National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April 2011 National Environment Protection (Assessment of Site Contamination) Measure April

Schedule B4

GUIDELINE ON

Site Specific Health Risk Assessments

The following guideline provides general guidance in relation to health risk assessment methodology in the assessment of site contamination.

This Schedule forms part of the National Environment Protection (Assessment of Site Contamination) Measure as varied 2011, and should be read in conjunction with that document, which includes a policy framework and assessment of site contamination flowchart.

This Schedule replaces Schedule B4 to the National Environment Protection (Assessment of Site Contamination) Measure 1999.

The National Environment Protection Council (NEPC) acknowledges the contribution of the National Health and Medical Research Council (NHMRC) and enHealth to the development of this Schedule.

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1 Introduction

1.1 Objectives

The objectives of this revised Schedule B4 guideline to health risk assessment methodology of the National Protection (Assessment of Site Contamination) Measure as varied 2011 are to:

- establish the fundamental principles of risk assessment as they relate to contaminated land decision making in Australia, which are protective of human health
- provide a framework for policy making and undertaking risk assessments that is transparent, logical and compatible with current scientific principles and practice
- provide a basis for deriving health investigation levels (HILs) presented in Schedule B7 guideline on health-based investigation levels
- provide a guide for deriving site-specific criteria which may act as clean-up levels.

The intended audience for this Schedule includes policy makers, risk practitioners, and regulators.

1.2 Overview of Schedule B4

This document provides an approach to conducting site-specific health risk assessments at contaminated sites. Due to the complexity of the risk assessment approach, a standard approach to all sites is not practicable and site-specific considerations will often need to be accounted for. This document is intended to provide a guide to assist the decision making process and, where possible, risk assessment procedures are recommended. The principles and guidelines in this Schedule are intended to assist in determining whether or not remediation is required at a site given the proposed land use.

1.3 Introduction to quantitative health risk assessment in contaminated land decision making

The Australian enHealth Council (enHealth 2010) defines risk assessment as 'the process of estimating the potential impact of a chemical, physical, microbiological or psychosocial hazard on a specified human population or ecological system under a specific set of conditions and for a certain timeframe'.

Quantitative (health) risk assessment is a step-wise process used to inform and assist the contaminated land decision-making process by modelling the dose or exposure of humans to observed site contamination. It is used to estimate, in a way that is adequately protective of health, the potential for site contamination to have a significant adverse effect on the health of those potentially exposed to it (referred to as exposed populations). This is achieved by modelling the dose that an individual may receive through incidental exposure to contaminated soil and/or water as a result of everyday activities. This estimated dose can then be compared against doses that are considered to result in no observable adverse impact to health, as published by authoritative bodies and health protection agencies.

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The quantitative risk assessment process, in the context of this guidance, is primarily designed to evaluate the long-term or chronic risks to exposed populations from contamination in the environment. Short-term or acute risks can be dealt with using similar means; however, normally this is not necessary because contamination severe enough to pose acute risks is sufficiently obvious that a complex assessment is not required.

This guidance is not intended to be used to assess the risks from occupational exposure to substances that may occur in an occupational setting or workplace. These risks are dealt with under occupational health and safety legislation and associated guidelines.

The quantitative risk assessment process can be adopted in reverse to estimate 'tolerable' concentrations of contaminants based on knowledge of site-specific factors and the nature of everyday activities that might occur on the site, currently, or after redevelopment to another land use. These concentrations can then be used to define management requirements or as remediation targets or site-specific clean-up criteria.

1.4 Site assessment process and terminology

The site assessment process for contaminated land is described in Schedule A of the National Environment Protection (Assessment of Site Contamination) Measure. Once the need for an assessment is triggered, a preliminary site investigation should be conducted using guidance outlined in Schedule B2 The objectives of the preliminary investigation are to identify the likely nature and extent of contamination at, and adjacent to, the site, and to provide a description of the physical setting.

Where there is a potential for soil and groundwater contamination to be present, then a detailed site investigation should be conducted. This involves the collection of soil, groundwater and soil vapour samples for field and laboratory analysis, and should be conducted using appropriate guidance such as that outlined in Schedule B2 and Schedule B3.

The laboratory results are then compared against appropriate guidance values, such as HILs.

1.4.1 Health investigation levels

HILs are defined as 'the concentration of a contaminant above which further appropriate investigation and evaluation will be required'.

The HILs are presented in Schedule B7. Levels marginally in excess of the HILs do not imply unacceptability or that a significant health risk is likely to be present. Similarly, levels less than the HILs may not imply acceptability or that a significant health risk does not exist for a sensitive sub-population (for example, people who have immuno-suppression or illness, people with pica (relatively common in some groups with severe or profound intellectual disability). Subject to an appropriate investigation and assessment process, a decision not to take further action or to take further action may be justifiable.

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HILs are not intended to be clean-up levels. The decision on whether clean-up is required and, if so, to what extent, should be based on site-specific assessment. Health risk assessment is one aspect of making the decision; however, other considerations such as practicality, timescale, effectiveness, cost and durability are also important.

1.4.2 Conceptual site model

In order to commence a risk assessment a preliminary understanding of the site and potential issues is necessary. Factors to be considered include:

- the typical and maximum concentrations of contaminants on site
- the vertical and horizontal distribution of the contaminants
- the physical and chemical properties of the contaminants and their likely mobility in the environment
- the people who may be exposed to the contaminants
- the means by which exposure could occur, and the frequency of exposure.

The understanding of the site is referred to as a conceptual site model. It may be presented by means of diagrams, tables and explanatory text.

1.4.3 The tiered approach

The human health risk assessment process for contaminated land is undertaken in stages or 'tiers' involving progressively more detailed levels of data collection and analysis. In this guidance, the tiers are referred to as Tier 1, Tier 2 and Tier 3. The approach provides for assessment at a level of complexity that is appropriate for the problem under consideration; the degree of health protection achieved is equal at each tier. As the amount of data and assessment detail increases and the conceptual understanding of site conditions (that is, the conceptual site model) is refined, the level of uncertainty decreases. In turn, the precision of the risk assessment process may be reduced.

A risk assessment progresses from Tier 1 to Tier 2 when uncertainty and risks at Tier 1 may be unacceptable, and further assessment is needed. Progression from Tier 2 to Tier 3 is similarly driven. Tier 3 provides more detailed and specific focus on risk-driving factors. It should be noted that the activities within the tiers may vary depending on the scale and complexity of the project.

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2 The Australian risk assessment framework

2.1 The enHealth framework

The Australian enHealth Council (enHealth) provides national leadership on environmental health issues, coordinates national policies and programs, and provides a pivotal link between international and environmental health stakeholders in Australia. It is also responsible for the implementation of the National Environmental Health Strategy.

In 2002, enHealth developed *Guidelines for assessing human health risks from environmental hazards* to provide a national approach to undertaking environmental health risk assessments, reprinting these in 2004 and updating in 2010. The guidelines present a general environmental health risk assessment methodology which has been adopted nationally to evaluate risks and establish standards for the protection of human health and the environment.

With respect to assessment of risks from contaminated land, the guidelines draw on other documents as follows:

- *Assessment and management of contaminated sites* (ANZECC & NHMRC 1992)
- National Environment Protection (Assessment of Site Contamination) Measure (NEPC 1999)
- SA Health Commission contaminated sites monograph series 1991, 1993, 1996 and 1998 (El Saadi & Langley 1991; Langley et al. 1993, 1996a, 1996b)
- Environment Protection and Heritage Council, *Proceedings of the 5th national workshop on the health and environmental assessment of site contamina*tion (Langley et al. 2003)
- *National Environment Protection (Ambient Air Quality) Measure, Report of the risk assessment task force* (NEPC 2000)
- National Research Council, *Science and decisions: advancing risk assessment* (National Research Council 2008)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health evaluation manual, Part A* (US EPA 1989)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health evaluation manual, Part B, Development of risk-based preliminary remediation goals* (US EPA 1991)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health evaluation manual, Part C, Risk evaluation of remedial alternatives* (US EPA 1991)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health Evaluation Manual, Part D, Standardized planning, reporting and review of Superfund risk assessments* (US EPA 1998)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health evaluation manual Part E, Supplemental guidance for dermal risk assessment* (US EPA 2004)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health evaluation manual Part F, Supplemental guidance for inhalation risk assessment* (US EPA 2009)
- US EPA, *Risk assessment guidance for Superfund, volume I, Human health evaluation manual, Supplement to Part A: Community involvement in Superfund risk assessments* (US EPA 1999)
- World Health Organisation, *IPCS Risk assessment terminology*, Harmonisation project document no. 1 (WHO 2004)
- World Health Organisation, *Principles of characterising and applying human exposure models*, Harmonisation Project Document no. 3 (WHO 2005)

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- World Health Organisation, *Part 1: Guidance document on characterising and communicating uncertainty in exposure assessment, and Part 2: Hallmarks of data quality in chemical exposure assessment*, Harmonisation project document no. 6 (WHO 2008)

The enHealth guidelines adopt a framework for evaluating risks that was developed by and for environmental health agencies, including the World Health Organization (WHO) and the United States Environmental Protection Agency (US EPA).

The framework comprises the following components:

- Issues identification
- Hazard assessment (often called toxicity assessment)
- Exposure assessment
- Risk characterisation
- Risk communication and management.

2.2 Risk assessment framework for contaminated sites

The risk assessment process for contaminated land is intended to achieve the following objectives:

- to determine tolerable levels of contaminants in soil and groundwater that are protective of public health and ecosystems
- to provide a consistent methodology for appraising and recording public health risks at contaminated sites
- to establish the baseline risks and determine whether site remediation is required
- to enable the comparison of potential health impacts of various remedial technologies.

The contaminated land risk assessment framework was originally outlined by NEPC in the Assessment of Site Contamination NEPM (NEPC 1999). The major difference between the framework originally outlined by NEPC and that of enHealth is the first step in the assessment process, which in enHealth (2010) is referred to as 'issues identification'. The term 'issues identification' is intended to establish the context for the risk assessment by a process of identifying the concerns that need to be addressed, such as 'what is causing the identified concern?', and 'why is the concern an issue?' Inclusion of the 'issues identification' as an explicit need is consistent with recent US conclusions that increased attention to scoping and planning of risk assessment is necessary (National Research Council 2008).

In this revised Schedule, the enHealth framework is adopted with minor additions to clarify the setting of the risk assessment framework in the contaminated land assessment process. The framework followed in this Schedule is illustrated in Figure 1. Detailed guidance for each stage of the process is provided in the body of this Schedule.

The risk assessment should be fully documented in order to ensure transparency, consistency in decision making and ease of understanding by interested parties. This means it should be supported with references to policy, scientific literature and other sources, including expert opinion. A risk assessment should also be subject to review and revision should significant new information become available.

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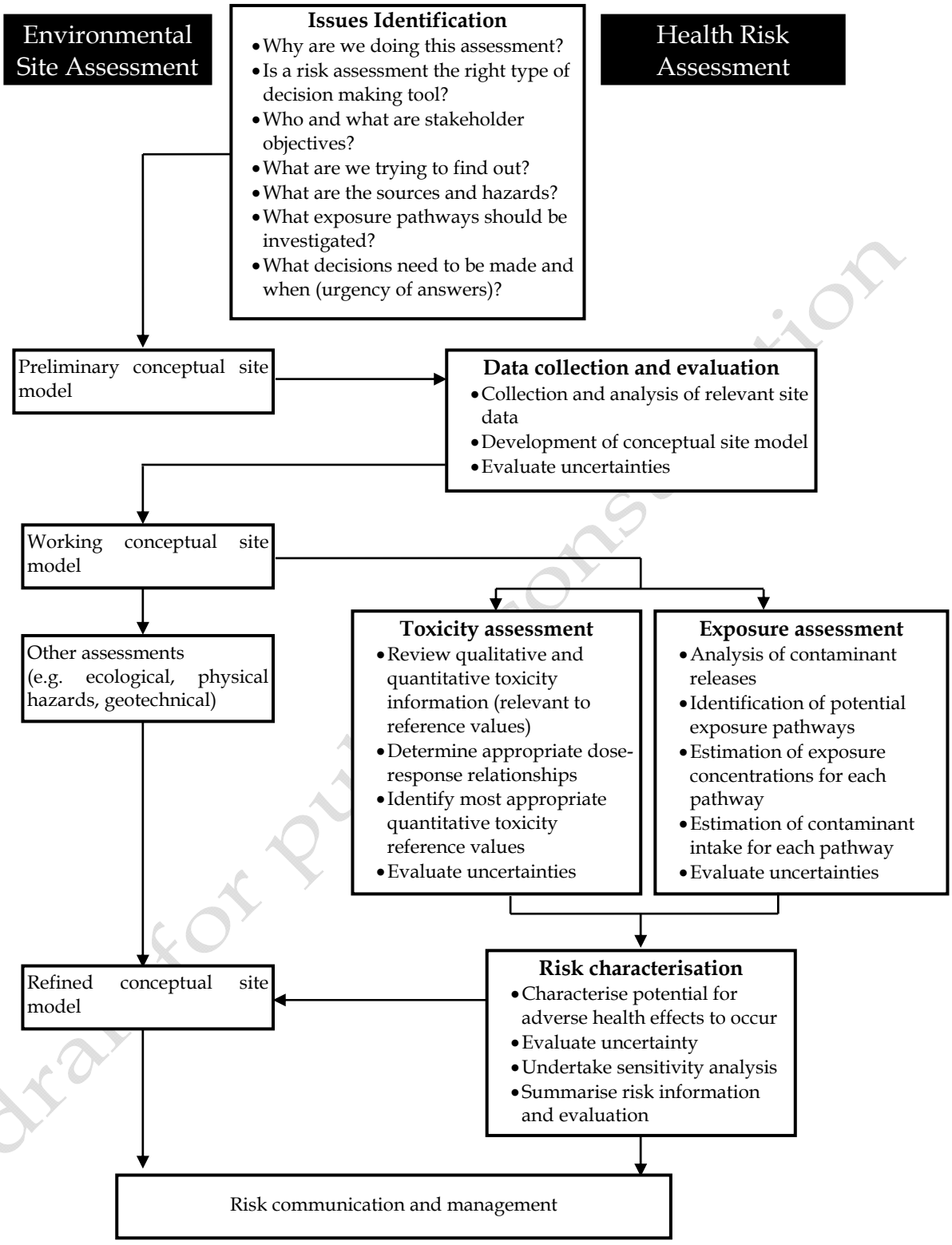

Figure 1. Risk assessment framework for contaminated sites

2.3 Fundamentals of the risk assessment approach

2.3.1 Issues identification

The issues identification stage of a contaminated land risk assessment is fundamental to the production of a useful output. Issues identification is a process of communication between stakeholders in the project, and its scope and complexity depends upon the scale of the project and the issues being dealt with. Issues identification covers both a planning and scoping phase and a problem formulation stage.

2.3.1.1 Planning and scoping

In the planning and scoping phase, a team of decision makers, stakeholders and risk assessors identifies the issue (or concern, problem or objective) to be assessed and establishes the goals, breadth, depth and focus of the assessment. The primary product of planning and scoping is a statement, with an explanation of why the assessment is being performed and what it will include and exclude, that is, how comprehensive it will be (NRC 2008).

Stakeholders in a contaminated land risk assessment are likely to be a subset of the following groups:

- regulators (environment protection agencies)
- local government (potentially several departments, e.g. planning, development control, road engineering, drainage, traffic, ecological issues)
- state and territory departments of health
- landowners
- tenants, land management companies
- local residents
- occupants of neighbouring properties
- water, sewerage, electricity, gas and telephone utilities
- other interested parties (e.g. non-governmental organisations, local interest groups)
- local politicians
- consultants.

The planning and scoping phase should be undertaken before work begins on the risk assessment. The steps recommended are:

- identify the stakeholders
- decide which of them it is appropriate to consult at this stage of the project.

Frame the answers to the following questions and discuss them with the stakeholders:

- Is a risk assessment the right type of decision-making tool?
- What is the issue that the proposed risk assessment is considering?
- Why is a risk assessment necessary?
- What do we want to find out?
- What are the hazards posing a risk?
- The source of the risks?
- What exposure pathways should be investigated?
- Who might be exposed and who will not be exposed?
- What decisions need to be made?

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- Timing of the assessment (urgency of answers)?
- Level of complexity/detail of risk assessment needed?

With the outcome of the consultation known, determine the objectives for the risk assessment. Where not all the stakeholders have been consulted, consideration of their likely objectives should be included.

2.3.1.2 Problem formulation

The problem formulation stage is where assessors and managers discuss how to conduct a risk assessment that covers the matters identified in the planning and scoping stage. Two critical products of the product formulation stage are a conceptual model that explicitly identifies the stressors, sources, receptors, exposure pathways and potential adverse human health effects that the risk assessment will evaluate, and an analysis plan (or work plan) that outlines the analytic and interpretive approaches that will be used in the risk assessment (NRC 2008).

2.3.2 Exposure pathways

The fundamental concept of risk assessment is that there must be an exposure pathway linking the source of contamination and the exposed population. Where this linkage exists, an assessment of the nature and significance of the exposure pathway is required to determine the level of risk.

2.3.3 Conceptual site model

A key concept behind all risk assessments is the definition of a suitable conceptual site model specific to each site. A conceptual site model is a site-specific description of the exposure pathway elements. The conceptual site model describes the source(s) of contamination, the pathway(s) by which contaminants may migrate through the various environmental media, and the populations (human or ecological) that may potentially be exposed.

A detailed conceptual site model should include information on the following:

- the contaminants: concentration, distribution and media in which they occur (soil, water, sediment or air) on and off the site as relevant
- physical characteristics of the environment: soil type, porosity, vadose zone thickness, groundwater gradient and velocity and hydraulic conductivity of the saturated zone on and off the site as relevant
- characteristics of the exposed populations: exposed populations may be people residing or working at the site or off-site areas, future occupiers of the site after redevelopment, or environmental populations on and off the site such as ecosystems in receiving environments such as natural surface waters.

A conceptual site model is generally a written description of the site that is accompanied by a schematic, graphical interpretation that depicts what is known or has been inferred about the site. It can also be presented as a flow diagram, as shown by the example in Figure 2.

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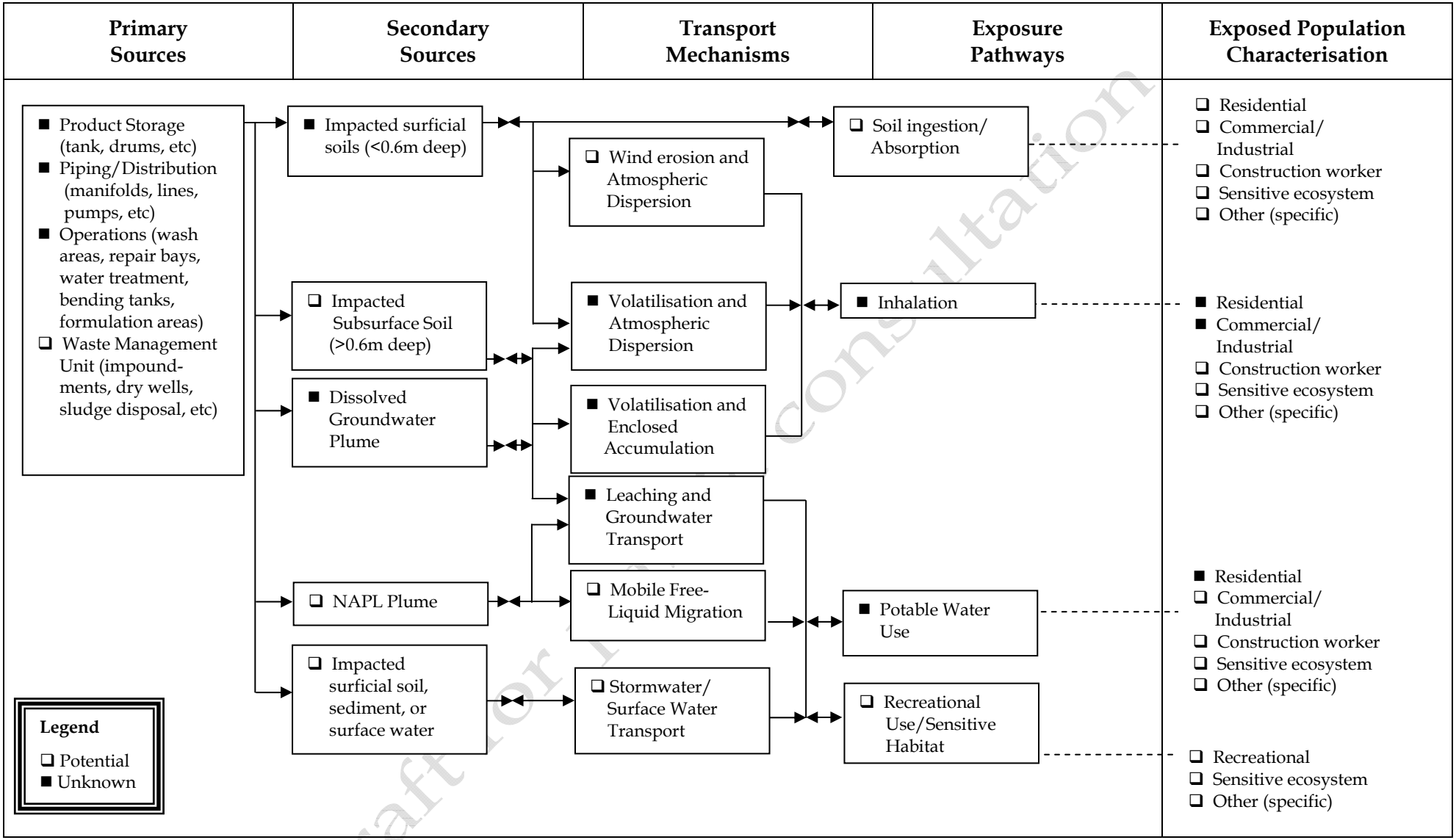

Figure 2. Conceptual Site Model flow diagram (modified from ASTM, 1995b)

A conceptual site model can inform the development of and be incorporated into the detailed scope for a human health risk assessment such as is shown in figure 3. This example deals with the scoping of a risk assessment for hazardous air pollutants (HAP) and persistent, bioaccumulative and toxic pollutants (PBT)

The schema identifies the sources, contaminants of concern (stressors), exposure pathways, potential receptors, and adverse human health effects that the risk assessment will address.

