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| The following Guideline provides general guidance in relation to Health-Based Soil Investigation Levels in the assessment of site contamination.  This Guideline forms part of the National Environment Protection (Assessment of Site Contamination Measure) 1999 and should be read in conjunction with that document, which includes a Policy Framework and Assessment of Site Contamination flowchart.  The National Environment Protection Council (NEPC) acknowledges the contribution of the National Health and Medical Research Council to the development of this Measure.  The monographs included in this Guideline were first published by the National Environmental Health Forum (NEHF) in 1996. These editions, revised July 1999, reflect changes made under the National Environment Protection Council Measure development process. The National Environment Protection Council extends its appreciation to the NEHF for their cooperation in allowing the NEPC to use and review these monographs. |

**7A**

**HEALTH-BASED SOIL INVESTIGATION LEVELS**

Paula Imray and Andrew Langley

National Environmental Health Forum Monographs

Soil Series No. 1

3rd edition

Revised July 1999

National Environmental Health Forum



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**Preface**

The National Environmental Health Forum has been established by the Directors of Environmental Health from each State and Territory and the Commonwealth with a secretariat provided by the Commonwealth Department of Health and Family Services.

The National Environmental Health Forum is publishing a range of monographs to give expert advice and guidance on a variety of important and topical environmental health matters. This publication is the first in the soil series. A list of published monographs, appears on page [iii**.**](#bookmark2)

The Directors of Environmental Health have agreed to the inclusion of this document by the National Environment Protection Council (NEPC) in the National Environment Protection (Assessment of Site Contamination) Measure 1999. During the development of the Measure, NEPC released a discussion paper, ‘Towards the Assessment of Contaminated Sites’ for an 8 week key stakeholder consultation period. The discussion paper proposed the inclusion of this document in the draft Measure. Submissions on the discussion paper suggested this was an appropriate inclusion.

In March 1999, NEPC released a draft Measure and Impact Statement for the Assessment of Site Contamination for a 12 week public consultation period. This document reflects changes made after consideration of public submissions on the draft Measure.

**Update**

In updating this edition there have been minor amendments to the text. In particular, attention has been drawn to soil eating behaviours which may indicate specific behavioural and environmental management measures to reduce exposures.

**Acknowledgments**

This publication has been made possible by considerable technical assistance, scientific editing and funding provided by each State and Territory Environmental Health Branch and the Commonwealth Department of Human Services and Health. Considerable assistance has been provided by reviewers from the States and Territories. Graphic design and layout assistance has been provided by Sandra Sowerby, Environmental Health Branch, South Australian Health Commission. The Department of Human Services (S.A.) library has assisted research and cataloguing for the document. Sue Newman, Belinda Kotowski, Julie Goodrick and Cheryl Orford provided word processing and organisational skills in the compilation of the document.

**Disclaimer**

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**PUBLISHED MONOGRAPHS**

The National Health Forum Monographs are published in series, each representing an area of interest or concern in public and environmental health.

The following list shows those published or in preparation, with the year of publication in parentheses.

*Water series*

1. Guidance for the control of Legionella (1996)
2. Guidance on water quality for heated spas (1996)
3. Rainwater tanks (1998)

*Soil series*

1. Health-based soil investigation levels (1996, 1998, 1999)
2. Exposure scenarios and exposure settings (1996, 1998, 1999)
3. Composite sampling (1996)

*Metal series*

1. Aluminium (1996)
2. Zinc (1997)
3. Copper (1997)

*Air series*

1. Ozone (1997)
2. Benzene (1997)
3. Nitrogen dioxide (1997)
4. Sulphur dioxide (1998)

*General series*

1*.* Pesticides in schools and school grounds (1997)

**HEALTH-BASED SOIL INVESTIGATION LEVELS**

Paula Imray

Queensland Department of Health

Andrew Langley

Department of Human Services (S.A.)

**1. INTRODUCTION**

The World Health Organization Environmental Health Criteria No.170 monograph *Assessing Human Health Risks of Chemicals: Derivation of Guidance Values for Health-based Exposure Limits* (1994) outlines general procedures for deriving guidance values for exposure to chemicals in environmental media. It is noted that guidance values are commonly derived for a representative general population with representative exposure conditions and should be adapted, as appropriate, at national and local levels.

The approach to deriving guidance values is based on the concept of a tolerable daily intake (TDI) which is a dose that humans may be exposed to every day throughout life without appreciable risk.

Guidance values are defined as:-

‘values, such as concentrations in air or water, which are derived after appropriate allocation of the Tolerable Intake (TI) among the possible different media of exposure. Combined exposure from all media at the guidance values over a lifetime would be expected to be without appreciable health risk. The aim of a guidance value is to provide quantitative information from risk assessment for risk managers to enable them to make decisions concerning the protection of human health.’ (WHO, 1994)

Deriving human Health-Based Investigation Levels (HILs) for soils is a particular example of deriving a guidance value for soil for each contaminant of concern. The general principles for deriving guidance values will be described firstly and then the process applied to develop health-based investigation levels for soils.

At the four National Workshops on Health Risk Assessment and Management of Contaminated Sites held to date, health-based investigation levels for various contaminants have been proposed. The basis for setting these will be reviewed and compared to the general method suggested above.

**2. PRINCIPLES FOR DERIVING GUIDANCE VALUES**

The critical steps involved in deriving guidance values are an evaluation of toxicity data in animals and humans and setting of tolerable intake (TI) levels based on the toxicity data, and allocation of the proportions of the TI to various exposure media.

**2.1 Establishing tolerable intakes**

The terms 'acceptable daily intake' (ADI), 'tolerable daily intake' (TDI), 'provisional tolerable weekly intake' (PTWI) and 'reference dose' (RfD) have similar definitions (see “[Glossary Of Terms](#bookmark16)”) and are expressions of the same concept: an estimate of the intake of a substance that over a lifetime is without appreciable health risk.

For practical purposes, toxic effects have been considered previously to be of two types, threshold or non-threshold, and tolerable exposures (for toxic effects other than cancer) and unit risks (for some cancer endpoints) have been derived.

Whilst this distinction implies mechanistic differences, it is not supported by scientific evidence. Thus, these Guidelines will not refer to threshold and non-threshold effects but to toxic effects other than cancer, and cancer toxic effects.

Where toxic effects have been considered to be of the threshold type, an uncertainty factor (also known as safety factor) has been applied to the no observable adverse effect level (NOAEL) in the most sensitive species to generate a TI. The magnitude of the uncertainty factor can vary from 10 to 10 000 depending on whether the data are from animals or humans and the quality of the study used for setting the NOAEL. Where a PTWI or an ADI has been set by the World Health Organisation or the National Health and Medical Research Council, that value should be used unless data, unavailable at the time the value was set, indicate an alternative value, endorsed by the relevant health agency, which should apply.

Where a TI is to be established on the basis of a NOAEL, a value for the uncertainty factor must be justified. Historically a factor of 100 has been applied to animal studies where adequate and appropriate studies are available. This factor of 100 is composed of a factor of 10 for interspecies variation and a second factor of 10 for intraspecies variation. Additional uncertainty factors have been incorporated to account for such things as the absence of a NOAEL, the absence of chronic data or other deficiencies in the data base. Where data from well conducted human studies were the basis for the safety evaluation, a factor of 10 has been considered appropriate (WHO, 1994). Other uncertainty factors have been proposed to take account of toxicokinetic or toxicodynamic data where these exist (Renwick and Walker 1993). Safety factors less than 10 have also been proposed as appropriate in some circumstances (Lewis *et al* 1990, Calabrese & Gilbert 1993).

TI values for some chemicals can be derived for different routes of exposure, eg. oral and inhalation. They may be based on the same or different critical effects. In cases where exposure via each route is considered to contribute to a combined dose to the target site(s) they are considered additive.

There is a need to appraise and account for all routes of exposure. If there is only one major route of exposure then the TI for that route should be used if there is confidence in the data base on which it was established. A more conservative TI should be used if there is uncertainty about the relative contribution of the various routes or media to total exposure.

Where toxicity is greater by one route than another, the TI for oral exposure (TIo) and TI for inhalation exposure (TIi) for similar effects may vary by 1 or 2 orders of magnitude. The guidance values should be derived considering each route of exposure, based on the respective TI values, and allocating a proportion of the TI for each route to the appropriate medium or media based on an appropriate exposure scenario.

Where there are route-specific effects, it is recommended that the guidance values be derived considering each route (for example, the oral and inhalation routes, based on the TIo and TIi, respectively), relating the TI for each route to the appropriate medium or media based on an appropriate exposure scenario. The 'critical' toxicological effect will need to be identified to determine which route-specific effect will govern the setting of the guidance value.

When the data base is limited such that only either a TIo or a TIi can be developed, the available TI should be allocated to various media based on an appropriate exposure scenario to determine the intake for each medium as the basis for a guidance value. Effects should be qualitatively similar and toxicokinetic data consistent with no route-specific effects at the site of entry if this approach is to be used. If any of these criteria are not met, a guidance value for an alternative route should not be derived from that data. If a TI is available for a route of exposure which does not make a significant contribution to total intake, do not derive guidance values for that route unless there is a critical route-specific effect that is somewhat independent of total intake eg skin sensitisation.

Guidelines for the Cancer Risk Assessment of Soil are currently being developed by the NHMRC Technical Working Party on Cancer Risk Assessment of Chemical Soil Contaminants and will be available as a separate document.

**3. BACKGROUND EXPOSURES AND ALLOCATIONS OF PROPORTIONS OF THE TI TO VARIOUS MEDIA**

An estimate should be made of the total intake that the population receives from various media (e.g. air, food, water, consumer products) based on exposure estimates for a consistent set of assumed or determined volumes of intake and representative concentrations in the general environment.

