



SOCIETY OF BROWNFIELD RISK ASSESSMENT

**Development of Acute Generic Assessment
Criteria for Assessing Risks to
Human Health from Contaminants in Soil**

Version 1.0

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PUBLICATION

This report is published by the Society of Brownfield Risk Assessment (SoBRA). It presents work undertaken by a SoBRA sub-group composed of volunteers listed in the Acknowledgments below. The publication presents a methodology for derivation of Acute Generic Assessment Criteria (AGAC) that risk assessors may choose to use to help in the assessment of acute health risks from short-term exposure to contaminants in soil. Worked examples for described scenarios are also provided in Section 6 of this report.

As set out in the text, it is imperative that users do not refer solely to the example AGAC presented in this report, but they read and understand their derivation and limitations as described in the supporting text presented herein. In writing this report it has been assumed that readers are already familiar with the use and limitations of Generic Assessment Criteria for assessing chronic health risks from long-term exposure to contaminants in soil.

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1 INTRODUCTION

1.1 Background

Most human health risk assessments for the evaluation of land contamination are focussed on chronic risks arising from long term exposure to specific substances. Because chronic risks often occur at lower doses than acute risks, they are often the key risk drivers. However, in some instances, the acute dose may also be an important consideration when assessing risk to human health from land contamination. The acute dose may be overlooked, for instance:

- Where the acute dose toxicity thresholds for the substance are very close to the chronic dose toxicity thresholds (e.g. free cyanide);
- Where averaging over long periods may overlook peaks of short-term exposure (particularly for scenarios where exposure is infrequent such that average long-term exposure is much less than peak exposure); and
- Where exposure occurs for only a short period (e.g. during maintenance works or site investigation).

There has been a lack of UK guidance and limited international guidance on assessing acute risks to human health from short term exposure to soil contamination. In order to address this lack of UK guidance, the Society of Brownfield Risk Assessment (SoBRA) created a subgroup. The subgroup's remit has been to research existing guidance and propose a methodology for deriving acute generic assessment criteria (AGAC) protective of human health for short-term exposure to contaminants in soil. This document presents the results of that work.

1.2 Scope of the Document

This document presents a methodology for deriving AGAC for various common short-term (less than 24-hour duration) exposure scenarios. These scenarios relate to two distinct receptor groups:

- Members of the public. The "critical" (i.e. most sensitive) receptor for this group will typically be a female child, which is consistent with CLEA residential and Public Open Space/allotments land-uses. Exposure scenarios considered are short-term oral, dermal or inhalation exposure to contaminants in soil whilst outdoors. For oral and dermal exposure, the scenario considered is direct contact with outdoor soil, whether this is in a residential garden, public

open space or on a site where trespass occurs when the site is not active. For inhalation exposure, the scenario considered for members of the public is exposure of a nearby off-site child to inhalation of dusts or vapours that have been released and migrated from excavation activities at the site (e.g. from construction or remediation) (i.e. off-site exposure from an on-site source). It has been assumed that members of the public (e.g. trespassers) would not be present on construction or remediation sites while dusts and vapours are being generated and therefore on-site inhalation exposure has not been considered. Note also that the subgroup considered that the acute risks from short-term inhalation of contaminants from undisturbed soils (e.g. in a residential garden, or public open space) were unlikely to be significant and therefore these risks have not been considered further; and

- Workers involved with excavations. The critical receptor for this group is assumed to be a female working adult. Exposure scenarios considered are short-term oral, dermal or inhalation exposure to contaminants in soil whilst working outdoors on-site. For oral and dermal exposure, the scenario considered is direct contact with outdoor soil on the assumption that personal protective equipment (PPE) is not worn. For inhalation exposure, the scenario considered is a worker inhaling dusts or vapours released from excavation activities, again without the use of PPE.

Section 2.6 discusses how the AGAC are intended to be applied. In summary, the AGAC for members of the public are intended to be compared with measured soil concentrations to determine whether or not risks from short-term exposure to outdoor soils are of potential concern under the assumed generic exposure scenarios. The AGAC for workers involved with excavations may provide useful information when planning intrusive works (e.g. ground investigation, remediation or construction activities). For example, they may be useful to highlight when particular attention should be paid to the management of acute health risks from contaminants in soil.

1.3 Limitations

This document has been developed to support assessment of toxicological risks caused by short-term exposure to contaminants in soil. It does not address acute risks to health associated with explosive or fire risks.

The information presented in this report is intended to aid the assessment of acute risks from contaminants in soil and inform risk management strategies accordingly. It is not intended to replace existing risk management strategies/procedures. For

example, comparison of AGAC with measured concentrations does not negate the need for monitoring and control of risks for workers involved in excavations on land affected by contamination.

Irrespective of the report, users should remain aware of their duties to ensure compliance with applicable regulations (e.g. Control of Lead at Work Regulations 2002 or Control of Asbestos Regulations 2012) and the need to minimise risk from exposure under the health and safety at work acts.

1.4 Reporting Structure

The report is divided into 6 sections:

- Section 2 provides the framework for assessing acute risks from contaminants in soil and outlines the proposed assessment process;
- Section 3 sets out the toxicological hazard screening assessment;
- Section 4 describes the derivation of acute reference criteria;
- Section 5 describes the exposure modelling and derivation of AGAC;
- Section 6 presents examples of AGAC derived using the framework; and
- Section 7 presents a list of references.

2 FRAMEWORK

2.1 Definition of Acute Exposure

There are a wide range of definitions for acute exposure. These range from one-off exposure lasting from a few seconds (e.g. ingestion of a bolus of soil) to 24 hours, to short-term repeated exposure of up to 14 days (such as a worker conducting a site investigation). For the purposes of this document acute exposure has been defined as an irregular (i.e. not repeated) exposure of up to 24 hours duration¹. In the case of acute inhalation exposure, the AGAC presented in this report are based on the assumption of exposure lasting for 30 minutes or less.

Toxicological impacts from acute exposure are generally short lived, such as nausea and vomiting, skin burns and irritation, headaches, vertigo and dizziness. In some cases, they can be more serious and include organ failure and even death. In the current assessment, the focus has been on substances considered toxic or harmful.

2.2 Overall Approach

The approach described herein conforms to what is described in the CLR11 document (Defra and Environment Agency, 2004) as a Generic Quantitative Risk Assessment (GQRA). GQRA involves the comparison of measured media concentrations with suitable risk based generic assessment criteria (GAC) in order to make an initial generic quantitative assessment of risks. In this report the GAC are referred to as acute generic assessment criteria (AGAC) and relate to the concentrations of contaminants in soil considered protective of human health for the acute exposure scenarios considered.

As with the Contaminated Land Exposure Assessment (CLEA) methodology for deriving GAC for chronic risks (Environment Agency 2009a, 2009b), the derivation of the AGAC involves (a) toxicological assessment to derive suitable health based guidance values (referred to herein as acute reference criteria) for relevant routes of exposure and (b) exposure assessment to determine the theoretical soil concentration at which short-term exposure via a given exposure scenario would equal the relevant acute risk

¹ It may be possible to extend the AGAC approach to longer exposure timescale through selection of the appropriate toxicological health based guidance values and adjustments to the exposure scenarios, but this is outside the current scope of this document.

health based guidance value. The derivation of acute reference criteria is described in Section 4. The calculation of AGAC is described in Section 5.

Unlike the CLEA methodology approach, rather than deriving GAC that account for all routes of exposure, separate AGAC are calculated for each relevant route of exposure. Relevant routes of exposure are determined by an initial hazard screening step. This involves the use of Classification, Labelling and Packaging (CLP) hazard codes to determine which routes of exposure (i.e. oral, dermal and/or inhalation) may be of concern for short-term (acute) exposure scenarios and is described further in Section 3.

2.3 Exposure scenarios

2.3.1 Exposure scenarios selected

The exposure scenarios that are considered in the current document and used as the basis for the AGACs relate to outdoor exposure scenarios that are commonly considered when assessing acute risk (Kowalczyk *et al*, 2013; NJDEP, 2012). The scenarios considered are not exhaustive and the appropriateness of the assessment should be considered on a case by case basis. The scenarios consider one route of exposure at a time, namely ingestion of soil, dermal contact with soil, and inhalation of dusts or vapours released from soil. Note: The scenarios do not include ingestion of dust, because a receptor is highly unlikely to ingest the mass of soil assumed in the AGAC calculations as dust.

The exposure scenarios are discussed further in Section 4. They can be divided into two types, (a) public and (b) occupational exposure:

a) *Public exposure*

The public exposure scenarios focus on a female child as the critical receptor. The three scenarios considered are:

- Ingestion of a one-off bolus dose of soil, e.g. whilst playing in a garden, public open space area or during trespass;
- Dermal contact with contaminants in soil, e.g. whilst playing in a garden, public open space area or during trespass; and

- Inhalation of dusts or vapours arising from excavation activities (e.g. during construction or remediation) on a nearby site².

b) Occupational exposure during construction, remediation or site investigation

The occupational exposure scenarios focus on a worker coming into direct contact with soil contaminants or inhaling dusts or vapours arising from the excavation of contaminated soils. Such a worker could be involved in remediation activities or could be a utilities worker who, for example, has accidentally excavated into contaminated soil beneath a cover system.

Note that (in the UK) the AGAC for the occupational exposure scenarios should not replace Health and Safety Executive (HSE) guidance on managing risk and controlling exposures, nor should they replace monitoring or other controls. However, they may help inform risk management planning for working on land affected by contamination. For example, they may be used to highlight potential acute risks prior to work commencement, or they may inform the design of appropriate cover systems.

2.3.2 Model Selection and Parameterisation

The algorithms selected for estimating exposure and deriving the AGAC are based on available methods and are discussed further in Section 5. In order to illustrate how AGAC may be derived, example parameter values have been selected and these are also discussed in Section 5. Note that the values selected are intended to represent “reasonable maximum exposure” but are for illustrative purposes only. The assessor should satisfy themselves whether these values are suitable for the situation they are assessing or whether an alternate approach is required.

2.3.3 Other Scenarios

The document focuses on risks that are relatively simple to model. It does not consider complex scenarios such as risks from transient vapours and gases entering houses and basements through services such as drains. It may be possible to extend the AGACs to other scenarios if the processes can be adequately modelled.

² As stated in Section 1.2 the risks from short-term inhalation of contaminants from undisturbed soils are unlikely to be significant.

2.4 Derivation of AGACs

The AGAC is the estimated soil concentration at which short-term exposure to the receptor is equal to the pre-determined acute reference criteria (i.e. the health-based guidance value). As such, provided that the actual soil concentration (discussed further in Section 2.5 below) is below the AGAC (and that exposure assumptions and parameters are suitably precautionary) then actual exposure should be less than the acute health-based guidance value. The significance of an exceedance of the AGAC will depend on the toxicological end-point used as the basis of the acute reference criteria, the uncertainty factors used to derive the acute reference criteria, and the level of precaution in the exposure modelling.

2.5 Use of AGACs

The AGACs relate to specific acute outdoor exposure scenarios and are based on simplified models. It is critical that the assessor considers the conceptual model for exposure to determine which (if any) of AGACs are suitable and to confirm that the assumptions made are appropriate for the scenarios selected.

When using AGACs as assessment criteria it is important to note that these relate to short term exposure to high concentrations of a substance that lead to acute effects. They do not relate to average exposure across a specific / defined area. Thus, AGACs should normally be compared with the maximum likely concentration that the individual may be exposed to, and not the average concentration within a specific area. The assessment should also consider the potential for undiscovered higher concentrations of contamination to be present, and the presence of "hotspots".

The AGACs do not assess risks from chronic exposure to contaminants in soil. These risks should be assessed separately where long-term repeated exposure could occur.

2.6 Caveats

2.6.1 Pica and Geophagia

Pica behaviour consists of repeated ingestion of non-nutritious substances (such as soil) over a period of time. Such repeated behaviour is outside the scope of this report. However, information on the amount of soil ingested in a single pica event has been considered to inform the parameter values used for assessing risks from ingestion of a one-off bolus of soil.