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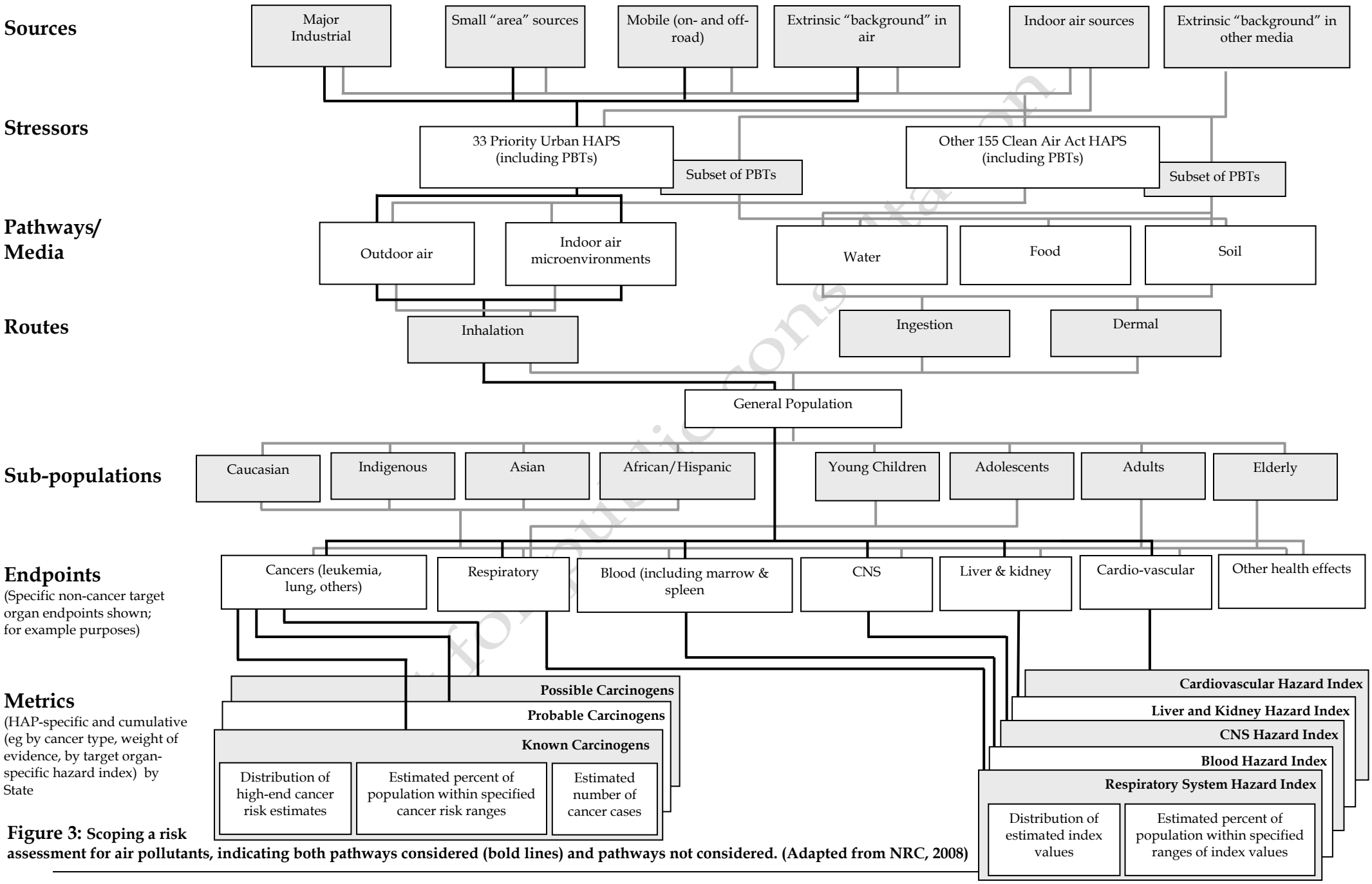

Figure 3: Scoping a risk assessment for air pollutants, indicating both pathways considered (bold lines) and pathways not considered. (Adapted from NRC, 2008)

2.4 The tiered approach

2.4.1 Fundamentals of the tiered approach

2.4.1.1 Tier 1

The Tier 1 (or screening level) assessment is the first stage of assessment at the site. It includes a comparison of known site data with published risk-based guidance levels, such as the HILs. The assessment provides an initial screening of the data to determine whether further assessment is required. HILs are Tier 1 guidance values which are designed to avoid significant risk to most exposed populations under a variety of circumstances (that should be understood). Exceedance of Tier 1 criteria is generally used to define the contaminants that require assessment at Tier 2. An assessment of the significance of exceedances may be necessary where they are marginal or present over a limited area. Where further assessment of contaminants exceeding Tier 1 criteria is not proposed, a clear and transparent explanation should be provided. A Tier 1, screening level, assessment is often bypassed where there are no appropriate risk-based guidance levels (including HILs).

2.4.1.2 Tier 2

A Tier 2 assessment is typically required when one or more contaminants are present at the site at levels that significantly exceed Tier 1 guidance criteria, if there are no appropriate Tier 1 criteria, or if there are unresolved and significant uncertainties identified in the Tier 1 assessment. Tier 2 assessment includes a site-specific risk assessment and the development of site-specific target levels for comparison with site data. Site-specific target levels are derived to be adequately protective of human health, but also to take into account site-specific conditions such as relevant exposure pathway linkages to avoid being unnecessarily conservative. Exceedance of Tier 2 criteria may result in a need for a Tier 3 assessment. As with Tier 1 exceedances, an assessment of the significance of exceedances may be necessary where they are marginal or present over a limited area. If Tier 2 criteria are exceeded, but further assessment (or action) is not proposed, the information and logic used to inform the decision should be documented clearly and transparently.

2.4.1.3 Tier 3

A Tier 3 assessment may be required where exceedance of Tier 2 site-specific target levels is judged to represent a potentially unacceptable risk to human health. The Tier 3 assessment typically focuses on the risk-driving contaminants in more detail, although studies aimed at reducing the uncertainties inherent in the modelling of exposure pathways are also common at Tier 3. This level of assessment may include statistical methods and mathematical modelling to assess the significance of the site contamination. The collection of additional data, such as soil vapour sampling, ambient air sampling, analysis of dust, biological monitoring and additional site investigations may be needed to support Tier 3 assessments in order to reduce uncertainties.

The tiered approach should provide a process for addressing site contamination methodically, with the level of complexity and cost proportional to the significance of the risk. Increased levels of site-specific data reduce uncertainties inherent in the assessment. It is important to note that for a given site the level of protection of exposed populations should remain the same regardless of the tier of assessment conducted.

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2.4.2 General risk assessment assumptions

Risk assessment is a tool to help risk managers make decisions about contaminated sites. Risk assessment should incorporate an appropriate level of health protection such that sensitive exposed populations are adequately protected. Because of the many uncertainties inherent in risk assessment, it is desirable that quantitative risk assessment methods should overestimate risk to some extent. Risk assessment allows for the fact that many variables are uncertain, and provides confidence that the conclusion will be health protective. It is also important, however, that the assessment is not excessively conservative, since this is likely to lead to unnecessary health concerns and to more remediation than is actually needed.

Risk assessors should transparently describe the assumptions and uncertainties used in a risk assessment.

Risk assessors should select exposure model inputs carefully, and consider the reasonableness of the exposure settings when taken together. Some examples of how assumptions may be applied to a model are:

- using an average (where there is sufficient data) and high-end estimate (e.g. upper 95th percentile or maximum) of the contaminant concentrations to represent the source
- choosing an appropriate sensitive exposed population for the conceptual site model, such as young children or pregnant women
- assuming that the behaviour of the sensitive population results in plausible high-end exposures (e.g. long exposure periods, exposure of large areas of the body, high activity rates)
- assuming that exposure to the population may occur by several pathways (e.g. a child who plays in contaminated soil also eats vegetables grown in this soil)
- using fate and transport models (e.g. vapour intrusion) which provide estimates of the amount of contamination that reaches the exposed populations.

Some exposure settings and assumptions (as in Tier 1 assessments) may not be realistic for the site under consideration as they are based on generic assumptions and parameters that are not going to be realistic for all sites. A Tier 2 assessment may be used to produce more site-specific values by amendment of the assumptions to reflect actual site conditions. Where available, data on biodegradation of contaminants and bioavailability of chemicals should be considered (by an appropriately qualified professional), and exposure factors (and assumptions) should reflect the scenarios under consideration. It is not necessary to assume that the HIL assumptions detailed in Schedule B7 prevail under all circumstances; both sites and the exposed populations may be very different from the HIL scenarios and this can and should be accounted for in a Tier 2 or Tier 3 assessment.

2.4.3 Risk assessment endpoints

There are two different approaches to risk assessment which can be referred to as 'forward' and 'backward' assessments.

In a 'forward' assessment, site data are assessed to estimate whether the observed contaminant concentrations pose a potentially significant health risk to exposed populations. Risks are expressed as hazard indices or as increased lifetime cancer risks and these risk estimates are used to support a decision regarding the acceptability of the risk.

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In the 'backward' assessment, the starting point of the assessment is the level of risk or exposure that is deemed to be acceptable for the site. The endpoint is a site-specific target level, which may be used for further assessment or to provide a basis for clean-up.

2.4.4 Deterministic versus probabilistic estimates

A deterministic approach means that variables input to an exposure model are expressed as single values or point estimates which are considered by the assessor to represent the best estimate of the value of the variable. The advantage of this approach is that it is simple and easily understood. Its potential disadvantage is that selection of many point estimates at the upper end of their likely ranges leads to compounding of the uncertainty. Sensitivity and uncertainty analysis are used to overcome this disadvantage and provide increased understanding and clarity on which values are risk-driving; this is itself a useful part of the risk assessment.

Probabilistic techniques can be used to overcome the potential for compounding conservatism and to provide increased understanding of the uncertainty inherent in the assessment results. Monte Carlo analysis and other probabilistic statistical techniques rely on the use of probability distribution functions instead of point estimates to represent the values of variables. A probabilistic exposure model is run over several thousand iterations, and values for each parameter are selected randomly from each distribution at each iteration. The output is also expressed as a probability distribution function.

The advantage of probabilistic methods is that it becomes possible to express the range of potential exposure and risk outcomes. This can aid decision making by increasing the level of understanding around how likely different risk outcomes may be. The disadvantage is that in order to generate meaningful output, a reasonable level of data and information on the shape of the probability distribution inputs is required. Often this information is not available, and in this case incorrect selection of a probability distribution can introduce error. Some variables are linked (for example, body weight and skin surface area) and if not carefully constructed, probabilistic models may be capable of generating some results that are physically impossible. Another disadvantage of probabilistic risk assessments is the difficulty in explaining this complex approach to the stakeholders who are involved in a site.

In summary, probabilistic techniques are generally not practicable for the majority of assessments at Tier 2 on the grounds that there is often insufficient data to support the method. When probabilistic techniques are used, the justification for the probability distribution functions selected as inputs should be clearly given, and dependent variables should be identified and linked.

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3 Data collection and data evaluation

3.1 Data collection

Data collection entails the acquisition and analysis of information about contaminants at a site that may affect human health and which will be the focus of the risk assessment. The purpose of data collection is to gather data that will improve the conceptual site model and hence enable a more site-specific assessment of risk to be made. Data will relate to not only contaminants (that is, the source) but the physical environment in which they are present (i.e the pathways). In some instances, it may also be appropriate to gather additional information about the potential exposed populations.

The elements of the data collection stage are described in more detail in Schedule B2. The general requirements for data collection in site investigation apply equally to data collected for the purpose of risk assessment. In accordance with Schedule B2, the following key components should form the basis of establishing the conceptual site model:

- setting data quality objectives
- establishing a site history
- detailing the site use, or proposed use
- reviewing local geology and hydrogeology
- establishing a sampling strategy
- undertaking appropriate analysis
- coherent presentation of data.

There are a number of issues in data collection which are specific to health risk assessment, and which are discussed below.

3.2 Source variables

There are a number of variables commonly required for health risk modelling, which can be measured on a site-specific basis. Site-specific data are always preferable to the use of literature or model default values, because environmental variables are prone to have large ranges. Choosing a worst-case value to account for uncertainty adds conservatism to the risk assessment. Getting site-specific data permits use of realistic values and provides more confidence in the risk assessment outcome.

3.2.1 Organic speciation

Several organic compounds are commonly analysed as groups of substances, for example, total petroleum hydrocarbons (TPHs), polycyclic aromatic hydrocarbons (PAHs) and phenols. Such tests may be useful for providing cost-effective estimates of the quantity of contaminant present, but are problematic in risk assessment because toxicity data are available only for specific substances. There are a number of ways to mitigate this problem (see below on TPH); however, it is always preferable to assess specific substances where possible.

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Chemical analyses providing detailed substance-specific breakdowns for many groups of organic compounds are commercially available. TPH and PAH speciations are particularly useful because the individual compounds within these groups have very different physicochemical and toxicological properties.

3.2.1.1 Metals speciation

The toxicity of heavy metals is largely dependent on the form in which they occur in the environment. A common example is chromium (Cr), where the environmentally stable Cr (III) oxidation state is relatively harmless, but the more oxidised Cr (VI) state is extremely toxic. For most heavy metals, the HIL assumes that the most toxic form is 100% of the contaminant present in soil. This is very unlikely to be true in most cases, and knowledge of the actual or probable form can be very useful at Tier 2.

Routine metal speciation analysis is commercially available in Australia for species and compounds of arsenic (As), selenium (Se), mercury (Hg), tin (Sn) and lead (Pb), while metal speciation of some other elements is routinely conducted internationally. At sites where potentially toxic metals are present, consideration should be given to whether speciated metal analysis of media is possible. Speciated metals data can be useful in refining the risk assessment process. Further discussion on the toxicity of metal species is provided in this Schedule.

3.2.1.2 Background concentrations

It is helpful to understand the prevailing background concentrations of chemical substances, since in some circumstances background concentrations may already exceed screening criteria. Guidance on the establishment of background concentrations is given in US EPA (2002b, 2002c).

Research also exists on the geochemical association between soil iron content and the naturally occurring concentrations of As, Cr, Co, Cu, Ni, Pb and Zn (Hamon et al. 2004). This research also provides a methodology for estimating the natural background metal concentration using the iron content. This is useful in circumstances where anthropogenic activities have increased metal concentrations (but not iron concentrations) over a wide area, where determination of background by sampling uncontaminated soil is not possible.

3.2.1.3 Vapour and particulate (dust) sampling

Direct sampling of ambient air, soil vapour and/or dust can be useful in the risk assessment process, as it provides actual data for inhalation exposure points as opposed to results obtained from fate and transport modelling applied to soil or groundwater data.

Air sampling may be useful in scenarios where human populations are potentially exposed to airborne contaminants over prolonged periods (for example, occupational or residential scenarios).

Australian standards for ambient air quality are provided by the NEPC (2003). Standard methods for sampling and analysis of ambient air and dust are available in various Australian standards (AS 3580 Series; AS 2800-1985; AS 2985-1987; AS 2986-1987; and AS 3640-1989) and US EPA TO Methods (including TO-15 and TO-17). Australian guidance on air sampling, analysis, assessment and modelling is available from the New South Wales Department of Climate Change and Water (NSW DEC 2005, 2007).

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Soil vapour sampling guidance is available from US EPA (2002 and associated updates and documentation), IRTC (2007) and Davis et al. (2009), and specific guidance on dust sampling for lead is available from the US EPA (1995a, 1995b) and ASTM (E1792-03 *Standard specification for wipe sampling materials for lead in surface dust)*.

3.2.2 Exposure pathway variables

3.2.2.1 Organic carbon content

The potential for a contaminant to volatilise from soil is strongly influenced by the compound's soil partition coefficient (K_d), which is a parameter that estimates the potential for the contaminant to adsorb to soil particles. The partition coefficient is a function of the compound's organic carbon partition coefficient (K_{oc}) and the fraction of organic carbon (F_{oc}) in the soil. The organic carbon partition coefficient represents the chemical partitioning between organic carbon and water in soil. The relationship between K_d, K_{oc} and F_{oc} is expressed by the following equation:

$$K_d = K_{oc} \times F_{oc}$$

Fraction of organic carbon is a soil property that can be measured, and which often has a significant effect on the risk assessment outcome for volatile and persistent contaminants. Samples of uncontaminated soil should be tested for F_{oc} such that the concentration of carbon measured is not influenced by the potential presence of organic contaminants. The number of samples required depends upon the inherent variability of the soil being characterised. Sufficient samples should be taken to represent each soil type being characterised.

3.2.2.2 Other key pathway parameters

Other key physical and chemical parameters that are required for the risk assessment process include the following:

- site topography and drainage direction
- depth to groundwater
- groundwater flow direction and velocity
- groundwater geochemical parameters including pH, reduction-oxidation potential, dissolved oxygen and major element chemistry
- presence of oxygen in the vadose zone
- soil type classification
- depth of contamination.

Data collection for the establishment of these variables is part of the site investigation process. Guidance on site investigation is provided in Schedule B2.

3.3 Data evaluation

The data evaluation stage is the first stage of the development of the conceptual site model. The level of effort expended and detail of reporting necessary should be proportionate to the amount of data available.

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The data evaluation steps include:

- assessing data quality
- rejecting unreliable data or those which fail to meet data quality objectives (DQOs)
- defining the source in three dimensions
- refining the understanding of pathways
- undertaking Tier 1 screening of chemical data against HILs or relevant criteria for those chemicals without HILs (checked with auditor/regulatory authority)
- identifying contaminants of concern.

3.3.1 Data quality assessment and data quality objectives

Guidance on data quality is presented in Schedule B2 and enHealth guidance, and should be followed when collecting data for risk assessment. Data quality and precision should be such that uncertainty in the risk assessment can be determined and minimized; data quality uncertainties should be explicitly discussed in the uncertainty analysis. Where a number of studies are being combined to provide definition of the source term, particular attention should be paid to the following.

3.3.1.1 Analytical methods

Use of different analytical and sample preparation methods can cause significant differences in results. For example, use of a 'clean-up' step (silica gel) in TPH analysis removes natural hydrocarbons such as soil humic acids, resulting in a much lower result in many cases. Guidance on analytical methods and their selection is given in Schedule B3.

3.3.1.2 Data quality objectives

Data collection is a vital and integral part of the risk assessment process. All data should be collected to meet pre-determined data quality objectives. In many instances, data from a preliminary or detailed site investigation may be available prior to commencing the risk assessment. In such cases, the assessor must determine whether the data quality objectives of any previous investigations are compatible with the objectives of the risk assessment and whether the original data quality objectives have been satisfactorily met.

3.3.1.3 Limits of detection

The detection limit of the analytical method used must be lower than the level at which the contaminant might become a concern (that is, lower than the HIL or Tier 1 screening level).

3.3.1.4 Density and distribution of samples

Under most circumstances, the data should adequately represent the source at the location where the population is likely to be exposed to it. Where sampling density guidelines — refer to Schedule B2 — cannot be followed, the effects of the lack of data on the risk assessment should be considered.