A proportion of the tolerable intake can be allocated to various sources of exposure to determine the intake or exposure from each medium. Guidance values can be developed from the intakes assigned to each medium. In some countries a fixed ratio is assigned to each medium: eg. Canada allocates 20% of the TDI, minus the estimated daily intake (EDI), to each of air, soil, food, water and consumer products. Where information on background exposures is adequate it would seem overly conservative and inflexible to allow only 20% of the (TDI - EDI) for each medium, when exposures from other media are well characterised. In such cases the IPCS

approach is to be used. The IPCS approach is to vary the proportions based on local data on background exposures for each substance (WHO, 1994).

The WHO Guidelines for Drinking Water Quality are derived using the TDI approach and, wherever possible, using data relevant to the proportion of the total intake normally ingested in drinking water (based on mean levels in air, food and drinking water). Where such information is not available, an arbitrary (default) value of 10% of the TDI is allocated to drinking water and used in the derivation of the guideline values.

The development of guidance values in the Environmental Health Criteria monographs is done for a clearly defined exposure scenario, based on the International Commission on Radiological Protection (ICRP) reference man (WHO, 1994). Aspects of this may need to be varied for local populations and conditions (e.g. body weight, fluid intake). For contaminated sites, 2 year old children have been regarded as the key target group to be used in criteria setting. It is recommended that where there are other age groups which are more sensitive or have differing exposure profiles, intake from each of the media should be also be estimated, using the Exposure Factors developed by Langley (1991, 1993) and Langley and Sabordo (1996) for the Australian environment where available. When unavailable, ICRP reference values or other justifiable values should be used. An Australian Human Exposure Factors Handbook will be available in 1998 as a separate document to provide standard exposure default values.

**4. INTERPRETATION AND USE OF GUIDANCE VALUES**

Guidance values incorporate assumptions about the general population exposure and the exposure scenario. Site- and context-specific considerations may make concentrations above the guidance values acceptable. Conversely, in specific situations such as where home grown produce is a significant part of the diet and the contaminant is taken up by plants, the guidance value may not be sufficiently protective of health. Other factors such as costs and ease and effectiveness of control may allow a variety of risk management strategies to be developed.

**5. HEALTH-BASED INVESTIGATION LEVELS FOR CONTAMINATED SITES**

Health-based investigation levels for contaminated sites are an example of guidance values for a particular medium, soil. A 'residential' land use setting is employed for deriving the guidance value and values are based on a default exposure scenario for a 2 year old child because the typical behaviour pattern of this age group gives them the greatest exposures to contaminants in soil. There are relatively higher soil ingestion rates, and dermal and inhalational exposures relative to body weight for a 2 year old compared to older age groups.

**6. SITE ASSESSMENT 1**

There are two prerequisites for comparison of soil test results with defined soil criteria. The first prerequisite is a uniform soil sampling methodology which provides an appropriate amount of information about the distribution and level of contaminants on a piece of land. The second is a uniform approach to data analysis to enable a meaningful interpretation of sampling results.

Site-specific evaluation of the available data and proposed land use will be required to determine whether single, occasional or typical values in excess of the investigation level will prompt the further investigation.

Levels slightly in excess of the investigation levels do not imply unacceptability or levels likely to pose a significant health risk (See [Figure 7-I)](#bookmark8).

Once the further investigation(s) is (are) completed, a site-specific health risk assessment will be required to determine the presence of health risk and, if present, its nature and degree.

Final assessment of the degree of contamination should take into account any uncertainties arising from the sampling and analytical methodologies.

Overt health effects would not be expected to occur until contamination is present at levels well in excess of response levels.

The nature of the response required to protect human health will depend on the assessment of risk associated with a given level of contamination. Where the risk is assessed as being relatively low, the response may simply involve informing occupants of the site so that they are aware of hazards arising from, for example, pica behaviour in children. In cases where there is a relatively high risk, complex soil treatment may be required.

More specifically, the nature of the response will be modulated by factors including:

1. Land use e.g. residential, agricultural/horticultural, recreation or commercial/ industrial.
2. Potential child occupancy.
3. Potential environmental effects including leaching into groundwater.
4. Single or multiple contaminants.
5. Depth of contamination.
6. Level and distribution of contamination.
7. Bioavailability of the contaminant(s) e.g. related to speciation, route of exposure.
8. Toxicological assessment of the contaminant(s) e.g. toxicokinetics, carcinogenicity, acute and chronic toxicity.
9. Physico-chemical properties of the contaminant(s).

1 incorporates ANZECC/NHMRC Guidelines text (from Langley & El Saadi, 1991)

1. State of the site surface e.g. paved, grassed or exposed.
2. Potential exposure pathways.
3. Uncertainties with the sampling methodology and toxicological assessment.

Where a site specific assessment is being carried out with a view to defining response levels, consideration should also be given to the possible risk associated with mixtures of contaminants, since in some circumstances such risks may necessitate a more or less extensive response than would be required to deal with a single contaminant.

A uniform approach should be applied in undertaking such assessments.

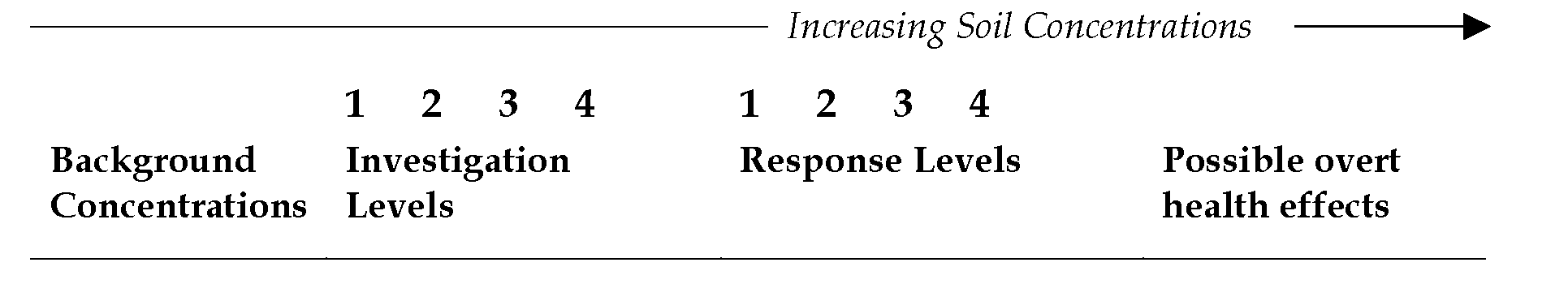
**7. NATURE OF SOIL CRITERIA**

[Figure 7-I](#bookmark8) details the relationship between soil criteria and soil concentrations.

Different response levels are intended to be used for different exposure situations (e.g. residential, recreational, or commercial/industrial land uses). There may be situations, such as with cadmium, where plant uptake rather than direct human exposures is the limiting factor and the order in [Figure 7-I](#bookmark8) may be 2,1,3,4.

**Figure 7-I**

**The relationship of soil criteria levels for Substance X.**



**Proposed Land Uses:**

1. Residential
2. Recreational
3. Residential (minimal exposure)
4. Commercial/Industrial

(Figure not to scale, sequence of '1234' will vary from substance to substance. For example, for another substance, the sequence may be 2134).

*(adapted from ANZECC/NHMRC, 1992, p36)*

When dealing with substances which are considered to have possible effects at very low doses (eg. some carcinogens), a specific approach will need to be established to derive the investigation and response levels. The NHMRC Working Party on the

Cancer Risk Assessment for Environmental Contaminants will establish Guidelines for Cancer Risk Assessment.

**8. USING INVESTIGATION LEVELS**

Investigation levels provide a trigger to assist in judging whether a detailed investigation of a site is necessary.

When assessing the environmental/health significance of levels of contamination above an investigation level, the following factors should be considered: potential ground water contamination; land use; the history and nature of the contamination; evidence of potential contamination from site inspection; the local background levels; the problems of the presence of multiple contaminants; and the size of the site. Exposure pathways will be more diverse for a larger site.

Separate health and environmental investigation levels have been established to take into account the different sensitivities of humans and other components of the environment. Site specific decisions need to be made to determine whether health or environmental levels (or both) should be applied.

**9. DETERMINATION OF HEALTH INVESTIGATION LEVELS (ANZECC/NHMRC, 1992)**

Similar principles will be used for determining HILs for contaminants with and without cancer toxic effects.

Investigation levels will be determined taking into account:

**A.** The bioavailability of a substance. The bioavailability should be assumed to be 100% if specific information is not available;

**B.** The Provisional Tolerable Weekly Intake (PTWI) or Acceptable Daily Intake (ADI) as determined by the World Health Organisation/Food and Agricultural Organisation (1987, 1994), or Guideline Dose (GD) for cancer toxic effects as determined by national health advisory bodies;

**C.** Other potential sources of the substances that comprise a proportion of the PTWI or ADI, or GD (e.g. background levels of the substance in food, water, air; and the amount of exposure through these routes)**.**

The total exposure to a substance 'X' can be represented by the equation:

**Exposure to substance X = Background Exposures (eg. from food and water)**

**+**

**Exposures from contaminated soil by ingestion,**

**inhalation and skin absorption)**

**= Background Exposures**

**+**

**Amount of substance absorbed from soil.**

= BE

+

(Sing x Cing x Bing + Sinh x Cinh x Binh + Sskin x Cskin x Bskin)

= BE + SEsoil

BE = Background Exposures (eg. from food and water).

Sing = Amount of soil ingested.

Sinh = Amount of soil/dust inhaled and retained.

Sskin = Amount of soil on skin.

Cing = Concentration of substance in soil ingested.

Cinh = Concentration of substance in soil/dust inhaled and retained.

Cskin = Concentration of substance in soil on skin.

Bing = Bioavailability, ie. percentage absorbed, of substance when ingested.

Binh = Bioavailability of substance when inhaled.

Bskin = Bioavailability of substance when on skin.

SEsoil = Substance exposure from soil."