Geophagia is the ingestion of soil as a cultural practice. This is rare in the UK and is also outside the scope of this report.

2.6.2 Mixtures

The toxicological effects of mixtures of chemicals have not been assessed in this report. In such cases specific consideration should be given to additive effects if contaminants have the same mechanism/mode of action. Similarly, mechanisms such as one substance increasing the mobility of another through the skin or to the lungs are not considered.

2.6.3 Physical Form

The current assessment is focussed on soil bound substances only. It excludes, for instance, non-aqueous phase liquids (NAPL) and buried canisters of substances. The behaviour of NAPL is different to soil bound contaminants, particularly in relation to skin adherence and vapour release. As a result, the AGACs derived using the methodologies presented herein are not suitable for assessing risks from acute exposure to NAPL.

2.6.4 Strong Acids and Bases

The methodology does not examine extremes of acidity/alkalinity (pH). OECD (2002) guidelines indicate that substances exhibiting pH extremes such as ≤ 2.0 and ≥ 11.5 may cause localised corrosive effects such as irritation and burns. Corrosive effects due to low or high pH are outside the scope of this document and should be considered separately.

2.6.5 Toxicological Endpoints

The approach described herein focuses on substances identified as harmful or toxic according to their CLP classification. Acute toxic effects can range from relatively mild, reversible symptoms such as nausea, dizziness or local irritation (cough, shortness of breath or skin irritation) to more serious and potentially irreversible effects such as organ toxicity, pulmonary oedema and death. All toxicological adverse effects observed following an acute chemical exposure should be identified and taken into consideration in the derivation of the acute reference criteria.

For the purpose of deriving acute reference criteria and AGAC it is important to identify and record the critical effect being considered as the point of departure because this forms the basis of the reference criteria. As with chronic exposure, the most sensitive adverse effects should generally form the basis of the acute reference concentration or dose. Note, however, that the toxicological end-point selected as the basis of the acute reference criteria should be appropriate for the regulatory regime in which the assessment is conducted. For example, the assessor may choose to base

the acute reference criteria on a more serious adverse health effect for an assessment conducted under Part 2A of the 1990 Environmental Protection Act (assessing significant possibility of significant harm) than they would for an assessment conducted under the Planning regime (for determining suitability for use).

In some cases where chemicals have a strong odour, the odour can lead to effects such as headaches and nausea rather than these symptoms being a true toxicological effect (e.g. HPA, 2014). Assessment of such effects can be complex, taking into account the factors such as odour threshold, intensity, hedonic tone (pleasantness/offensiveness), recognition and odour quality or character. Such assessment is outside the scope of this document.

3 TOXICOLOGICAL SCREENING ASSESSMENT

Hazard information can be used initially to screen out a substance, or one or more exposure routes for a substance, where risks from acute exposure are unlikely to be of concern. This section describes an approach that uses substance hazard phrases (where available) to identify the substances and corresponding routes of exposure for which it may be beneficial to derive AGACs.

3.1 Applicable Hazard Codes

The Classification, Labelling and Packaging of Substances and Mixtures Regulation 1272/2008 (CLP) provides the criteria to assess the physical, human health and environmental hazards of substances. The CLP defines 63 different hazard codes and precautionary statements, the following of which are considered applicable to acute exposure scenarios:

Ingestion

- H300: Fatal if swallowed
- H301: Toxic if swallowed
- H302: Harmful if swallowed
- H303: May be harmful if swallowed
- H304: May be fatal if swallowed and enters airways
- H305: May be harmful if swallowed and enters airways

Dermal contact

- H310: Fatal in contact with skin
- H311: Toxic in contact with skin
- H312: Harmful in contact with skin
- H313: May be harmful in contact with skin
- H314: Causes severe skin burns and eye damage
- H315: Causes skin irritation
- H316: Causes mild skin irritation
- H317: May cause an allergic skin reaction

Inhalation

- H330: Fatal if inhaled
- H331: Toxic if inhaled
- H332: Harmful if inhaled
- H333: May be harmful if inhaled
- H334: May cause allergy or asthma symptoms or breathing difficulties if inhaled
- H335: May cause respiratory irritation
- H336: May cause drowsiness or dizziness

In addition, the following hazard codes/precautionary statements for “specific target organ toxicity” (STOT) (single exposure) (SE) are also applicable when assessing risks from short-term exposure. More detailed review of the toxicology may be required to determine the route of exposure to which the code relates for a specific substance.

- H370 (STOT SE1): Causes damage to organs
- H371 (STOT SE2): May cause damage to organs

For substances that have not been classified for acute toxicity under CLP a conservative screening assessment can be undertaken by completing an initial toxicological review using the same methodology as set out in the CLP Regulation to determine the likely CLP hazard codes/precautionary statements for a substance.

3.2 Identifying Hazard Information

There are various potential sources of hazard code information. In order of preference these are:

- Table 3.1 of Annex VI of the Classification, Labelling and Packaging (CLP) Regulation 1272/2008. Available at <http://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:02008R1272-20170101&from=EN> ;
- Harmonised classifications in the Classification and Labelling Inventory maintained by the European Chemical Agency (ECHA). The inventory’s search page enables you to find classification entries: Available at: <http://echa.europa.eu/web/guest/information-on-chemicals/cl-inventory-database> ; and
- Self-classifications in the Classification and Labelling Inventory maintained by ECHA.

For some substances such as heavy metals and anions, there are a number of entries related to the form of the metal present. For example, for zinc there are entries for zinc oxide or zinc sulphate. It is important to consider the worst case for each exposure route.

Other potential sources of hazardous properties information include:

- Information generated in accordance with REACH, available at <https://echa.europa.eu/information-on-chemicals/registered-substances>;
- The Pesticide Properties Database, available at <https://sitem.herts.ac.uk/aeru/ppdb/en/> ; and
- Manufacturer's safety data sheet (e.g. for spills of known substances).

4 DERIVATION OF ACUTE REFERENCE CRITERIA

Acute reference criteria are required in order to derive the AGACs for a substance. As discussed above, acute reference criteria are only required for the routes of exposure where risks from acute exposure to that substance are a plausible concern.

In line with the CLEA Science Report SR2 (EA, 2009a) approach for deriving health criteria values (HCVs) for chronic risk, it is recommended that a literature review is first conducted to identify possible acute reference criteria and/or toxicological information from which an acute reference criterion could be derived. Guidance on potential sources of information for acute reference criteria are given in Section 4.2 below.

Once the literature review has been conducted, the assessor will then need to choose an appropriate acute reference criterion for each applicable route of exposure. Where none are available, consideration must be given to deriving one from available toxicological information. Guidance on selecting appropriate acute reference criteria are given in Section 4.2. Further information on deriving acute reference criteria *de novo* is provided in Section 4.3.

The results of the literature review, and selection and justification of the acute reference criteria should then be clearly documented. An example completed proforma with this information is presented in Appendix A.

4.1 Definition of Acute Reference Criteria for Each Route of Exposure

The acute reference criteria being developed depend on the route of exposure and are defined below.

- The acute oral reference dose (ARF_{D_{oral}}) is an estimate of the dose (via ingestion) over a short period (up to 24 hours) that is without appreciable risk of adverse health effects occurring (relevant to the objective of the assessment being undertaken). This will typically be expressed in milligrams or micrograms of substance per kilogram bodyweight (mg or µg/kg bw). The toxicological end points of acute oral exposure being considered include both localised effects such as nausea and vomiting as well as systemic effects such as organ effects;
- The acute dermal reference dose (ARF_{D_{dermal}}) is an estimate of the dose (via the skin) over a short period (up to 24 hours) that is without appreciable risk of adverse health effects occurring (relevant to the objective of the assessment being undertaken). This will typically be expressed in milligrams or

micrograms of substance per square centimetre of skin area (mg or $\mu\text{g}/\text{cm}^2$). For the purposes of this document the toxicological end points considered for the acute dermal reference dose are irritant³ or allergic⁴ contact dermatitis rather than systemic dermal toxic effects. This is because short-term exposure causing systemic effects is likely to be dominated by the oral route of exposure; and

- The acute inhalation reference concentration (ARfC_{inh}) can be defined as an estimate of the concentration in inhaled air over a short period (typically 15 to 30 minutes) that is without appreciable risk of adverse health effects occurring (relevant to the objective of the assessment being undertaken). This will typically be expressed as milligrams or micrograms of substance per cubic metre of air (e.g. in mg/m^3) or in parts per million (ppm) in air. The toxicological end points of acute inhalation exposure being considered include both localised effects such as respiratory irritation as well as systemic effects such as effects on organs.

4.2 Data Sources and Hierarchy

Acute toxicological criteria can be found in a number of UK and international sources. In line with the approach adopted in CLEA (EA, 2009a), authoritative UK derived values should normally be given preference, followed by authoritative international bodies. Examples of UK authoritative bodies include the Health and Safety Executive (HSE), Public Health England (PHE) and Public Health Wales (formerly the Health Protection Agency – HPA), the Committee on the Toxicity (COT) of Chemicals in Food, Consumer Products and the Environment, the Environment Agency and the Food Standards Agency (FSA). Examples of applicable authoritative bodies from outside the UK include the European Food Standards Agency (EFSA), the World Health Organization (WHO), the US Agency for Toxic Substances and Disease Registry (ATSDR), the US Environmental Protection Agency (USEPA), the US National Institute

³ Irritant contact dermatitis is an inflammation of the skin that results in localised redness, swelling and scaling that usually appears immediately following exposure. It occurs in response to direct chemical damage that causes the release of inflammatory mediators from skin cells.

⁴ Allergic contact dermatitis occurs following exposure to a skin allergen, manifesting as a widespread rash or skin lesions, redness, inflammation and swelling that occur several days after exposure. Examples of skin allergens are nickel, cobalt and hexavalent chromium, plastics and resins such as epoxy resins, formaldehyde and isocyanate (European Agency for Safety and Health at Work, 2003).

for Occupational Safety and Health (NIOSH), the Dutch National Institute for Public Health and the Environment (RIVM), the Australian National Industrial Chemicals Notification and Assessment Scheme (NICNAS) and Health Canada.

When using data from such sources it is important to cross reference to the original sources where appropriate i.e., if reports from other authoritative bodies are referenced. It is critical to note that where new toxicological data are available, such data should normally take precedence regardless of the jurisdiction.

Care must be taken to fully understand the derivation of acute reference criteria to ensure they are appropriate to use for the derivation of AGAC. For example, acute exposure guidance levels (AEGs) signify concentrations above which adverse effects may occur, in contrast with other acute reference values that signify concentrations or doses below which adverse effects are unlikely to occur. Moreover, different acute reference values are based on different receptors. Workplace exposure limits (WEL) are based on healthy adults whereas AEGs are based on nearly all members of the general public, including sensitive individuals (such as young people).

Preference should be given to acute reference criteria derived by authoritative bodies, but where these do not exist, acute reference criteria may have to be obtained from other sources (e.g. scientific papers or on-line reports) or derived from toxicological data. Further guidance on deriving the acute reference criteria *de novo* is given in Section 4.3 below.

Useful sources of acute reference criteria specific to each route of exposure are discussed below.

4.2.1 Acute Oral Reference Dose

A source of authoritatively derived ARfDs for oral exposure is the US ATSDR Minimum Risk Levels (MRLs). An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. MRLs are derived for acute (<14 days), intermediate (14 – 365 days) and chronic duration (>365 days) exposure. MRLs for acute exposure are most relevant to the setting of the ARfD_{oral}.

The USEPA drinking water health advisories (USEPA, 2018) may also provide a useful authoritative basis for setting the ARfD for oral exposure. Health advisories are estimates of acceptable drinking water levels for chemical substances over a defined exposure period based on health effects information. The USEPA set health advisories for specific substances for 1-day and 10-day exposure. The One-Day health advisory is likely to be the most relevant to the setting of the oral ARfD for the exposure

scenarios considered. This is defined as the concentration of a chemical substance in drinking water that is not expected to cause any adverse non-carcinogenic effects for up to one day of exposure. The One-Day health advisory is intended to protect a 10 kg child consuming 1 litre of water per day.