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3.3.2 Three dimensional source definition

Understanding the source is a critical part of risk assessment. There are many factors that control the risk to health from soil; the contaminant concentration is only one of them. The conceptual site model should include a detailed description of the source, bringing together information from the site history, soil and geological information, the depth and extent of the source, and the chemical data.

Site history should provide information on how the contaminants were released to the soil, and the form in which they were likely to have been released. It may also provide information on the length of time the contaminants have been on the site. This information permits judgements to be made on the likely form and mobility of the contaminants. Site history should also allow judgements to be made on where the contamination is likely to be located.

Contaminants may not be uniformly distributed through the soil profile; they may be associated with a particular soil stratum, such as a layer of imported fill material, or a layer of clay that preferentially adsorbs a contaminant. If the distribution or depth of the contamination is not characterised correctly, the risk can be overestimated or underestimated. The risk of exposure to contaminant vapours derived from soil or groundwater contamination is often driven by the soil type and porosity, the depth of the contamination and the presence of organic carbon in the soil profile. Therefore, by understanding which soil stratum is impacted, key parameters such as soil type, soil depth and F_{oc} can be obtained from the appropriate zone, and a more accurate assessment of the risk can be made.

3.3.3 Refining the exposure pathways

Site investigation data may either introduce or rule out exposure pathways in the conceptual site model. For example, establishing the groundwater flow direction with improved certainty might show that a pathway to a potentially exposed population does not exist because that exposed population proves to be located upgradient of the source. Detailed guidance is available in Schedule B2.

Guidance on physical hazards such as inhalation of asbestos fibres, risk of fire or explosion from flammable gases and risk of exposure to asphyxiating atmospheres (for example, methane, carbon dioxide, carbon monoxide) is beyond the scope of this Schedule. Appropriate guidance should be followed for the assessment of physical hazards.

Exposure pathway assessment should lead to a clear conclusion on which pathways are considered viable and which are not, with reasoning and appropriate evidence. Where viable pathways cannot be assessed in the risk assessment process, appropriate controls for mitigating the risk should be provided and documented.

3.3.4 Tier 1 screening

Tier 1 screening involves comparison of site analytical results with appropriate screening criteria. In Australia, the HILs will be used for Tier 1 screening to provide a rapid assessment of whether the site contamination may pose any significant risk to health. Should contaminant concentrations at a site occur at levels that are below the HILs, this implies that for the majority of the people in the population there is no significant health risk from contamination and that remedial action may not be required to protect health.

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For contaminants where HILs are not available, the methodology set out in Schedule B7 could be adopted to create HIL equivalent screening criteria. Sources of toxicity reference values should be considered in the same way as has been done for those chemicals with HIL (same hierarchy and other considerations). Exposure scenarios should be used as laid out in Schedule B7. This should only be undertaken by suitably qualified professionals. The resulting values may be used in the same way as HILs. All assumptions and calculations should be transparent, clearly referenced and justified.

Tier 1 values may be adopted from external peer reviewed sources only if the assumptions used to generate the values are equivalent or more conservative than those used to develop the HILs and are suitable for use. In these cases, the relevance of the values and assumptions included in the development of the proposed Tier 1 values must be clearly justified and referenced.

Exceedances of the HILs must be identified and considered. HIL exceedances do not imply that a risk is necessarily present but that further assessment may be justified. HILs are not intended to indicate a clear demarcation between 'acceptable' and 'unacceptable'. Marginal exceedances may not require quantitative Tier 2 risk assessment to conclude that further assessment is not necessary. The magnitude of the exceedance should be considered in the context of the conceptual site model (that is, whether the exposure pathways are plausible and whether exposure will result in harm).

Background concentrations may also be an important consideration at the Tier 1 screening stage. If it can be clearly demonstrated that site concentrations are consistent with natural regional background levels (natural or anthropogenic), this can provide evidence that site contamination has not significantly increased the background level and further assessment is not justified. However, it is important to note that background concentrations in some cases may present a risk: for example emissions from motor vehicles can result in higher exposure to residents in a transport corridor. Residences adjacent to refuelling stations may be exposed to high hydrocarbon background. Naturally elevated background can result from highly mineralised geologic environments, resulting in enrichments of potentially toxic trace metals or from anthropogenic inputs such as atmospheric deposition in highly industrialised areas that are often found in major Australian cities. In such cases, consultation with local environment regulators and health protection agencies would be appropriate.

Where site concentrations exceed Tier 1 screening criteria, a 'no further assessment' conclusion should be clearly justified by considering aspects such as the following:

- whether sufficient sampling has been undertaken for adequate confidence that the results are representative of the site
- whether the 95th upper confidence limit (UCL) of the arithmetic mean of the samples exceeds the Tier 1 screening criterion, and whether the standard deviation exceeds 50% of the Tier 1 screening criterion (these exceedances would indicate significance)
- whether any single value exceeds 250% of the screening criterion and, if it does, whether this value may represent a 'hotspot' that requires further consideration
- subject to the above, statistical analysis of the data may indicate that overall exposure is unlikely to constitute a risk
- professional judgement on the significance of the impact, such as whether exposure to the contamination is plausible under normal site conditions.

Guidance on how to calculate the 95% upper confidence limit and standard deviation is provided in the New South Wales sampling design guidelines (NSW DEC 1995) and is also available in statistics textbooks (for example, Gilbert 1987).

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Note that in applying the above guidance it is essential that the contaminant distribution is reviewed prior to applying the statistical tests, and an appropriate data set selected for calculation of averages and standard deviations.

This means that localised areas or volumes of significantly differing contaminant concentration ('hotspots') must be identified and removed from the data set. Hotspots may be identified using statistical tests for the identification of 'outliers', for example, Rosner's test as described by Gilbert (1987) which assumes that the data is normally distributed, which must be demonstrated – other statistical tests may be relevant for non-normally distributed data, or by judgement on the basis of analytical results and site history knowledge. Hotspots must not be ignored by 'averaging' them away.

The end-point of the Tier 1 screening is the selection of the contaminants of potential concern that require further assessment. If the Tier 1 screening assessment concludes that there are no contaminants with plausible pathways to exposed populations, then the assessment is complete.

Further assessment after Tier 1 screening may comprise either additional assessment at Tier 2 (that is, risk assessment as described by the remainder of this guidance). Alternatively, additional data collection may be required.

Contamination at a site will also be evaluated by looking at media other than soil, such as air, groundwater or surface water. This data must also be analysed in order for it to be used in the appropriate exposure scenario at a site. There is less guidance available on the characteristics of such data and indicators of its sufficiency. An initial review of these other types of data is required to determine if the sampling and analysis methods used were appropriate and if the detection limits achieved were appropriate given the toxicity reference values.

Groundwater data being used to assess human exposure may consider the maximum (preferably using the 95%UCL of the average) and average at the site, off site (as relevant) or for specific bores that may be relevant for the assessment of exposures relevant to the use of extracted groundwater.

If air data or soil vapour data is available for the site, then the use of that data needs to be considered within the context of the conceptual site model and the activities at the site that may affect the presence of the chemicals in the air. Consideration of both a reasonable maximum and a relevant average case should be developed where possible.

Averages should be calculated using half the detection limit for results that were below detection limits.

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4 Exposure assessment

4.1 Introduction

Exposure assessment involves the estimation of the magnitude, frequency, extent and duration of exposures to contaminants. In the data evaluation stage, the conceptual site model was refined to produce an understanding of the source(s), pathway(s) and exposed population(s) that require assessment at Tier 2. In the exposure assessment stage, the conceptual site model is used to generate a numerical representation of the exposure pathways that can be modelled quantitatively; these are the model input values. The input values are then used to model exposure point concentrations and estimate intakes of contaminants by the exposed populations.

To promote consistency and transparency in making exposure assessment assumptions, the Environmental Health Committee (enHealth) has established a framework for exposure assessment, set out in enHealth (2010). This section follows the framework, adding details specific to contaminated land assessment.

Exposure assessment modelling methods have largely been developed for use in contaminated land risk assessment by the US EPA, and in the following section there are many references to US EPA guidance documents. These documents are extensive, well referenced and provide model algorithms and guidance on their use. Adoption of the modelling methodology in this Schedule does not imply endorsement of the US guidance; the US methods are to be adapted to meet Australian policy objectives and environmental circumstances.

In the following sections, the derivation of model input values generally using point estimates (that is, single value estimates) is discussed. A more complete discussion of the use of probability distributions as input values (for example, Monte Carlo method) was provided above. There may be circumstances where the use of probabilistic methods is appropriate in Tier 2 assessments, and it is not the intention of this Schedule to discourage the use of probabilistic models where their use can be beneficial to the outcome of the assessment.

The key elements of exposure assessment, as applied to contaminated land risk assessment, are to:

- determine input values for contaminant concentrations
- determine input values for pathways
- determine input values for exposed populations
- estimate exposure concentrations
- estimate chemical intake
- collect data to test predictions in stages 4 and 5 (where relevant).

Stages 1, 2 and 3 comprise the translation of the conceptual site model into modelling terms as described. Stages 4 and 5 are achieved using a quantitative health risk assessment model. Stage 6 is a test of the model.

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4.2 Exposure settings

4.2.1 Defining model inputs to represent the contaminant source

The data evaluation stage should provide a good understanding of the source. Consideration needs to be given as to how the contaminant concentration will be applied in a Tier 2 human health risk assessment; that is, what values will be used as the input concentrations representative of site conditions. Note that, depending on the complexity of the site, there may be more than one 'source' requiring further assessment. Note also that the source may be in soil, water, separate phase liquid or vapour. Sources in different physical forms generally should be assessed separately and require separate input values.

There are a number of options for choosing the value to use as an input concentration. The most appropriate method will depend on the data set, and different methods may be required for different source areas or contaminants, since these may show very different distributions. If a series of data over time are available, consideration of trends will be needed; this is particularly important for groundwater sources. Some commonly used approaches are described herein; however, more sophisticated statistical methods may be used if the data set is suitable. Whatever approach is used to define the source term, it should be clearly explained and justified.

Maximum observed contaminant concentration. This generally provides a conservative assessment because if estimated risks from the maximum concentrations are not significant, then the site should be suitable for use under the conceptual site model considered. Maximum concentration is often suitable for groundwater sources where trends are poorly defined. However, a maximum concentration may not be representative of the source as a whole and may result in a significant overestimation or underestimation of risk if the data are extremely limited.

Mean concentration. The mean contaminant concentration can be a suitable input concentration provided that it can be shown that it adequately represents the source being considered. It is important that small areas of high concentrations or hot-spots are not ignored by averaging with lower values from other parts of the site. The mean value may be more representative of the source as a whole than the maximum, and may provide a better estimation of the actual concentration that a population would be exposed to over a period of time.

95% upper confidence limit (UCL) of the arithmetic mean contaminant concentration. This provides a 95% confidence level that the true population mean will be less than, or equal to this value. The 95% UCL is a useful mechanism to account for uncertainty in whether the data set is large enough for the mean to provide a reliable measure of central tendency. Note that small data sets result in higher 95% UCLs. Further guidance on the use of 95% UCLs can be found in NSW DEC (1995) and US guidance.

Monte Carlo (or other probabilistic) techniques, as discussed earlier in this Schedule.

Source input values will normally be soil, groundwater or soil vapour data. In more detailed assessments, more specific sources may be defined, such as dust, bore water or ambient air.

Considerations in this section are also relevant to contaminant source input values derived using fate and transport or other exposure point estimation methods. These are described elsewhere in this Schedule.

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4.2.2 Exposure pathway input values

This stage involves describing the physical environment in terms of the input values that will be used to represent exposure pathways in the model. The scenario being modelled should clearly relate to the existing or proposed land use for which decisions on contamination are required. Depending on the pathways modelled, a number of variables will need to be defined, for example, soil type, soil properties, depth to groundwater, soil and vapour sources, climactic variables and building characteristics and dimensions. Schedule B7 provides a complete list of the exposure pathway variables used to derive the HILs, together with justification of the values selected.

Exposure pathway input parameters used to conduct a site-specific risk assessment should be the same as those used to derive the HILs, provided that the conceptual site model is similar to the HIL exposure scenarios. Where the conceptual site model is different from the HIL scenarios, it is likely that some alternative physical setting variables will be required.

Each variable used in a model should be clearly referenced and justified. Some commercially available models do not permit amendment of all the variables listed and the user must rely on the default values supplied with the software. Where this is the case, it should be demonstrated that the model defaults are applicable to the site. It should be appreciated that models developed for the use in other countries may incorporate assumptions that are not justified in the Australian environment. Reference should be made to the physical setting assumptions outlined in Schedule B7 for guidance on values likely to be suitable for Australian sites. Site-specific data from site investigations should be used wherever possible.

4.2.3 Exposed population input values

The purpose of this part of the risk assessment is to determine the characteristics and behaviour of the critical exposed populations. Exposed populations may relate to the current or proposed future use of the site, and it should be made clear which land use assumptions are being made. Exposed populations may be located at some distance from the site, with pathways involving transport via groundwater, surface water or wind. Exposed populations may also be linked to the site via the food chain, for example, consumers of fish, meat or agricultural products which may be affected by site contaminants.

The physical characteristics and behaviour patterns representative of the exposed population must be selected for modelling. This is essentially a common sense exercise, which does not normally require any specific assessment methodology or data.

The main considerations are described below.

Physical characteristics: selection of representative values for physical aspects such as age, life expectancy, body weight and respiration rate must be made. It is not expected that risk assessors will be generally required to generate these assumptions (refer to those listed in B7 and/or enHealth (2010) where relevant); considerable uncertainty is involved and variations in assumptions can have a significant impact on the risk assessment outcome. Exposed population physical characteristics should be sourced from applicable Australian guidance on exposure assessment (enHealth 2010). Schedule B7 provides the values that have been selected to derive the HILs, which are primarily sourced from enHealth (2010).

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Exposed population behaviour: exposure assessment requires the development of a model behaviour pattern that is judged to represent an exposed population. Some data on Australian behaviour patterns are available (for example, see EPHC 2004, enHealth 2010). Important considerations in contaminated land risk assessment include factors such as the distribution of hours spent indoors and outdoors, amount of time spent in the location where exposure is predicted, level of physical activity, the nature of work or leisure activities, and the exposure duration. In selecting values to represent exposed population behaviour, it is important to consider the following:

Plausible high-end exposure – is the recommended approach to judging receptor population behaviour. The likelihood of the modelled scenario should be considered, and behaviours which might reasonably apply to real people should be selected for modelling.

Consistency – Schedule B7 provides behavioural and exposure duration assumptions for four standard exposure scenarios. Where site-specific assessments are essentially considering the same exposed populations in similar circumstances to the standard scenarios, the Schedule B7 behavioural assumptions should be adopted. This promotes consistency between site-specific risk assessments. Where the exposed population differs significantly from the standard scenarios, amendments to behavioural assumptions can be made and should be clearly justified. Note that amending the commercial/industrial and public open space scenarios may be a common requirement, since activities within these land uses are prone to be variable.

Exposure via food and drink – the standard scenarios described in Schedule B7 provide limited consideration of exposure via food and drink because most Australians do not source a significant proportion of their food or water from their own property.

Allocation of background exposure – related to the above point is the extent to which the exposed population is exposed to the contaminants under consideration as part of their daily lives. As well as presence in food and drink, contaminants may be present in the air, in consumer products, in household goods and in building materials. It is therefore necessary to consider the extent to which the risk assessment will allow for this exposure. At a screening level, simple (but appropriately justified) assumptions may be adopted. In more detail, background exposure can be considered on a site-specific basis depending on the site location and land use, as well as considering the contaminants individually.

It is recommended that in designing the exposure assessment, worst-case scenarios (particularly those where many 'high-end' assumptions are compounded) should generally be avoided. The sensitivity and uncertainty of the assumptions adopted in the exposure assessment should be considered.

4.3 Exposure point concentrations

An exposure point concentration is the estimate of the concentration of the source contaminant in the medium that the population is exposed to, at the location where exposure is predicted to occur.

It is preferred that, where possible, exposure concentrations are derived from direct measurements in the relevant media (soil, bore water, indoor air, fruit and vegetables).

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However, under some circumstances it is not practical to measure concentrations directly, and in these cases exposure point concentrations are typically estimated using computer models.

The most commonly used exposure point estimation methods are:

- vapour intrusion modelling used to estimate vapour concentrations in ambient air from measured soil vapour, soil, groundwater and separate phase product data
- particulate modelling used to estimate the concentrations of dust generated from surface soil
- groundwater fate and transport modelling, used to estimate groundwater concentrations at an exposed population location where direct measurement is not possible, or to predict future groundwater concentrations at an exposed population location
- plant uptake modelling, used to estimate concentrations of contaminants in crops and vegetables from soil and groundwater data
- animal uptake modelling, used to estimate concentrations of contaminants in fish and meat.

These methods are described herein, with the exception of groundwater fate and transport modelling for which guidance is presented in Schedule B2. It should be noted that the level of uncertainty associated with the use of any model for the purpose of estimating exposure concentrations should be carefully considered and discussed.

4.4 Exposure point concentrations - volatiles

4.4.1 Introduction

Volatile substances are those that are capable of changing from liquid to vapour phase (that is, volatilising) under ambient conditions. While there are a few definitions of what may be considered of significance with respect to volatilisation, a volatile substance can be defined as having a Henry's law constant of greater than or equal to 10^{-5} atm/m^3/mol and its vapour pressure greater than 1 mmHg at room temperature (NJ DEP 2005). These two factors more specifically address the key measures of volatility.

Vapours may arise primarily from three processes:

- by desorption from soil organic matter, described by the sorption coefficient and the F_{oc} in the soil
- from groundwater plumes, described by the Henry's law partition coefficient between water and air
- from non-aqueous phase liquids, depending on the vapour pressure of the volatile compounds in the non-aqueous phase and the mole fraction of the compound of interest (US EPA 2000).

To assess exposure to volatiles, it is necessary to estimate the concentration of the vapour in the air that the exposed population breathes. The most appropriate approach to the quantification of these exposures is to utilise direct measurements of indoor or ambient air. Vapours in ambient air are relatively easy to sample; however, the collection and interpretation of these data can be difficult.

An indoor air sampling program may be expensive if many samples over a reasonably long period are needed to get representative results. In homes and workplaces, gaining access can be difficult and may lead to unnecessary concern on the part of the occupants.

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Depending on the volatile compounds considered, ambient air results may be difficult to interpret since many other sources in addition to the site soil and groundwater can be present. For these reasons, ambient air measurements are not generally available. When they are available they may be unsuitable as a means to assess risks associated with soil and groundwater source alone, because of the inability to distinguish whether a soil or groundwater derived component is present or not.

Where direct measurements are not available, indoor and outdoor ambient air contaminant concentrations can also be predicted by modelling from measured soil vapour concentrations. Soil vapour measurement is the preferred route in most situations where a vapour issue is considered likely to exist.

In the absence of measured soil vapour concentrations, it is also possible to model the generation of vapour from soil, groundwater and separate phase liquids. This procedure adds another level of uncertainty to the process, and may lead to inaccurate results.

The uncertainties associated with the use of a model for these purposes should be well understood and discussed in relation to the nature of the volatile contaminants assessed.

Where unresolved uncertainties or unacceptable risks are predicted by modelling vapour concentrations, direct measurement of soil vapour and/or exposure concentrations indoors and outdoors should be obtained.

4.4.2 Indoor air concentrations:

The direct measurement of indoor air concentrations is appropriate where practical (taking care with any data collected in indoor air to minimise background influences).

Alternatively, indoor air concentrations can be modelled (estimated) using an attenuation factor (refer to US EPA 2008 for a range of values that could be considered), a model such as the Johnson and Ettinger (1991) model, or another appropriate (justified) model. The Johnson and Ettinger (1991) model is a one-dimensional 'heuristic' analytical solution to model convective and diffusive vapour transport into indoor spaces. It provides an estimated attenuation coefficient that relates the vapour concentration in the indoor space to the soil vapour concentration at the source of contamination (US EPA 2004a). A vapour attenuation factor, 'alpha' (α), is calculated, which is the ratio of the concentration of a chemical vapour in an indoor scenario relative to that measured in the soil. This model has been updated and modified since 1991 (Abreu & Johnson 2005, 2006) and it is also described in Davis et al. (2004, 2009a). Inputs to the model include chemical properties of the contaminant, saturated and unsaturated zone soil properties, and structural properties of the building (US EPA 2004a).

