived Soil IL (mg/kg)</th>
<th>Calculated from Derived Criteria Exposure Concentration in Air (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methamphetamine</td>
<td>0.57</td>
<td>0.0003</td>
<td>0.0011</td>
<td>0%</td>
<td>1</td>
<td>5</td>
<td>3.5E-05 2.6E-04 7.4E-08</td>
</tr>
<tr>
<td>MDMA</td>
<td>0.57</td>
<td>0.004</td>
<td>0.014</td>
<td>0%</td>
<td>1</td>
<td>62</td>
<td>4.7E-04 3.5E-03 9.9E-07</td>
</tr>
<tr>
<td>Pseudo/Ephedrine</td>
<td>1</td>
<td>1.3</td>
<td>4.55</td>
<td>50%</td>
<td>1</td>
<td>6074</td>
<td>4.6E-02 6.0E-01 9.6E-05</td>
</tr>
<tr>
<td>Chloroform</td>
<td>0.0005</td>
<td>0.015</td>
<td>0.14</td>
<td>50%</td>
<td>1</td>
<td>240</td>
<td>1.8E-03 1.2E-05 5.3E-02</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>0.0005</td>
<td>0.0012</td>
<td>1</td>
<td>20%</td>
<td>1</td>
<td>122</td>
<td>9.2E-04 6.1E-06 2.3E-02</td>
</tr>
<tr>
<td>Ammonia</td>
<td>0.01</td>
<td>0.029</td>
<td>0.1</td>
<td>20%</td>
<td>1</td>
<td>1878</td>
<td>1.4E-02 1.9E-03 2.4E-02</td>
</tr>
<tr>
<td>Iodide</td>
<td>0.14</td>
<td>0.01</td>
<td>0.001</td>
<td>60%</td>
<td>1</td>
<td>2</td>
<td>1.8E-05 3.3E-05 3.9E-04</td>
</tr>
<tr>
<td>Bromide</td>
<td>0.01</td>
<td>0.1</td>
<td>0.0007</td>
<td>10%</td>
<td>1</td>
<td>2</td>
<td>1.4E-05 1.8E-06 6.3E-04</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.1</td>
<td>0.00002</td>
<td>0.0001</td>
<td>50%</td>
<td>1</td>
<td>0.6</td>
<td>4.3E-06 5.7E-06 9.0E-09</td>
</tr>
<tr>
<td>N-Methylformamide</td>
<td>0.6</td>
<td>0.0086</td>
<td>0.03</td>
<td>0%</td>
<td>1</td>
<td>127</td>
<td>9.7E-04 7.6E-03 2.0E-06</td>
</tr>
<tr>
<td>Methylamine</td>
<td>0.03</td>
<td>0.00086</td>
<td>0.003</td>
<td>0%</td>
<td>1</td>
<td>69</td>
<td>5.2E-04 2.0E-04 4.6E-04</td>
</tr>
<tr>
<td>Nitroethane</td>
<td>0.03</td>
<td>0.089</td>
<td>0.31</td>
<td>0%</td>
<td>1</td>
<td>4491</td>
<td>3.4E-02 1.3E-02 1.4E-01</td>
</tr>
<tr>
<td>Boron</td>
<td>0.01</td>
<td>0.2</td>
<td>0.005</td>
<td>60%</td>
<td>1</td>
<td>8692</td>
<td>6.6E-02 8.6E-03 1.4E-04</td>
</tr>
<tr>
<td>Mercury</td>
<td>0.01</td>
<td>0.00071</td>
<td>0.001</td>
<td>25%</td>
<td>1</td>
<td>62</td>
<td>4.7E-04 6.2E-05 9.8E-07</td>
</tr>
<tr>
<td>Lithium</td>
<td>0.01</td>
<td>0.002</td>
<td>0.007</td>
<td>0%</td>
<td>1</td>
<td>233</td>
<td>1.8E-03 2.3E-04 3.7E-06</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>0.03</td>
<td>0.1</td>
<td>0.35</td>
<td>0%</td>
<td>1</td>
<td>6319</td>
<td>4.8E-02 1.9E-02 1.2E-01</td>
</tr>
<tr>
<td>Benzene</td>
<td>0.0005</td>
<td>0.004</td>
<td>0.03</td>
<td>10%</td>
<td>1</td>
<td>99</td>
<td>7.5E-04 4.9E-06 2.1E-02</td>
</tr>
<tr>
<td>Toluene</td>
<td>0.03</td>
<td>0.08</td>
<td>5</td>
<td>10%</td>
<td>1</td>
<td>5612</td>
<td>4.2E-02 1.7E-02 8.0E-01</td>
</tr>
<tr>
<td>Ethylbenzene</td>
<td>0.03</td>
<td>0.091</td>
<td>22</td>
<td>0%</td>
<td>1</td>
<td>8187</td>
<td>6.2E-02 2.4E-02 1.1E+00</td>
</tr>
<tr>
<td>Xylenes (total)</td>
<td>0.03</td>
<td>0.179</td>
<td>0.87</td>
<td>0%</td>
<td>1</td>
<td>5065</td>
<td>3.8E-02 1.5E-02 6.1E-01</td>
</tr>
<tr>
<td>Naphthalene</td>
<td>0.13</td>
<td>0.02</td>
<td>0.003</td>
<td>10%</td>
<td>1</td>