*(ANZECC/NHMRC, 1992, p37)*

Qualifications to setting the Health-based Investigation Levels are:

* 'In setting an investigation level guideline, total exposure to substance X, (i.e. the sum of the background exposure and the substance exposure from soil) should not exceed the ADI or PTWI, (or GD) i.e., BE+SEsoil < ADI or PTWI, (or GD).'
* The degree to which exposures at a proposed investigation level guideline are below the ADI or PTWI, or GD will be set by national health advisory bodies and will depend on factors such as: the nature of the adverse effects, the completeness of toxicological data, exposure variability within a population and the relative sizes of BE and SEsoil.
* It should be recognised that '...short-term exposure to levels exceeding the PTWI is not a cause for concern provided the individual's intake averaged over longer periods of time does not exceed the level set' (WHO, 1989, p9).

Different levels of bioavailability will occur between soil ingested, inhaled or in contact with skin.

The health investigation level guideline will be set by national health advisory bodies.

A variable percentage of the TI will be allowed for exposure to contaminated soil. This is consistent with the IPCS approach and that used in the four Australian workshops.

When the PTWI/ADI is used for establishing investigation levels for individual contaminants, the basis for the level set should be sought from appropriate World Health Organisation documents (e.g. WHO 1987, WHO 1989). This information should include target organ(s) and effect(s) (e.g. nature, reversibility, severity, LOAEL for most significant toxic effect); bioavailability; and safety factors accounting for variations in human sensitivity and extrapolations from animal studies. Similarly, when a GD derived using the NHMRC Guidelines for the Cancer Risk Assessment of Soil Contaminants is used, the basis for the derivation should be fully documented. Guideline Doses for soil contaminants with cancer toxic effects will be determined by national health advisory bodies or their appointees.

If no PTWI, ADI, or GD is available a specific approach acceptable to the relevant health agencies will need to be determined using WHO (1994) for non carcinogens, or NHMRC Cancer Risk Assessment for Environmental Contaminants (in press) for substances with cancer toxic effects and used for calculations.

It is considered that these methods for determining investigation levels will protect the entire population with few exceptions. Where a significant proportion of the population demonstrates allergic sensitisation to a substance (eg nickel) this will need to be considered in criteria setting. People who may have unusual sensitivity to contaminants may need to be considered in a site assessment.

**10. HEALTH INVESTIGATION LEVEL GUIDELINES**

Investigation levels based on health considerations were established (ANZECC/NHMRC 1992) using a risk assessment approach for lead, cadmium, arsenic and benzo(a)pyrene, which are frequently occurring and toxicologically important contaminants. Further soil criteria are detailed in [Table 11-A.](#bookmark12)

Further investigation level guidelines will be proposed as sufficient toxicological information becomes available and the present guidelines may also be subject to change as more information becomes available.

The levels should not be interpreted rigidly. Two sites with similar distributions of concentrations and median lead levels of 290 and 310 will not be significantly different. The proposed land use, distribution of contaminants and the frequency distribution of elevated levels will all be very important in interpreting the results for a site.

The proposed health-based soil investigation levels are detailed in [Table 11-A](#bookmark12) using the exposure settings detailed in the following section. Environmental and aesthetic matters will also need to be considered in the evaluation of a site.

**11. EXPOSURE SETTINGS**

The following exposure settings (Taylor and Langley 1996) are based on several conservative assumptions and are used to provide a 'tiered' set of soil criteria for different exposure settings:

**A.** Standard' residential with garden/accessible soil (home-grown produce contributing less than 10% of vegetable and fruit intake; no poultry): this category includes children’s day-care centres, kindergartens, pre-schools and primary schools.

**B.** Residential with substantial vegetable garden (contributing 10% or more of vegetable and fruit intake) and/or poultry providing any egg or poultry intake. 2

**C.** Residential with substantial vegetable garden (contributing 10% or more of vegetable and fruit intake); poultry excluded. 2[2](#bookmark11)

**D.** Residential with minimal opportunities for soil access; includes dwellings with fully and permanently paved yard space such as high-rise apartments and flats.

**E.** Parks, recreational open space and playing fields; includes secondary schools.

**F.** Commercial/Industrial: includes premises such as shops and offices as well as factories and industrial sites. It is assumed that thirty years is the duration of exposure.

Where land is used predominantly for one purpose, but contains within it a more “sensitive” use, then the exposure setting relevant to that more sensitive use must be adopted for that particular parcel of land. For example, if an industrial site is also used for residential purposes such as a caretaker’s residence, or there is an on-site creche within a commercial facility, then the appropriate residential setting “A” should be used for areas of the site that may give rise to soil exposure.

There are numerous qualifications and constraints to the use of these soil criteria and Taylor and Langley (1999) must be examined closely before these settings are used.

Guidelines for agricultural land will need to be determined by appropriate agencies e.g. agricultural, environmental and health agencies.

2 In the context of establishing exposure settings specifically for different land uses and to derive soil investigation criteria, it is considered appropriate for these particular settings to adopt a threshold of 10% domestic food production. The majority of households with vegetable gardens would not reach this level of food production. The threshold of 10% is an indicative rather than an absolute value. Where this value is likely to be exceeded the site should be assessed as a 'C' scenario.

**Table 11-A**

**Proposed Health-baseda Soil Guidelines for Individual Substances**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Substance** | **Health-based Investigation Levelsb (mg/kg)** | | | | | **Health-based Response Levels (mg/kg)** |
|  | **A** | **Bc** | **Cd** | **D** | **E** | **F** |
| Aldrin+ Dieldrin | 10 |  |  | 40 | 20 | 50 |
| Arsenic (total) | 100 |  |  | 400 | 200 | 500 |
| Benzo(a) pyrene | 1 |  |  | 4 | 2 | 5 |
| Beryllium | 20 |  |  | 80 | 40 | 100 |
| Boron | 3000 |  |  | 12000 | 6000 | 15000 |
| Cadmium | 20 |  |  | 80 | 40 | 100 |
| Chlordane | 50 |  |  | 200 | 100 | 250 |
| Chromium(III)ef | 12% |  |  | 48% | 24% | 60% |
| Chromium (VI) | 100 |  |  | 400 | 200i | 500 |
| Cobalt | 100 |  |  | 400 | 200 | 500 |
| Copper | 1000 |  |  | 4000 | 2000 | 5000 |
| Cyanides (complexed)g | 500 |  |  | 2000 | 1000 | 2500 |
| Cyanides (free) g | 250 |  |  | 1000 | 500 | 1250 |
| DDT**+**DDD+DDE | 200 |  |  | 800 | 400 | 1000 |
| Heptachlor | 10 |  |  | 40 | 20 | 50 |
| Lead | 300 |  |  | 1200 | 600 | 1500 |
| Manganese | 1500 |  |  | 6000 | 3000 | 7500 |
| Methyl mercuryh | 10 |  |  | 40 | 20 | 50 |
| Mercury (inorganic)[e](#bookmark12) | 15 |  |  | 60 | 30 | 75 |
| Nickel | 600 |  |  | 2400 | 600[i](#bookmark12) | 3000 |
| Polycyclic aromatic | 20 |  |  | 80 | 40 | 100 |
| hydrocarbons (PAHs) |  |  |  |  |  |  |
| PCBs (total) | 10 |  |  | 40 | 20 | 50 |
| Phenol j | 8500 |  |  | 34000 | 17000 | 42500 |
| Total petroleum |  |  |  |  |  |  |
| hydrocarbonsk |  |  |  |  |  |  |
| >C16-C35 aromatics | 90 |  |  | 360 | 180 | 450 |
| >C16-C35 aliphatics | 5600 |  |  | 22400 | 11200 | 28000 |
| >C35 aliphatics | 56000 |  |  | 224000 | 112000 | 280000 |
| Zinc | 7000 |  |  | 28000 | 14000 | 35000 |

a A draft methodology for the derivation of Ecological Investigation Levels is available from Environment Australia.

b See exposure settings detailed in Section 11 and Taylor and Langley (1998)

c Site and contaminant specific: on-site sampling is the preferred approach for estimating poultry and plant uptake. Exposure estimates may then be compared to the relevant ADIs, PTWIs and GDs.

d Site and contaminant specific: on-site sampling is the preferred approach for estimating plant uptake. Exposure estimates may then be compared to the relevant ADIs, PTWIs and GDs.

e Need to ensure valency state by site history/ analysis/ knowledge of environmental behaviour

f Soil discolouration may occur at these concentrations

i Skin contact resulting in exacerbation of pre-existing skin sensitisation is the critical effect and recreational use is considered the same as residential use because of the skin contact opportunities

g See Cyanides (free and complexed) pg 29. The nature of the cyanides on a site must be assessed. To use the HIL for complexed cyanides, no more than a five per cent of free cyanides should be present (and *vice versa* for free cyanides).

h Need to ensure form of substance by site history/ analysis/ knowledge of environmental behaviour

j Odours and skin irritation may occur at lower, as yet undetermined, concentrations. PVC pipes may be affected at high concentrations with possible adverse effects on the water therein.

k These HILs refer to the noncarcinogenic component and should be used according to the two-stage framework detailed on p. 35.

Notes to [Table 11-A:](#bookmark12)

1. The health-based soil criteria do not necessarily take into account environmental and aesthetic concerns, which may impact greatly upon remediation and management decisions. Therefore whilst an investigation level for commercial land use may be contemplated that is five times higher than that for residential land with garden, this may not be an acceptable investigation threshold from the perspective of protecting particular species or the ecosystem.
2. For residential settings, it is assumed that 70 years is the duration of exposure. However for many contaminants (particularly those for which ADIs or PTWIs have been established) exposures over a much shorter period during childhood tend to dictate investigation criteria.
3. Highly volatile substances are excluded from consideration in this table.
4. Changes in concentration over time in a soil stratum may occur as the result of factors such as breakdown, volatilisation and leaching. This may be relevant to the assessment of long term exposures.
5. These values must only be used where there has been adequate characterisation of a site (ie sufficient and appropriate sampling). The arithmetic mean must be compared to the values given in [Table 11-A.](#bookmark12) The relevance of localised elevated values must 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 values given in [Table 11-A](#bookmark12)
* no single value exceeds 250% of the relevant value given in [Table 11-A.](#bookmark12)

1. Some contaminants may be taken up by poultry. These include organochlorine pesticides and PCBs and some metals. If it is likely that poultry will be kept, the health-based criteria in Column A should not be used. Cross and Taylor (1996) provides further information.
2. The application of Investigation Levels and Response Levels to site management will be guided by the risk management process which will be driven by scientific, technological, social, political and economic factors.