The Johnson and Ettinger model as described by US EPA (2004a) is the most commonly used indoor vapour model. The US EPA model provides additional functionality permitting the estimation of soil vapour concentrations from soil, groundwater and phase separated liquid. It is provided at

<http://epa.gov/oswer/riskassessment/airmodel/johnson_ettinger.htm>

There are a number of ways in which this vapour model can be manipulated to improve the confidence in the outcomes from the model; however, confidence in any model output without corresponding data for the purpose of validation is not high. Additional guidance is provided in Davis et al. (2004, 2009a).

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4.4.3 Outdoor air concentrations

The direct measurement of outdoor air concentrations is appropriate where practical, taking care with any data collected to minimise background influences.

Alternatively, outdoor air concentrations can be estimated using models such as the Jury et al. (1983) model. The Jury model calculates the maximum flux of contaminant vapours from an infinite soil contaminant source via vapour phase diffusion. Chemical movement to the atmosphere is modelled via volatilization loss through a stagnant air boundary layer at the soil surface, making it appropriate for use in an outdoor exposure setting. The Jury (1983) model is widely accepted as an appropriate methodology for vapour modelling into outdoor air and has been applied by environment agencies in the United Kingdom (Environment Agency 2009) and United States (US EPA 1996) in the development of Tier 1 soil investigation levels. The Jury model is also recommended for estimating outdoor vapour concentrations in the Standard guide for risk-based corrective action (ASTM 2004).

The modelling of outdoor air exposures also needs to account for vapour dispersion, between the soil surface and breathing zone of potentially exposed populations. This can be done using an outdoor box model which may predict ambient vapour concentrations on the downwind edge of the area source at the breathing zone height, as described by ASTM (2004). Alternatively, vapour dispersion in a well mixed box may be estimated using calculated air dispersion factors, as described in US EPA (1996). Either method is considered appropriate.

4.4.4 Finite and infinite sources

The Johnson and Ettinger model is constructed as both a steady-state solution to vapour transport (infinite or non-diminishing source) and as a quasi-steady-state solution (finite or diminishing source) for soil contamination. A finite source model was not provided for groundwater since groundwater migration reduces the certainty of concentration attenuation.

In situations where the soil or non-aqueous phase liquid source of dissolved phase volatile groundwater contamination is no longer present, dissolved phase concentrations should diminish or attenuate as the contaminants volatilise (or biodegrade). Dissolved phase contamination therefore becomes 'finite'.

In circumstances where relatively small amounts of contaminants are present in the source zone, the infinite source assumption can give rise to a physically impossible output when a long period of time is modelled. Assessments should consider whether sufficient source exists to support the volatilisation modelled for the time period under consideration.

The finite source model can be used for site-specific risk assessment, provided that field evidence for the finite nature of the source is presented.

4.4.5 Biodegradation

There is a body of work that clearly shows that the concentration of petroleum hydrocarbon (TPH and benzene, toluene, ethylbenzene and xylene) vapours in well oxygenated, generally near surface soil can be significantly reduced by biodegradation (Davis et al. 2009b). However, this is generally not the case in less well oxygenated soil under large areas of hardstand. It is also not applicable to chlorinated solvents (except vinyl chloride where evidence suggests good biodegradation in the presence of oxygen) and other volatile contaminants.

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Davis et al. (2009b) recommend a process for incorporating biodegradation into vapour models where there is sufficient evidence to justify its use. This approach is considered appropriate for site-specific risk assessment.

The fundamentals of the Davis et al. (2009b) approach are:

1. For biodegradation to be applicable, the oxygen concentration in the soil gas must be 5% or greater at no less than 1 m below the ground.
2. An exposure reduction factor may be considered where the vapour source is greater than 2 m below the ground/base of a building.
3. Biodegradation can only be considered under buildings within a 7.5 m radius from the edge of the slab.

Where 1) is demonstrated, and where exclusions 2) and 3) do not apply, then a 10 fold reduction factor can be applied to vapour concentrations for sources greater than 2 m deep. For sources greater than 4 m deep, a 100 fold reduction factor may be considered, where the vapour source maximum is less than 100 mg/L.

Approaches to assessing and modelling biodegradation of petroleum hydrocarbon vapours are developing rapidly. Useful methods are likely to be developed and will come into general use. For example the American Petroleum Institute (API) in 2009 released *Biovapor,* a model permitting simulation of biodegradation on a site specific basis. It can be accessed and downloaded at <www.api.org/ehs/groundwater/vapor/index.cfm>.

Whilst it is not possible to provide a recommendation that this model is certainly an appropriate method for use in Australia, it is not the intent of this guidance to restrict useful development of techniques. Therefore, where new methods can be justifiably applied, their use should be considered.

4.4.6 Vapours from non-aqueous phase liquids

Non-aqueous phase occurs when the sorbed phase, aqueous phase, and vapour phase of a chemical have reached saturation in soil. Concentrations above this saturation limit (Csat) for all of the specified chemicals of a mixture result in a non-aqueous phase liquid or solid (US EPA 2000).

At contaminant concentration less than Csat, the equilibrium vapour concentration at the contaminant source is proportional to the soil concentration, according to the vapour modelling equation presented by Johnson and Ettinger (US EPA 2004a). When a non-aqueous phase is present however, the vapour concentration at the contaminant source is independent of the soil concentration but proportional to the mole fraction of the individual component of the non-aqueous phase mixture, according to Raoult's Law (US EPA 2000).

Raoult's Law states that 'the vapour pressure of each chemical component in an ideal solution is dependent on the vapour pressure of the individual component and the mole fraction of the component present in the solution'.

Therefore, as the number of components in a solution increases, the individual vapour pressures decrease as the mole fraction of each component decreases with each additional component.

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In order to calculate the mole fraction for mixtures, or a solution of compounds, it is necessary to know the concentrations of the individual components comprising the non-aqueous phase liquid. This cannot be achieved by estimating the proportion of components in non-aqueous phase liquid from dissolved phase results. A sample of the free phase liquid should be collected and analysed.

$$\text{Mole fraction} = \frac{\text{Number of moles of compound}}{\text{Total number of moles}}$$

$$\text{Number of Moles} = \frac{\text{Concentration of compound in solution}}{\text{Compound molecular weight}}$$

The saturation vapour concentration can therefore be calculated by:

$$C_{si} = \frac{X \bullet \rho \bullet MW}{R \bullet T}$$

where:

C_{si} = Saturation vapour concentration (g/cm^3)

X = Mole fraction of chemical in product

ρ = Vapour pressure of the chemical (mm Hg)

MW = Molecular weight of compound (g/mole)

R = Molar gas constant (62, 361 mmHg - cm^3/mole - k)

T = Absolute temperature (293 K for ambient conditions)

The US EPA (2004a) provides a guide to the NAPL-SCREEN and NAPL-ADV models, which should be used to calculate exposure point concentrations where non-aqueous phase liquid is present.

When using the Johnson and Ettinger model in a risk assessment, if the risk-based concentration is greater than Csat and the contaminant is a liquid or gas at the soil temperature, the final soil SSTL can be set equal to the Csat. The purpose of this approach is to eliminate the possibility of allowing a liquid residual phase to exist within the soil column, which may leach to the water table (US EPA 2004). Alternatively, potential risks associated with vapour derived from the non-aqueous phase can be estimated by applying Raoult's Law.

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4.5 Exposure point concentrations - particulates

The concentration of particulates (dust) relevant for different exposure scenarios can be determined using either a dust concentration factor or a soil particulate emission factor (PEF). The PEF approach is presented in the soil screening guidance (US EPA 1996) and supplemental soil screening guidance (US EPA 2002a). The methodology uses a fixed conservative soil particulate release rate combined with a box model to determine dust concentrations in air. The PEF assumes loosely packed surface soil so that a relatively large concentration of dust is entrained in air. The entrained soil particles are considered to mix in the ambient air breathing zone directly above the soil source. In combination with air dispersion models and site-specific meteorological data (wind rose), the predicted dust level at a specific location emanating from a specific source (for example, construction site) can be determined. It is noted, however, that this model does not address exposures associated with dust that is generated from dry, exposed soil, generated during active use of a site (for example, during use of dry sporting fields) or mechanically generated (such as during vehicle movements). Dust generated during these scenarios may be better estimated using a dust concentration (loading) relevant to the area and nature of activity assessed.

4.6 Exposure point concentrations - food consumption

Ingestion of contaminated food is a potential pathway for humans to be exposed to toxic chemicals and, in some risk assessments, may be a significant pathway requiring quantitative assessment. Guidance on how to calculate the mean daily (contaminant) intake from ingestion of contaminated food is provided in the following hierarchy of sources:

- Environmental health risk assessment (enHealth 2010)
- Australian total diet study (formerly the Australian market basket survey), Food Standards Australia and New Zealand. Reports are accessible at < www.foodstandards.gov.au/educationalmaterial/australiantotaldiets1914.cfm>
- Exposure factors handbook (US EPA 1997)
- UK Contaminated land exposure assessment model: technical basis and algorithms (DEFRA/Environment Agency 2002) and updated technical background (Environment Agency 2009).

Patterns in food consumption vary between individuals and groups of individuals and therefore the likelihood exists that some groups of people will have a different diet than the population as a whole (Cross & Taylor 1996). Australian national food consumption data, including percentage of each food type or group consumed on a daily basis are available in enHealth (2010) and have been compiled from a number of dietary surveys. Additional guidance including contaminant intake algorithms for different produce is available in the US EPA exposure factors handbook (US EPA 1997) and through the UK Environment Agency (2006). enHealth (2010) strongly recommends that Australian dietary survey data is used in exposure assessments, as overseas diets are likely to be of less relevance; however, the algorithms from overseas guidance are applicable to Australia. When using the dietary survey data, it is important to consider the limitations of the survey conducted, the site-specific situation being addressed, and to use the most appropriate survey data that fits the scenario in question.

When assessing contaminant intake from food consumption, it is important to consider not only the percentage of a particular food group in the diet, but also the percentage of the food group that could potentially have been grown/reared in a contaminated environment.

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4.6.1 Fruit and vegetable consumption

In circumstances where home-grown fruit and vegetable consumption is likely to be significant (for example, more than 10% of the diet), the consumption of garden vegetables grown in soil on a contaminated site is likely to represent the main potential transfer of soil contamination to adults and children. An assessment of exposure from this pathway depends on three critical factors: how much contamination is likely to be accumulated by garden vegetables from the surrounding soil, how much home-grown produce is likely to be consumed by those in the household, and how much contamination in food is absorbed by the human body (Paustenbach 2000).

Limited published information is available on the percentage of Australian households having domestic fruit and vegetable gardens, and on the percentage of home-grown fruit and vegetables consumed in comparison to those purchased. However, health risk assessments are site-specific and therefore this information may be obtained through discussion with the site owner/occupier at the conceptual site model development stage. In the absence of site-specific information, Cross and Taylor (1996) provide a summary of information currently available.

Contaminant uptake behaviour varies markedly between plant species and for different contaminants. Ideally, the concentration of contaminants in home-grown vegetables and fruits should be measured directly on a site-specific basis, but this is often impractical in contaminated land assessments. In the absence of site-specific contaminant uptake data, the chemical concentrations in the edible portions of fruits and vegetables can be predicted from the soil-to-plant concentrations factors (CF), which describe the relationships between the concentrations of contaminants in the soil and plant contaminant concentrations, on a fresh weight basis.

A methodology to estimate contaminant intake from fruit and vegetables for home grown vegetable intakes of up to 10% of the diet is provided in Schedule B7.

It is recommended that caution be employed in the evaluation of potential human health risks associated with ingestion of fruits and vegetables using generic predictions of plant uptake. Data are limited, and the methods for predicting plant uptake of contaminants from soil and groundwater data are not well validated, particularly for many organic substances. Further site-specific investigations are likely to be justified in situations where the consumption of home-grown produce is likely to constitute a significant portion (>10%) of the diet of a household or where commercial production of vegetables and/or fruit may occur.

4.6.2 Poultry, meat and fish consumption

The calculation of intake concentrations from the consumption of poultry, meat and fish is complicated and suffers from significant uncertainties. In order to estimate contaminant concentrations in the potential food source using soil, groundwater and surface water concentrations, the following information is required as a minimum:

- an understanding of the diet of the exposed population
- an estimation of contaminant concentrations in the food source consumed by the livestock or fish
- an estimation of the relative percentage of time spent roaming and feeding in the contaminated area

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- an accounting for assimilation rates of the contaminant into the food source and bioaccumulation within the higher organism.

Data are scarce for each element, and there is currently no generic methodology available for estimating contaminant levels in animals resulting from their exposure to contaminated soil or water.

If the exposure of farm livestock has already occurred, direct measurement of the contaminant levels in the meat would be the most practical means to estimate intakes of potential exposed populations. Some studies have been conducted on fish and shell fish and equations for estimating contaminant concentrations in fish from water concentrations are available in ANZECC and ARMCANZ (2000) and Arnot and Gobas (2003). Additional algorithms on uptake in humans from consumption of contaminated fish are also provided in the ANZECC and ARMCANZ (2000) guidelines.

In the absence of directly measured contaminant concentrations in fish, the ANZECC and ARMCANZ method is recommended to estimate the likely concentration of contaminant uptake into the fish tissues and subsequently the mean daily intake dose from human consumption. Implementing the ANZECC and ARMCANZ methodology involves:

- measuring the contaminant concentrations in the water environment that the fish inhabit
- estimating the uptake of contaminant into the edible portion of the fish tissue (allowing for the percentage of time the fish may spend in the contaminated section of the environment)
- estimating potential human consumption of fish caught within the contaminated area.

4.7 Estimation of contaminant intake

4.7.1 Introduction

Contaminant intake is estimated for each chemical and pathway separately. The recommended methodology generally follows US EPA (1989). There are two basic approaches which have been developed on the basis of the way in which chemicals cause toxicity:

Threshold toxicity is exhibited by chemicals where there is an exposure level below which no toxic effect is thought to occur. Threshold substances are generally considered to include most non-carcinogenic chemicals and non-genotoxic carcinogens.

Non-threshold toxicity is exhibited by chemicals where there is considered to be no dose below which no adverse effect will occur. In theory, any level of exposure could result in a response. Genotoxic carcinogens comprise this group.

For threshold chemicals, the estimated daily intake can be compared with a threshold toxicity reference value (TRV) which is a value representing a dose that will cause no adverse effect over a lifetime of exposure. Note that, as applied here, the TRV means any appropriate measure of tolerable daily intake, and includes doses derived by various bodies for different applications. Reference doses can be tolerable daily intakes (TDI), usually from drinking water guidelines (for example, Australian or WHO) or acceptable daily intakes (ADI) typically used in food and drug guidance, or US EPA reference doses (RfD).

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Threshold TRVs are often available for ingestion and inhalation exposure routes, and occasionally for dermal exposure. Since the TRV for different exposure routes can be quite different, each TRV is only applied to the relevant pathway. Consequently, estimated intakes are only summed to the extent that they correspond to the same exposure route (that is, all ingestion pathways added, all inhalation pathways added, but ingestion intake not added to inhalation intake) when estimating risk by comparing intake to the TRV. The relative scarcity of TRVs for the dermal exposure route is usually accommodated by adding the dermal intake to the ingestion intake where a dermal TRV cannot be sourced. Once hazard/risk quotients and indices are calculated, summing of all pathways can and should be conducted.

For non-threshold chemicals, intakes are estimated on a daily basis, multiplied by the exposure duration (ED) and then divided by the averaging time (AT). AT is typically a value representing a lifetime. This results in a daily intake which is multiplied by a non-threshold TRV — such as a cancer slope factor (CSF) or unit risk factor (URF) — to produce a measure of excess lifetime cancer risk.

Doing the calculations in this fashion assumes that a low dose over a long period causes equivalent effects to a single high dose – that is, that it is the daily average exposure each day over the whole lifetime that is relevant to the development of cancer.

4.7.2 Ingestion intakes

Methodologies and algorithms for estimating ingestion intakes are available for the pathways listed below. Note that the data applied within these methods should be sourced from Australian publications such as enHealth 2010 as far as possible. Details of the data used to generated the HILs are given in Schedule B7.

- Incidental ingestion of soil and indoor dust (US EPA 1989)
- Ingestion of soil attached to home grown vegetables (DEFRA/Environment Agency 2002)
- Eating home-grown fruit and vegetables (DEFRA/Environment Agency 2002)
- Eating poultry, meat or fish (US EPA 1989)
- Drinking contaminated water (US EPA 1989)
- Incidental ingestion of surface water while swimming (US EPA 1989)
- NHMRC Guidelines for managing risks in recreational water (2008).

The equations available to estimate these intakes are generally of the form shown below and detailed in Table 1 for the direct soil ingestion pathway (US EPA 1989). All currently available exposure models for contaminated land assessment use equations of this form, although there may be variations in detail.

$$\text{Intake (mg/kg-day)} = \frac{C_S \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

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Table 1. Variables description for soil ingestion intake calculation

Variable	Units	Description
C_S	mg/kg	Concentration in soil
IR	mg soil/day	Soil ingestion rate
CF	10^{-6} kg/mg	Unit conversion factor
FI	-	Fraction ingested from contaminated source
EF	Days/year	Exposure frequency
ED	years	Exposure duration
BW	kg	Body weight
AT	days	Averaging time

Values for the variables in Table 1 (and similar variables for other ingestion pathways) would be derived during the stages of exposure assessment described earlier in this section.

4.7.3 Dermal intakes

Dermal intakes can be estimated for the following pathways:

- dermal contact with soil and dust
- dermal contact with separate phase liquid
- dermal contact with chemicals in water (swimming, showering, bathing or incidental contact).

Dermal contact with vapour phase contaminants is not generally assessed since it is likely to be insignificant in comparison with other pathways (US EPA 1989).

Dermal absorbed dose or dermal intake is estimated using the concept of absorbed dose per event (US EPA 2004b). The overall absorbed dose depends on the number of events, the adherence factor (AF) and the fraction of contaminant absorbed (ABS).

It is noted that there are significant uncertainties inherent in the estimation of both AF and ABS. US EPA (2004b) provides estimates of ABS for eleven substances or groups of substances and provides guidance on the treatment of uncertainty for the assessment of substances where specific ABS values are not available. AF is dependent on soil type, activity type and exposed population age, and values are provided for a range of circumstances in Exhibit C-2 in Appendix C of US EPA (2004). Because soil type is so important to the value of AF, additional guidance is provided for sediment (although the method is the same). Wet soil and fine grained soil have much higher values of AF than dryer and coarser soil.

The equation for dermal intakes is published as follows and detailed in Table 2.

$$\text{Dermal intake (mg/kg-day)} = \frac{DA_{event} \times EF \times EV \times ED \times SA}{BW \times AT}$$

$$DA_{event} = C_S \times CF \times AF \times ABS$$

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Table 2. Variables description for soil dermal contact intake calculation

Variable	Units	Description
DA_{event}	mg/cm²-event	Dermal absorbed dose per event per unit exposed skin area
EF	Days/year	Exposure frequency
EV	Events/day	Event frequency
ED	years	Exposure duration
SA	cm²	Skin surface area available for contact
BW	kg	Body weight
AT	days	Averaging time
C_S	mg/kg	Concentration in soil
CF	10^{-6} kg/mg	Unit conversion factor
AF	mg/cm²-event	Adherence factor of soil to skin
ABS	-	Dermal absorption fraction

4.7.4 Inhalation intakes

Inhalation intakes can be estimated for the following pathways:

- inhalation of vapours in indoor air
- inhalation of vapours in outdoor air
- inhalation of dust particles.

The mechanism for deriving the vapour and dust concentration in air for the above pathways is described in an earlier section of this Schedule. The quantification of intakes or exposures via inhalation can be undertaken on the basis of an intake (US EPA 1989) or an exposure concentration (US EPA 2009).