The New York State Departments of Environmental Conservation and Health (NYSDEC/NYS DH, 2006) also list ARfDs for oral exposure for some chemical substances (namely barium, cadmium, copper, cyanide, nickel, pentachlorophenol and phenol).

4.2.2 Acute Dermal Reference Dose

Published authoritative acute reference criteria for dermal exposure are few and far between. Moreover, peer reviewed dermal toxicity and dermal irritation data tend to be far more limited than for the oral and inhalation routes of exposure.

NYSDEC/NYS DH (2006) list ARfDs relevant to irritant contact dermatitis from dermal exposure for some substances (including nickel, chromium VI, phenol and specific semi-volatile organic compounds). Some dermal data are collated during REACH assessments and are available on the ECHA dissemination pages⁵.

4.2.3 Acute Reference Concentration

The exposure model used in the AGACs for the inhalation route of exposure is related to short term release of vapours and dusts arising from excavation work. Such works are likely to result in short-term “bursts” of dust and vapours and it is assumed that exposure duration will typically be of the order of 30 minutes or less. As discussed further below the choice of ARfC should be made with this in mind.

Occupational exposure

For occupational exposure in the UK there are already defined WELs for specific substances. These are listed in the HSE’s EH40 document (HSE, 2018) and are legally binding. They are set in order to help protect the health of workers and cannot be adapted readily to evaluate or control non-occupational exposure. They may therefore be considered appropriate for an adult construction worker/engineer/maintenance worker. They are not appropriate for assessing potential risks to the general public.

The EH40 document gives WELs for both short-term (15 minute) and long-term (8 hr) exposure durations for most substances listed. According to the EH40 document the

⁵ See <https://echa.europa.eu/>

short-term exposure limits (STELs) are set at a sufficiently low level to prevent effects such as eye irritation. The short-term exposure limits are considered appropriate for the exposure scenario under consideration. Note that where a short-term exposure limit is not specified, EH40 recommends that a value of three times the long-term exposure limit is used as a guideline to control short-term peaks in exposure.

For substances where WELs are not available, the USEPA Acute Exposure Guideline Levels (AEGLs) (see below) or ATSDR acute MRLs for inhalation exposure may provide a suitable alternative.

General public exposure

The USEPA have developed AEGLs for the general public, including susceptible individuals (see <http://www.epa.gov/aeql>) and their significance in relation to adverse effects was discussed earlier in Section 4.2.

AEGLs are available for five separate exposure periods, ranging from 10 minutes to 8 hours (comparable to the exposure range of the WELs). More specifically, AEGLs have been developed for exposure periods of 10 minutes, 30 minutes, 1 hour, 4 hours and 8 hours. As with WELs, they are expressed as ppm or mg/m³. The 10-minute and 30-minute exposure periods are considered to be most relevant to the exposure scenario under consideration.

For each exposure period, three AEGLs have been developed, which are distinguished by the varying degrees of severity of toxic effects. The USEPA defines the three categories of AEGLs as follows:

- AEGL-1 is the airborne concentration above which it is predicted that the receptor could experience notable discomfort, irritation, or certain asymptomatic non-sensory effects. These effects are not disabling and are transient and reversible upon cessation of exposure;
- AEGL-2 is the airborne concentration above which it is predicted that the receptor could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape; and
- AEGL-3 is the airborne concentration above which it is predicted that a receptor could experience life-threatening health effects or death.

Whilst the AEGL-1 concentrations are considered to be set at a similar risk level to the WELs, exposure to airborne concentrations below AEGL-1 are defined by the USEPA as representing exposure levels that can still produce mild and progressively increasing but transient and non-disabling odour, taste, and sensory irritation or certain asymptomatic, non-sensory effects. As a result, they may not always be as health

protective as the WELs. In general, where WELs exist and are more health protective than the AEGLs, the WELs should be used.

For substances where AEGLs are not available the ATSDR acute MRLs for inhalation exposure may provide a suitable alternative.

4.3 Derivation of acute reference criteria from toxicity data

In the absence of acute reference criteria derived by an authoritative body, acute reference criteria may be derived (*de novo*) from available toxicological data. This section provides a brief overview of how this could be achieved. **Note that it is strongly recommended that acute reference criteria are derived by experienced toxicologists.**

As with the derivation of chronic reference criteria, a point of departure, such as a no adverse effect level (NOAEL) for the most sensitive toxicological endpoint (i.e. the toxic effect that occurs at the lowest concentration or dose) is divided by appropriate uncertainty factors.

Care must be taken to ensure the appropriate toxicity study is selected. Most acute toxicity studies are designed to identify levels that cause death (LD50 values) or severe toxicity for the purposes of classification and labelling of substances. Such studies are not appropriate to determine a NOAEL for critical effects (European Commission (EC), 2001). However, if no other acute toxicity data are available, LD50 data could be used to derive acute reference criteria as long as appropriately conservative uncertainty factors are used. Newer studies carried out according to OECD guidelines rely more on observational signs of toxicity so may be of more use in deriving a point of departure (EC, 2001).

Subacute toxicity studies, including 14-day range finding and 28-day studies, are often used as a basis of acute reference criteria. Such studies may be the most adequate source of data, particularly if acute endpoints have been measured (EC, 2001). Subchronic studies, such as 90 day studies, may be used if it can be demonstrated that there is no difference between acute and subchronic effects, when there are no acute data or when effects are seen early on in the study i.e. following a short term exposure. Epidemiological data may also be used if robust exposure monitoring data can be provided.

To derive the acute reference criteria, the appropriate toxicological study and the critical toxicological endpoint must be identified. The critical endpoint is the most relevant adverse health effect that occurs at the lowest dose. This selection should be based on the quality of the study, exposure period and type, site, severity and

incidence of the toxicological effect. From such data the point of departure i.e. NOAEL, lowest adverse effect level (LOAEL) or benchmark dose (BMD) should be determined. Care must be taken to ensure that the effects on which the point of departure is based are relevant to humans (i.e. some toxicological effects occur in laboratory test species that cannot physiologically occur in humans and therefore would not be a suitable point of departure).

Suitable uncertainty factors are then applied to the point of departure to extrapolate from the animal data to the human population by accounting for inter- and intra-species differences. Factors such as the quality of the study and severity of endpoint should be considered when deriving uncertainty factors.

The CLEA SR2 report (EA, 2009a) provides information on the selection of uncertainty factors to use for chronic exposure but not acute. Alternate appropriate guidance should be consulted for acute exposure, for example Renwick (2000). In the absence of alternative guidance being available it should be noted that a default uncertainty factor of 100 would normally be used to derive chronic reference criteria, based on a 10-fold factor for both interspecies and intraspecies variability.

When reviewing toxicological data, it is important to consider whether there could be differences in sensitivity between groups of individuals (e.g. adults and children, gender) in order to derive acute reference criteria that are protective of sensitive individuals. For example, for substances that are commonly used in industry, human toxicological data are more likely to be from studies involving adults than children (who may be more sensitive to toxicological effects). Any differences between adults and children or gender should be incorporated into the acute reference criteria so that they are protective of sensitive individuals.

5 EXPOSURE ASSESSMENT AND CALCULATION OF AGAC

As discussed in Section 2.3.1, AGACs have been derived for a number of exposure scenarios. These include exposure scenarios where members of the public are the critical receptor and occupational exposure scenarios where workers involved with excavations are the critical receptor. Separate AGAC are derived for each receptor type and each route of exposure (identified as applicable following the hazard screening discussed in Section 3). The methodologies and assumptions for deriving AGAC for each exposure scenario are discussed below.

5.1 Oral Exposure

5.1.1 Exposure scenarios

The following exposure scenarios have been considered for the oral route of exposure:

1. **General public exposure.** The AGACs for this scenario are based on the assumption of a young female child who ingests a single bolus dose of soil. This could include any land use where children have access to soil, including residential use and open space scenarios. This scenario could also be applied to a child trespasser (although an older child than that discussed below should likely be selected). The potential for cumulative effects from multiple bolus doses has not been assessed. Instead a conservative estimate of the mass of soil ingested has been applied that is considered as being representative of a single large dose (or potentially the sum of several smaller doses over a short duration). If, during the literature review of toxicity data, it becomes apparent that a substance is prone to cause cumulative effects then potential sub-chronic exposure should be considered further.

The default CLEA residential female child has been considered, with exposure via a single bolus dose. Time averaging across the ages of 0 – 6 years old has not been conducted as this is not appropriate for acute exposure. Rather, parameter values for a 1 to 2-year old female child have been selected as representative of a reasonable worst case for this scenario; and

2. **Occupational exposure.** The AGACs for this scenario are based on the assumption of an adult commercial/ industrial worker who is exposed to contaminated soil during their work, such as a construction, utility, site investigation or remediation worker. The default CLEA female commercial

worker has been considered, with exposure over the working day as the duration.

5.1.2 Model Selection

The AGAC for the oral route of exposure has been calculated using the following equation taken from the Center for Environmental & Human Toxicology, University of Florida (2005). This equation is also consistent with that used to derive the New York State soil clean-up objectives for acute oral exposure (NYSDEC/ NYSDH, 2006) and with the method used by Kowalczyk *et al* (2013) for assessing acute risks from cyanide in soil.

$$AGAC_{oral} = \frac{BW \cdot ARfD_{oral}}{MS_{ing} \cdot RBA_{ing}} \times 1000 g \cdot kg^{-1}$$

Where

AGAC_{oral} = acute generic assessment criteria for oral exposure (mg.kg⁻¹)

BW = body weight (kg)

ARfD_{oral} = acute oral reference dose (mg.kg⁻¹(body weight))

MS_{ing} = mass of soil ingested in the short-term period being considered (g)

RBA_{ing} = relative bioavailability of ingested soil (relative to toxicity study) (fraction)

Note that this method does not account for:

- Background exposure to non-soil sources (as would be the case for assessing chronic risk for non-threshold substances). Background exposure is likely to be negligible relative to the ARfD_{oral} and can therefore be discounted.
- Exposure via the consumption of homegrown produce. This exposure pathway is considered by the subgroup as unlikely to cause an unacceptable acute risk; and
- Additive or synergistic effects with other substances, for example those which may affect the same target organ or alter the chemical form of the substance assessed.

5.1.3 Exposure parameters for soil ingestion

The exposure parameters used for the calculation of the AGAC_{oral} are discussed below:

Mass of Soil Ingested

Children

For the child receptor, potential pathways include direct hand to mouth transfer (including intentional ingestion but not pica or geophagia as discussed in Section 2.6), mouthing of objects with attached soil, indirect hand to mouth transfer, and other accidental ingestion of soil. For acute exposure scenarios, mouthing and intentional ingestion of soil are likely to be the most significant and are discussed further below:

- **Mouthing behaviour.** This is common in most children and refers to the tendency of children to explore objects by placing them in their mouths. Mouthing is particularly prevalent for 0 to 2-year olds and then reduces sharply (USEPA, 2011). The USEPA (2011) refer to studies by Stanek *et al* (1998) and Calabrese *et al* (1997) that report daily mouthing or ingestion of sand/stones in 6% and soil in 4% of the 528 children studied. For frequency per month these percentages rise to 27% of children mouthing or ingesting sand/stones and 18% mouthing or ingesting soil. It is therefore clear that this pathway is potentially significant.

Measurement of the mass of soil ingested by these behaviours has been attempted, but the results are not clear. Reported ingestion rates for twelve non-pica children are up to 3,581 mg.d⁻¹ depending on the measurement methodology utilised (Calabrese *et al*, 1997), although the standard deviations are up to 1,056 mg.d⁻¹ indicating considerable variability between children.

The USEPA (2011) recommend an upper 95th percentile value of 200 mg.d⁻¹ as representative of average long-term combined soil and dust ingestion from all pathways in non-pica children. An estimate of the maximum daily rates, which would be more appropriate for acute risk assessment and may be much greater, is not provided.