These investigation level guidelines have specific definitions and relate to specific sampling, extraction and analytical techniques. As such, they should not be compared with other tables of values which have different definitions or use different sampling, extraction and analytical techniques. Details of the derivation of these HILs can be found in the Proceedings of the National Workshops (El Saadi O & Langley AJ 1991; Langley A & van Alphen M 1993; Langley AJ, Markey BR & Hill HS, 1996; Langley A, Imray P, Lock W & Hill H 1998.). A brief summary of the key assumptions in the derivation of these HILs is given in Appendix 1.

**12. RELATED ISSUES**

**12.1 Homegrown Produce**

The report by Cross and Taylor (1996) *Human Exposure to Soil Contaminants Through the Consumption of Home-grown Produce* assesses this important indirect pathway of human exposure to soil contaminants. Where contamination occurs on residential land and home-grown produce contributes to the food intake the HILs set in the general manner may not provide the anticipated margin of protection if there is significant uptake of the contaminant into produce. In these instances, exposure to contaminants via home-grown produce may have the potential to demand lower HILs. The degree of uptake by produce is highly variable and depends on factors such as the specific contaminant, the type of produce, the soil type, coexistent chemical components of the soil and the growing conditions.

It is evident that there remain many uncertainties about assessment of exposure through contaminant uptake into home-grown produce and subsequent ingestion, where residential subdivision of contaminated land has occurred. The situation is complex because of large site-to-site variations in conditions influencing contaminant uptake. Given the large number of factors which influence uptake by plants (e.g. soil type, pH, and balance of other ions), it is suggested that measuring contaminant levels in produce from the particular site is likely to be the best means of determining potential exposure. Estimates of exposure may need to take into account food preparation methods, dietary habits and seasonal variation.

For residential sites where more thorough exposure assessments are indicated, contaminant levels in produce should be determined using sampling, preparation and analytic procedures comparable to those in the Australian Market Basket Survey (AMBS). Whilst residents should, in principle, be able to grow produce that meets the Australian Food Standards Code (AFSC), this Code is directed towards produce grown commercially using 'Good Agricultural Practice' but it also has regard for international trade agreements and, although a useful yardstick, is not necessarily based on toxicological considerations. Food preparation and analytical techniques used in the AFSC are not always comparable to those for the AMBS.

**12.2 Soil Eating Behaviours**

Consistent soil eating behaviour (geophagia) is considered rare although intermittent eating of unusual substances (pica) including soil is more common. There should be an awareness of these behaviours and specific behavioural and environmental management measures may be indicated to reduce the exposures if a particular individual is identified with these behaviours.

**13. EXPOSURE DURATION AND EXCEEDANCES OF THE TOLERABLE INTAKE**

The issue of the significance of exceedances of the TI has been discussed by Langley and Sabordo (1996). Appropriate durations of exposure need to be assessed so that transient (short-term) and important exposures are not obscured by the use of average estimates, eg. average lifetime exposure. The duration and magnitude of exceedances of the TIs must be obvious in exposure assessments.

WHO documents (1987) state 'Because in most cases, data are extrapolated from lifetime animal studies, the ADI relates to life-time use and provides a margin of safety large enough for most toxicologists not to be particularly concerned about short-term use at exposure levels exceeding the ADI, providing the average intake over longer periods does not exceed it.' Further information on this was provided in 1989 when it was added that 'It is impossible to make generalisations concerning the length of time during which intakes in excess of the PTWI would be toxicologically detrimental. Any detrimental effect would depend on the nature of the toxicity and the biological half-life of the chemical concerned' (WHO, 1989).

In considering potential exceedances of the TI (ADI or PTWI) Renwick and Walker (1993) propose three questions:

1. What proportion of the population should be allowed to exceed the ADI?
2. To what extent can the ADI be exceeded without any real concern?
3. How long does the person need to exceed the ADI before there is a cause for real concern?

The significance of any minor excursions of intake above the TI can best be put into context by consideration of the data on which the TI was based.

When the TI was based on a NOAEL from animal studies the following should be considered. The precision of the NOAEL depends on the sensitivity of the toxicological end point of the observed effect. This is influenced by the group size studied, the incidence of the lesion in control and test animals, inter-animal variability and the type of effect (eg. gross histopathology or enzyme induction). The increment between doses employed in the studies is also important. When there are very large increments between doses the NOAEL demonstrated by the study can be significantly lower than the actual or absolute NOEL.

Thus the significance of an exceedance of the TI can only be assessed on a substance-specific basis and by reference to the toxicological data (e.g. dose-response patterns, timing of exposure and severity of effects), the basis of the NOAEL, and the magnitude and duration of the exceedance.

**14. EVALUATION OF MIXTURES**

The 1992 Australian and New Zealand Guidelines recognised the issue of mixtures and stated that:

'all toxicity data are derived from studies utilising pure chemicals. The reduction in activity of a toxicant in a soil matrix or synergistic effects of multiple toxicants being present have not as yet been evaluated.'

A number of approaches for risk assessment of mixtures are described in the literature and include: the additivity of risks at low concentration (US EPA model), the comparative potency model, and various techniques based on short-term tests and the use of biomarkers (Shaw and Moore 1996).

One of the important interactive effects affecting toxicity is the influence of components in a mixture on the metabolic activation and detoxification enzymes. Enzymes such as cytochrome P-450 can have their activity induced or reduced by various compounds.

The US EPA has recognised that the applicability of additivity decreases as the number of components in a mixture increases.

A relative potency or comparative potency method allows a risk estimate for a mixture on the assumption that there is a constant relative potency between different mixtures across different bioassay systems. Toxic equivalence factors (TEFs) have been used to assess relative potency of closely related chemicals such as dioxins and PCB congeners. Structure activity relationships have aided the development of TEFs.

The International Agency for Research on Cancer has suggested a risk assessment strategy with complex mixtures involving epidemiological studies on populations exposed to complex mixtures; assessment of human exposures and relevant biological effects; and experimental approaches *in vitro* and *in vivo*, such as studies using extracts of complex mixtures or assays of biomarkers.

The properties of metals in alloys are different from their chemical components (Dresher and Poirier, 1997).

The potential importance of toxicological interactions of soil contaminants is recognised in Australia. Such interactions must be considered in the evaluation of sites where a mixture of contaminants is present.

**15. REFERENCES**

Addison J, Davies LST, Robertson A, Wiley RJ (1988), The release of dispersed asbestos fibres from soils. Report No. TM/88/14. Edinburgh, Institute of Occupational Medicine.

ANZECC/NHMRC (1992). Australian and New Zealand Guidelines for the Assessment and Management of Contaminated Sites. Canberra: Australian and New Zealand Environment and Conservation Council/National Health and Medical Research Council.

ATSDR (1994). Toxicological Profile for Zinc (Update). Atlanta, GA; Agency for Toxic Substances and Disease Registry.

Calabrese EJ, Gilbert CE (1993). Lack of total independence of Uncertainty Factors (UFs): implications for the size of the total Uncertainty Factor. *Regulatory Toxicology & Pharmacology*, 17: 44-51.

Cross SJ, Taylor ER (1996). Human Exposure to Soil Contaminants through the Consumption of Home-grown Produce. Contaminated Sites Monograph series No. 6. Adelaide; South Australian Health Commission.

Dresher WH, Poirier RH (1997). Metallic Alloys and Mixtures: Definitions, Behaviour and Characteristics with Special Reference to the Environment. Ottawa, International Council on Metals and the Environment.

El Saadi O, Langley AJ eds (1991). The Health Risk Assessment and Management of Contaminated Sites. Proceedings of a National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Monograph No. 1. Adelaide; South Australian Health Commission.

Hodgson E, Mailman RB, Chambers JE (1988). Macmillan Dictionary of Toxicology. London; Macmillan Reference Books.

IRIS. Integrated Risk Information System. US Environmental Protection Agency, Washington, DC. 1994.

Langley A (1991). Exposure Pathways. In: El Saadi O and Langley AJ eds. The Health Risk Assessment and Management of Contaminated Sites. Proceedings of a National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Monograph No. 1. Adelaide; South Australian Health Commission.

Langley A (1993). Refining Exposure Assessment. In: Langley AJ, Van Alphen M eds. The Health Risk Assessment and Management of Contaminated Sites. Proceedings of the Second National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Monograph No. 2. Adelaide: South Australian Health Commission.

Langley AJ, El Saadi O eds (1991). Protocol for the Health Risk Assessment and Management of Contaminated Sites. Summary of a National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Monograph No. 1. Adelaide: South Australian Health Commission.

Langley AJ, Van Alphen M eds (1993). The Health Risk Assessment and Management of Contaminated Sites. Proceedings of the Second National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Monograph No. 2. Adelaide: South Australian Health Commission.

Langley AJ and Sabordo L (1996). Exposure Factors in Risk Assessment. In: Langley AJ, Markey BR and Hill HS eds. Proceedings of the Third National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Sydney, 1995. Monograph No. 5.. Adelaide: South Australian Health Commission.

Langley AJ, Markey BR & Hill HS, eds (1996). The Health Risk Assessment and Management of Contaminated Sites. Proceedings of the Third National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Sydney, 1995. Monograph No. 5. Adelaide: South Australian Health Commission.

Langley A, Imray P, Lock W & Hill H, eds. (1998) The Health Risk Assessment and Management of Contaminated Sites. Proceedings of the Fourth National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Brisbane, 1996. Monograph No. 7. Adelaide: South Australian Health Commission.

Lewis SC, Lynch JR, Nikiforov AI (1990). A new approach to deriving community exposure guidelines from "No-Observed-Adverse-Effect Levels". *Regulatory Toxicology and Pharmacology*, 11: 314-330.

Renwick AG, Walker R, (1993). 'An analysis of the risk of exceeding acceptable or tolerable daily intake.' *Regulatory Toxicologically and Pharmacology*, 18: 463-480.