The equation for inhalation intakes is published as follows and detailed in Table 3.

$$\text{Inhalation intake (mg/kg-day)} = \frac{C_A \times InhR \times ET \times B \times EF \times ED}{BW \times AT}$$

The estimation of intake as an exposure concentration can also be undertaken in accordance with guidance from US EPA (2009). Although the exposure point estimation is complex, the estimation of intake is simple. The equation for intake estimation in indoor air is given below, adapted from the US EPA (2009), and the variables are described in Table 3. For threshold chemicals, EC, the estimated exposure concentration, is compared to the inhalation specific toxicity reference value for assessment of chronic or subchronic risks. For non-threshold chemicals, excess cancer risk is calculated by multiplying the URF by the EC. Note that a method for the assessment of acute exposure risk is also provided, in which the CA is compared directly to the appropriate toxicity reference value. When sourcing inhalation toxicity reference values using the hierarchy of data sources listed in Table 4, the inhalation value may be an air quality guideline.

$$\text{EC } (\mu g/m^3) = \frac{C_A \times ET \times B \times EF \times ED}{AT}$$

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Table 3. Variables description for vapour inhalation intake calculation

Variable	Units	Description
InhR	m^3/hour	Inhalation rate relevant for receptor and activity
ET	hours	Exposure time
B	--	Bioavailability
EC	µg/m^3	Exposure concentration (a time-weighted average concentration)
C_A	µg/m^3	Concentration in air (exposure point concentration which has been measured or modelled)
ET	Hours/day	Exposure time
EF	Days/year	Exposure frequency
ED	years	Exposure duration
BW	kg	Body weight
AT	hours or days	Averaging time

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4.8 Unique considerations in exposure modelling

4.8.1 Blood lead modelling

There is a substantial body of evidence that links environmental exposure to lead to uptake into the blood stream. The impact of lead on cognitive processes, especially of children, is also well understood. Several studies have been undertaken at Port Pirie in South Australia showing the relationships between maternal blood lead and pregnancy outcome and children's abilities at various age groups following environmental exposure to lead (e.g Country Health SA 2007 at <www.publications.health.sa.gov.au/envh>). Studies suggest that an increase of 10 µg/dL of lead in blood (PbB) can lead to an IQ decrease of 1 to 5 points and recent studies show that there may be no lower threshold on the effect of lead in blood (ATSDR 2007).

A risk assessment technique has been developed to assess the uptake of lead into the blood, which effectively applies an *a priori* uptake factor to an estimated dose to estimate blood lead concentration. The US EPA integrated exposure uptake biokinetic model (IEUBK) for lead in children is widely known and used in this respect. In addition, the US EPA adult lead methodology may be an appropriate tool to assist in the assessment of adult lead exposures. Both of these tools are available at

< www.epa.gov/superfund/lead/products.htm>.

This approach with the appropriate justifications is considered suitable at Tier 2 for assessing risks from lead.

The US EPA continues to develop a research-oriented biokinetic model, the all-ages lead model, which is designed to potentially replace the integrated uptake biokinetic model for lead in children and includes a full population age range (0 – 90 years) and updated uptake and biokinetic modules. This model is, however, still under peer review and has not been formally approved for application in contaminated land assessments. Information on the all-ages lead model is available at:

<http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=139314>.

4.8.2 Bioavailability and bioaccessibility

In contaminated land health risk assessment, we are commonly estimating the intake of a chemical in soil, and comparing this to a tolerable daily intake or reference dose. The intake of contaminant is estimated from the intake of soil, using soil concentration data which nominally represents the 'total' concentration of the substance in soil. The toxic effect of a contaminant actually depends upon the 'uptake' or absorbed dose of contaminant, that is, the amount that gets into the bloodstream after being swallowed or inhaled. Bioavailability is a generic term meaning the proportion of the intake of a substance which is absorbed into the body. The rate at which the substance is absorbed into the body is also often known as 'bioavailability'.

The literature on bioavailability rarely discusses soil specifically, and literature definitions of the term 'bioavailability' are variable. In contaminated land risk assessment it is useful to separate 'bioavailability' into two distinct elements:

- Is the substance able to move from the soil into the gut or lung? (this is often referred to as the oral or inhalational *bioaccessibility*)
- Once released from the soil, is the substance able to enter the bloodstream and be taken up by the body organs? (we refer to this as *bioavailability* because this is generally what toxicological texts mean by it, as they are not usually referring to soil contaminants).

The TDI has usually been derived from animal experiments, and will generally apply to intake of the pure substance, rather than from the substance in soil (note that this should be reviewed for all chemicals of concern to determine relevance).

Since TDIs are generally derived from direct oral or inhalation administration of the chemical to an animal (or humans, in relatively few cases), they intrinsically account for *bioavailability* as defined above. However, because they rarely, if ever intrinsically account for the soil matrix, *bioaccessibility* is not accounted for.

It is usually not necessary to account for bioavailability (as defined above) in the oral and inhalation pathways, because the TRV incorporates it already. The derivation of the TRV should be understood, with reference to how the experimental dose was administered. In cases where doses have been injected, for example, there would be case for introducing a factor to represent the bioavailability of the substance when administered orally or by inhalation.

The dermal pathway has a well-established mechanism for considering bioavailability because lack of dermal reference doses means that the dermal dose is compared to the ingestion reference dose. The dermal dose is estimated by applying a factor ABS (dermal absorption fraction described elsewhere in this Schedule) to modify the applied dose in soil to calculate the absorbed dose. It represents the proportion of the contaminant in soil which is considered to be absorbed into the bloodstream through the skin.

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Because the dermal dose then represents the absorbed dose rather than applied dose it is necessary to modify the TRV because it is based on data that uses applied dose rather than absorbed dose. This is done by applying the gastro-intestinal absorption factor (GAF) to the TRV, which reduces the toxicity reference value by the bioavailability of the substance.

In the oral and inhalation pathways, it should in theory always be reasonable to introduce a factor to allow for bioaccessibility, since this is almost never intrinsically part of the reference dose. Unfortunately, data are limited and so it is not appropriate to introduce this factor except where more generic bioavailability values are available which at present is limited to arsenic and lead.

Bioaccessibility of contaminants in soil is complicated, highly variable and difficult to predict. This is because it depends strongly on the nature of the soil matrix (for example, organic carbon, potential particle size etc.) and on environmental conditions, particularly redox potential. HILs are derived using 100% bioaccessible assumptions with the exception of lead and arsenic. This is because bioaccessibility is variable and not readily predicted on a generic basis.

The UK developed and validated a physiologically based extraction test (PBET) methodology for testing oral bioaccessibility of arsenic in soil (Environment Agency & British Geological Survey 2002a, 2002b) and used it to permit derivation of an adjustment factor for arsenic on a site-specific basis. The PBET method is also widely used to estimate bioaccessibility of lead (for example, Ruby 2004). The US EPA (2005b) found good correlation between bioaccessibility tests and in-vivo bioavailability studies for lead. It is generally accepted that use of PBET tests has limitations, and that the results are prone to be very variable between sites.

This means that it is not considered possible to estimate bioaccessibility factors that can be applied generically to a substance. It is also likely that use of a single PBET test method for many metals will produce results of varying reliability for different metals.

A detailed review was carried out by Ng et al. (2010) as part of the review of this NEPM. It concluded that physiologically based extraction procedures were acceptable for use at Tier 2 to estimate the bioaccessibility of arsenic and lead. Currently it is considered that there is no reliable in-vitro method for any other contaminant; however, further research may provide adequate validation in future.

In-vivo methods are available and are likely to be more reliable and less conservative than in-vitro methods; however, these are expensive and not generally likely to be practical for the resolution of contaminated land issues.

It is recommended that site-specific assessment of bioaccessibility for As and Pb be carried out using in-vitro tests. The recommended methods are either the solubility bioavailability research consortium (SBRC) in-vitro assay (Kelley et al. 2002) or the PBET in-vitro assay (Ruby et al. 1996).

There do not appear to be any validated analytical methods for estimating inhalation bioaccessibility and given that chemical vapours tend to cross the lung membranes fairly easily, assuming 100% bioaccessibility is probably appropriate.

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4.8.3 The approach to total petroleum hydrocarbons

Petroleum products have a high degree of variability in their physical properties and chemical compositions. Products such as gasoline, diesel, fuel oil and jet fuel each have their own chemical signatures and the composition of the same product can vary depending on where it was distilled and the source of its crude oil. This makes environmental assessment of these products difficult and an approach has been developed that can assess the broad and varying range of compounds in a uniform manner.

Internationally recognised publications on the composition and assessment of petroleum hydrocarbons are available from the TPH Criteria Working Group (TPHCWG 1997a, 1997b, 1998). These documents present criteria for breaking total petroleum hydrocarbons (TPH) down into aromatic and aliphatic fractions with associated data available on the physical chemistry and toxicity of each fraction. It is based on choosing a relevant substance in each fraction and assuming that all the chemicals that are included in that fraction have the same toxicity as the surrogate chemical.

The TPHCWG approach was developed to provide a consistent and transparent method for dealing with petroleum hydrocarbons in risk assessment. The alternative to adopting the TPHCWG approach is either to assume that the risks from petroleum mixtures can be adequately assessed using indicator substances such as benzene and benzo(a)pyrene, or to attempt to analyse for individual substances and assess each one separately.

The former approach is practical; however, it creates problems in determining transparent clean-up criteria for the bulk of the TPH, since absence of benzene and benzo(a)pyrene does not obviously ensure absence of risk. The latter approach would be rigorous, but given that there are thousands of compounds in TPH mixtures, it would not be practical. The TPHCWG method is therefore the recommended approach.

Analytical data for soil, groundwater and phase separated hydrocarbons can be obtained on the aromatic-aliphatic composition of TPH compounds in terms of carbon numbers. If these are grouped into their corresponding TPHCWG fractions, concentrations representing each group can be modelled as source term input values.

TPHCWG does not provide inhalation reference values for carbon numbers greater than C_{16}. With respect to the inhalation pathway for TPH C_{16}-C_{36} (aliphatic and aromatic fraction), TPHCWG (1998) states that:

'[t]here are no appropriate data available for the development of RfCs [inhalation reference concentrations] in this carbon range. Also, the development of an inhalation RfC from this fraction was determined to be inappropriate because the compounds in this carbon range are not volatile and inhalation will not be a relevant exposure pathway'.

The Cooperative Research Centre for Contamination Assessment and Remediation of the Environment (CRC CARE) has drafted a technical report on the development of health screening levels (HSLs) for petroleum hydrocarbons in soil and groundwater (Friebel & Nadebaum 2009). In terms of soil contamination, the document provides HSLs for benzene, toluene, ethyl benzene, xylenes, naphthalene, benzo(a)pyrene and TPH fractions (C_6-C_{10}, >C_{10}-C_{16}, >C_{16}-C_{34} and >C_{34}) for different soil types (sand, silt and clay), land-use scenarios (residential, commercial/industrial, recreational and maintenance workers) and depths (0 – <1 m, 1 – <2 m, 2 – <4 m and >4 m). These TPH HSLs were derived using TPHCWG aromatic and aliphatic fractions.

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The CRC CARE document (Friebel & Nadebaum 2009) provides useful screening levels for sites where the key contaminants are petrol or diesel, and where phase separated hydrocarbon is not present. In these circumstances, the HSLs are equivalent to HILs and may be used in the same way. Where other products (for example, aviation fuels, fuel oils, kerosene) are present, analysis of the aromatic and aliphatic fractions separately is necessary to determine the composition, since this can have a significant impact on the risk.

Where phase separated hydrocarbon is present, a site-specific assessment including analysis of the aromatic and aliphatic fractions is likely to be necessary.

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5 Toxicity assessment

5.1 Introduction

Toxicity assessment is typically divided into two activities:

- *hazard identification* – the process of understanding the health effects that contaminants can have
- *dose-response assessment* – the process of making a quantitative link between the degree of exposure to a chemical and the effect realised.

These descriptors can have widely varying meanings, depending on the scope and purpose of the risk assessment. The sections herein describe the processes as they apply to assessing risks from contaminated land, with particular focus on site-specific decision making.

Further and more general guidance on toxicity assessment is provided in enHealth (2010).

Ideally, such assessments need to be undertaken by an appropriately qualified toxicologist. This is definitely required if any attempt is made to develop a TRV directly from toxicity data in the literature rather than use a TRV already developed by a government authority.

5.1.1 Sources of toxicity information

Toxicity assessment in contaminated land risk assessment is primarily a literature-based research exercise. For contaminants which have an HIL, the review results are presented in Schedule B7. In many risk assessments, reference to the appropriate review in Schedule B7 will provide adequate information to inform the toxicity assessment. In cases where no HIL is presented, or where the risk assessor is aware that more recent information is available, the toxicity review should be compiled and reviewed by an appropriately qualified professional from reliable peer-reviewed sources.

In principle, risk assessments would ideally be based on research that has been carried out, peer reviewed and recommended by Australian health authorities as appropriate for Australian circumstances. In practice, there is limited Australian-specific information available, and Australian health standards for air quality and drinking water (for example, NEPC 2004; NHMRC 2004) are also largely based on international data sources, in particular, WHO publications.

There are a number of readily available web-based authoritative sources of toxicity information, which are designed for the purpose of informing risk assessments. In general, published Australian data and the classification of carcinogens as reported by the International Agency for Research on Cancer (IARC) should be used in risk assessments when available, but other data may be used where appropriately justified. Data sources listed in Table 4 are considered to provide information which is compliant with Australian requirements for setting public health standards. They should generally be referred to in the order listed in Table 4; however, where there are good reasons (such as relevance of studies and currency) to select one source over another, these should prevail over rigid application of the hierarchy with the appropriate justifications.

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Table 4. Sources of information for toxicity assessment

Reference	Description
1. **World Health Organization** sources, for example, WHO air quality and NHMRC drinking water quality guidelines, and Environmental Health Criteria series documents. Documents from the International Programme on Chemical Safety (IPCS) and Joint FAO/WHO Expert Committee on Food Additives (JECFA) are included in this group.	Australia is a party to the WHO process and has incorporated Australian material into a variety of Environmental Health Criteria documents. Australian drinking water guidelines (ADWGs) use WHO guidance as a primary resource. Most WHO documents are available on the web at <www.inchem.org>.
2. International Agency for Research on Cancer (IARC) documents	IARC provides classification for carcinogens. The IARC classifications should be the primary source for carcinogen classification and should generally be used to determine whether chemicals will be considered as carcinogens for the purposes of the risk assessment.
3. National Health and Medical Research Council documents e.g. Australian drinking water guidelines (ADWGs)	NHMRC documents are a useful source of Australian-specific information. ADWG in particular should be checked to ensure that Australian circumstances are understood.
4. National Environmental Protection Council documents e.g. NEPM (Air Toxics) and NEPM (Ambient Air Quality)	As above, provide useful information on Australian circumstances, although for a more limited range of contaminants than ADWGs. NEPC publications can be accessed at <http://www.ephc.gov.au> The fifth national workshop on the health risk assessment and management of contaminated sites was published by NEPC and is available at <http://www.ephc.gov.au/taxonomy/term/60>
5. Other Australian Government sources of toxicity criteria	The Australian Government publishes acceptable daily intakes and acute reference doses for agricultural and veterinary chemicals. These can be accessed at <http://www.health.gov.au/internet/main/publishing.nsf/Content/ocs-adi-list.htm> <http://www.health.gov.au/internet/main/publishing.nsf/Content/ocs-arfd-list.htm>.
6. South Australia Health	South Australia Health published a series of workshops on risk assessment of contaminated land (1st to 4th National Workshops on the health risk assessment and management of contaminated sites, between 1991 and 1996). They can be accessed at <http://www.publications.health.sa.gov.au/envh/> This website also provides a number of other publications that are useful for risk assessments.
8. US Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels and toxicological reviews	ATSDR information is often based on the same sources as the IRIS database, but is independent of the US EPA. Its toxicological reviews are available on the web. ATSDR provides minimal risk levels (MRLs) for threshold effects which can be used in the same way as RfDs. MRLs can be accessed at <http://www.atsdr.cdc.gov/mrls/index.html>.

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Reference	Description
9. Other governmental sources of information on chemicals and risk assessment, e.g. NICNAS priority existing chemical reports, US EPA IRIS database, UK, Dutch and New Zealand guidance	Useful for general toxicity information, and may provide support data such as bioavailability/ bioaccessibility, estimates for background concentrations, methods for considering groups of chemicals, mixtures, cumulative effects etc. Should be accompanied by robust justification. Toxicity criteria should only be derived from these when none are available from any source listed above.
10. Other sources of peer reviewed toxicity criteria including other US EPA sources such as the regional screening levels, the PPRTV or HEAST tables on which the regional screening values are based or state-based US agencies such as California EPA, OEHHA etc.	Other sources of toxicity criteria suitable for use in risk assessment include US EPA's provisional peer reviewed toxicity values (PPRTV), California Environmental Protection Agency (Cal EPA) toxicity values, and the US EPA Superfund health effects assessment summary tables (HEAST).
11. Peer reviewed journals	In general single source papers from journals should not be used to derive toxicity information for contaminated land risk assessment. They do not normally comply with the requirement for having carried out extensive literature review, and budgets for site-specific risk assessment will not normally support such review. Where information sourced directly from journals is used, robust justification should be provided.

5.1.2 Sources of physical and chemical data

Physical and chemical data are necessary inputs to the exposure modelling process, and also provide useful information on fate and transport of the contaminant. These data are generally experimentally derived, and some values may vary considerably between sources. The source of physical and chemical data should always be quoted in risk assessments, and the effects of uncertainty considered. As an aid to consistency and transparency in Australian risk assessment, the following sources, in order, are recommended for the selection of physical and chemical data. These sources are those used to derive the HILs — see Schedule B7.

- ORNL (Oak Ridge National Laboratory) risk assessment information system, <http://risk.lsd.ornl.gov/index.shtml> (this database of physicochemical factors was completely reviewed in 2009 and so the outputs are now more consistent/reliable)
- ATSDR (Agency for Toxic Substances and Disease Registry) toxicological profiles
- Peer reviewed journals.

5.2 Hazard identification

Hazard identification is defined by enHealth (2010), following Health Canada (1999) as the process of determining:

- what types of (adverse) health effects might be caused by the agent (contaminant)
- how quickly the adverse health effects might be experienced and their duration.

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In contaminated land health risk assessment, these will generally be determined with reference to the data sources listed in Table 4. The health effects of a substance are likely to have been researched using a number of methods and the data sources adopt a weight of evidence approach in recommending which research to rely on. The reviews provided by the data sources are carried out by expert panels, and the level of expertise and effort expended in scrutinising the primary sources of information cannot generally be reproduced in a site-specific risk assessment. A brief explanation of the methods used to derive information on health effects is given here. The reader is referred to enHealth (2010) for more detail.

Information on health effects is usually generated by studies using animals (in-vivo studies), studies involving people (epidemiological studies) or laboratory experiments using cells or tissue rather than live animals (in-vitro studies). Relevant and reliable data on people are available for only a very few chemicals, and therefore most health effect information is derived by extrapolation from animal and laboratory experiments. This produces considerable uncertainty, since animals and people will not necessarily have similar responses to an agent.

Further uncertainty in contaminated land health risk assessment is introduced by the fact that toxicological research usually focuses on evaluating a specific substance, and does not account for the complexities introduced by the substance's presence in soil or in mixtures of substances.

Health effects can be broadly separated into acute and chronic effects. The distinction between acute and chronic exposure risks relates to the duration of exposure and timing for the onset of any health effects. Acute health effects occur within minutes, hours or days of a relatively short period of exposure, whilst chronic health effects occur as a result of prolonged or repeated exposures over many days, months or years and symptoms may not be immediately apparent.

5.2.1 Acute effects

Acute toxicity information on chemicals is widely available because it is used for the classification of manufactured chemicals for supply. Studies of acute effects using animal experiments may produce information on oral, dermal and inhalation toxicity, skin and eye irritation and skin sensitisation.

Standard protocols are available; for example, OECD test guidelines (OECD 1998), resulting in a high degree of standardisation of available information. Acute reference doses are also available for some substances from the Australian Government (for example, pesticides – Table 4) and from the US EPA's IRIS database. Another source of guidelines covering acute effects is the US NOAA public exposure guidelines which can be found at:

<http://response.restoration.noaa.gov/topic_subtopic_entry.php?RECORD_KEY%28entry_subtopic_topic%29=entry_id,subtopic_id,topic_id&entry_id(entry_subtopic_topic)=659&subtopic_id(entry_subtopic_topic)=24&topic_id(entry_subtopic_topic)=1>.

The soil HILs, and most contaminated land risk assessments, focus on chronic effects. This is because in most circumstances soil contamination capable of causing acute health effects would be self-evidently unacceptable. However, acute effects can be important during remediation works (for example, inhalation of vapours resulting in dizziness) and should not be ignored in hazard identification.

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There can also be potential for physical hazards, for example, fire, explosion, and subsurface gas accumulation leading to asphyxiating atmospheres. These are managed by techniques beyond the scope of this Schedule; however, the hazard identification process should acknowledge them.

5.2.2 Chronic threshold effects

Chronic threshold effects cover all kinds of chronic toxicity other than cancer as well as those compounds that exhibit 'threshold' toxicity as described for non-genotoxic carcinogens. This may include sub-chronic effects (medium term, for example, less than 10% of a lifetime), effects on reproduction, or development of the foetus.

When assessing hazards, attention should be given to the derivation of threshold reference values and the relevance of that derivation to the environmental contaminants under consideration. Toxicity assessment should seek to identify hazards that are relevant to the form of the contaminant in soil, water or air and should not assume 'worst-case' toxicity on the basis of substances or exposure pathways that are implausible, for example, where the effect was based on an occupational study looking at the inhalation of metal fumes. Since the metal in soil would not be capable of causing fumes, this hazard would be inapplicable and could be reasonably discounted.