In February 2012 the Office of Environmental Health Hazard Assessment (OEHHA) Scientific Review Panel (SRP) for California released a draft report that summarises previous studies of soil ingestion rates, predominately studies completed in the USA and Netherlands. The report concludes that a 95th percentile value of 400 mg.d⁻¹ is appropriate for assessing long-term exposure to children of 0-2 and 2-6 years old (excluding pica behaviour).

- **Intentional ingestion.** The USEPA (2011) report that the intentional ingestion of soil by children is 'relatively common' being most prevalent in 1 to 3-year old children, and refer to a 2001 ATSDR study which estimates that 33% of

children ingest more than 10,000 mg of soil one or two days a year when describing the range of potential short term one off doses.

Soil pica behaviour (i.e. the repeated intentional ingestion of soil) is outside the scope of this report but estimates of the amount of soil ingested by soil pica children may provide a reasonable worst case for the one-off ingestion of a bolus of soil. USEPA (2011) also recommends a value of 1,000 mg.d⁻¹ for soil-pica behaviour in children and notes that this value may be appropriate for acute exposures. However, they also acknowledge that literature estimates as high as 10,000 mg.d⁻¹ have been reported with a maximum reported rate for one individual of 41,000 mg.d⁻¹. OEHHA (2012) recommend a mean soil-pica ingestion rate of 5,000 mg.d⁻¹ for children of all ages.

The Health Protection Agency (HPA, now Public Health England) applied a value of 5,000 mg.d⁻¹ for assessing acute risks to children using an area of public open space (Kowalczyk *et al*, 2013). This was based on literature review and was considered to be protective of both accidental and intentional ingestion. RIVM also applied a soil ingestion rate of 5,000 mg.d⁻¹ when deriving Dutch soil screening criteria for cyanide (RIVM, 2001).

Based on the above an acute soil ingestion rate of 5,000 mg.d⁻¹ is proposed for deriving AGAC for oral exposure to the child receptor. This is in line with the approach adopted by the HPA for assessing acute risks to children in a public open space and is within the range of rates recommended by other authoritative bodies.

Adult Worker

For the adult worker, potential pathways include indirect hand to mouth transfer (e.g. from eating and smoking after handling contaminated soils) and ingestion of soil derived airborne dust. Some literature sources differentiate between 'contact intensive' and 'non-contact intensive' workers with the former having greater ingestion rates. Workers involved with excavations would be regarded as contact intensive.

The Environment Agency (2009b) recommend an average daily soil and dust ingestion rate of 50 mg.d⁻¹ for assessing chronic risks from long-term exposure to adults, but note that the evidence base for adult soil ingestion is much smaller than for children. This value (based on a single study) is also recommended by the USEPA (2011) who note that confidence levels for soil and dust ingestion rates for adults are low.

Using data from the same studies as the USEPA, the OEHHA (2012) also advised a mean value of 50 mg.d⁻¹ and an upper 95th percentile value of 200 mg.d⁻¹ for assessing chronic risks to adults from long-term exposure.

An adult soil and dust ingestion rate of 480 mg.d⁻¹ was suggested by Hawley (1985) for adults involved in outdoor activities, although this was based on estimates relating to soil and dust levels on hands, extent of mouthing and frequencies of activities rather than more direct measurements (USEPA, 2014). This value has been adopted by the US military for soil ingestion during each field day, and also for activities such as construction or landscaping. The United Nations (UN) adopted a value of 1,000 mg per military field day (UNEP/UNCHS Balkans Task Force, 1999).

The USEPA (2014) Adult Lead Methodology (ALM) states that a plausible range for adult contact-intensive exposure is 50-200 mg.d⁻¹ and advises that there is reasonable support for use of 100 mg.d⁻¹ as a chronic value for contact intense exposure. The ALM also notes that the Office of Solid Waste and Emergency Response (OSWER) recommends an upper bound of 330 mg.d⁻¹ based on Stanek *et al* (1998) (one of the studies referred to by USEPA and OEHHA). OEHHA (2012) note that the value of 330 mg.d⁻¹ was based on an upper 95th percentile value which was significantly affected by a single unusually high sample within the study. This may have reflected 3-4 days of exposure and was therefore considered unreliable for the purposes of estimating long-term soil ingestion rates.

The New Zealand Ministry for the Environment (2011) differentiates between Commercial/ Industrial Indoor and Outdoor workers, and recommends long-term average soil ingestion rates of zero and 50 mg.d⁻¹, respectively.

The limited data available suggest that it may be appropriate to have two sets of ingestion rates for commercial adults: one for non-contact intensive activities and one for contact intensive activities. An ingestion rate of 200 mg.d⁻¹ is proposed for non-contact intensive activities, based on the upper 95th percentile value recommended by OEHHA (2012) for assessing chronic risk and upper end of recommended range from ALM (USEPA, 2014). In the absence of empirical data for intensive activities, an ingestion rate of 400 mg.d⁻¹ is proposed for contact intensive activities, derived from a factor of two applied to the 95th percentile for non-contact intensive activities. This latter value is within the same order as values commonly used for construction workers and has been adopted for the derivation of the adult worker AGACs for the oral route of exposure.

Body Weight

Body weight will vary with age and gender. For the calculation of AGAC for the child receptor it is proposed that a body weight of 10kg is used. This is approximately equal to the average body weight of a 1 to <2 year old female child used in the CLEA model for age class 2 (9.8kg – EA, 2009b).

For the calculation of AGAC for the adult worker receptor it is proposed that a body weight of 70kg is used. This is equal to the average body weight of a 16 to <65 year old female used in the CLEA model for age class 17 (EA, 2009b).

Relative Bioavailability

For the derivation of AGAC the relative bioavailability will normally be assumed to be 1 (100%). For derivation of site-specific assessment criteria there may be justification to reduce the relative bioavailability if there is evidence that the bioavailability of the contaminant in soil is less than the bioavailability of the contaminant in the studies used as the basis of the acute reference dose. Note that bioavailability tends to be greater under fasted conditions and this should be accounted for where a bioavailability of less than 100% is considered.

5.2 Dermal Exposure

5.2.1 Exposure Scenarios

The following exposure scenarios have been considered for the dermal route of exposure:

- General public exposure. The AGACs for this scenario are based on exposure via dermal contact, with soil being left on the skin of a young child for several hours. This could include any land use where children have access to soil, including residential use and open space scenarios. This scenario could also be applied to a child trespasser (although an older child than that discussed below should likely be selected); and
- Occupational exposure. The AGACs for this scenario are based on exposure via dermal contact with soil being left on the skin of an adult commercial/ industrial worker who is exposed to contaminated soil during their work, such as a construction, utility, site investigation or remediation worker. It is assumed that the worker is not wearing PPE.

Dermal contact may occur with soils adhered to the face, hands, arms, legs or other areas of exposed skin.

5.2.2 Model Selection

As discussed in Section 4.1, for the purposes of calculating the AGAC, the toxicological end-point is assumed to be contact dermatitis. The risk of acute adverse systemic effects from diffusion through the skin into the bloodstream has not been considered

because acute systemic risks are more likely to be driven by the oral route of exposure.

The model proposed for calculation of the dermal AGAC is shown below. This method was used by the Environment Agency (2009c) for assessing risk of contact dermatitis for the nickel Soil Guideline Value (SGV) and has also been used to derive the New York State soil clean-up objectives for dermal exposure to irritants in soil (NYSDEC/NYS DH 2006). The method was also used by Kowalczyk *et al* (2013) for assessing risks from contact dermatitis with chromium VI in soil.

$$AGAC_{dermal} = \frac{ARfD_{dermal}}{ABS_d \times AF} \times 10^6 \text{ mg.kg}^{-1}$$

Where

ARfD_{dermal} = Skin reference dose based on irritant contact dermatitis or patch test threshold for no effect level following exposure to allergen (mg.cm⁻²)

AF = Soil to skin adherence factor (mg.cm⁻²)

ABS_d = Dermal absorption fraction (fraction)

5.2.3 Exposure parameters for dermal contact

The exposure parameters used for the calculation of AGAC_{dermal} are described below:

Dermal Absorption Fraction

The dermal absorption factor is the percentage of a substance in the applied soil that is absorbed by the skin. These are chemical specific, and it is proposed to use the absorption factors listed in the Environment Agency (2009b) CLEA SR3 report where available. These are generally based on exposure of up to 24 hours (USEPA, 1992a) and hence are suitable for acute exposure assessment.

Dermal Soil to Skin Adherence Factor

The dermal soil to skin adherence factor is the mass of soil adhered to each square centimetre of exposed skin. It is effectively a measure of how dirty the skin is and is expected to vary with receptor type and activity as well as soil type and moisture content.

Child

The USEPA estimated the 95th percentile mass of soil on skin for children playing in wet and dry soil of 3.3 mg.cm⁻² and 0.4 mg.cm⁻², respectively (USEPA, 2004). There are studies which show higher soil adherence rates (such as children playing in mud) which imply complete covering of the skin with more than a single layer of soil. Such

layers of soil are much less likely to remain on the skin for a long period without either being washed off or falling off. In addition, due to the need for migration of contaminants through the soil to the skin surface the percentage of the substance (the dermal absorbed dose) in the soil from thicker layers of soil is likely to be lower than for a monolayer (i.e. layer of soil one particle thick).

The soil loading that corresponds to a monolayer has not been well established and is likely to vary according to the soil density and the distribution of particles by size. The USEPA (1992a) estimated soil loading for a monolayer to be 8 mg.cm^{-2} . This was based on the assumptions of average particle diameter of $100 \text{ }\mu\text{m}$, particle density of 1500 mg.cm^{-3} and particles being tightly packed. However, the USEPA noted that such tight packing was not consistent with the available data. Based on judgement and unpublished experimental observations, the USEPA identified 5 mg.cm^{-2} as their best estimate of the loading for a monolayer, below which the flux begins to decline (USEPA, 1992a).

For the purposes of deriving the AGAC for the child receptor a soil to skin adherence factor of $5 \text{ mg soil.cm}^{-2}$ is proposed as a reasonable worst case.

Adult Worker

The USEPA (2004) reviewed soil adherence factors for commercial/industrial workers and report 95th percentile soil adherence factors ranging from 0.1 to 0.9 mg.cm^{-2} for most workers. The reported 95th percentile soil adherence factors for construction workers, utility workers and heavy equipment workers were 0.3 , 0.9 and 0.7 mg.cm^{-2} , respectively. Workers involved with pipe laying in wet soils were reported to have significantly higher soil adherence factors, with a 95th percentile of 13.2 mg.cm^{-2} .

For the purposes of deriving the AGAC for the adult worker involved with excavations the 95th percentile value of 0.9 mg.cm^{-2} is considered a reasonable worst case.

5.3 Inhalation Exposure

5.3.1 Exposure Scenarios

The following exposure scenarios have been considered for the inhalation route of exposure:

- General public exposure. The AGACs for this scenario are based on exposure to an off-site child via inhalation of dusts or vapours generated from excavation activities on the site. The child could be playing in an adjacent garden or public open space. This scenario would also be protective of members of the public simply walking past the site. It has been assumed that trespassing is unlikely

to occur when excavation works are actually underway and the on-site scenario for a child is unrealistic.

- Occupational exposure. The AGACs for this scenario are based on exposure to an on-site worker from inhalation of dusts or vapours released during excavation activities (see Figure 5.1 below). Such activities include trial pitting as part of a site investigation, remediation excavation and installation or repair of underground utilities. It is assumed that exposure occurs whilst the worker is stood next to the excavation at the surrounding ground level to the excavation. Note that this scenario does not include the assessment of risks due to entering confined spaces, i.e. the trial pit or excavation.