Shaw G and Moore M (1996). Chemical Mixtures and Soil Contamination- Issues for Risk Assessment of Carcinogens. In: Langley AJ, Markey BR and Hill HS (eds) Proceedings of Third National Workshop on the Health Risk Assessment and Management of Contaminated Sites. Sydney, 1995. Monograph No. 5.

Sloof W et al, eds (1989). Integrated Criteria Document Copper. Report No. 758474009. Bilthoven, National Institute of Public Health and Environmental Protection.

Taylor R and Langley AJ (1998). Exposure Settings and Exposure Scenarios. National Environmental Health Forum Soils Monograph No. 2. 2nd edition. Adelaide: South Australian Health Commission.

TPHCWG (1997) A Risk-based Approach for the Management of Total Petroleum Hydrocarbons in Soil. A Technical Overview of the Petroleum Hydrocarbon Risk Assessment Approach of the TPH Criteria Working Group. Final Draft March 1997

Turczynowicz L (1993). The Assessment and Management of Gasworks Sites. In: Langley AJ, Van Alphen M eds. The Health Risk Assessment and Management of Contaminated Sites. Proceedings of the Second National Workshop on the Health

Risk Assessment and Management of Contaminated Sites. Monograph No. 2. Adelaide: South Australian Health Commission.

US EPA (1990). Guidance on remedial actions for Superfund sites with PCB contamination. Washington, DC. Office of Emergency and Remedial Response.Technical Report Series 776. Geneva; World Health Organization.

World Health Organization (1972). Evaluation of certain food additives and the contaminants mercury, lead, and cadmium. Sixteenth report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). World Health Organization Technical Report Series 505. Geneva; World Health Organization.

World Health Organization (1987). Environmental Health Criteria 70. Principles for the safety assessment of food additives and contaminants in food. International Programme on Chemical Safety (IPCS) in cooperation with the Joint FAO/WHO Expert Committee on Food Additives (JECFA). Geneva; World Health Organization.

World Health Organization (1989). Evaluation of certain food additives and contaminants. Thirty third report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). World Health Organization Technical Report Series 776. Geneva; World Health Organisation.

World Health Organization (1989a). Guidelines for predicting dietary intake of pesticide residues. Joint UNEP/FAO/WHO Food Contamination Monitoring Programme. Geneva, World Health Organization.

World Health Organization (1993). Guidelines for drinking water quality. 2nd edition.Volume 1. Recommendations. Geneva, World Health Organization.

World Health Organization (1994). Environmental Health Criteria 170. Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. International Programme on Chemical Safety (IPCS). Geneva; World Health Organization.

**GLOSSARY OF TERMS**

**ADI** Acceptable Daily Intake. The daily intake of a chemical which, during a lifetime, appears to be without appreciable risk, on the basis of all the facts known at the time. It is expressed in milligrams of the chemical per kilogram of body weight.

*WHO, 1989a*

**ATSDR** Agency for Toxic Substances and Disease Registry, US Department of Health & Human Services.

**EDI** Estimated Daily Intake. A prediction of the daily intake of a pesticide residue based on the most realistic estimation of residue levels in food and the best available food consumption data for a specific population. The residue levels are estimates taking into account known uses of a pesticide, the range of contaminated commodities, the proportion of a commodity treated, and the quantity of contaminated homegrown or imported commodities. The EDI is expressed in milligrams of the residue per person.

*WHO, 1989a*

**GAP** Good Agricultural Practice. 'The officially recommended use of [pesticides], under practical conditions, at any stage of production, storage, transport, distribution, or processing of food, agricultural commodities, or animal feed, bearing in mind the variations in requirements within and between regions. This takes into account the minimum quantities necessary to achieve adequate control, applied in such a manner that the amount of residue is the smallest practicable and which is toxicologically acceptable.'

*WHO, 1989a*

**GD** Guideline Dose. An estimate of the daily human dose (milligrams per kilogram per day) of a chemical which, during a lifetime of exposure is likely to be of negligible risk of cancer, on the basis of all the information known at the time. The guideline dose is derived by regulatory authorities using cancer risk assessment according to guidelines developed by national health advisory bodies.

**GV** Guidance values. 'Values, such as concentrations in air or water, which are derived after appropriate allocation of the Tolerable Intake (TI) among the possible different media of exposure. Combined exposure from all media at the guidance values over a lifetime would be expected to be without appreciable health risk. The aim of a guidance value is to provide quantitative information from risk assessment for risk managers to enable them to make decisions concerning the protection of human health.'

*WHO, 1994*

**HIL** Health Investigation Level. The concentration of a contaminant (arrived at using appropriate sampling, analytical and data interpretation techniques) above which further appropriate investigation and evaluation will be required. The

investigation and evaluation is to ascertain: the typical and extreme concentration of contaminant(s) on the site; the horizontal and vertical distribution(s) of the contaminant(s) on the site; the physico-chemical form(s) of the contaminants; and the bioavailability of the contaminant(s).

**ICRP** International Commission on Radiological Protection.

**IPCS** International Programme on Chemical Safety.

**MRL** Minimal Risk Level. An estimate of daily human exposure to a dose of a chemical that is likely to be without appreciable risk of adverse non-cancerous effects over a specified duration of exposure.

*ATSDR,1994*

**NHMRC** National Health and Medical Research Council.

**NOAEL** No Observable Adverse Effect Level. 'Greatest concentration or amount of a substance, found by experiment or observation, which causes no detectable adverse alteration of morphology, functional capacity, growth, development or life span of the target organism under defined conditions of exposure. Alterations of morphology, functional capacity, -growth, development or life span of the target may be detected which are judged not to be adverse.'

*WHO, 1994*

**NOEL** No Observable Effect Level. 'Greatest concentration or amount of a substance, found by experiment or observation, which causes no alterations of morphology, functional capacity, growth, development or life span of target organisms distinguishable from those observed in normal (control) organisms of the same species and strain under the same defined conditions of exposure.'

*WHO, 1994*

**PTWI** Provisional Tolerable Weekly Intake. The Tolerable Intake expressed as a weekly amount. The term was established by WHO (1972) for several heavy metals which 'are able to accumulate within the body at a rate and to an extent determined by the level of intake and by the chemical form of the heavy metal present in food'

*WHO, 1989*

**RfD** Reference Dose. An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure (mg/kg/day) to the general human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime of exposure. It is derived from the No Observed Adverse Effect Level (NOAEL) or Lowest Observed Adverse Effect Level (LOAEL) by application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgement of the entire database of the chemical. It is expressed in units of milligrams of contaminant per kilogram body weight per day

*IRIS, 1994*

**TDI** Tolerable Daily Intake. The TI expressed as a daily amount.

**TEF** Toxic Equivalence Factor.

**TI** Tolerable Intake. 'An estimate of the intake of a substance which can occur over a lifetime without appreciable health risk. It may have different units depending upon the route of administration.'

*WHO, 1994*

**TIo** Tolerable Intake by the oral route.

**Tii** Tolerable Intake by the inhalation route. 'Though not strictly an 'intake'... generally expressed as airborne concentrations (i.e. µg or mg per m³)'

*WHO, 1994*

**Toxicodynamics** The study of the relationship between toxicant concentrations and effects with an emphasis on the mechanism(s) of action

*Hodgson et al, 1988*

**Toxicokinetics** The study of the absorption, distribution, metabolism and excretion of toxicants by living organisms

*Hodgson et al, 1988*

**US EPA** United States Environmental Protection Agency.

**WHO** World Health Organization.

**APPENDIX 1**

**Health Investigation Levels**

Since the first National Workshop on Health Risk Assessment and Management of Contaminated Land held in 1991, there have been three subsequent workshops at which there have been developments in the procedures for deriving Health Investigation Levels and the application of these procedures to generate levels for a number of common contaminants.

This brief summary draws out key assumptions in deriving these Health Investigation Levels but those looking for background information, references and details of the rationales by which these Health Investigation Levels were developed should refer to the proceedings of the first, second, third and fourth National Workshops (Contaminated Sites Monographs 1, 2, 5 and 7 respectively) which are referenced on pages 14 and 15.

The following list gives the Workshop and Monograph reference for each contaminant or contaminant group.

|  |  |  |
| --- | --- | --- |
| **Substance** | **Workshop Date** | **Monograph Number** |
| Aldrin, Dieldrin, Chlordane & Heptachlor | 1993 | 2 |
| Arsenic (total) | 1991 | 1 |
| Asbestos | 1993 | 2 |
| Benzo (a) pyrene | 1991 | 1 |
| Beryllium | 1995 | 5 |
| Boron | 1996 | 7 |
| Cadmium | 1991 | 1 |
| Chromium (VI) | 1993 | 2 |
| Chromium (III) | 1993 | 2 |
| Cobalt | 1996 | 7 |
| Copper | 1993 | 2 |
| Cyanides (complexed) | 1993 | 2 |
| DDT | 1993 | 2 |
| Lead | 1991 | 1 |
| Manganese | 1995 | 5 |
| Mercury (inorganic, methyl) | 1995 | 5 |
| Nickel | 1995 | 5 |
| Phenol | 1993 | 2 |
| Polychlorinated biphenyls (PCB) - total | 1993 | 2 |
| Total petroleum hydrocarbons | 1996 | 7 |
| Zinc | 1995 | 5 |

For some of these substances biodegradation, volatilisation and complex formation with soil components will occur over time and this may need to be considered in the evaluation of a site.

**Health-based Investigation Levels for Soil**

**Aldrin, Dieldrin, Chlordane and Heptachlor**

The organochlorine (cyclodiene) termiticides aldrin, dieldrin, chlordane and heptachlor were reviewed together in view of the similarities in chemistry and toxicity. Aldrin and dieldrin have not been used in Australia since 1992 but both had previously been used as termiticides in domestic premises and were widely used in agriculture. The use of chlordane and heptachlor as termiticides continued until 30 June 1995 except in the Northern Territory where use continued until 31 October 1997.