Hazard identification for these compounds should detail the following:

- the IARC classification (classification table presented in this Schedule)
- what kinds of chronic toxic effect might result, and what kind of studies this conclusion is based on
- differences in effects between oral, dermal or inhalation exposure
- whether there may be any specific susceptible groups in the population
- the reliability of the available information.

5.2.3 Chronic cancer effects

Chemicals may cause cancer in a wide variety of ways, termed modes of action. There is flexibility in defining modes of action, which can be described at almost any level of complexity, reflecting the extent of chemical-specific information available and the needs of the risk assessment (Clewell 2005; Lambert & Lipscomb 2007). It is now known that several modes of carcinogenic action exist, including mutagenicity, mitogenesis, inhibition of cell death, cytotoxicity with reparative cell proliferation, and immune suppression (US EPA 2005a; Butterworth 2007). Although it represents a simplification, for the purposes of risk assessment, cancer-causing chemicals (carcinogens) are generally divided into two types, genotoxic and non-genotoxic.

Genotoxic carcinogens are defined as chemicals for which there is adequate evidence of the potential to interact with and/or modify the functions of genetic material and which has the ability to induce tumours via a mechanism involving direct damage to DNA (Butterworth 1990; IARC 2006). For genotoxic carcinogens, it is assumed that no level of exposure is entirely safe and even at extremely low levels some damage to the genetic material may increase the chance of developing cancer.

This is known as a non-threshold (or linear) dose-response relationship and the application of a threshold — based on 'no observable adverse effect level' (NOAEL) — is not considered appropriate.

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Non-genotoxic carcinogens are chemicals that induce tumours via a mechanism which does not involve direct damage to genetic material (IARC 2006). For non-genotoxic carcinogens, it is assumed that a threshold dose can be determined below which no toxic or carcinogenic effects are seen (that is, a non-linear dose-response relationship can be established). A common approach for determining a safe dose for chemicals that exhibit a threshold or non-linear dose-response relationship is the selection of a NOAEL (or other point of departure) from relevant animal or human studies.

Complexity is added by the uncertainty in whether some chemicals are carcinogenic or not, leading to difficulty for the risk assessor in deciding which approach to consider. Different sources of information may provide different views on whether a substance is a carcinogen or not. The US EPA in particular regards a wide range of chemicals as potential carcinogens and this view is not always shared by health authorities in other countries. The IARC classification is summarised in this Schedule.

Table 5. IARC classification for carcinogens

Group	Description
1	Agent[1] is carcinogenic to humans
2A	Probable human carcinogen, an agent for which there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals
2B	Possible human carcinogen, an agent for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals
3	Not classifiable as to their carcinogenicity to humans
4	Probably not carcinogenic to humans
Note: 1.IARC defines an agent as 'specific chemical groups, groups of related chemicals, complex mixtures, occupational or environmental exposures, cultural or behavioural practices, biological organisms and physical agents.. (IARC 2006)	

For contaminated land health risk assessment, it is recommended that the IARC classification (IARC 2006) should be applied as follows:

- categories 1, 2A and 2B should be treated as carcinogens
- category 3 substances can only be treated as non-carcinogens given the paucity of information but it should be recognised that this classification doesn't mean cancer is ruled out and it is subject to change
- category 4 should be treated as non-carcinogens.

In some circumstances, a chemical may have a carcinogenic classification but lack toxicity criteria to assess the cancer risk. In these circumstances, it is recommended that the chemical be assessed using a threshold approach (that is, ignoring the cancer risk) in modelling. The possibility of cancer effects should be identified and evaluated, even though a numerical expression of risk cannot be derived.

There may also be circumstances where the data relating to the classification of a substance as a carcinogen is not relevant to soil contamination. This would apply, for example, where the cancer classification was based on an occupational study where inhalation of metal fumes had been found to cause cancer. Since the metal in soil would not be capable of causing fumes, this hazard would be inapplicable and could be reasonably discounted.

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Hazard identification for carcinogens should detail the following:

- the IARC classification
- whether the chemical is considered genotoxic or non-genotoxic
- whether a threshold or non-threshold approach is to be used, and the reasons for the choice
- what kinds of cancer might result, and what kind of studies this conclusion is based on
- differences in effects between oral, dermal or inhalation exposure
- whether there may be any specific susceptible groups in the population
- the reliability of the available information.

5.3 Dose-response assessment

5.3.1 Overview

The dose-response assessment looks at establishing a quantitative relationship between the exposure to a chemical and the effect realised. For the purposes of contaminated land health risk assessment, dose-response assessment comprises selection of suitable reference values from the authoritative data sources listed in this Schedule.

The term toxicity reference value refers to measures of tolerable dose which are derived by expert panels of behalf of government or international bodies responsible for public health standards. The TRVs recommended have been developed for use in risk assessment or in setting public health standards. They incorporate allowances for uncertainty in the studies upon which they are based, and generally also accommodate (where possible) safety factors to provide for particularly sensitive groups in the population. The information provided to support the TRVs invariably provides a description of the process followed.

In health risk assessment, chemicals have generally been divided into two groups that are assessed by different methods (threshold and non-threshold). The methodological distinction was made on the basis that genotoxic carcinogens were considered to have no exposure level beneath which no effect would be realised (termed 'non-threshold') whereas non-carcinogens (and carcinogens with non-genotoxic effects) were considered to have a 'threshold' exposure below which there would be effectively no health risk. This approach has been the basis of the US EPA's risk assessment methodology (US EPA 1989, 2005), and consequently tends to dominate the availability and nature of dose-response data.

It is recognised that these approaches have significant limitations, and that a more unified approach would be sensible. Concern has also been expressed in the international risk assessment community that the current approach both underemphasises non-cancer effects, and provides an unrealistic 'bright line' divide between possible harm and safety (National Research Council 2008). Future developments in dose-response assessment are likely to move towards a unified approach covering all chemicals, and perhaps a single mechanism to express risk.

Currently, the threshold and non-threshold approaches still dominate risk assessment practice, and the TRVs associated with each are described below.

5.3.2 Threshold toxicity reference values

The US EPA's IRIS database describes the toxicity criteria for 'threshold' substances as follows:

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'Oral reference doses and inhalation reference concentrations (RfDs and RfCs, respectively) for effects known or assumed to be produced through a nonlinear (possibly threshold) mode of action. In most instances, RfDs and RfCs are developed for the non-carcinogenic effects of substances'.

For threshold chemicals, TRVs reflect a measure of tolerable daily exposure. The threshold is typically selected from a point of departure in the dose-response curve below which adverse effects are not observed (NOAEL) and includes a range of safety (or uncertainty) factors. There are a number of terms that are used by different agencies to define a threshold TRV. Most commonly these include an ADI (acceptable daily intake), TDI (tolerable daily intake), TC (tolerable concentration in air), RfD (reference dose), RfC (reference concentration), MRL (minimal risk level) and REL (reference exposure level) as noted in Table 6.

Table 6. Threshold toxicity reference values

Toxicity criterion	Units	Meaning
Acceptable daily intake ADI	mg/kg-day	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 (WHO 1994). The term ADI is generally used for chemicals such as pesticides which are deliberately used on food or crops. ADI is very similar conceptually to RfD; the different terminology arises from the measure having been defined by a different body.
Tolerable daily intake TDI	mg/kg-day	An estimate of the intake of a substance which can occur over a lifetime without appreciable health risk (WHO, 1994). TDI is generally used when a chemical is a food or environmental contaminant. Like ADI, TDI is conceptually similar to RfD.
Reference dose RfD	mg/kg-day	An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive sub-groups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used (IRIS). Generally used in non-cancer health assessments.
Reference concentration RfC	mg/m^3	An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive sub-groups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used (IRIS). Generally used in non-cancer health assessments.

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In contaminated land health risk assessment the estimated exposures from oral (ingestion), dermal and inhalation routes are compared to the corresponding TRV. This process is described in the risk characterisation section of this Schedule.

5.3.3 Cancer toxicity reference values

The US EPA's IRIS database describes the TRVs for 'non-threshold' substances that it presents as follows:

'Descriptors that characterize the weight of evidence for human carcinogenicity, oral slope factors, and oral and inhalation unit risks for carcinogenic effects. Where a nonlinear mode of action is established, RfD and RfC values may be used'.

For non-threshold chemicals, TRVs reflect a cancer risk value commonly referred to as a cancer slope factor (CSF) or unit risk factor (URF).

CSFs are typically calculated for genotoxic carcinogens (that is, non-threshold compounds). A slope factor is a plausible upper-bound estimate of the probability of a response per unit of intake of a chemical over a lifetime. The slope factor is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen (US EPA 1989). This approach is also known as the 'linear' approach which implies a proportional (linear) relationship between risk and dose at low doses. CSFs assume that there is no level of exposure to carcinogenic chemicals that does not pose a finite probability, however small, of generating a carcinogenic response.

A URF is an expression of carcinogenic potency in concentration terms, such as probability of cancer per 1.0 µg/L of drinking water or probability of cancer per 1.0 µg/m^3 or ppm in air. Generally, the drinking water URF is derived by converting a CSF from units of mg/kg-day to units of µg/L, and an inhalation URF is developed directly from a dose-response analysis using equivalent human concentration already expressed in units of µg/m^3 (US EPA 2005a). Derivation of URF often assumes a standard intake rate (for example, inhalation of 20 m^3 of air per day or ingestion of 2L of water per day) and body weight (for example, 70 kg). When a theoretical upper-bound cancer risk estimate is calculated using a URF instead of a CSF, it is often termed the unit risk.

Unit risk factors can be converted to slope factors as follows:

CSF(inh) = UR(inh) x 1000 x 70/20

where

CSF (inh) = inhalation cancer slope factor (mg/kg-day)

UR(inh) = inhalation unit risk factor (reported as per µg/m^3) which is converted to a CSF assuming 70kg body weight and 20m^3/day inhalation volume.

Benchmark doses are typically calculated for carcinogenic substances that are considered to be non-genotoxic (that is, threshold compounds). A benchmark dose is defined as the dose that corresponds to a specified change in adverse response compared to the response in untreated animals (Crump 1995).

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The dose is associated with a given incidence (for example, 1%, 5% or 10% incidence) of effect, the benchmark risk, based on the best fitting dose-response curve in the region of the dose-response relationship where biologically observable data are available (Filipsson et al. 2003; enHealth 2010). Although the benchmark dose has mainly been used for risk assessment of non-cancer end-points, this approach can also be applied for cancer end-points. When selecting benchmark doses, recent Australian guidance recommends adopting a 10% incidence (NEPC 2009).

Increasingly, the benchmark dose approach for cancer risk assessment is being adopted globally in recognition of the identified difficulties and uncertainties associated with the low-dose extrapolation method (that is, generation of cancer slope factors). A benchmark dose is applied in the same way as a threshold TRV.

There is often a choice to be made in selection of dose-response data for carcinogens. Recommendations are summarised below. For a more detailed treatment of the methodologies applied to assessing cancer risks, refer to the review of cancer risk methodologies at <www.ephc.gov.au>.

- Benchmark dose data where available from the listed sources should be adopted in preference to cancer slope factors or unit risks.
- Where appropriate benchmark dose data are not available, cancer slope factors (for genotoxic carcinogens) and threshold TRVs (for non-genotoxic carcinogens) should be used. When using threshold TRVs for a substance with an IARC classification of 1, 2A or 2B, care should be taken to check that the carcinogenic effects were considered in the derivation of the threshold TRVs.
- In the event that the threshold TRVs does not consider carcinogenic effects, and no other cancer toxicity criterion is available, the substance should be assessed using the available threshold TRVs, noting the potential additional cancer risk as a weight-of-evidence factor for decision making purposes.
- The process for choosing appropriate dose-response data for carcinogens in site-specific risk assessments is illustrated in Figure 4 of the review of cancer risk methodologies (on the EPHC website: <www.ephc.gov.au>).

5.4 Other considerations in toxicity assessment

5.4.1 Absence of information

It will not normally be necessary for risk assessors to derive toxicity reference values for use in site-specific risk assessment. This should only be considered when criteria have not been developed by sources listed in this Schedule and should only be undertaken by a suitably qualified toxicologist.

In cases where the potential site contaminants (that is, known or suspected to be present in the site soil) have no TRVs, but are not detected above the lowest detection limit that can be achieved using available analytical methodology, it is considered acceptable to assume that no additional assessment is necessary. Note that 'available' in this context would include internationally available. The extent of effort expended in procuring a suitable analysis should be proportional to the probability of the contaminant's presence and severity of suspected toxic effects.

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Where derivation of new TRVs is required, an extensive and robust literature review undertaken by appropriately qualified scientists is expected. The procedure for establishing toxicity reference values is set out in enHealth (2010) and should be followed. Note that the process requires a high degree of expert judgement.

5.4.2 Early-life susceptibility

Special consideration has been advocated by the US EPA for assessing risks associated with early-life exposure to mutagenic carcinogens (refer to enHealth 2010 and US EPA 2005 for further information). US legislation also mandates the application of an additional 10x safety/uncertainty factor in the derivation of an RfD for pesticides where studies indicate developmental neurotoxicity or other toxic effects that could be associated with early-life susceptibility.

With respect to non-threshold reference values, the US EPA guidance recommends that individual compounds are assessed to determine whether there is a mutagenic mode of action and whether the potential for early life-time susceptibility should be considered. Where relevant, the following can be applied:

- If chemical-specific data on susceptibility from early-life exposures are available and incorporated within the derived TRVs (e.g. the oral slope factor for vinyl chloride), then these should be used where appropriate without any further adjustment.
- If chemical-specific data is not available, then adjustment factors are applied to the calculation of risks associated with early-life exposures. The adjustment factors include a ten-fold adjustment for exposures during the first 2 years of life, a three-fold adjustment for exposures from ages 2 to less than 16 years of life and no adjustment for exposures for ages 16 years and older.

While Australian environmental health authorities have not enunciated specific policies relating to applying these US early-life risk assessment strategies, additional precaution tends to be applied on a case-by-case basis when justified by relevant data. In other words, the US early-life risk assessment policies are not automatically adopted in Australia.

5.4.3 Metal speciation

A chemical 'species' is the specific form of an element defined by its oxidation (valency) state and/or complex or molecular structure. Some of these structural levels are more important for risk assessment than others. In particular, valency state and inorganic and covalent organometallic speciation are of great importance in determining the toxicity of metals and metalloids (WHO 2006a). Elements occur in soil in either the solid phase or in the soil solution. In the solid phase, ions can be bound to soil components by means of ion exchange or surface complexes or they can occur as minerals or be co-precipitated as minerals in soil. In the soil solution they can occur as free ions or complexes.

Standard chemical analysis provides a measure of 'total' metal in soil or water, expressed as a concentration of the elemental form. This is not particularly informative as a means of assessing how toxic the soil or water could be.

Further difficulty is introduced because toxicological research rarely focuses on the metal species most likely to be present in soil. Typically, the focus is on the most toxic forms, and on those that are of particular health concern as a result of their presence in food, consumer products or in the workplace. This means that the available TRVs for metals and metal compounds may significantly overestimate the toxicity of the metals in soil and water.

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Some examples of variations in toxicity with chemical species include the following:

- Cr(III) is considered to have low toxicity while Cr(VI) is carcinogenic
- inorganic As(III) compounds are carcinogenic while arsenobetaine is essentially non-toxic
- inorganic tin (Sn) compounds are considered essential for plants and some animals but tributyltin (TBT) is an endocrine disruptor.

The chemical species of a metal can affect its toxicokinetics by influencing its absorption, distribution, biotransformation and elimination. It is therefore important that risk assessments should consider the species rather than the elemental constituent in order to create meaningful data.

Some typical elemental species in soil are summarised in Table 7.

Table 7. Elemental species that may influence the toxicity of elements in soil (WHO, 2006)

Element	Aerobic soil	Anaerobic soil
Arsenic	$Ca_3(AsO4)_2$, $Mg_3(AsO_4)_2$, As_2O_5	As_2S_3
Cadmium	$Cd(OH)_2$, $CdCO_3$	CdS
Chromium	$Cr(OH)_3$ (low to neutral pH)	$Cr(OH)_3$
Lead	PbO, $PbCO_3$, $Pb_3(CO_3)(OH)_2$	PbS
Mercury	$HgCl_2$, HgO, $Hg(OH)_2$	HgS
Nickel	NiO, $NiCO_3$, $Ni(OH)_2$	NiS

The speciation of metals in soil and water can be determined to some extent by chemical analysis; however, this is expensive and the data obtainable are limited. Assumptions regarding speciation will normally have to made using available understanding of the site conditions. It is recommended that where analytical information is not available, risk assessments considering metals should account for metal species using a reasonable worst-case assumption regarding the presence of toxic forms of the metal. TRVs should then be selected for relevance to the assumption presented.

The reasonable worst-case assumption should be derived from the following:

- knowledge of the soil type, organic carbon content and moisture content
- the pH and redox conditions
- the form that the metal was in when it was released to the environment.

Other indicators such as iron content and the presence of other contaminants which may influence metal speciation may also be useful.

The reasonable worst-case assumption should be to assume that the metal in soil is the most toxic form that could be present given the understanding of the conditions. Forms that are not stable under environmental conditions, or that would require implausible soil processes to produce, should be discounted.

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An alternative means of determining elemental speciation of aqueous solutions is through geochemical equilibrium speciation modelling. Examples of such models include MINTEQA2 (Allison, Brown & Novo-Gradac 1991) and PHREEQC (Parkhurst & Appelo 1999). Where reliable analytical methods cannot be found to conduct relevant elemental speciation analyses, geochemical modelling methods can be used to predict species in solution and phases that are likely to precipitate from solution. The accuracy of such methods is largely dependent on the accuracy of the input data. Such geochemical modelling methods are applicable to both soil solutions and aqueous environments.

Typical input data required to run geochemical models include basic geochemical field parameters (pH, electrical conductivity, reduction oxidation potential, dissolved oxygen and temperature), major ion chemistry (sodium, potassium, calcium, magnesium, ammonium, chloride, carbonate, bicarbonate, sulfate, nitrate, nitrite, phosphate), iron, manganese and the trace metal chemistry of the soil solution. The essential data needed to perform a speciation calculation are the temperature, pH, and the concentration of elements and element valency states.

Where there is reasonable evidence to suggest that a metal contaminant may be present as a species that is less toxic than the toxicity assumed by the HIL, analytical or modelling methods may be useful in characterising the geochemistry of the particular environment. In this way, a more accurate prediction of the toxicity of the metal contaminant can be made.

5.5 Introduction

The final step of the health risk assessment process is risk characterisation. In this step, information from the data collection, exposure and toxicity assessments is summarised and integrated into quantitative and qualitative expressions of risk. Risk characterisation conveys the risk assessor's judgement as to the nature and existence of (or lack of) human health risks, in an informative and useful manner for decision makers.

Risk characterisation can be considered a three step process:

- risk estimation
- risk evaluation
- sensitivity and uncertainty analysis.

The aim of risk characterisation is to:

- identify the key health endpoints that have driven the risk assessment
- provide a description of the assumptions used and the effect of these on the final risk estimate
- quantify risks from individual and multiple chemicals
- assess risks for each exposure pathway and for all the exposure pathways summed together
- describe the risks to individuals and populations in terms of extent and severity of probable harm
- provide a description of uncertainty and where uncertainty arose during each stage of the risk assessment process and the effects of these on the final risk estimate
- assess the sensitivity of the results to the input parameters
- communicate key risk information to the risk manager.

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5.6 General risk characterisation principles

There are a number of principles which form the basis for the risk characterisation process, including the following:

- Human health risk assessments should be undertaken according to methods outlined in this Schedule and appropriate supporting documents, e.g. enHealth (2010).
- Risk assessments should be transparent. The nature and use of default values and methods, assumptions and professional or policy judgements in the risk assessment should be clearly identified. Conclusions drawn from the evidence should be separated from professional or policy judgements.
- Risk characterisation should include a summary of the key issues and conclusions of each of the other components of the risk assessment, as well as describing the nature and likelihood of adverse health effects. The summary should include a description of the overall strengths and limitations (including uncertainties) of the assessment and conclusions.
- Risk characterisation (and risk assessments) should be consistent in general format, but recognise the unique characteristics of each specific situation.
- Risk characterisation is not complete unless a discussion of uncertainty and sensitivity is provided.
- Risk characterisation is a key component of risk communication.
- Health risk assessments must be undertaken with an appreciation that the health risk assessment is part of a larger assessment that also encompasses ecological risk assessment.
- To protect public health and the environment, an appropriate degree of conservatism must be adopted to account for uncertainties.
- Actions should always adequately protect public health and the environment, putting these responsibilities before all other considerations.