<td>151</td>
<td>1.1E-03 1.9E-03 2.2E-03</td>
</tr>
<tr>
<td>TPH C6-C9</td>
<td>0.03</td>
<td>5</td>
<td>0.7</td>
<td>20%</td>
<td>1</td>
<td>1848</td>
<td>1.4E-02 5.5E-03 5.6E-01</td>
</tr>
<tr>
<td>TPH C10-C14</td>
<td>0.13</td>
<td>0.03</td>
<td>0.2</td>
<td>20%</td>
<td>1</td>
<td>958</td>
<td>7.2E-03 1.2E-02 2.9E-02</td>
</tr>
<tr>
<td>TPH C15+</td>
<td>0.13</td>
<td>0.03</td>
<td>0.105</td>
<td>20%</td>
<td>1</td>
<td>1170</td>
<td>8.9E-03 1.5E-02 1.8E-05</td>
</tr>
</tbody>
</table>
### Table E3  Derivation of ILs Outdoor Soil – Non-Threshold Carcinogenic Effects - Residential Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd) (unitless)</th>
<th>Non-Threshold Slope Factor (SF) (mg/kg/day)^1</th>
<th>Non-Threshold Unit Risk (mg/m^3)^1</th>
<th>Target Risk</th>
<th>Derived Soil IL (mg/kg)</th>
<th>Calculated from Derived Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Oral Intake (mg/kg/day)</td>
<td>Dermal Intake (mg/kg/day)</td>
</tr>
<tr>
<td>Benzene</td>
<td>0.0005</td>
<td>0.035</td>
<td>0.006</td>
<td>1E-05</td>
<td>8</td>
<td>7.7E-06</td>
</tr>
<tr>
<td>Safrole and Isosafrole</td>
<td>0.1</td>
<td>0.22</td>
<td>0.063</td>
<td>1E-05</td>
<td>1</td>
<td>1.0E-06</td>
</tr>
</tbody>
</table>

1. Unit risk is the ratio of the dose to the body surface area.
### Table E4  Summary of Exposure Assumptions – ILs Outdoor Soil – Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Assessment of Exposure to Soil Outdoors</th>
<th>Abbrev.</th>
<th>units</th>
<th>Parameter</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil Ingestion Rate</td>
<td>IRA</td>
<td>mg/day</td>
<td>25</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Surface Area of Skin</td>
<td>SAₕ</td>
<td>cm²/day</td>
<td>4700</td>
<td>Assume 24% of body surface area gets dirty as per enHealth (2003)</td>
</tr>
<tr>
<td>Soil to Skin Adherance Factor</td>
<td>AF</td>
<td></td>
<td>0.5</td>
<td>Default as per enHealth (2002)</td>
</tr>
<tr>
<td>Fraction of Day (where soil remains on skin) - adults</td>
<td>FA</td>
<td>hours</td>
<td>0.5</td>
<td>Assume adults wash after 12 hours</td>
</tr>
<tr>
<td>Time Spent Outdoors</td>
<td>ETo</td>
<td>hours</td>
<td>2</td>
<td>Assumed</td>
</tr>
<tr>
<td>Time Spent Indoors</td>
<td>ETi</td>
<td>hours</td>
<td>8</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Particulate Emission Factor</td>
<td>PEF</td>
<td>m³/kg</td>
<td>5.0E+07</td>
<td>Default assumed based on 50 ug/m³ respirable dust (enHealth 2002) where 100% is derived from the site</td>
</tr>
<tr>
<td>Ratio of air concentrations indoors to outdoors</td>
<td>Cfi</td>
<td></td>
<td>0.75</td>
<td>Assume concentrations of volatiles and dusts indoors are 75% of those outdoors (outdoor sources) as per NEPM (1999)</td>
</tr>
<tr>
<td>Bioavailability</td>
<td>Oral</td>
<td>Bo</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td></td>
<td>Dermal</td>
<td>Bd</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td></td>
<td>Inhalation</td>
<td>Bi</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Absorption Fraction</td>
<td>Oral</td>
<td>ABSO</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td></td>