The WHO has set ADIs for these compounds and the US EPA has established Reference Doses and carcinogenic potency factors.

Background exposure of the general population occurs via ingestion of adventitious residues in the diet, and inhalation of vapour. Although the concentrations of cyclodienes in food and water have decreased in recent years, there is concern about produce not sampled by the Australian Market Basket Survey. Additionally, low levels of exposure from air are continuing, albeit at reducing levels.

Soil HILs were calculated taking into account ingestion, inhalation and dermal absorption of soil contaminants.

An additional safety factor of 3 was used because of the uncertainties about the bioavailability estimates and levels of exposure in the future and because the data on which these estimates are based are limited.

Thus, the suggested guidelines, based on exposure for a child are 10 mg/kg soil for aldrin and dieldrin combined for a standard residential exposure scenario. In comparison, the Dutch human 'tox C' value (concentration that would provide exposure equal to the TDI) is 13 mg/kg for aldrin and 5.2 mg/kg for dieldrin (van den Berg, 1993).

In the case of heptachlor, the HIL is 10 mg/kg and it is possible that exposure to this concentration in soil may lead to daily intakes above the ADI in some circumstances such as recent soil treatment. Therefore the value of 10 mg/kg for a standard residential exposure scenario is subject to the residence not having been treated in the previous 12 months. This is to allow for the added exposures that may occur from house treatments. (Except for the Northern Territory this became irrelevant on 30 June 1996 due to the prohibition of use after 30 June 1995.).

In the absence of data about air levels of chlordane after application a conservative value of 10 mg/kg was suggested (DiMarco, 1993, p 162). An investigation value of 50 mg/kg for a standard residential exposure scenario has been derived based on the ADI calculations.

Plant and poultry uptake warrant consideration as these may be relevant exposure pathways for these termiticides.

**Arsenic**

Arsenic is widely distributed - both naturally and anthropogenically - in the environment and contamination often occurs as a result of agricultural and timber preservation activities. The WHO PTWI for arsenic is 0.015 mg/kg/wk and there is a narrow margin of safety between the PTWI and intakes reported to have toxic effects in epidemiological studies.

For children, dietary exposure to arsenic could account for 50% of the PTWI based on information from Australian Market Basket Surveys.

Absorption of arsenic by oral, dermal and inhalation routes was assessed and the contribution from ingested soil accounts for 90% of the total exposure from contaminated soil. Based on a 2.5 year old child, body weight 13.2 kg and ingesting 100 mg soil per day, the HIL for arsenic was determined to be 100 mg/kg (equivalent to 40% of the PTWI) for a standard residential exposure scenario.

**Asbestos**

The major health risk is from inhaled asbestos fibres. Friable materials pose the greatest health risks as they will more likely give rise to airborne asbestos fibres.

No relationship between soil levels and air levels can be predicted for an asbestos-contaminated site. The Addison et al study (1988) shows trends using a laboratory test system but the huge amount of variability which may exist on contaminated sites makes it very difficult and probably inappropriate to apply results from this study to contaminated sites.

Since asbestos left undisturbed is not considered to present a risk to health from ingestion, there is no scientific basis for setting an 'acceptable' level in soil related to ingestion. The risks depend on the potential for disturbance and generation of airborne asbestos which may be inhaled.

The Addison et al study (1988) showed that under conditions that generate dust to approximately 5 mg/m2, asbestos in dry soil at 0.001% (w/w homogeneous sample) gives rise to > 0.01 f/ml in air.

On a contaminated site, release of fibres will be reduced when asbestos is in the form of manufactured products such as asbestos-cement sheets, pipes or boards, or if there is a high moisture content in the soil.

Appropriate site-specific measurements on a site are warranted if there are sufficient concerns based on site conditions and the nature of the asbestos.

**Benzo (a) pyrene**

Benzo(a)pyrene is a PAH found in soil from both naturally-occurring and anthropogenic sources. Natural background is due to production by plants while contamination results from activities such as coal gasification, petroleum refining, coke production, iron and steel founding, and combustion of fossil fuels and other organic matter.

Some PAHs are classified as IARC Group 2A and 2B carcinogens and the procedure for these compounds, in the absence of an ADI/PTWI, was to establish their carcinogenic potency, and then estimate a dose associated with a particular increased lifetime cancer risk. The two exposure routes of inhalation and ingestion are both considered important toxicologically. Estimates of background exposure from water, food and air were made and the major source of exposure for most people was shown to be dietary. From PAH contaminated soil the ingestion route of exposure is most significant. A HIL of 1 mg/kg for a standard residential exposure scenario was proposed which was estimated to give an exposure of 0.8 ng/kg/day, and to be nearing a significant contribution to the dietary estimates.

**Beryllium**

Beryllium, the lightest metal, is a ubiquitous trace element in the environment (average concentration in soil 2.8 to 5 mg/kg). It has minor widespread industrial uses, particularly as an alloy with copper and in ceramics as BeO. Absorption of beryllium and beryllium compounds is substantial after inhalation, less than 1% by the oral route and even less by the dermal route.

Occupational beryllium exposure has been associated with acute and chronic lung diseases. The acute disease arises from inhalation exposure to high levels of soluble beryllium salts (eg sulfate, chloride) and BeO and may lead to chronic disease. Chronic disease is associated with long-term inhalation of dust particles containing beryllium. There is an immunological component and a variable latency period which depends on the beryllium species. Dermatological effects may also occur on skin contact.

Beryllium and its compounds are classified as Group 1 carcinogens by IARC and Group 2B by the US EPA. They may cause lung cancer following inhalation. Based on human data the US EPA has derived an oral slope factor of 4.3 mg/kg/day and an RfD for non-neoplastic endpoints from soluble beryllium salts of 5 µg/kg/day. A TDI of 1 µg/kg/day is proposed, based on a NOAEL of 0.54 mg/kg/day in a rat study and a safety factor of 500. In setting the HIL, inhalation of contaminated dust is not considered to contribute significantly to exposure. Calculations of oral and dermal exposure routes indicate that for a 13.2 kg infant, ingesting 100 mg soil/day, and allowing for background exposure from diet, a HIL of 20 mg/kg for a standard residential exposure scenario is appropriate.

**Boron**

Boron is a nonvolatile solid metalloid element that occurs widely in nature at low concentrations. It is ubiquitous in rocks, soils and water. Boron is an essential nutrient for plants and is probably an essential trace element for humans. It does not bioaccumulate in humans.

People can be exposed to boron in food (mainly vegetables, fruits, nuts and legumes). Food content varies from 0.16 mg/kg in red meat to 160 mg/kg in quinces. The estimate of adult average daily ingestion for Australia is 2.23 mg/day. Drinking water levels in South Australia have a reported range of 0.02 - 0.18 mg/L in metropolitan areas and 0.02 - 1.3 mg/L in country areas. Boron concentrations in rocks range from 5 mg/kg in basalts to 100 mg/kg in shales and average 10 mg/kg in the earth’s crust. Most of the earth’s soils have <10 mg/kg and average 10 - 20 mg/kg.

Chronic human exposures have caused anorexia, weight loss, vomiting, mild diarrhoea, skin rash, alopecia, convulsions and anaemia and, in high dose animal studies, gonadal injury and fetal effects.

A health-based investigation level of 3000 mg/kg is considered appropriate based on a 30% contribution of the difference between estimated background exposures and a tolerable intake (derived, in the absence of a WHO ADI, from animal studies for non-cancer effects with the application of safety factors).

**Cadmium**

Cadmium is widely distributed in the environment at very low levels; contamination occurs from industrial, mining and agricultural activities. The WHO PTWI for cadmium is 7 µg/kg/wk. A relatively small margin of safety exists between the PTWI and exposures that produce deleterious effects. Estimated exposure from background sources is about 30% of the PTWI. Since cadmium is a cumulative heavy metal, body burden increases with age.

Soil ingestion is the major route of exposure to cadmium in soil, with inhalation of contaminated dust contributing more than dermal contact with soil. Based on a soil ingestion rate of 100 mg/day for a 2.5 year old child, a HIL of 20 mg/kg is proposed, which is 19% of the PTWI (Langley, 1991), for a standard residential exposure scenario.

**Chromium**

Chromium occurs naturally in soils at levels generally below 100 mg/kg. In soil it is usually present as Cr (III) while Cr (VI) occurs rarely in nature. It is an essential trace element. Chromium is used in leather tanning, chrome plating, wood preservation, chrome alloy, paints and pigments.

The US EPA chronic RfD for Cr (III) is 1 mg/kg/day, based on a NOAEL of 1.46 mg/kg/day from a chronic feeding study in rats. Assuming a child has a body weight of 12 kg and ingests 100 mg soil/day, a Health Investigation Level of 120 000 mg/kg for Cr (III) is proposed for a standard residential exposure scenario. At this concentration, soil discolouration is likely and, based on aesthetic considerations, a lower level of chromium (III) may be considered desirable.

It has been recommended that skin hypersensitivity to chromium be considered in the risk assessment of chromium contaminated soils. Contact dermatitis can develop after relatively short periods of contact to chromium compounds and reported positive rates to Cr (VI), as potassium dichromate are 4 to 20% of the groups tested. There may be a threshold for effect based on a dose-response relationship shown experimentally. A Health Investigation Level of 100 mg/kg for Cr (VI) for a standard residential exposure scenario is proposed to provide a 10 fold safety margin over the 'likely threshold concentration for skin sensitivity' suggested by Sheehan *et al* (1991).

The inhalation exposure route is considered to be unimportant and the cancer risk from inhalation at a concentration of 100mg/kg is negligible. Concentrations considerably higher than this could be tolerated for response levels.

It should be recognised that Cr (VI) is generally unstable in the environment and will be usually expected to transform readily to Cr (III) except in some specific situations such as high concentrations of Copper-Chrome-Arsenate timber preservatives.