5.7 Risk estimation

Risk estimation combines the estimated intakes calculated in the exposure assessment with the TRVs (threshold and non-threshold where relevant) from the toxicity assessment to produce numerical indices of likely health effect. The risk estimation methodology differs for threshold and non-threshold compounds due to the different modes of chemical effect.

5.7.1 Threshold risk estimation

For threshold compounds, the intake for each exposure pathway is divided by the appropriate threshold TRV (allowing for intakes from other sources where relevant) to produce a simple ratio, termed a hazard quotient (HQ) or risk quotient (RQ). The HQs for all exposure pathways for each contaminant can be summed to produce a total hazard index (HI) or risk index (RI); however, there are limitations to this approach.

$$\text{Hazard quotient (HQ)} = \frac{\text{Intake (mg/kg/day)}}{\text{Threshold TRV (mg/kg/day)}}$$

$$\text{Hazard index (HI)} = \Sigma \text{ Hazard quotients}$$

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The HQs for all exposure pathways for all contaminants should be summed to produce a total HI, unless evidence is available to show this is not necessary. When summing these HQs, the following should be taken into consideration:

- HIs should be calculated separately for chronic, sub-chronic and shorter-duration exposures.
- HIs should be calculated separately for genotoxic (where benchmark dose data are available) and non-genotoxic groups of chemicals.
- Ideally, HIs should be categorised into groups of chemicals that induce the same type of effects or that act by the same mechanism of action. However, this process is not simple and requires a good understanding of the toxicology of the chemicals concerned and must only be undertaken by an appropriately qualified toxicologist. If this segregation is not performed carefully, an underestimate of the true hazard could result. When toxicological information is lacking or unclear, it should be assumed that the chemicals act by the same mechanism of action and hence summation of the HQs is appropriate.
- HIs should represent the exposure pathways that have the potential to expose the same individual or subpopulation, making sure to consider areas of highest exposure for each pathway for both current and future land uses. All exposure pathways should be summed unless information is available that indicates the same individual or subpopulation cannot be exposed by a particular pathway(s).

5.7.2 Non-threshold risk estimation

Where non-threshold TRVs are adopted (that is, assuming a linear low-dose relationship), risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the carcinogen. The estimated intake for each exposure pathway and non-threshold TRV are multiplied to produce pathway-specific estimates of increased lifetime cancer risks (ILCR).

However, for those carcinogens where appropriate benchmark dose data are available, the risk estimation method outlined above for threshold compounds applies.

ILCR = Intake (mg/kg/day)xTRV(mg/kg/day)$^{-1}$

ILCR = exposure concentration (mg/m^3)xTRV(mg/m^3)$^{-1}$

ILCR estimates from all pathways should be summed to produce a total increased lifetime cancer risk for each contaminant assessed. ILCR estimates should also be summed across different contaminants. When combining ILCR estimates, the US EPA (1989) identifies several limitations which should be considered.

These include:

- As each non-threshold TRV is an upper 95th percentile estimate of potency, and because upper 95th percentiles of probability distributions are not strictly additive, the total cancer risk estimate might become artificially more conservative as risks from a number of different carcinogens are involved.
- It will often be the case that substances with different weights of evidence for human carcinogenicity are included. The cancer risk equation for multiple substances sums all carcinogens equally, giving as much weight to class 2 as to class 1 carcinogens. In addition, non-threshold TRVs derived from animal data will be given the same weight as non-threshold TRVs derived from human data.
- The action of two different carcinogens may not be independent.

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In practice, it will often be the case that there is insufficient information to make a well-informed decision as to whether it is reasonable or not to sum ILCRs across either pathways or contaminants (see also Mixtures section of this Schedule.) It is recommended that a precautionary approach (set out below) should be followed under most circumstances. Where more information is available, a decision to assess contaminants or pathways as independent and non-additive should be supported with reference to the toxicology of the contaminants concerned by appropriately qualified toxicologists.

- ICLR estimates should normally be summed across pathways unless specific evidence is provided that the cancer end-points and/or modes of action of the contaminant are clearly different for different pathways and that the same person cannot be exposed by the different pathways.
- ICLR estimates should normally be summed across contaminants unless specific evidence is provided that the cancer end-points and/or modes of action of the contaminant are clearly different for different contaminants.
- ILCR estimates should only be summed where they relate to an exposed population that could plausibly be exposed to all of the contaminants / pathways that are added.
- It is recognised that synergistic (that is, more than additive) effects are possible; however, the practical difficulties of quantifying the synergy in a contaminated land risk assessment are significant. Unless evidence for synergistic effects is available, the potential for synergistic effects may be omitted from the assessment. Additive effects are much more common and are covered in risk assessment by summing risks across chemicals and pathways.

5.8 Risk evaluation

5.8.1 Threshold risk evaluation

The threshold HQ assumes that there is a level of exposure below which it is unlikely for sensitive populations to experience health effects. If the exposure level does not exceed the threshold (that is, HQ less than 1), then it is reasonable to conclude that no adverse health effects are likely to be realised. If the exposure level exceeds the threshold (that is, HQ greater than 1) then further consideration is necessary. A HQ greater than 1 does not automatically imply that an unacceptable risk is present but does indicate that further action(s) (investigation or risk management) are warranted.

It is generally considered that the greater the HQ, the greater the level of concern. HQ ratios should not be interpreted as statistical probabilities; for example, a HQ of 0.001 does not imply that there is a one in one thousand chance of the effect occurring (US EPA 1989).

5.8.2 Non-threshold risk evaluation- acceptable level of cancer risk

When using non-threshold TRV as dose-response criteria, the recommended acceptable incremental lifetime risk of developing cancer arising from exposure to carcinogens in soil is 1 in 100,000 (10^{-5}). The HILs were developed using 1×10^{-5} as an acceptable risk value.

The concept of 'acceptable' risk is subjective and variable; consequently there is no global consensus on a level of theoretical cancer risk that is considered 'acceptable' (Hrudy & Krewski 1995). The WHO (2000) considers that the decision on the acceptability of a risk should be made by national authorities within the framework of risk management. The drinking water guidelines recommended by NHMRC and WHO are associated with an excess cancer risk of 10^{-6} (NHMRC 2004) and 10^{-5} (WHO 2006) respectively.

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In the 1970s, the US Food and Drug Agency adopted a risk level of 1 in 1,000,000 (10^{-6}) as the incremental cancer risk for carcinogenic residues in foods that was considered to be 'essentially zero' (Kelly 1991). It is understood that the origin of this 'essentially zero' risk level was purely arbitrary and was applied to decision making about animal drug residues and not contaminated sites regulation.

However, since then, the 10^{-6} risk level has become commonplace in the regulation and management of environmental contaminants in soil with apparently no sound scientific, social, economic, or other basis for its selection (Kelly 1991). The concept of 'zero risk' is based on the assumption that only the absence of the chemical (zero exposure) poses no risk and depends on the ability to detect a chemical which becomes increasingly impracticable for ubiquitous environmental chemicals.

Although a 1 in 1,000,000 (10^{-6}) cancer risk has been the most frequently used target risk level for risk management decision making of environmental (including soil) contamination situations, many agencies identify a range of increased cancer incidence risks, ranging from 1 in 10,000 (10^{-4}) to 1 in 1,000,000 (10^{-6}) (WHO 2000; WHO 2006b). As discussed, the acceptable risk range depends on the situation and circumstances of exposure.

In the US, the 10^{-6} cancer risk level is applied as a 'point of departure'; final risk-based decision making considers other factors such as technical feasibility and economics. Therefore, final risk-based objectives may equate to a theoretical upper-bound cancer risk of significantly less than 10^{-6}. The Dutch intervention levels for soil are based on increased cancer risks of 10^{-4} (de Bruijn et al. 2001).

5.9 Risk evaluation of mixtures

Contaminated land studies frequently involve assessing the health risks associated with soil where a number of different chemical contaminants are present. In contrast, toxicological studies usually assess the effects of a single chemical. The risk assessor faces a difficulty in determining whether the effects of the mixture might be additive, greater than additive (synergistic) or less (antagonistic) (Priestly 2009). It is possible that such effects are not important at the low doses common in environmental exposure, leading to the concept of an 'interaction threshold' below which the effects of mixtures are insignificant (Hamm et al. 2005). Additive effects are being found to be more common than synergism. Despite the limitations in the data, risk assessors have been incorporating additivity into their assessments for some time. This is done by adding all risks from all chemicals together to get total risk for a site and also by adding risks from all pathways. There are likely to be situations where the chemicals cause quite different effects by quite different mechanisms and so it is possible that summing risks in this way can overestimate risks in such situations. However, most sites have a mix of chemicals that are similar because they have arisen due to the particular activities on a site.

Priestly (2009) has reviewed all the available approaches to the risk evaluation of mixtures including summing of risk quotients as already described, which are summarised below:

Hazard quotient approach uses the ratio of the estimated exposure to the measure of acceptable exposure (the hazard quotient) for each component of the mixture and adds them to produce a hazard index which is the expression of the likely acceptability of the mix. This approach is widely used in Australian risk assessment. It is recommended for the assessment of petroleum hydrocarbons by the UK Environment Agency (Environment Agency 2005).

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The approach suffers from a fundamental limitation, which is the inherent assumption that the components summed have a common mode of action, or at least a common end-point. Where this is not the case, the components are theoretically toxicologically independent.

This HQ approach is recommended as a screening tool for most Tier 2 risk assessments. It is particularly useful for the assessment of petroleum hydrocarbon mixtures, and other mixtures where the assumption that substances are likely to have similar toxicological effects can be justified.

Where the HI for the mixture is less than unity (HI<1), a conclusion that exposure is likely to be within acceptable bounds may be made. When HI>1 it does not necessarily indicate that a site poses an unacceptable risk but it does indicate that either further consideration should be applied, or risk management actions should be recommended. A HI >1 does not necessarily indicate unacceptable risk.

Further consideration would normally include assessment of the modes of action that the mixture components might exhibit. This Schedule provides guidance on how to apply the HQ approach.

Summation of non-threshold risk estimates (ICLRs) applies similar logic to the HQ approach. It is common to assess risk from genotoxic carcinogens in terms of a numerical estimate of increased probability of developing cancer over a lifetime. It is possible to sum these risks from components of a mixture to develop a combined estimate of total risk. The approach suffers from exactly the same limitation as the HQ approach, in that it assumes a similar mode of action or end-point, which may be unjustified.

This approach is recommended as a screening approach for all genotoxic carcinogens in a mixture. Where the sum exceeds the acceptable risk, the components should be assessed in more detail to look at whether the mode of action or end point justifies summing the risk. Where modes of action and/or end-points are clearly different, carcinogens should not be summed.

Assessment of representative mixtures by direct in-vivo or in-vitro experiments. This approach is clearly limited by the number of variations in relative concentration that could be tested, and also by other complexities of environmental contamination such as weathering and degradation. It is not likely that this approach will be widely applicable to contaminated land health risk assessment.

Toxicity equivalence factor (TEF) approach in which the potential effects of a group of similar substances are estimated relative to a single member of the group. The components of the mixture are assumed to contribute to the toxicity in a similar way, and their relative effect is calculated in proportion to their concentration in the mixture by adjustment using a relative potency factor. The approach has limitations in that the assumption of similar mode of action may be unjustified in a variety of ways. The key limitation in a practical sense is that it is only available where a well-established set of relative potency factors exists for the components of the mixture. Currently its use is limited to carcinogenic PAHs, dioxins, PCBs (polychlorinated biphenyls) and some endocrine disrupting chemicals. This approach is recommended for carcinogenic PAHs, and guidance on how to apply it is provided in the toxicity profile for benzo(a)pyrene in Schedule B7.

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Component elimination or simplification approach may be used in circumstances where it is not reasonable to assume a common mode of action for components in a mixture. In this case, the components are assumed toxicologically independent. The outcome of this approach is to assume that the overall risk is no greater than the risk posed by the riskiest component of the mixture. This method should be applied where it can be shown that adding HQs or ILCRs is not reasonable.

5.10 Uncertainty and sensitivity analysis

5.10.1 Uncertainty analysis

Uncertainty in health risk assessment is the lack of knowledge about the correct value such as a specific exposure measure or estimate (enHealth 2010). Uncertainty is distinguished from variability, which refers to true differences in attributes due to diversity or heterogeneity; variability cannot be reduced by further measurement or study, although it can be better characterised (NRC 2008).

Both uncertainty and variability contribute to uncertainty in the estimation of risk and should be adequately assessed in a risk assessment. Such consideration needs to be done transparently so that all users of a risk assessment can understand the approach taken.

An analysis of the uncertainty in the risk assessment is important because:

- Information from different sources carries different kinds of uncertainty and knowledge of these differences is important when uncertainties are combined for characterising risk.
- The risk assessment process, with management input, involves decisions regarding the collection of additional data (versus living with uncertainty). In the risk characterisation, a discussion of the uncertainties will help to identify where additional information/data could contribute significantly to reducing uncertainties in risk assessment.
- A clear and explicit statement of the strengths and limitations of a risk assessment requires a clear and explicit statement of related uncertainties (US EPA 1995c).
- Characterising uncertainty in risk informs the stakeholders about the range of possible risks from an exposure. Risk estimates may sometimes diverge widely (NRC 2008).
- Characterising the uncertainty in risk associated with a given decision informs the decision maker about the range of potential risks that result from the decision (NRC 2008).

Uncertainty analysis is generally a qualitative process; however, in some cases it can be semi-quantitative or quantitative.

The first step should be a consideration of the conceptual site model and what aspects of that model are uncertain and how that uncertainty has been accounted for.

The second most important part of the uncertainty assessment is an evaluation of the uncertainty and variability in the site characterisation data. Site characterisation data will always be limited by time, site constraints and budgets. However, the risk estimates based on even quite limited data can be fit for purpose if the exposure concentrations are a long way below (or above) toxicity reference values which indicate that the risks are either very low or very high. Decision making based on such uncertain but quite clear results is straightforward. Where risks are close to or slightly above unacceptable (the 'grey' zone), the issue of the uncertainty and variability in the site characterisation data becomes much more important and so the uncertainty assessment needs to be more detailed.

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When assessing risks, uncertainty can arise from missing or incomplete information, be incorporated into the scientific theory affecting the ability of a model to make predictions, and result from uncertainty affecting a particular parameter, for example, sampling errors. Such uncertainty has the potential to cumulatively overestimate or underestimate risk during an assessment. An assessment of uncertainty is a part of the health risk assessment process and consequently must be addressed for each step of the risk assessment and for its cumulative effect from all of the steps.

There are three broad types of uncertainty (US EPA 1992):

- *Scenario uncertainty* is uncertainty arising from missing or incomplete information such as descriptive errors, aggregation errors, errors in professional judgement, and incomplete analysis.
- *Parameter uncertainty* is uncertainty affecting a particular parameter such as measurement errors, sampling errors, variability, and use of generic or surrogate data.
- *Model uncertainty* is uncertainties in scientific theory affecting the ability of a model to make predictions.

NRC (2008) provides a detailed evaluation of the techniques currently provided for in US EPA guidance and concludes that although a number of usable methodologies are provided, it is unclear what level of detail is required to capture and communicate key uncertainties. A further comment is that quantitative methods suffer from the difficulty in sensibly quantifying all uncertainties, and that the apparent precision of quantitative analysis for some uncertainties may distract attention from other, possibly equally important but unquantifiable, uncertainties.

In most health risk assessments for contaminated land projects, it is unlikely that quantitative uncertainty analysis (for example, US EPA 2001) will provide value given the effort required to undertake it. A clear qualitative analysis is considered sufficient in most cases to provide the communication of the effects of uncertainty that is necessary.

Further discussion and guidance regarding uncertainty is provided in enHealth (2010). A useful example of an uncertainty analysis table is also provided in enHealth (2010). NRC (2008) and WHO (2008) provide useful guidance on the principles to be adopted for uncertainty analysis; these have been adapted for specific relevance to contaminated land risk assessment herein:

- Risk assessments should provide qualitative (as a minimum) or quantitative description of uncertainty and variability consistent with available data. The information required to conduct detailed uncertainty analysis may not be available in many situations.
- Sensitive sub-populations should be considered to the extent that they are not covered by the selected toxicity criteria (generally they will be).
- The uncertainty analysis should seek to communicate which uncertainties are most important to the conclusions of the risk assessment.
- The level of detail of the uncertainty analysis should be commensurate with the scope of the risk assessment.
- Uncertainty analysis should be expressed in terms that can be understood by the risk manager and other stakeholders.
- Uncertainty and variability should be kept conceptually separate.

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The combination of uncertainty in the scientific data and assumptions (the 'inputs') and inability to validate assessment results directly or to isolate and evaluate the impact of a resulting decision (the 'outputs') creates a situation in which decision makers, the scientific community, the public, industry and other stakeholders have little choice but to rely on the overall quality of the many processes used in the conduct of risk assessment to provide some assurance that the assessment is aligned with societal goals (NRC 2008).

5.10.2 Sensitivity analysis

Sensitivity analysis is a type of uncertainty analysis that aims to provide a level of quantification of the effects of uncertainty and variability in a model. It is a part of the uncertainty analysis described above, and should be undertaken when a risk assessment is conducted using a deterministic exposure model. Sensitivity analysis is capable of providing a quantitative estimate of uncertainty in input variables whose range can be reasonably estimated. It cannot accommodate model uncertainty, and its ability to analyse other uncertainties (as opposed to variabilities) is limited to the risk assessor's ability to quantify them. Sensitivity analysis should be undertaken in addition to uncertainty analysis. The general principles outlined above are applicable.

Sensitivity analysis is the process of changing one variable while leaving the others constant and determining the effect on the output. The procedure involves fixing each uncertain quantity, one at a time, at its credible lower-bound and then its upper bound (holding all other at their medians), and then computing the outcomes for each combination of values (US EPA 1992). It can be used to test the effects of both uncertainty and variability in input values.

Sensitivity analyses can be used to identify important input variables (or groups of variables) and develop bounds on the distribution of exposure or risk. A sensitivity analysis can also estimate the range of exposures or risk that result from combinations of minimum and maximum values for some parameters and mid-range values for others (US EPA 1989).

All risk assessments where conclusions are derived using modelling should incorporate a sensitivity analysis and describe the variability in the model outputs generated by plausible variation in the inputs. Note that some input variables may be connected and unable to vary independently. Monte Carlo models, where inputs are described by probability distribution functions, provide probability distribution function outputs. The Monte Carlo method reduces the requirement for sensitivity analysis but may not eliminate it, depending on the model used.

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6 Risk communication and management

Detailed guidance on risk communication and management is provided in Schedule B8 and the risk assessor must be aware of how the risk assessment will be used as this is an essential step following the conclusion of a risk assessment.

6.1 Risk communication

Risk communication is the process of informing people about potential hazards to their person, property, or community. From the risk management perspective, the purpose of risk communication is to help affected communities understand the processes of risk assessment and management, to form scientifically valid perceptions of the likely hazards, and in some cases to inform decisions about how risk should be managed. There should be a clearly defined functional separation between risk assessment and risk management. In the US, many stakeholders believe that the current process for developing and applying risk assessments lacks credibility and transparency (NRC 2008). Although there does not appear to be specific research on this issue related to contaminated land risk assessment in Australia, the same may well be true.

The investigation, management and remediation of contaminated sites may give rise to a range of community concerns. These may be based on actual or perceived environmental risks and loss of amenity or nuisance. When planning a communication strategy, the factors relevant to the timing and extent of consultation should be identified. The extent of communication will vary, but should include all stakeholders in the vicinity of the site who may be physically affected by the site assessment or remediation or by loss of amenity or nuisance, as well as those who may not be physically affected but have concerns about the contamination (the broader community).

There are a number of underlying principles that should be considered in risk communication. These have been set out in various publications, including *Core Values for the Practice of Public Participation* (International Association of Public Participation 2000), and propose that the community be provided with information and the opportunity to input to decisions which may affect their wellbeing. The community should also be advised about the proposed plan for risk communication. Planning for risk communication should therefore commence during the site investigation stage.

The communication plan should be flexible and identify and manage new information as it becomes available and provide ongoing dialogue with the community.

Guidance on risk communication is provided in Schedule B8, and by enHealth (enHealth, 2010), as well as by state regulators (for example, Western Australia Department of Environmental Protection's *Community Consultation*, 2002) and international agencies (US EPA 2007b).

6.2 Risk management

One of the key objectives of risk assessment is usually to support a decision about what to do about the contamination present on a site. For effective risk management, it is important that the potential risk management actions are considered at the planning stage (that is, issues identification). Failure to adequately plan is likely to result in a risk assessment that does not fully meet the needs of the risk manager and other stakeholders.