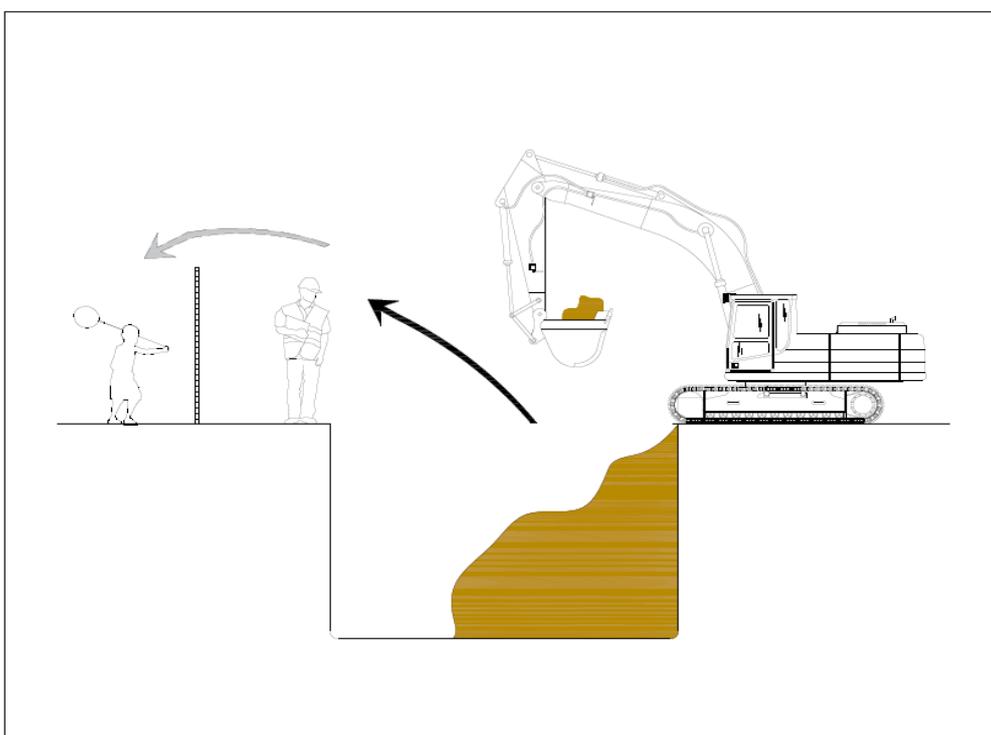


Figure 5.1: Example of inhalation exposure scenario for adult worker (or child in vicinity of excavation)

For these scenarios it is assumed that peak exposure to dust or vapours in air would only be for a short period i.e., no greater than 30 minutes duration. This period reflects either a period of main dust release while moving dusty material, or an initial spike in vapour release as an excavation is first opened.

Note that the AGAC calculated in this report are based on the assumption of trial pit sized excavations (1.8 m² x 3.0 m). For larger excavations, the assessor should amend the excavation dimensions accordingly to ensure the AGAC are protective.

5.3.2 Model Selection

In order to assess the potential acute risks from the release of dust or volatile organic carbon (VOC) vapours, the concentrations at the “point of exposure” need to be estimated. This value can then be compared to the acute reference dose in air (ARfC) in order to assess the level of risk to the receptor. For the purposes of deriving the AGAC, the calculations are conducted in reverse to estimate the theoretical soil concentration at which the point of exposure concentration equals the ARfC.

The methods used to estimate the point of exposure concentration typically comprise two components: (1) a component to estimate the emission rate of contaminant to air (in units of g.d^{-1}); and (2) a “box model” component to estimate the concentration of contaminant in a box of air adjacent to the excavation arising from the emission. These are discussed in the sections below. Note that the method presented herein does not account for the reduction in concentrations due to dispersion away from the area of excavation.

Box Model

A simple box model is used to estimate the average concentration of contaminant in a box adjacent to the excavation. This assumes that there is a constant stream of air through the box into which the emitted contaminants mix.

$$C_{air} = \frac{ER \times 1000}{W \times h \times u}$$

Where

C_{air} = Concentration in air within the box (mg.m^{-3})

ER = Emission rate of VOCs to air (g.s^{-1})

W = Width of box perpendicular to wind direction over which emissions occur (m)

h = Mixing zone height (m)

u = Wind speed (m.s^{-1})

Emissions Model - VOCs

There are few recognised techniques for estimating the release of VOCs following ground disturbance. Modelling of the partitioning of contaminants from soil to air/soil vapour has been discussed in detail within the literature, and is subject to much debate. This is mainly due to the differences found between predicted concentrations of compounds in the gaseous phase when compared to the results from empirical studies. It is generally recognised that there are limitations and simplifications

involved when modelling is taking place in a complex and heterogeneous medium such as soil (CIRIA, 2009). Calculation methods can comprise highly complex models or relatively simple equations. Monitoring may help provide confidence in the emissions model, provided the uncertainties of the monitoring are taken into account.

The method developed by the USEPA (1992b) is considered suitable for the purposes of estimating the AGAC for inhalation of VOCs. This method assumes that VOCs are released directly from air filled pore space within the excavated material and from diffusion of VOCs from pores within the excavated soil to ambient air. The overall emission rate is given by the following equation:

$$ER = ER_{ps} + ER_{diff}$$

Where

ER = Emission rate of VOCs to air (g.s⁻¹)

ER_{ps} = Emission rate of VOCs to air released directly from pore space (g.s⁻¹)

ER_{diff} = Emission rate of VOCs to air from diffusion (g.s⁻¹)

The equations used for estimating the emission rates from pore space and diffusion are given below. Note that default values proposed by USEPA (1992b) are given in square brackets.

Emission rate from pore space

$$ER_{ps} = \frac{P \times MW \times 10^6 \times \theta_a \times ExC \times Q}{R \times T}$$

Where

P = Partial vapour pressure of VOC (mm Hg)

MW = Molecular weight of VOC (g.mol⁻¹)

θ_a = Air filled porosity of soil (fraction)

ExC = Soil gas to atmosphere exchange constant [0.33]

Q = Excavation rate (m³.s⁻¹)

R = Gas constant [62,361 mmHg.cm³.g⁻¹.mol⁻¹.K⁻¹]

T = Soil temperature (K)

This equation assumes that the VOC is at saturation vapour pressure. If this is not the case, the equation will tend to over-estimate the release rate of vapours. For concentrations below saturation an equation assuming equilibrium soil gas concentration is likely to be more appropriate, i.e.:

$$ER_{ps} = \frac{K_{aw}}{K_{sw}} \times C_{soil} \times \theta_a \times ExC \times Q$$

Where

K_{aw} = air water partition coefficient, ambient temperature ($\text{cm}^3 \cdot \text{cm}^{-3}$)

K_{sw} = total soil-water partition coefficient ($\text{cm}^3 \cdot \text{g}^{-1}$)

C_{soil} = soil concentration ($\text{mg} \cdot \text{kg}^{-1}$)

As described in the CLEA SR3 report (EA, 2009b), the total soil water partition coefficient is given using the equation below:

$$K_{sw} = \frac{\theta_w + (K_{oc} \cdot f_{oc} \cdot \rho_{soil}) + (K_{aw} \cdot \theta_a)}{\rho_{soil}}$$

Where

θ_w = Water filled porosity of soil (fraction)

K_{oc} = Organic carbon partition coefficient ($\text{cm}^3 \cdot \text{g}^{-1}$)

f_{oc} = Fraction of organic carbon in soil (fraction)

ρ_{soil} = Soil bulk density ($\text{g} \cdot \text{cm}^{-3}$)

Emission rate from diffusion

$$ER_{diff} = \frac{C_s \times 10^4 \times A}{\left(\frac{\theta_a}{K_{eq} \times k_g} \right) + \sqrt{\frac{\pi \times t}{D_e \times K_{eq}}}}$$

Where

C_s = Mass loading in bulk soil ($\text{g} \cdot \text{cm}^{-3}$)

A = Emitting surface area (m^2)

t = Time to achieve best fit curve (s) [60]

K_{eq} = Equilibrium coefficient [dimensionless]

k_g = Gas phase mass transfer coefficient ($\text{cm} \cdot \text{s}^{-1}$) [0.15]

D_e = Effective diffusivity in air ($\text{cm}^2 \cdot \text{s}^{-1}$)

R = Gas constant ($\text{mmHg} \cdot \text{cm}^3 \cdot \text{mol}^{-1} \cdot \text{K}^{-1}$) [62361]

T = Soil temperature (K)

The mass loading in bulk soil is given by:

$$C_s = C_{soil} \times \rho_{soil} \times 10^{-6}$$

The equilibrium coefficient can be calculated using the following equation:

$$K_{eq} = \frac{K_{aw}}{K_{sw}} \times \frac{\theta_a}{\rho_{soil}}$$

The effective diffusivity in air is calculated using:

$$D_e = \frac{D_a \theta_a^{3.33}}{\theta_t^2}$$

Where

D_a = Diffusivity coefficient in air ($\text{cm}^2.\text{s}^{-1}$)

θ_t = Total porosity of soil (fraction)

Emission Rate Model - Dust

The USEPA (1995) has also developed a handbook for soil emission factors (AP42) for a variety of construction activities. The following empirical equation is provided (in Section 13.2.4.3) for estimating dust emission from aggregate handling operations:

$$E = \frac{k \times 0.0016 \times (u / 2.2)^{1.3}}{(M / 2)^{1.4}}$$

Where

E = Mass of dust particles emitted per tonne of material transferred (kg.tonne^{-1})

k = Particle size multiplier (dimensionless)

u = Wind speed (m.s^{-1})

M = Material moisture content (%)

For dust particles less than 10 μm in diameter (i.e. respirable particles), the AP42 document (Section 13.2.4.3) suggests a particle size multiplier of 0.35.

This equation can be used to estimate an emission rate by multiplying by the excavation rate, soil density and contaminant concentration in soil, i.e.:

$$ER_{dust} = \frac{k \times 0.0016 \times (u / 2.2)^{1.3}}{(M / 2)^{1.4}} \times Q \times \rho_{soil} \times C_{soil} \times 10^{-3}$$

Where

ER_{dust} = Emission rate of contaminant as respirable dust ($g.s^{-1}$)

Q = Excavation rate ($m^3.s^{-1}$)

ρ_{soil} = Soil bulk density ($g.cm^{-3}$)

C_{soil} = Soil concentration ($mg.kg^{-1}$)

The USEPA also provide methods for estimating emission factors for a range of other activities, such as bulldozing, grading and truck loading. These methods are not directly relevant to the scope of this document and therefore the reader is referred to the AP42 document if further details on these methods are required.

5.3.3 Conversion to acute generic assessment criteria for soil

In order to derive the inhalation AGAC, the above equations must be rearranged to determine the soil concentration at which the predicted point of exposure concentration equals the ARfC. The reader is reminded that the scenarios being assessed only relate to excavation of soil.

For substances generating VOCs

$$AGAC_{soil} = \frac{ARfC_{inh} \times W \times h \times u}{\left(\left[\frac{K_{aw}}{K_{sw}} \times Q \times \theta_a \times ExC \times 10^3 \right] + \left(\frac{A \times 10 \times \rho_{soil}}{\left[\frac{\theta_a}{K_{eq} \times k_g} + \sqrt{\frac{\pi \times t}{D_{eff} \times K_{eq}}} \right]} \right) \right)}$$

Where:

$AGAC_{inh_VOCs}$ = acute generic assessment criteria for inhalation of VOCs ($mg.kg^{-1}$)

$ARfC_{inh}$ = acute reference concentration in air ($mg.m^{-3}$)

$$K_{eq} = \frac{K_{aw}}{K_{sw}} \times \frac{\theta_a}{\rho_{soil}} \quad D_e = \frac{D_a \theta_a^{3.33}}{\theta_t^2}$$

For substances generating dust

$$AGAC_{inh_dust} = \frac{ARfC_{inh} \times W \times h \times u \times (M/2)^{1.4}}{k \times Q \times \rho_{soil} \times 0.0016 \times (u/2.2)^{1.3}}$$

Where:

$AGAC_{inh_dust}$ = acute generic assessment criteria for inhalation of dust ($mg.kg^{-1}$)

$ARfC_{inh}$ = acute reference concentration in air ($mg.m^{-3}$)

For the purposes of deriving the AGAC presented in this report it has been assumed that a 1.8 m² x 3.0 m deep trial pit is excavated over a 30-minute period and that the receptor is immediately downwind of the excavation and the resulting stockpile located adjacent to the trial pit. The trial pit area assumes that the excavation is 3.0 m long and 0.6 m wide. The model assumes that there are emissions from the base and sidewalls of the trial pit (area of 23.4 m²), and also from the surface of the resulting stockpile. The stockpile is assumed to be a regular cone with a diameter of 4.0 m, height of 1.7 m and surface area of 16.4 m². Thus, total surface area for emissions is assumed to be 39.8 m². Note that dimensions of the cone were calculated from the in-ground volume of the excavation of 5.4 m³ multiplied by a soil bulking factor of 1.3 following excavation and an assumed reasonable stockpile diameter. Applying different shapes to the stockpile such as rectangular or triangular based prisms with reasonable dimensions made little difference to the calculated emitting area for the trial pit stockpile scenario.