**Cobalt**

Cobalt has similar properties to iron and nickel. Its minerals have been used as colouring agents for pottery, glass and jewellery for several thousand years, continuing to the present day. More recent industrial uses include the production of ‘hard metal’ cutting and drilling tools (with tungsten carbide), and the manufacture of high-strength, specialised alloys with widespread applications, including surgical prostheses. It is found at 1 - 40 mg/kg in soils and in variable amounts in plants and animals. Cobalt is a component of vitamin B12 which is essential for the production of red blood cells. Humans obtain all their vitamin B12 from dietary sources. Total background exposure for adults from food, air and water is about 1µg/kg/day.

Gastrointestinal absorption ranges from 18 - 97% depending on the form of cobalt and nutritional status. Absorption is increased in iron deficiency states. It is widely distributed in organs and tissues and crosses the placenta in pregnant animals. Inhaled cobalt is deposited in the lung and cleared with a long half-life. Elimination of absorbed cobalt is mainly via urine (60 - 70%), and the terminal half-life for clearance from the blood in humans has been estimated at about two years.

Occupational inhalational exposure to cobalt has been associated with hard-metal pneumoconiosis and occupational asthma. Lung function impairment has been reported following exposure to air levels of 0.007 - 0.893 mg cobalt/m3 but most studies have been confounded by the presence of other metals including tungsten

and arsenic. However, lung disease has been identified in particular occupations where cobalt appeared to be the only metal present. Cardiomyopathy and thyroid function changes have also been reported following inhalational exposure.

Cobalt sulfate added to beer as a foam stabiliser was associated with cardiomyopathy in several studies from North America and Europe in the 1960s. Effects were seen from 0.04 mg cobalt/kg/day but high alcohol consumption and poor nutritional status confound interpretation. Cobalt (generally 0.5 - 1 mg/kg/day) has been used therapeutically for the treatment of some forms of anaemia. Skin contact causes dermatitis in sensitive people.

IARC classify cobalt and cobalt compounds as group 2B: possibly carcinogenic to humans. This is based on sufficient evidence for carcinogenicity of metal powder and cobalt oxide in animals, and inadequate or limited data for other salts and humans. Cancer was seen locally following injection or intra-tracheal instillation in animals. These routes are not considered pertinent for environmental exposure and cancer is not taken as an end-point for the present risk assessment.

The available data are inadequate to derive a specific PTDI with confidence. However, a PTDI range of 1 - 5 µg/kg/day is derived based on a surrogate LOAEL of 0.5 mg/kg/day from human therapeutic studies, and a combined safety factor range of 100 - 500. A health investigation level for soil of 100 mg/kg is recommended based on total exposure from soil contributing a fraction of the estimated background intake of cobalt from food.

**Copper**

Copper (Cu) is a common element in the earth's crust and an essential trace element for normal growth and development. It has valencies of 0, + 1, + 2 and less frequently + 3 and + 4, although it mainly appears in the divalent form.

Its concentration in soil averages from 2 to 128 mg/kg. It is used in the production of copper wire and wares, copper pipes, alloys (Cu - tin as bronze and Cu - zinc as brass) and pesticides (e.g. copper oxides, copper sulfate, and copper chrome arsenate).

Young children especially those younger than one year, are the most sensitive groups to copper toxicity. However, since soil intake by children younger than one year is generally negligible (Sheehan et al 1991), the target group selected for health risk assessment should be based on the actual or anticipated occupants of the site (Soong & Emmett, p 242 - 3). For setting the HIL the 2 year old child will be considered the receptor.

The problems of setting a health investigation level for copper stem from the absence of an identified NOAEL for Cu, a small margin of safety represented by the LOAEL to daily intake ratio and a large variability in the Cu concentration in drinking water associated with the use of copper piping in domestic water supplies (Soong and Emmett, 1993).

The following assumptions are used:

* Long-term intake of 0.17 mg/kg bw/day is considered as a safe level (Sloof et al 1989). Using 0.17 mg/kg/day as the TDI for a two year old child weighing 12 kg, the 'tolerable' intake of Cu from all sources is calculated to be 2.04 mg/day.
* Daily copper intake from Australian food by a two year old child is estimated to be 1.1 mg/day, based on the 95% percentile energy intake (National Food Authority 1992). Normal daily copper intake from water by a two year old child is estimated to be 0.05 mg, based on an intake of 1 litre of water containing 0.05 mg Cu/L.
* The 'allowable' intake of Cu from soil is then 2.04 mg - 1.15 mg (food and water) = 0.89 mg Cu/day.

Given an intake of 100 mg of soil/day, an intake of 0.89 mg Cu/day will result from a soil concentration of 8 900 mg/kg. Applying a safety factor of 10 to provide a margin of safety as there is a likelihood of greater intake of Cu from drinking water in many households (and rounding the number up) gives a HIL of 1 000 mg/kg for a standard residential exposure scenario.

The figure of 1 000 mg/kg as a preliminary health investigation level for Cu needs to be applied with caution to all sites as the general level of copper from drinking water is likely to be raised in homes with copper pipes or having a bore water supply. The health response level for Cu in the soil will need to be determined on a site-specific basis taking into account intake from other sources.

**Cyanides (free and complexed)**

Toxicity of free cyanides is well described but data on the toxicity of complexed cyanides is limited. The evidence suggests that the acute toxicities of ferri- and ferrrocyanide complexes are low with their toxicity dependent on any release of free cyanide.

Following the publication of the paper by Turczynowicz (1993) in the Proceedings, a review of cyanide was received which established a TDI of 0.012 mg/kg/day for cyanide by WHO (1993) based on a study using free and dissociable potassium cyanide in solution rather than complexed cyanides.

In determining the soil concentration of free and dissociable, or complexed cyanides it is important that the appropriate analytical procedures are employed. The technique for measuring free and dissociable cyanides also measures thiocyanates. The reader is referred to Appendix IV of the Gasworks Sites chapter (Turczynowicz, 1993) in the Proceedings of the Second National Workshop.

Based on a subchronic study demonstrating a NOAEL for complexed cyanides of 25 mg/kg/day and applying a safety factor of 1 000, a TDI of 0.025 mg/kg/day was derived (Turczynowicz, 1993).

Assuming a soil ingestion rate of 100 mg/day for the child then a health investigation level of 500 mg/kg complexed cyanide is recommended for a standard residential exposure scenario. (Turczynowicz, 1993, p 284)

Under certain chemical conditions the generation of hydrogen cyanide gas may occur and this should be considered in site assessments. Current exposure models are insufficient to quantify gas generation and exposure in the breathing zone of confined or open areas and ambient air monitoring may need to be considered.

This HIL has been developed in the context of the assessment of gasworks sites where the cyanides are considered to be predominantly in the form of complexed cyanides. Typically one form of cyanide predominates. There is insufficient information to provide a general HIL for mixtures of free and complexed cyanides where more than a few percent of the total cyanides are as free cyanides: appropriate site-specific adjustments will need to be made. The nature of the cyanides on a site must be assessed. If the cyanides on a site are predominantly free cyanides (eg an electroplating site) the HIL derived from the TDI of 0.012mg/kg/day in WHO (1993) should be used. This HIL derived for free cyanides is 250 mg/kg, calculated taking 13.2 kg as the body weight of a 2.5 year old and allowing 20% of the TDI from exposure to contaminated soil.

**DDT**

At the second workshop it was recommended that the existing ADI be reviewed by the NHMRC Committee on Toxicity and an Australian ADI be set for the purpose of establishing a Health Investigation Level. That review led to an NHMRC ADI of 0.002 mg/kg/day. The exposure routes of inhalation and dermal absorption were thought to make only a very small contribution to exposure from contaminated soil and the soil HIL was calculated based on ingestion exposure. Background exposure to DDT was estimated.

The level of 200 mg/kg was recommended as a suitable investigation level for human health for a standard residential exposure scenario. DDT has been classified as a Group 2B carcinogen by IARC. Plant and poultry uptake warrant consideration as these may be relevant exposure pathways. DDT breaks down to DDE and DDD.

**Lead**

The environment is pervasively contaminated by lead, particularly in urban areas where sources of lead are densely concentrated, but rural and remote areas may also be contaminated (Maynard, 1991).

Two approaches for setting investigation levels for lead were utilised and the results compared. Based on the WHO PTWI for lead of 25 µg/kg/wk for children and allowing that soil may contribute 52% of the PTWI an HIL of 306 mg/kg was calculated. Estimates of exposure to lead from other sources such as air, food and drinking water were made. The diet is generally considered to be the largest component of background exposure to lead. It has been estimated that a child of 2

years on average ingests 115 - 205 µg/wk. In deriving the HIL, the value for soil intake/day was 80 mg/d (note: most other calculations assumed this to be 100 mg/day) and the child's body weight was 13.2 kg.

The other approach is based on the notion of a safe blood lead level - ie, using a level of concern, with or without a safety factor, or some existing achievable basal blood lead value and then applying uptake and biokinetic modelling to obtain soil criteria. Higher values for acceptable levels of lead in soil were generated by this approach and the more conservative estimate of the HIL recommended.

The HIL set for a standard residential exposure scenario for lead is 300 mg/kg.

**Manganese**

Manganese is the tenth most abundant element in the earth's crust and often is associated with iron in the natural environment. Compounds of manganese are naturally occurring and the general population is exposed to low levels of manganese by ingesting food and drinking water, inhaling ambient air and soil contact. Manganese is an essential trace element required for functioning of many enzyme systems including pyruvate carboxylase, phosphatase, and lipid and mucopolysaccharide synthetases.

Manganese deficiency interferes with normal growth, bone formation and reproduction in a number of animal species. Excessive exposure to manganese is commonly by inhalation in the occupational setting. Symptoms of chronic excess exposure are neurological. Mild cases show mental instability; moderate intoxication causes clumsiness, speech disorders, difficulty in walking; and severe poisoning cases show tremors, gait disturbance, mask-like face, ataxia and other motor disturbances. Neurological effects also result from oral ingestion of manganese compounds.