<td>Inhalation</td>
<td>ABSi</td>
<td>1</td>
<td>Assume to be 100% for all chemicals of interest</td>
</tr>
<tr>
<td>Body weight</td>
<td>Adults</td>
<td>BWₕ</td>
<td>70</td>
<td>Default for adults from USEPA considered relevant for Australian population</td>
</tr>
<tr>
<td>Exposure Frequency</td>
<td>EF</td>
<td>days/year</td>
<td>240</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Exposure Duration</td>
<td>EDₕ</td>
<td>years</td>
<td>30</td>
<td>As per enHealth (2002) and NEPM (1999)</td>
</tr>
<tr>
<td>Averaging Time (noncarcinogenic)</td>
<td>ATₙₑ</td>
<td>hours</td>
<td>ED³365²4</td>
<td>Calculated based on ED (converted to hours)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>days</td>
<td>ED³365</td>
<td></td>
</tr>
<tr>
<td>Averaging Time (carcinogenic)</td>
<td>ATₙₑ</td>
<td>hours</td>
<td>613200</td>
<td>Based on lifetime of 70 years (converted to hours)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>days</td>
<td>25550</td>
<td></td>
</tr>
</tbody>
</table>
### Table E5  Derivation of ILs Outdoor Soil – Threshold Effects - Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd)</th>
<th>Tolerable Daily Intake (TDI) (mg/kg/day)</th>
<th>Tolerable Concentration in Air (TC) (mg/m³)</th>
<th>Background Intake (B) (% of TDI or TC)</th>
<th>Target HI</th>
<th>Derived Soil IL (mg/kg)</th>
<th>Calculated from Derived Criteria Oral Intake (mg/kg/day)</th>
<th>Dermal Intake (mg/kg/day)</th>
<th>Exposure Concentration in Air (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methamphetamine</td>
<td>0.57</td>
<td>0.0003</td>
<td>0.011</td>
<td>0%</td>
<td>1</td>
<td>46</td>
<td>1.1E-05</td>
<td>2.9E-04</td>
<td>2.0E-07</td>
</tr>
<tr>
<td>MDMA</td>
<td>0.57</td>
<td>0.004</td>
<td>0.014</td>
<td>0%</td>
<td>1</td>
<td>613</td>
<td>1.4E-04</td>
<td>3.9E-03</td>
<td>2.7E-06</td>
</tr>
<tr>
<td>Pseudo/Ephedrine</td>
<td>1</td>
<td>1.3</td>
<td>4.55</td>
<td>50%</td>
<td>1</td>
<td>57659</td>
<td>1.4E-02</td>
<td>6.4E-01</td>
<td>2.5E-04</td>
</tr>
<tr>
<td>Chloroform</td>
<td>0.0005</td>
<td>0.015</td>
<td>0.14</td>
<td>50%</td>
<td>1</td>
<td>732</td>
<td>1.7E-04</td>
<td>4.0E-06</td>
<td>6.8E-02</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>0.0005</td>
<td>0.0012</td>
<td>1</td>
<td>20%</td>
<td>1</td>
<td>2860</td>
<td>6.7E-04</td>
<td>1.6E-05</td>
<td>2.3E-01</td>
</tr>
<tr>
<td>Ammonia</td>
<td>0.01</td>
<td>0.029</td>
<td>0.1</td>
<td>20%</td>
<td>1</td>
<td>12248</td>
<td>2.9E-03</td>
<td>1.4E-03</td>
<td>6.5E-02</td>
</tr>
<tr>
<td>Iodine</td>
<td>0.14</td>
<td>0.01</td>
<td>0.001</td>
<td>60%</td>
<td>1</td>
<td>6</td>
<td>1.3E-06</td>
<td>8.7E-06</td>
<td>4.0E-04</td>
</tr>
<tr>
<td>Bromide</td>
<td>0.01</td>
<td>0.1</td>
<td>0.0007</td>
<td>10%</td>
<td>1</td>
<td>4</td>
<td>1.0E-06</td>
<td>4.8E-07</td>
<td>6.3E-04</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.1</td>
<td>0.00002</td>
<td>0.0001</td>
<td>50%</td>
<td>1</td>
<td>7.5</td>
<td>1.8E-06</td>
<td>8.2E-06</td>
<td>3.3E-08</td>
</tr>
<tr>
<td>N-Methylformamide</td>
<td>0.6</td>
<td>0.0086</td>
<td>0.03</td>
<td>0%</td>
<td>1</td>
<td>1250</td>
<td>2.9E-04</td>
<td>8.3E-03</td>