Emissions are assumed to occur into a box with length of 10 m in the direction of wind flow which has a speed of 1 m.s⁻¹ (i.e. a light breeze). The height of the mixing zone is taken as 1.0 m for a child and 2.0 m for an adult. It should be noted that the model calculates the width of the box by dividing the area by the length, thus making the box longer reduces the width and therefore produces more conservative AGAC. The value of 10 m has been selected as a conservative estimate for deriving AGAC. In specific scenarios where the distance between working area and receptors is well defined then the assessor could consider reducing this value.

As noted in Section 5.3.1, these parameters assume a typical trial pit sized excavation. Assessors of larger excavations such as on remediation projects should amend the dimensions accordingly.

In relation to soil properties for the vapours a sandy loam soil has been assumed, which is the soil type assumed for calculation of the SGVs. It should be noted that the USEPA dust model is only calibrated for soil with a 1 to 4.8% moisture content and clay/silt fraction of 0.44 to 19%. The upper end value of 4.8% for moisture content has been used because this is likely to be a reasonable worst case for UK soils.

5.3.4 On Site Measurements

An estimation of the total concentrations of VOCs likely to be generated from the disturbance of a soil source can also be made through on-site measurements. Available methods include flux boxes, borehole gas monitoring and the 'fluff' test (CIRIA, 2012). The use of other on-site measurement techniques to measure actual

dust or vapour released may be beneficial for validating the results of emission and dispersion modelling.

5.4 Summary of exposure parameters

The exposure parameters selected for calculation of the AGAC presented in this report are summarised in the tables below. It is imperative that the assessor checks whether these parameters are suitable for the scenario they are considering before using the AGAC presented in this report.

Table 5.1 - Summary oral exposure parameters for child receptor

Parameter	Value	Units	Justification and reference
Soil ingestion rate	5,000	mg.d ⁻¹	Value adopted by Kowalczyk <i>et al</i> (2013) and recommended by RIVM (2001) and OEHHA (2012) for assessing acute risks to children. Within the range of measured short-term soil ingestion rates for children given by USEPA (2011). Assumes no pica behaviour.
Exposure duration/frequency	1	Day/ Single event	Approach adopted in CLP Regulations and in USEPA for assessing acute risks
Body weight	10	Kg	Based on CLEA body weight of 9.8 kg for 1-year to 2-year old female children (EA, 2009b) and common practice when assessing risks to children (USEPA, 2011).

Table 5.2 - Summary of typical exposure for workers involved with excavations

Parameter	Value	Units	Justification and reference
Ingestion rate Non-contact intensive activities	200	mg.d ⁻¹	Upper 95 th percentile value recommended by OEHHA for assessing chronic risk (OEHHA, 2012) and upper end of recommended range from ALM (USEPA, 2014). Assumes no pica behaviour and no contact-intensive behaviour such as digging. Likely to be conservative.
Ingestion rate Contact intensive activities	400	mg.d ⁻¹	Factor of two applied to value selected for non-contact intensive activities and of same order as values of 330 – 480 mg/d commonly used in risk assessments for construction workers.
Exposure duration/frequency	1	Working day mg.d ⁻¹	Approach adopted in CLP Regulations and in USEPA for assessing acute risks.
Body weight	70	Kg	Based on CLEA body weight for a female worker (EA, 2009b).

Table 5.3 - Summary of dermal exposure parameters for a child

Parameter	Value	Units	Justification and reference
Dermal Soil to Skin Adherence factor	5	mg.cm ⁻²	Conservative estimate based on USEPA (2004) 95 th percentile estimate for children playing in wet soil and USEPA (1992) estimate for monolayer thickness.
Dermal Absorption factors	Chemical specific	Unitless	Recommend using same values as used for assessing chronic risks

Table 5.4 - Summary of dermal exposure parameters for workers involved with excavations

Parameter	Value	Units	Justification and reference
Dermal Soil to Skin Adherence factor	0.9	mg.cm ⁻²	Based on 95th percentile soil adherence for Utility workers USEPA (2004).
Dermal Absorption factors	Chemical specific	Unitless	Recommend using same values as used for assessing chronic risks

Table 5.5 - Key parameters for modelling VOC and dust emission and dilution

Parameter	Value	Units	Justification and reference
Box Model for Air Dilution			
Width of box perpendicular to wind direction (W)	5	m	Based 4m diameter stock pile with an allowance of 1m width for the trial pit (0.6m + 0.4m stand off from stockpile)
Mixing zone height for box model (h)	1 for child 2 for adult	m	Values from EA (2009b)
Wind speed (u)	1	m.s ⁻¹	Minimum value given in US EPA offices of Air and Radiation Research and Development (1997)
Excavation rate [volume of disturbed soil surface area] (Q)	0.003	m.s ⁻¹	Assumed that a 3m deep trial pit is excavated, with a surface area of 1.8m ² in 30min.

Table 5.5 (Continued) - Key parameters for modelling VOC and dust emission and dilution

Emission Rate Model for Vapours			
Surface area of which emissions occur (A)	39.8	m ²	Comprised of total emitting area from trial pit and stockpile as set out below.
	23.4	m ²	Trial pit: Assumes excavation is 0.6m wide (2ft JCB bucket), 3.0m long (ground investigation trial pit) and 3.0m deep. Includes emissions from side walls of pit with contamination present from ground surface
	16.4	m ²	Stockpile: Assumes regular cone of 4.0m diameter and 1.7m height
Air-filled porosity of soil (θ_a)	0.2	Unitless	CLEA default value for sandy loam soil (EA, 2009b)
Water-filled porosity of soil (θ_w)	0.33	Unitless	CLEA default value for sandy loam soil (EA, 2009b)
Total porosity of soil (θ_t)	0.53	Unitless	CLEA default value for sandy loam soil (EA, 2009b)
Bulk density of soil (ρ_{soil})	1.21	g.cm ⁻³	CLEA default value for sandy loam soil (EA, 2009b)
Soil gas to atmosphere exchange constant (ExC)	0.33	Unitless	USEPA (1997) suggested value for dry sandy soil
Gas-phase mass transfer coefficient (k_g)	1.5	cm.s ⁻¹	USEPA (1997) suggested value
Time since start of excavation of soil of interest (t)	60	s	USEPA (1997) suggested value
Soil temperature (T)	283	K	CLEA default value for sandy loam soil (value is not directly used in equation, but is used to calculate Ksw) (EA, 2009b)
Fraction of Organic Carbon (foc)	0.0058	Fraction	Assumed 1% soil organic matter
Emission Rate Model for Dust			
Bulk density of soil (ρ_{soil})	1.21	g.cm ⁻³	CLEA default value for sandy loam soil (EA, 2009b)
Moisture content	4.8	%	Upper end of moisture content for model calibration (USEPA, 1995). (Sandy loam is generally wetter)
Particle size multiplier (k)	0.35	Unitless	Value in AP42 for dust particles less than 10 μ m in diameter (USEPA, 1995)

6 USE OF FRAMEWORK TO DERIVE AGAC

The framework has been used to derive AGAC values for the following contaminants:

- Arsenic;
- Benzene;
- Cadmium;
- Free Cyanide;
- Lead;
- Phenol;
- Trichloroethylene (TCE); and
- Vinyl Chloride (VC).

A summary of the assessment and the calculated AGACs for each contaminant are presented below. An example spreadsheet is presented in Appendix 1.

Note that the AGACs presented in the following table are based on the exposure scenarios described in Sections 5.1.1, 5.2.1 and 5.3.1. Users should have read and be familiar with the entire contents of this report, in particular Sections 2.5 and 2.6, to understand the applicability of these AGACs for their assessment.

Acute Generic Assessment Criteria - Arsenic

Summary of AGAC (mg/kg)

	Child	Adult
Oral	80	7,000
Dermal	Not Derived	Not Derived
Inhalation	7,000,000	14,000,000

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw	0.04	Armstrong CW, Stroube RB, Rubio T, Siudyla EA, Miller GB. Outbreak of fatal arsenic poisoning cause by contaminated drinking water. Arch Environ Health 39:276-290 (1984).	Based on Armstrong study in which contaminated well water was drank for 1 week in 1980s. The LOAEL was 0.2 so the aRfD would be 0.04 by applying a UF of 5 for use of LOAEL for throat irritation, nausea and vomiting and 1 for human variability.
Acute dermal reference dose	mg/cm ²	n/a	n/a	No acute dermal data
Acute inhalation reference concentration - child	mg/m ³	0.3	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on 3 x 8hr WEL for arsenic as a guide to STEL. This is lower than the AEGL-2 (30 mins) of 0.67 mg/m ³ for arsine (there is no AEGL-1). Note that CalEPA acute oral reference exposure level (REL) ⁶ of 0.0002 µg/m ³ not used as this is based on fetal effects from 4hr inhalation exposure to pregnant mice over 4 days of gestation period and is not considered relevant to scenario being modelled.
Acute inhalation reference concentration - adult	mg/m ³	0.3	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on 3 x 8hr WEL as a guide to STEL

⁶ Air Toxics Hot Spots Risk Assessment Guidelines Technical Support Document For the Derivation of Noncancer Reference Exposure Levels, June 2008

Acute Generic Assessment Criteria - Benzene

Summary of AGAC (mg/kg)

	Child	Adult
Oral	47	4,100**
Dermal	14,000,000**	79,000,000**
Inhalation	120	240

Notes:

**Likely to exceed soil saturation limits. AGAC is not applicable where free product is present, see section 2.6.3.

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw/	0.0235	USEPA Benzene health advisory, Office of Drinking Water, March 31, 1987	USEPA Health advisory for 10 day drinking water standard - Based on Haematological impairment (including severe leukopenia) in rats. Sprague-Dawley rate inhaled benzene for 6hrs per day 4 days per week with review after second week. Uncertainty factor of 100 for inter and intraspecies variation with route to route extrapolation
Acute dermal reference dose	mg/cm ²	7.1	New York State Brownfield Cleanup Program, Development of Soil Cleanup Objectives, Technical Support Document. September 2006. Appendix C-1	Based on Mouse Ear Swelling test in mice from Gad SC, Dunn BJ, Dobbs DW, et al. 1986. Development and validation of an alternative dermal sensitization test: the mouse ear swelling test (MEST). Toxicol Appl Pharmacol. 84:93-114.
Acute inhalation reference concentration - child	mg/m ³	9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL. This is lower than the AEGL-1 (30 mins) of 237 mg/m ³
Acute inhalation reference concentration - adult	mg/m ³	9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL

Acute Generic Assessment Criteria - Cadmium

Summary of AGAC (mg/kg)

	Child	Adult
Oral	140*	12,000
Dermal	Not derived	Not derived
Inhalation	1,800,000	3,500,000

Notes * slightly below the C4SL for residential without plant uptake in gardens of 150mg/kg.