There is no clear toxicity threshold for manganese in humans and dietary intakes range from 2 to 50 mg/day. The US EPA has set a chronic oral reference dose of 0.14 mg/kg/day. It was assumed that dietary intake was 2.5 mg/day, drinking water contained 0.01 mg/L and the child's soil intake was 100 mg/day. A HIL of 1 500 mg/kg is proposed for a standard residential exposure scenario based on calculations that this level would result in an incremental exposure of approximately 10% over an adequate Mn intake received from food and drinking water. This value does not include manganese present as the organic compounds MCT and MMT which need to be assessed separately.

**Mercury**

Mercury has three valency states (Hg0, elemental; Hg2+, mercuric; and Hg+, mercurous) and is found in the environment in the form of various inorganic and organic complexes and as the elemental metal. Toxicity of mercury depends on the form with alkyl > (aryl and alkoxyl) > inorganic in decreasing order of toxicity. The

effects of methyl mercury are on the CNS and prenatal stages are most susceptible to methyl mercury exposure. The Joint FAO/WHO (JECFA, 1988) has established a Provisional Tolerable Weekly Intake (PTWI) of 300 µg total mercury of which no more than 200 µg should be methylmercury. The USEPA has set a reference dose (RfD) for chronic oral exposure at 0.3 µg/kg/day, which is a similar estimate of acceptable exposure.

Dietary intake and exposure to mercury from dental amalgams appear to be the major sources of mercury exposure for the general population, contributing an estimated 50% of the PTWI. If 20% of the PTWI is allowed for exposure to contaminated soil, the health-based soil investigation level may be set at 15 mg/kg for inorganic mercury for a standard residential exposure scenario.

The health-based soil investigation level for a standard residential exposure scenario could be set at 10 mg/kg methyl mercury where the site is small and bioconcentration of organic mercury in an aquatic food chain is not feasible. Where large scale contamination of lakes, rivers or bays is under assessment much more rigorous standards would be needed to protect against bioaccumulation.

**Nickel**

Nickel is a natural part of the environment. Levels of nickel in soils range from about 4 to 80 mg/kg. The general population is exposed to nickel via food, drinking water, smoking, air and by skin contact with soil, water, and metals containing Ni or plated with nickel.

Sensitisation to nickel is clinically important with 10 - 15% of females being sensitised. It is apparently the commonest allergen in boys and girls.

The US EPA has derived a chronic oral RfD based on a chronic feeding study in rats. The NOAEL was 5 mg/kg/day and an uncertainty factor of 300 was applied to set the RfD at 0.02 mg/kg/day.

The contribution allowed from exposure to contaminated soil was 30% of this dose. For non-dermatitis related effects a level of 1 440 mg/kg would not be expected to cause adverse effects.

Elicitation of an allergic reaction in a sensitised person will occur at concentrations lower than concentrations required for induction of allergic contact dermatitis in non-sensitised individuals. A soil HIL of 600 mg/kg for a standard residential exposure scenario should apply to prevent the elicitation of allergic contact dermatitis in the sensitised population, and this will also be protective for non-sensitised persons (Turczynowicz & Sabordo, 1995).

**Phenol**

Exposures to phenol by all routes - soil ingestion, dermal absorption and inhalation -were considered in deriving the HIL for phenol. Based on the US EPA oral reference

dose for phenol, 0.6 mg/kg/day, and allowing 25% of this dose to come from soil a HIL of 8 500 mg/kg for a standard residential exposure scenario was calculated.

It is recommended that further modelling be undertaken to establish a phenol level in soil based on aesthetic and/or ground water pollution potential for Australian conditions. The low odour thresholds for phenols may demand investigation levels below the HIL on aesthetic grounds. Skin irritation may possibly occur at as yet unidentified lower concentrations than the proposed HIL. Given the physico-chemical properties of phenol it is unlikely to persist in high concentrations for long durations unless there is a continuing source.

If phenols are detected using a generic testing method, then further information will be required due to significant differences in toxicity among the substituted phenolic compounds. For example, there is a 1 000 fold difference in US EPA oral reference doses between phenol and 2,6 - dimethylphenol (where phenol is 1 000 times less toxic than 2,6 - dimethylphenol).

**Polychlorinated Biphenyls (PCBs)**

PCBs are thermally and chemically very stable and comprise a group of 209 possible discrete compounds. In Australia, PCBs were mainly used in electrical components as insulators, heat transfer or hydraulic fluids. They are persistent in the environment and accumulate in biological systems and biomagnify in the food chain.

Background exposure is mainly from food although intake from this source has been declining since restriction of PCBs in the 1970s and would seem to be negligible based on recent Australian Market Basket Survey results.

The WHO has not set an ADI or TDI for PCBs. The US EPA suggests 0.0001 mg/kg/day is a minimal risk level based on a NOAEL of 0.0105 mg/kg/day for neonatal toxicity in monkeys. Based on a NOAEL of 0.0125 mg/kg/day for Arochlor 1016 and a safety factor of 100 a TDI of 0.0001 mg/kg/day was derived. When soil is assumed to be the only source of PCB exposure and allowing the bioavailability of PCBs in soil to be 30% oral, 10% dermal and 50% by inhalation route (US EPA, 1990), a guidance value of 18 mg/kg was obtained.

The HIL proposed is 10 mg/kg for a standard residential exposure scenario which incorporates an additional uncertainty factor of almost 2 due to uncertainty in the NOAEL on which the TDI is based.

**Total Petroleum Hydrocarbons**

Total Petroleum Hydrocarbons (TPHs) are a diverse range of chemicals derived from crude petroleum with a ubiquitous distribution within our community. They are frequently encountered on contaminated sites and their particular physico-chemical properties result in the potential for exposure from air, (ground)water and soil.

Vehicle or heating fuels are the most commonly encountered TPH contaminants and, in common with many solvents, are a mixture of hydrocarbon compounds of variable proportions. Vehicle fuels usually include benzene (in petrol) and additives (anti-knock lead, scavenging detergents, anti-oxidants) according to the grade and type of fuel.

The risk assessment of mixtures such as TPHs involves difficulties due to: the limitations in data about toxicology, environmental fate and transport; the absence of standard analytical procedures; and problems with the modelling of exposures to volatile compounds. The Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG,1997) has reviewed data about toxicology, fate and transport for fraction-specific TPHs and has established reference doses for inhalation and ingestion of TPHs based on equivalent carbon fractions. These data have been reappraised and appropriate modelling used to derive health-based investigation levels for this monograph.

TPHCWG (1997) proposes two approaches used in a hybrid framework. The first approach examines the presence of indicator chemicals which are carcinogenic substances. This generally includes substances such as benzene and polycyclic aromatic hydrocarbons such as benzo (a) pyrene which, if detected, require an appraisal using other HILs. If indicator chemicals are not present then the second stage is used. The second stage is the TPH fraction stage which assigns toxicity criteria to well-defined fractions based on the available toxicity data for constituents, mixtures, solvent streams and whole product.

The absence at this time of a standard Australian method for dealing with carcinogenic soil contaminants and the complexities of modelling multiple exposure pathways for volatile and environmentally mobile TPHs currently preclude the establishment of HBILs for the TPH fractions that exhibit complex environmental behaviour pathways and/or are carcinogenic. In the first instance HBILs have been derived for >C16-C35 aromatics, >C16-C35 aliphatics and >C35 aliphatics based on their limited environmental mobility and low volatility. These are 90 mg/kg for >C16-C35 aromatics, 5600 mg/kg for >C16-C35 aliphatics and 56000 mg/kg for >C35 aliphatics.

Further work is being undertaken to derive HBILs for the remaining TPH fractions that exhibit more complex environmental behaviours and/or are carcinogenic.

**Zinc**

Zinc is one of the most common elements in the earth's crust. It is found in the air, soil, and water and is present in all foods. Zinc is an essential element needed by the body in small amounts. Either too little or too much zinc can be harmful to health.

Zinc is one of the most abundant trace metals in humans. It is found in nearly all tissues and tissue fluids, and is a co-factor in over 200 enzyme systems.

The Recommended Daily Allowance (RDA) for zinc is 15 mg/day for men and 12 mg/day for women. The Minimal Risk Level (MRL) is an estimate of daily human

exposure to a dose of a chemical that is likely to be without appreciable risk of adverse non-cancerous effects. An MRL of 0.3 mg/kg/day has been derived for exposures of intermediate duration. Due to a lack of adequate long-term studies in humans or animals, the intermediate MRL of 0.3 mg/kg/day has been adopted as the chronic MRL.

Considering the direct exposure route of soil ingestion for a 2 year old child, body weight 10 kg and daily soil ingestion rate of 100 mg/day, the allowable level of zinc in soil would be 7 000 mg/kg. Direct dermal absorption of zinc from soil would be very low. Indirect routes of uptake such as via water contamination or uptake into fruits and vegetables have not been evaluated. It is recommended that the health-based investigation level for zinc in soil be set at 7 000 mg/kg for a standard residential exposure scenario.

**Comparison of assumptions in deriving HILs**

A range of conservative assumptions have been used in developing the Health Investigation Levels. The variations detailed below are considered, given the nature and magnitude of the conservative assumptions, not to have practical significance nor to require adjustment of the Health Investigation Levels. Hereafter, standard assumptions will be used unless there is substance- specific information to justify alternative values.

* Soil ingestion has been assumed to be 100 mg/day, except in the case of lead where 80 mg/day was used. However, the lead HIL includes added conservatism by using an exposure component, the lead PTWI, which is considered not to result in a net increase in body burden of lead.
* Bioavailability was mostly assumed to be 100%, although this varied for organochlorines and PCB where various availabilities were given to different routes based on data.
* All exposure routes were considered but in most cases oral exposure predominated. For Ni and Cr(VI) skin sensitivity reactions formed the basis of the HIL. Dermal and inhalational uptake was significant for organochlorines, PCBs and phenol.
* The percentage of the TI allowed from exposure to contaminated soil has varied more than other assumptions. For lead and PCB 50% of PTWI was allowed, for arsenic 40% and in the range of 15 to 25% for cadmium, phenol, complexed cyanides, beryllium and mercury. These variations are largely explained by the contributions of other background factors, especially food.
* A weight of 13.2 kg was used generally for the weight of the target child, but occasionally 12 kg and, for zinc, 10 kg.