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Risk management decisions are often taken by a group of stakeholders (for example landowner, project managers, legal advisors and regulators), few of whom are expert in risk assessment. It is important that the results of risk assessment, including the consideration of uncertainty, are presented in a way that can be understood by non-specialists.

One of the key considerations in risk management is the extent to which remediation is needed in order to adequately mitigate the risk. Poor understanding of the risk assessment process and the inherent uncertainties can lead to lack of confidence on the part of stakeholders, often resulting in a decision to remediate to a higher standard than necessary (for example to the HILs).

Making a risk management decision requires informed consideration of the risks in the context of the site and broader stakeholder issues that may include practicality and community acceptability, cost-benefit, time scales for remediation, and technical feasibility.

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8 Appendices

8.1 Appendix 1: Structure of a risk assessment report

8.1.1 Introduction

Health risk assessment reports should be clear and transparent in their development; stating the objective of the assessment, setting the scene (conceptual site model summary), and clearly identifying the data sources and assumptions that the assessment has been based upon. A clear visual image of the site, contaminant source locations, potential exposed populations and exposure pathways present at the site should be conveyed;for example, site plans and schematic conceptual site model diagrams. The risk assessment report should provide a systematic approach for characterising the nature and magnitude of the risks associated with environmental health hazards with justification for decisions made throughout the report provided at each stage.

8.1.1.1 General

The objectives of this section are to:

- provide guidance to consultants on how to report Tier 2 quantitative health risk assessments
- provide guidance to regulatory agencies/recipients reviewing risk assessment reports on what information to expect and the level of detail required based on individual situations.

Regulatory bodies should seek further information from consultants where reports:

- are not clear and transparent
- do not present a logical framework for the decisions and assumptions made in conducting the assessment.

8.1.1.2 Key principles

Quantitative health risk assessments should follow the guidance provided in Schedules B4 and B7 and should be divided into five key steps listed below to ensure logical reporting of the assessment:

1. issues identification
2. data collection and evaluation (development of a conceptual site model)
3. exposure assessment
4. toxicity assessment
5. risk characterisation (and development of site-specific target levels, if required).

The level of detail required in a risk assessment report should be appropriate to the complexity of the site and individual scenario requiring assessment. In order to make this judgment, the risk assessor and reviewer should consider:

- whether the objectives of the risk assessment have been clearly defined
- whether the conceptual site model fully represents the site conditions and complexity
- whether the information obtained from the site assessment is robust and sufficiently characterises the current contamination status of the site, including contaminant source areas (refer to Schedule B2 for details on how to conduct an effective site assessment)

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- whether the available data has been appropriately interpreted and full justification provided as to how the data has been used within the assessment — Schedule B2 also provides information on data assessment
- whether the exposure scenarios and settings selected adequately represent the relevant land uses and potentially exposed populations.

8.1.1.3 Interpretation of data

Interpretation of site data and selection of input data used in the risk modelling is paramount to the outcome of the risk assessment and requires professional judgement. In the selection of appropriate representative input data, the assessor must consider the conceptual site model, and make it clear how the input values and modelling strategy (for example, use of fate and transport models) relate to the conceptual site model.

The risk assessment must also consider detection limits and their appropriateness in relation to the screening criteria and/or toxicity of a substance. The risk assessor must justify the reasoning behind the input values chosen for any risk assessment.

8.1.1.4 Use of subjective terms

The report language should be objective and avoid the use of subjective terms such as 'heavy/medium/light contamination' which can lead to confusion. In many parts of the risk assessment, expert judgement is necessary. It should be made clear where this is the case, and all assumptions should be identified and explained.

8.1.1.5 Specific

Further information on what is required and what to include in the five-step process that comprises the fundamentals of the risk assessment are presented in the following sub-sections.

8.1.1.6 Issues identification

Issues identification should be part of the introductory section of the risk assessment. Information on the following should be provided:

- the nature of the problem (that is, why this assessment is being carried out)
- the stakeholders (including off-site receptors) and their objectives (as far as possible)
- the objectives of the risk assessment (what the risk assessment is trying to determine)
- an outline of the risk management decisions that need to be made.

The relationship between the risk assessment and the risk management process should be made clear.

8.1.1.7 Data collection and evaluation (development of a conceptual site model)

The data collection section relies on a well-designed site assessment developed with an understanding of potential exposure pathways/routes associated with past and present land use in mind. It is assumed here that the site investigation itself is presented as a separate document (or report section) which need not be repeated in the risk assessment; however, the raw data relied on in the risk assessment should be included as an appendix.

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The data collection section should include the following:

- identification of the data used in the risk assessment
- consideration of the data quality objectives and whether these are met by the data available
- identification of any significant data gaps.

The data evaluation section should include:

- summary of the conceptual site model
- selection of and justification for Tier 1 screening criteria
- explanation of any fate and transport modelling used at Tier 1 (e.g. groundwater fate and transport to estimate groundwater quality at an off-site receptor for comparison with drinking water standards)
- identification of any need for site zoning to consider specific source areas separately (e.g. hotspots, or areas where different land uses apply)
- explanation of the basis on which the site results are screened (e.g. comparison of 95% upper confidence levels of each zone to the screening criteria)
- Tier 1 screening
- identification of and justification for contaminants of concern for Tier 2 assessment
- identification of critical exposed populations (including off-site receptors) and pathways for Tier 2 assessment
- identification of and justification for any insignificant exceedances of screening criteria that will not be assessed at Tier 2.

8.1.1.8 Exposure assessment

This section should include a clear discussion on the exposure scenarios likely to occur at the site, and whether the site-specific situation fits into the exposure scenarios characterised in NEPM Schedule B7. If these scenarios are not applicable or representative of the exposure scenario under assessment, an explanation together with behavioural and lifestyle assumptions and site assumptions should be provided within the report.

Software and mathematical algorithms used to calculate the contaminant intake should be referenced. It is only necessary to present equations if the risk assessment uses a method that is not published in full (for example, if amendments to algorithms are made). An explanation of why the model or approach selected is appropriate should be given.

All input variables should be presented and justified. Use of default assumptions must be justified.

All reasonable efforts should be employed to validate exposure models (model uncertainty) with field data (for example, soil vapour data to inform outputs from the model developed by Johnson and Ettinger (US EPA 2004a). where possible.

8.1.1.9 Toxicity assessment

In the hazard identification, a brief summary of the potential adverse effects of the contaminants of concern should be given. The summary should concentrate on the potential effects that are relevant to the contaminant in the context of the site and the exposure scenarios. Lengthy reviews of toxicology are not generally required. Clear presentation and referencing for physical and chemical properties is also required.

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Dose-response assessment involves selection of appropriate toxicity reference values. If toxicity reference values used are adopted from the relevant Schedule B7 appendix, then no additional explanation is required and a reference is sufficient. If toxicity reference values are selected for substances not included in Schedule B7, then explanation and justification of a similar order to that presented in Schedule B7 should be given.

8.1.1.10 Risk characterisation

This section needs to present the quantitative estimates of risk calculated through modelling and must provide an evaluation of the overall quality of the assessment and the degree of confidence the risk assessor has in the estimates of risk and conclusions drawn.

Risk characterisation should include a summary of key issues and conclusions, as well as describing the likelihood of adverse health effects. The summary should include a description of the assumptions made when conducting the risk assessment, together with the limitations and uncertainties associated with the risk assessment.

The conclusions should be presented in language that can be understood by non-specialists. The significance of the quantitative risk estimates should be explained in the context of the objectives of the project and the risk management decisions that need to be made.

8.1.1.11 Uncertainty

Uncertainty analysis should identify sources of uncertainty in the risk assessment and quantify them as far as possible. A tabular presentation such as that given in enHealth (2004), Table 16, is considered likely to be suitable for many circumstances. The uncertainty analysis should be specific to the assessment undertaken; a generic appraisal of the uncertainties inherent in all risk assessments is not sufficient. The uncertainty analysis should identify the impact that the uncertainty may have on the outcome (using the sensitivity analysis where possible) and identify those uncertainties that are not included in the sensitivity analysis.

8.1.1.12 Sensitivity analysis

A sensitivity analysis should present the key quantifiable uncertainties and provide plausible ranges for each. The effect on the model outcome should be stated for each uncertainty (or set of related uncertainties). Commentary on the significance of uncertainties and variability should be given. A tabular format may be appropriate; an example is provided below.

Table 8. Example format for presentation of sensitivity analysis results

Variable	Range		Risk outcome		Sensitivity level / Comment
	Min	Max	Min	Max	

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9 Shortened forms

ADI	acceptable daily intake
ANZECC	Australian and New Zealand Environment and Conservation Council
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zealand
ASTM	American Society for Testing and Materials
CDI	chronic daily intake
CRC CARE	Cooperative Research Centre for Contamination Assessment and Remediation of the Environment
CSF	cancer slope factor
HIL	health investigation level
LOAEL	lowest observable adverse effect level
MDI	mean daily intake
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NOAEL	no observable adverse effect level
PAH	polycyclic aromatic hydrocarbons
RfC	reference concentration
RfD	reference dose
SSTL	site specific target level
TDI	tolerable daily intake
TPH	total petroleum hydrocarbons
TPHCWG	TPH Criteria Working Group
US EPA	United States Environmental Protection Agency
UR	unit risk factor
WHO	World Health Organisation

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10 Glossary

Acceptable daily intake (ADI) is the estimated maximum amount of a chemical expressed on a per kg body mass basis, to which individuals in a sub-population may be exposed daily over their lifetimes without appreciable health risk.

Acceptable risk is a risk management term. The acceptability of risk depends on scientific data, social, economic and political factors, and the perceived benefits arising from exposure to an agent.

Acute exposure is contact between an agent and a target occurring over a short time, generally 14 days or less, with a single or repeated dose. Other terms such as 'short-term exposure' and 'single-dose' are also used.

Adverse effect is change in the morphology, physiology, growth, development, reproduction, or life span of an organism, system, or sub-population that results in an impairment of functional capacity, an impairment of the capacity to compensate for additional stress, or an increase in susceptibility to other influences.

Aliphatic is a hydrocarbon compound that does not contain a benzene ring. Aliphatic compounds may be straight, branched or cyclic chains of carbon atoms. They may include double or triple bonds. Carbon atoms in the chain are also generally bonded to hydrogen atoms but other elements, for example, chlorine, sulphur and nitrogen can also be present.

Aromatic is a hydrocarbon containing one or more benzene rings.

Background level is the amount of agent in a medium (for example, water or soil) that is not attributed to the sources(s) under investigation in an exposure assessment. Background level(s) can be naturally occurring or the result of human activities.

Bioaccessibility is the fraction of a contaminant in soil that is soluble in the gastrointestinal tract or lung and available for absorption.

Bioavailability is a generic term meaning the amount of a contaminant that is absorbed into the body following dermal contact, ingestion or inhalation.

Cancer is a disease of heritable, somatic mutations affecting cell growth and differentiation; that is, genetic alterations incurred in the first damaged cells are acquired in subsequent cells after cell division within the same individual.

Cancer slope factor is the plausible upper-bound estimate of the probability of a response per unit of intake of an agent over a lifetime.

Carcinogen is a cancer-causing agent.

Chemical of potential concern is an agent that is potentially site-related and whose data are of sufficient quality to be judged as potentially causing an adverse health effect.

Chronic exposure is a continuous or repeated exposure contact between an agent and a target for a duration of three months or greater.

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Clean-up level is a concentration of contaminant in soil or water derived for the purpose of providing an acceptable standard for remediation. May be risk based or modified by considerations of feasibility, practicality, acceptability, timescale and cost.

Concentration is the amount of material or agent dissolved or contained in unit quantity in a given medium or system.

Conceptual site model is a description of a site including the environmental setting, geological, hydrogeological and soil characteristics together with nature and distribution of contaminants. Potentially exposed populations and exposure pathways are identified. Presentation is usually graphical or tabular with accompanying explanatory text.

Contact volume is a volume containing the mass of agent that contacts the exposure surface.

Contaminant is any chemical existing in the environment above background levels and representing, or potentially representing, an adverse health or environmental risk.

Contaminated land is land that is affected by chemicals that occur at concentrations above background or local levels and which is likely to pose an immediate or long-term hazard to human health or the environment. The affected land may be within a specific site and/or adjacent off-site land.

Critical effect is the adverse effect(s) judged to be the most appropriate for determining the tolerable intake.

Data quality objectives (DQOs) involve the establishment of the amount, nature and quality of data required to complete a specific risk assessment.

Dose is the total amount of a chemical administered to, taken up by, or absorbed by an organism, system, or sub-population.

Dose-response curve is the graphical representation of a dose-response relationship.

Dose-response is the relationship between the amount of chemical administered to, taken up by, or absorbed by an organism, system, or sub-population and the change developed in that organism, system, or sub-population in reaction to the agent.

Effect is change in the state or dynamics of an organism, system, or sub-population caused by exposure to a chemical.

Expert/professional judgement is the opinion of an authoritative person on a particular subject.

Exposed population comprises the people who may be exposed to the contaminant. Synonymous with 'receptor'.

Exposure assessment is the evaluation of the exposure of an organism, system, or sub-population to a chemical (and its derivatives).

Exposure is the concentration or amount of a particular chemical that reaches a target organism, or system, or sub-population in a specific frequency for a defined duration.

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Exposure concentration is the exposure mass divided by the contact volume or the exposure mass divided by the mass of contact volume, depending on the medium.

Exposure duration is the length of time over which continuous or intermittent contacts occur between a chemical and the exposed population.

Exposure event is the occurrence of continuous contact between chemical and exposed population.

Exposure frequency is the number of exposure events within an exposure duration.

Exposure model is a conceptual or mathematical representation of the exposure process.

Exposure pathway is the means by which a contaminant makes contact with the exposed population.

Exposure route is the way in which an agent enters a target after contact (for example, ingestion, inhalation or dermal absorption).

Exposure scenario is a set of conditions or assumptions about sources, exposure pathways, concentration of contaminants involved, and exposed population (that is, numbers, characteristics, habits) used in the evaluation and quantification of exposure(s) in a given situation.

Genotoxic chemicals are those for which there is adequate evidence of the potential to interact with, and/or modify the function of genetic material and which has the ability to induce tumours via a mechanism involving direct damage to DNA.

Hazard identification is the identification of the type and nature of adverse effects that a contaminant has an inherent capacity to cause to an exposed population.

Hazard indices/index (HI) is the sum(s) of at least two hazard quotients. It is noted that the WHO is moving towards the use of risk indices/index (RI).

Hazard is the inherent property of a contaminant or situation having the potential to cause adverse effects when a population may be exposed to that contaminant.

Hazard quotient (HQ) is the ratio of the mean daily intake to the reference dose or tolerable daily intake for threshold exposure. It is noted that the WHO is moving towards the use of risk quotient (RQ).

Health investigation levels involve screening criteria based on health risk, presented in Schedule B7.

Intake is the total amount of contaminant (or dose) taken into the body by the exposure route.

Non-aqueous phase liquid (NAPL) is an agent that is insoluble or only slightly soluble in water that exists as a separate liquid phase in environmental media. The free liquid phase of an agent, that is, not dissolved in water or adsorbed to soil.

Non-genotoxic carcinogen is an agent which induces tumours via a mechanism which does not involve direct damage to genetic material (DNA).

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Pica is a behaviour exhibited occasionally by young children, characterised by the deliberate ingestion of non-nutritive substances, such as soil.

Reference dose is an estimate of the daily exposure dose that is likely to be without deleterious effect even if continued exposure occurs over a lifetime. Equivalent in meaning to tolerable daily intake and acceptable daily intake.

Remediation is the **c**leaning up of contaminated land.

Response is the change developed in the state of dynamics of an organism, system, or sub-population in reaction to exposure to an agent.

Risk assessment is a process intended to calculate or estimate the risk to a given target organism, system, or sub-population, including the identification of attendant uncertainties, following exposure to a particular contaminant, taking into account the inherent characteristics of the agent of concern as well as the characteristics of the specific target system.

Risk characterisation is the qualitative and, wherever possible, quantitative determination, including attendant uncertainties, of the probability of occurrence of known and potential adverse effects of an agent in a given organism, system, or sub-population, under defined exposure conditions.

Risk communication is the interactive exchange of information about health and environmental risks amongst risk assessors, managers, news media, interested groups, and the general public.

Risk estimation is the quantification of the probability, including attendant uncertainties, that specific adverse effects will occur in an organism, system, or sub-population due to actual or predicted exposure.

Risk evaluation is the establishment of a qualitative or quantitative relationship between risks and benefits of exposure to a chemical, involving the complex process of determining significance of the identified hazards and estimated risks to the system concerned or affected by exposure. Risk evaluation is an element of risk management. Risk evaluation is synonymous with risk-benefit evaluation.

Risk management is a decision-making process involving consideration of political, social, economic, and technical factors with relevant risk assessment information relating to a hazard to determine an appropriate course of action.

Risk is the probability of an adverse effect in an organism, system, or sub-population caused under specific circumstances by exposure to a contaminant.

Safety factor is the composite (reductive) factor by which an observed or estimated no-observed-adverse-effect-level (NOAEL) is divided to arrive at a criterion or standard that is considered safe or without appreciable risk.

Safety involves the practical certainty that adverse effects will not result from exposure to an agent under defined circumstances. It is the reciprocal of risk.

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Screening criteria are concentration values used in screening. Usually published for the purpose by an authoritative body (for example, HILs) or derived according to a specified methodology. Screening criteria are available for soil, groundwaters, surface waters and sediments.

Screening is the process of comparison of site data to screening criteria to obtain a rapid assessment of contaminants of potential concern.

Sensitive groups are populations with both susceptibility and vulnerability factors.

Sensitivity analysis is the process of changing one variable while leaving the others constant and determining the effect on the output. The procedure involves fixing each uncertain quantity, one at a time, at its credible lower bound and then its upper bound (holding all other at their medians), and then computing the outcomes for each combination of values. It can be used to test the effects of both uncertainty and variability in input values.

Site specific target levels are risk-based concentration values derived using Tier 2 or Tier 3 exposure modelling. May be used as criteria for further assessment or as clean-up levels.

Source is the contaminant that is considered to represent a potential risk requiring assessment.

Sub-chronic exposure is a contact between an agent and a target of intermediate duration between acute and chronic. Different bodies vary on their definitions of the duration of 'sub-chronic' exposure, since it varies with species. US EPA uses up to 10% of an organism's lifetime (enHealth 2010), however between 3-6 months is often used when discussing sub-chronic exposure to people.

Susceptibility refers to intrinsic biological factors that can increase the health risk of an individual at a given exposure level. Examples of susceptibility factors include genetic factors, late-age and early-life, prior or existing disease.

Threshold is the dose or exposure concentration of an agent below which a stated effect is not observed or expected to occur.

Tier 1 evaluation is a risk-based analysis comparing site data with generic published screening criteria for various property uses (for example, residential, commercial and industrial). This tier has the lowest data requirement, generic exposure assumptions, and applies the most conservative criteria.

Tier 2 evaluation is a site-specific assessment in which risks to potentially exposed populations are assessed using site-specific data on pathways, land uses and the characteristics of the exposed populations. A Tier 2 evaluation usually involves the use of a quantitative exposure model. A Tier 2 evaluation is more complex than a Tier 1 evaluation and requires more site-specific information. As a result, a health protective effect will be achieved with a lower level of conservatism.

Tier 3 evaluation is a further step from a Tier 2 evaluation and looks in more detail at specific risk-driving factors. This often involves additional data collection, and may incorporate more sophisticated modelling techniques.

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Tolerable daily intake is analogous to *acceptable daily intake*. The term 'tolerable' is used for substances that are not deliberately added, such as contaminants in food and water.

Toxicity criteria are measures of tolerable intake or acceptable risk, such as reference doses and cancer slope factors.

Toxicity is the inherent property of a chemical to cause an adverse biological effect.

Uncertainty is a lack of or incomplete information or knowledge.

Unit risk is the plausible upper-bound estimate of the probability of a response from a chemical over a lifetime expressed in units of concentration for a specified medium.

Uptake is the amount of contaminant that enters the body through a barrier such as the skin, lungs or gut lining. Uptake is generally less than intake because not all the contaminant that enters the lungs or gut, or contacts the skin, is absorbed.

Vadose zone is the portion of the sub-surface between the water table and the ground surface, also termed the unsaturated zone. Soil pore space in the vadose zone is only partially occupied by water, which is held in place by capillary forces and adhesion to soil particles.

Variability describes true differences in attributes or values due to diversity or heterogeneity.

Vulnerability refers to human populations at higher risk due to environmental factors; examples of vulnerability factors include poverty, malnutrition, poor sanitation, climate change, and stress associated with mental health diseases.

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