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw	0.07	Health Canada. Draft Proposal for Cadmium Guideline in Children's Jewellery. July 2011.	The basis for the MRL is a study by Borzelleca et al. (1989) in which Sprague-Dawley rats were administered with cadmium chloride via drinking water for 10 days. A NOEL of 7.3 mg /kg was identified and uncertainty factor of 100 was applied for inter and intraspecies variation.
Acute dermal reference dose	mg/cm ²	No data	-	-
Acute inhalation reference concentration - child	mg/m ³	0.075	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL. This is lower than the AEGL-1 (30 min) of 0.13 mg/m ³
Acute inhalation reference concentration - adult	mg/m ³	0.075	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL

Acute Generic Assessment Criteria – Free Cyanide

Summary of AGAC (mg/kg)

	Child	Adult
Oral	24	2100
Dermal	Not derived	Not derived
Inhalation	380	1,400

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw	0.012	Environment Agency (EA). Contaminants in Soil: Collation of Toxicological Data And Intake Values For Humans. Inorganic Cyanide. Tox 5. 2002	EA Tolerable Daily Intake (TDI) based on study where potassium cyanide was administered for 24 weeks by gavage to swine. LOAEL based on increased ambivalence and longer response time to various stimuli. The EA indicated that ingestion of a bolus dose of cyanide equivalent to the TDI would not be expected to cause any acute toxicity. EFSA have derived an Acute Reference dose for cyanide based on a study of humans fed cyanogenic glycosides (CNGs) but this only applied to foods containing CNGs as the main source of cyanide (EFSA Panel on Contaminants in the Food Chain (CONTAM), 2016. Acute health risks related to the presence of cyanogenic glycosides in raw apricot kernels and products derived from raw apricot kernels.)
Acute dermal reference dose	mg/cm ²	No data	n/a	No authoritative data for NOAEL or LOAEL.
Acute inhalation reference concentration - child	mg/m ³	2.8	AEGL-1, 10 & 30 min.	Board on Environmental Studies and Toxicology. Acute Exposure Guideline Levels (AEGLs) for Selected Airborne Chemicals Volume 2. 2002. Adopted as lower than the UK WEL.
Acute inhalation reference concentration - adult	mg/m ³	5	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition). STEL 15 min	UK authoritative occupational health acute exposure limit.

Acute Generic Assessment Criteria – Lead

Acute generic assessment criteria were considered for lead. A review of the available toxicological data for lead indicated that most of the thresholds related to blood lead levels. For instance, the UK Surveillance of Elevated Blood Lead in Children (PHE, 2018) used a threshold of 10µg/dL as a reporting threshold to assess the potential for raised blood lead levels, and the Control of Lead at Work Regulations 2002 (HSE, 2002) sets an action level as a blood-lead concentration of -

- (a) in respect of a woman of reproductive capacity, 25 µg/dL;
- (b) in respect of a young person, 40 µg/dL; or
- (c) in respect of any other employee, 50 µg/dL;

The current AGAC methodology does not address calculating blood lead concentrations following short term exposure. We are aware that dynamic physiologically-based pharmacokinetic (PBPK) models such as the Leggett model (Pounds & Leggett, 1998) may allow assessment of the effects of short-term exposure on blood lead, but consideration of the accuracy and appropriateness of the models are outside the scope of this document.

As part of our review we identified some data on environmental sources that may inform acute thresholds for lead in soil. We note that for most incidents of lead poisoning the exposure time is not clear, and the recorded incidents may have occurred following repeated exposure over days or weeks. There is also uncertainty on the source concentrations, bioavailability of lead and amounts ingested. Some of the data identified include:

- Cases of lead poisoning from snooker and pool chalk cube including
 - A 3 year, 9-month old child with pica presented with a blood lead concentration of 36 µg/dL from sucking a snooker chalk cube with 7,200 mg/kg of lead (Dargan *et al*, 2000).
 - A 28-month old girl known to have eaten at least one pool cue chalk cube had blood lead concentrations of 22 to 35 µg/dL from pool cue chalk with 4,030 mg/kg of lead (Miller *et al*, 1996).
 - A 27-month old boy who had blood lead concentrations of up to 25.6 µg/L had been seen to have pool cue chalk in his mouth on several occasions. This was found to contain 7,000 mg/kg of lead (Miller *et al*, 1996).(Note that other possible sources were also present here).
- Case of lead in a 5-year old (11.6 µg/dL to 20.4 µg/dL) where poisoning originated from turmeric spice (23 mg/kg of lead). It was estimated that assuming a 5 g dose per day (representative of an acute soil ingestion pica-like dose and of the family’s daily spice

use), the 5-year old could have been consuming 4.6 µg/kg bw per day, approaching double the maximum normal dietary intake (CHAPD, 2016).

- Case of lead poisoning from opium in an adult with blood lead level of 114 µg/dL. The adult patient presented to a hospital with a 4-day history of migratory colicky (cramping) abdominal pain, absolute constipation, nausea, vomiting and anorexia. As a result, the patient was treated with chelation therapy. Although the lead composition of the opium is unknown, the patient smoked approximately 10 g per week (CHAPD, 2016).
- A review of incidents of lead poisoning from environmental sources by the HPA in 2008 (CHAPD, 2008) noted that the main source of lead exposure in children was paint. However, in many cases this was not considered to be 'true' lead paint (20-50% lead) but rather 'non-lead' (0.1-1%) paint. Lead in paint can have high bioaccessibility (e.g. due to lead acetate and lead carbonate).

Until the 1950s, UK paint may have contained up to 50% lead by weight (500,000 mg/kg), which is potentially capable of causing lead poisoning in a small child if they ate just a single chip. Leaded paint at these concentrations may still be found in non-remediated Victorian properties. Voluntary agreements and legislation, such as the 1968 British Standard to label paint with lead concentrations less than 1.0 mg/cm² or 0.5% by weight as 'low-lead paint' and the eventual prohibition of any added lead in 1992 (except in specialist paints), have considerably reduced the likelihood of exposure to lead from paint.

PHE has arrangements with specialist laboratories to notify Health Protection Teams (HPT) within PHE of results where the blood lead level is greater than 10 µg/dL (0.48 µmol/l) in children under 16 years old. The treating physician may then be contacted by the HPT.

Symptoms are often not evident until blood lead concentrations are approximately 50 µg/dL (2.4 µmol/l), although there is evidence that lead has deleterious health effects at blood lead concentrations considerably lower than this. The developing nervous system is particularly susceptible and exposures that correspond to a blood lead level as low as 2 µg/dL have been reported to cause developmental lead neurotoxicity (EFSA, 2010).

Chelation therapy is often carried out on children where blood lead exceeds 50 µg/dL (2.4 µmol/l). Management of cases below this limit normally involves removal from exposure.

The PHE compendium of chemical hazards document on lead (PHE, 2017) and the ATSDR toxicological profile for lead (ATSDR, 2007) contain more detailed information on the adverse health effects associated with acute exposure to lead.

References for Lead

Agency for Toxic Substances and Disease Registry (ATSDR), 2007. Toxicological Profile for Lead. August 2007. Available at <https://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>

Chemical Hazards and Poisons Division (CHAPD), 2008. Lead poisoning cases associated with environmental sources. Chemical Hazards and Poisons Report. Issue 11, January 2008, p16.

Chemical Hazards and Poisons Report (CHAPD), 2016. Unusual cases of lead poisoning in the UK. Chemical Hazards and Poisons Report, Issue 26, April 2016, p73.

Dargan P.I., Evans P.H., House I.M. & Jones A.L., 2000. A case of lead poisoning due to snooker chalk. Arch Dis Child. December 2000. 83(6):519-20.

EFSA (European Food Safety Authority) Panel on Contaminants in the Food Chain (CONTAM), 2010. Scientific Opinion on Lead in Food. EFSA Journal 2010; 8(4): 1570

Health and Safety Executive (2002). Control of lead at work (Third edition) Control of Lead at Work Regulations 2002. Approved Code of Practice and guidance

Miller M.B., Curry S.C., Kunkel D.B., Arreola, P., Arvizu, E., Schaller, K. and Salmen D., 1996. Pool cue chalk: a source of environmental lead. Pediatrics 1996;97:916-7

Pounds J.G, & Leggett R.W., 1998. The ICRP age-specific biokinetic model for lead: validations, empirical comparisons, and explorations. Environ Health Perspect. 106 Suppl 6:1505-11.

Public Health England (PHE), 2017. Guidance on Lead: health effects, incident management and toxicology. Information on lead, for use in responding to chemical incidents. Available at <https://www.gov.uk/government/publications/lead-properties-incident-management-and-toxicology>

Public Health England (PHE), 2018. Surveillance of Elevated Blood Lead in Children (SLiC). A British Paediatric Surveillance Unit analysis

Acute Generic Assessment Criteria - Phenol

Summary of AGAC (mg/kg)

	Child	Adult
Oral	2,000	175,000**
Dermal	Not derived	Not derived
Inhalation	73,000**	320,000**

Notes:

**Likely to exceed soil saturation limits. AGAC is not applicable where free product is present, see section 2.6.3.

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw	1	ATSDR Acute oral reference dose. Toxicological Profile for Phenol. September 2008	Agency for Toxic Substances and Disease Registry (ATSDR) is considered to be sufficiently authoritative. Thresholds based on pregnant Sprague-Dawley rats study dosed by gavage in water on gestation days (GDs) 6–15. BMDL derived based on decreased maternal weight gain and UF for inter and intra species variability. Limited non-fatal oral data identified following literature search.
Acute dermal reference dose	mg/cm ²	n/a	n/a	Phenol is not a sensitizing agent, and therefore the dermal methodology proposed within this assessment (which is based on the Nickel SGV) is not appropriate. Information on non-fatal dermal exposure is presented within the EA SGV report, from which the commercial SGV was calculated. (Environment Agency 2009 Soil Guideline Values for phenol in soil Science Report SC050021 / Phenol SGV). This uses a different methodology to that in the current report.
Acute inhalation reference concentration - child	mg/m ³	16	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Short term exposure limit (STEL) chosen as more conservative than AEGL-1 which is 73 mg/m ³ for 10min and 30min exposure (
Acute inhalation reference concentration - adult	mg/m ³	16	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Short term exposure limit (STEL)

Acute Generic Assessment Criteria - Trichloroethene (TCE)

Summary of AGAC (mg/kg)

	Child	Adult
Oral	Not derived	Not derived
Dermal	No data	No data
Inhalation	8,000	16,000

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw	n/a	The European Chemicals Agency (ECHA)	Oral risks screened out as TCE not considered acutely toxic under CLP regulation.
Acute dermal reference dose	mg/cm ²	n/a	No data identified	No data is identified
Acute inhalation reference concentration - child	mg/m ³	820	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL. This is lower than the AEGL-1 (30 mins) of 967 mg/m ³
Acute inhalation reference concentration - adult	mg/m ³	820	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL

Acute Generic Assessment Criteria – Vinyl Chloride

Summary of AGAC (mg/kg)

	Child	Adult
Oral	Not derived	Not derived
Dermal	Not derived	Not derived
Inhalation	98	220

Toxicity Basis

	Units	Value	Reference	Rationale
Acute oral reference dose	mg/kg/bw	n/a	n/a	DEFRA state in their review that no studies on the effects of ingested vinyl chloride have been found.
Acute dermal reference dose	mg/cm ²	n/a	n/a	Effects from dermal exposures are unlikely as vinyl chloride is not well absorbed across the skin (ATSDR).
Acute inhalation reference concentration - child	mg/m ³	23.4	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on 3 x 8hr WEL as guide to STEL. This value is lower than the AEGL-1 (30 min) of 800 mg/m ³
Acute inhalation reference concentration - adult	mg/m ³	23.4	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on 3 x 8hr WEL as guide to STEL

Comments

An acute risk for inhalation is not required under risk phases (because there are no applicable hazard codes for short term inhalation exposure for vinyl chloride (see Section 3 of main report)). However, an AGAC has been generated using available data.

7 REFERENCES

Calabrese, E.J., Stanek, E.J., Pekow, P., 1997. Soil ingestion estimates for children residing on a Superfund site. *Ecotoxicol Environ Saf.* 36:258–268

Center for Environmental & Human Toxicology University of Florida, 2005. Technical Report: Development of Cleanup Target Levels (CTLs) For Chapter 62-777, F.A.C. 2005.