<td>5.5E-06</td>
</tr>
<tr>
<td>Methylamine</td>
<td>0.03</td>
<td>0.00086</td>
<td>0.003</td>
<td>0%</td>
<td>1</td>
<td>625</td>
<td>1.5E-04</td>
<td>2.1E-04</td>
<td>1.8E-03</td>
</tr>
<tr>
<td>Nitroethane</td>
<td>0.03</td>
<td>0.089</td>
<td>0.31</td>
<td>0%</td>
<td>1</td>
<td>19946</td>
<td>4.7E-03</td>
<td>6.6E-03</td>
<td>2.7E-01</td>
</tr>
<tr>
<td>Boron</td>
<td>0.01</td>
<td>0.2</td>
<td>0.005</td>
<td>60%</td>
<td>1</td>
<td>153684</td>
<td>3.6E-02</td>
<td>1.7E-02</td>
<td>6.7E-04</td>
</tr>
<tr>
<td>Mercury</td>
<td>0.01</td>
<td>0.00071</td>
<td>0.001</td>
<td>25%</td>
<td>1</td>
<td>1529</td>
<td>3.6E-04</td>
<td>1.7E-04</td>
<td>6.7E-06</td>
</tr>
<tr>
<td>Lithium</td>
<td>0.01</td>
<td>0.002</td>
<td>0.007</td>
<td>0%</td>
<td>1</td>
<td>5773</td>
<td>1.4E-03</td>
<td>6.4E-04</td>
<td>2.5E-05</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>0.03</td>
<td>0.1</td>
<td>0.35</td>
<td>0%</td>
<td>1</td>
<td>35809</td>
<td>8.4E-03</td>
<td>1.2E-02</td>
<td>2.8E-01</td>
</tr>
<tr>
<td>Benzene</td>
<td>0.0005</td>
<td>0.004</td>
<td>0.03</td>
<td>10%</td>
<td>1</td>
<td>291</td>
<td>6.8E-05</td>
<td>1.6E-06</td>
<td>2.6E-02</td>
</tr>
<tr>
<td>Toluene</td>
<td>0.03</td>
<td>0.08</td>
<td>5</td>
<td>10%</td>
<td>1</td>
<td>47160</td>
<td>1.1E-02</td>
<td>1.6E-02</td>
<td>2.8E-00</td>
</tr>
<tr>
<td>Ethylbenzene</td>
<td>0.03</td>
<td>0.091</td>
<td>22</td>
<td>0%</td>
<td>1</td>
<td>113602</td>
<td>2.7E-02</td>
<td>3.8E-02</td>
<td>6.5E+00</td>
</tr>
<tr>
<td>Xylenes (total)</td>
<td>0.03</td>
<td>0.179</td>
<td>0.87</td>
<td>0%</td>
<td>1</td>
<td>16207</td>
<td>3.8E-03</td>
<td>5.4E-03</td>
<td>8.3E-01</td>
</tr>
<tr>
<td>Naphthalene</td>
<td>0.13</td>
<td>0.02</td>
<td>0.003</td>
<td>10%</td>
<td>1</td>
<td>414</td>
<td>9.7E-05</td>
<td>5.9E-04</td>
<td>2.6E-03</td>
</tr>
<tr>
<td>TPH C8-C9</td>
<td>0.03</td>
<td>1.7</td>
<td>0.7</td>
<td>20%</td>
<td>1</td>
<td>4388</td>
<td>1.0E-03</td>
<td>1.5E-03</td>
<td>5.6E-01</td>
</tr>
<tr>
<td>TPH C10-C14</td>
<td>0.13</td>
<td>0.03</td>
<td>0.2</td>
<td>20%</td>
<td>1</td>
<td>6671</td>
<td>1.6E-03</td>
<td>9.6E-03</td>
<td>8.8E-02</td>
</tr>
<tr>
<td>TPH C15+</td>
<td>0.13</td>
<td>0.03</td>
<td>0.105</td>
<td>20%</td>
<td>1</td>
<td>14363</td>
<td>3.4E-03</td>
<td>2.1E-02</td>
<td>6.3E-05</td>
</tr>
</tbody>
</table>
### Table E6  Derivation of ILs Outdoor Soil – Non-Threshold Carcinogenic Effects - Commercial/Industrial Use

<table>
<thead>
<tr>
<th>Compound</th>
<th>Dermal Absorption Fraction (ABSd)</th>
<th>Non-Threshold Slope Factor (SF) (mg/kg/day)</th>
<th>Non-Threshold Unit Risk (mg/m³)⁻¹</th>
<th>Target Risk</th>
<th>Derived Soil IL (mg/kg)</th>
<th>Oral Intake (mg/kg/day)</th>
<th>Dermal Intake (mg/kg/day)</th>
<th>Criteria Exposure Concentration in Air (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>0.0005</td>
<td>0.035</td>
<td>0.006</td>
<td>1E-05</td>
<td>40</td>
<td>4.2E-06</td>
<td>1.0E-07</td>
<td>1.6E-03</td>
</tr>
<tr>
<td>Safrole and Isosafrole</td>
<td>0.1</td>
<td>0.22</td>
<td>0.063</td>
<td>1E-05</td>
<td>6</td>
<td>5.8E-07</td>
<td>2.7E-06</td>
<td>1.5E-04</td>
</tr>
</tbody>
</table>