CIRIA, 2009. The VOCs Handbook. Investigating, assessing and managing risks from inhalation of Volatile Organic Compounds (VOCs) at land affected by contamination. CIRIA C682.

CIRIA, 2012. Remediating and mitigating risks from volatile organic compound (VOC) vapours from land affected by contamination. CIRIA C176.

European Commission, 2001 Health & Consumer Protection Directorate-General 7199/VI/99 rev. 5 05/07/2001 Draft Guidance Document Guidance For The Setting Of An Acute Reference Dose (ARfD)

https://ec.europa.eu/food/sites/food/files/plant/docs/pesticides_ppp_app-proc_guide_tox_acute-ref-dose.pdf

Defra and Environment Agency, 2004. Model Procedures for the Management of Land Contamination. Contaminated Land Report 11. Environment Agency, September 2004. ISBN: 1844322955.

Environment Agency (EA), 2009a. Updated Human health toxicological assessment of contaminants in soil Science Report – SC050021/SR2 Environment Agency, January 2009. ISBN: 978-1-84432-858-1.

Environment Agency (EA), 2009b. Updated Technical Background to the CLEA Model. Science Report: SC050021/SR3. Environment Agency, January 2009. ISBN: 978-1-84432-856-7.

Environment Agency (EA), 2009c. Soil Guideline Values for nickel in soil. Science Report SC050021 / Nickel SGV. Environment Agency, March 2009.

European Agency for Safety and Health at Work, 2003. FACTS40 Skin sensitisers <https://osha.europa.eu/en/tools-and-publications/publications/factsheets/40>

Hawley JK. 1985. Assessment of health risk from exposure to contaminated soil. *Risk Anal* 5(4): 289-302

Health Protection Agency (HPA), 2014. Heating oil incidents: action card for public health practitioners v1.0.

https://webarchive.nationalarchives.gov.uk/20140714112439/http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1284475799316

Health and Safety Executive (HSE), 2018. EH40/2005 Workplace Exposure Limits (third edition). <http://www.hse.gov.uk/pubns/books/eh40.htm>

Kowalczyk G., Brown, M., Twigg, R., Welfare W. and Macklin Y., 2013. Contaminated land: can acute exposure be a significant health risk? Two case studies and associated risk assessment methods. *Environmental Science Process and Impacts*, 2013, 15, 1859-1865. August 2013.

New Jersey Department of Environmental Protection (NJDEP), 2012. Response to Charge Question on Development of Health-Based Acute Criteria Summary Report of the NJDEP Science Advisory Board, February 2012.

New York State Department of Environmental Conservation (NYSDEC) and the New York State Department of Health (NYSDH), 2006. New York State Brown-field Cleanup Program, Development of Soil Cleanup Objectives, Technical Support Document. September 2006.

New Zealand Ministry for the Environment, 2011. Methodology for Deriving Standards for Contaminants in Soil to Protect Human Health. ME1055. June 2011.

Organisation for Economic Co-operation and Development (OECD), 2002. OECD Guideline For The Testing Of Chemicals Acute Dermal Irritation/Corrosion 404 Adopted: 24th April 2002.

Office of Environmental Health Hazard Assessment (OEHHA), 2012. Air Toxics Hot Spots Program Risk Assessment Guidelines Technical Support document for Exposure Assessment and Stochastic Analysis, Draft SRP Report 2012.

Renwick, 2000. Renwick AG The use of safety or uncertainty factors in the setting of acute reference doses *Food Addit Contam.* 2000 Jul;17(7):627-35.

RIVM, 2001. Risk assessment of historical soil contamination with cyanides; origin, potential human exposure and evaluation of Intervention Values. H.W Kostner. RIVM Report 711701019. January 2001.

Stanek, E.J., Calabrese, E.J., Mundt, K., Pekow, P. and Yeatts, K.B., 1998. Prevalence of soil mouthing/ingestion among healthy children aged 1 to 6. *J Soil Contam* 7(2):227-242.

UNEP/UNCHS Balkans Task Force (BTF), 1999. The potential effects on human health and the environment arising from possible use of depleted uranium during the 1999 Kosovo conflict. 1999.

USEPA, 1992a. Environmental Assessment, Dermal Exposure Assessment: Principles and Applications EPA/600/8-91/011B January 1992.

USEPA, 1992b. Estimation of Air Impacts for the excavation of contaminated Soil' EPA-450/1-92-004, March 1992.

USEPA, 1995. US EPA offices of Air and Radiation Research and Development AP 42, Fifth Edition Compilation of Air Pollutant Emission Factors, January 1995.

<https://www.epa.gov/air-emissions-factors-and-quantification/ap-42-compilation-air-emission-factors>

USEPA, 1997. Air Emissions for the Treatment of Soils Contaminated with Petroleum Fuels and Other Substances, Offices of Air & Radiation and research & Development. EPA-600/R-97-116. October 1997.

USEPA 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)

USEPA, 2011. Exposure Factors Handbook. EPA/600/R-090/052F. 2011

USEPA, 2014. Adult Lead Methodology <http://www.epa.gov/superfund/lead/almfaq.htm>
Last updated 25/11/2013. Accessed 12/01/2014.

USEPA, 2018. 2018 Edition of the Drinking Water Standards and Health Advisories. EPA 822-F-18-001. Office of Water U.S. Environmental Protection Agency. Washington, DC. March 2018

APPENDIX 1
EXAMPLE SPREADSHEET FOR
DERIVATION OF AGAC

Substance Name	Benzene
CAS Number	71-43-2

Risk and Hazard Phrases	http://ec.europa.eu/enterprise/sectors/chemicals/doc Reference	
where possible refer to http://ec.europa.eu/enterprise/sectors/chemicals/documents/classification/index_en.htm and check main report and adaptations		
Source	Value	Meaning
CLP-Regulation (EC) No 1272/2008	H225	Highly flammable liquid and vapour
CLP-Regulation (EC) No 1272/2008	H350	May cause cancer
CLP-Regulation (EC) No 1272/2008	H340	May cause genetic defects
CLP-Regulation (EC) No 1272/2008	H372	Causes damage to organs through prolonged or repeated exposure
CLP-Regulation (EC) No 1272/2008	H304	May be fatal if swallowed and enters airways
CLP-Regulation (EC) No 1272/2008	H319	Causes serious eye irritation
CLP-Regulation (EC) No 1272/2008	H315	Causes skin irritation

Toxicity Data

	Value	Reference
Acute oral reference dose	mg.kg(bw) ⁻¹ .d ⁻¹ 0.0235	USEPA Health advisory for 10 day drinking water standard
Acute dermal reference dose	mg.cm ⁻² 7.1	New York State Brownfield Cleanup Program Development of Soil Cleanup Objectives. Technical Support Document Appendix C-1. Method for Deriving Soil Cleanup Objectives (SCOs) for Soil Contaminants Based on Toxicity Data for Irritant Contact Dermatitis (Non-Allergic Skin Irritation).
Acute inhalation reference concentration - child	mg.m ⁻³ 9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)
Acute inhalation reference concentration -worker	mg.m ⁻³ 9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)

Chemical specific Exposure parameters

Oral			
Relative bioavailability oral (soil:tox)	-	1	Assumes that absolute bioavailability of contaminant in soil = absolute bioavailability of contaminant in form used to derive ARfD Default - assume 1
Dermal			
Dermal absorption fraction	-	0.1	CLEA default for VOCs- Updated technical background to the CLEA model, Environment Agency 2009
Inhalation			
Dust or vapour		Vapour	
If vapour:			
Diffusivity in air	cm ² .s ⁻¹	0.0877	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Air water partition coefficient at ambient temperature	-	1.16E-01	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Organic carbon partition coefficient	cm ³ .g ⁻¹	1.83	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007

Output		Child	Adult
AGAC			
Oral	mg/kg	47	4113
Dermal	mg/kg	14200000	78888889
Inhalation	mg/kg	119	238

Substance Name **Benzene**
CAS Number **71-43-2**

Toxicological summary

	Value	Reference	Rationale
Acute oral reference dose	mg/kg bw/day 0.0235	USEPA Health advisory for 10 day drinking water standard	USEPA Health advisory for 10 day drinking water standard - Based on hematological impairment (including severe leukopenia) in rats. Sprague-Dawley rats inhaled benzene for five per day 4 days per week with review after second week. Uncertainty factor of 10 for inter species variation and 10 for intraspecies variation with route to route extrapolation
Acute dermal reference dose	mg/cm ² 7.1	New York State Brownfield Cleanup Program Development of Soil Cleanup Objectives. Technical Support Document Appendix C.1. Method for Deriving Soil Cleanup Objectives (SCOs) for Soil Contaminants Based on Toxicity Data for Irritant Contact Dermatitis (Non-Allergic Skin Irritation).	Based on Mouse Ear Swelling test in mice from Gad SC, Dunn BJ, Dobbs DW, et al. 1996. Development and validation of an alternative dermal sensitization test: the mouse ear swelling test (MEST). Toxicol Appl Pharmacol. 84:93-114.
Acute inhalation reference concentration - child	mg/m ³ 9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8hr WEL as guide to STEL. This is lower than the AEG1-1 (30 mins) of 237 mg/m3
Acute inhalation reference concentration - adult	mg/m ³ 9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)	Based on three times 8 hr WEL as guide to STEL

Human Health Hazard Profile - References

Organisation	Route of exposure	Health criteria type	Value	Units	Point of departure	Value	Units	Uncertainty factor	UF description	Species	Description	Target organ/Critical Effect	Reference	Web Link	Date Web Checked
Environment Agency	Inhalation	Not applicable	-	-	NOAEL	9600	mg/m ³	-	-	Human	Short-term exposures to 9,600 mg/m3 can be tolerated for 30 minutes.	No adverse effects	EC, 2003. In 'Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans Benzene, Science report: SC050021 March 2009'	http://web.archive.org/web/20140328044227/http://uk.environmental-agency.gov.uk/Archives/03050021.pdf	Dec-16
	Inhalation	Not applicable	-	-	NOAEL	80	mg/m ³	-	-	Human	No clinical signs of toxicity were recorded in workers exposed at an average of 80 mg/m3 for six hours	No adverse effects	EC, 2003. In 'Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans Benzene, Science report: SC050021 March 2009'		
	Inhalation	Not applicable	-	-	LOAEL	800-1600	mg/m ³	-	-	Human	Inhalation of 800-1,600 mg/m3 results in vertigo, drowsiness, headache and nausea	Vertigo, drowsiness, headache, nausea	Clayton and Clayton, 1994. In 'Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans Benzene, Science report: SC050021 March 2009'		
	Inhalation	Not applicable	-	-	LOAEL	4800	mg/m ³	-	-	Human	Higher concentrations (4,800 mg/m3) cause euphoria followed by giddiness, headache, nausea, staggered gait and, with continued exposure, unconsciousness	Giddiness, headache, nausea, staggered gait, unconsciousness	Clayton and Clayton, 1994. In 'Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans Benzene, Science report: SC050021 March 2009'		
WHO IPCS Environmental Health Criteria	Inhalation	Not applicable	-	-	LOAEL	4800	mg/m ³	-	-	Human	It has been estimated that exposure to benzene concentrations of 4800 mg/m3 (1500 ppm) for 60 min causes serious symptoms.	Serious symptoms of illness (Symptoms not reported)	Gerarde HW (1960). Toxicology and biochemistry of aromatic hydrocarbons. In 'WHO IPCS Environmental Health Criteria 150, 1963.'	http://www.inchem.org/documents/ehc/ehc/ehc150.htm	Dec-16
	Inhalation	Not applicable	-	-	LOAEL	1600	mg/m ³	-	-	Human	It has been estimated that exposure to benzene concentrations of 1600 mg/m3 (500 ppm) for 60 min leads to symptoms of illness.	Symptoms of illness			
	Inhalation	Not applicable	-	-	LOAEL	160-480	mg/m ³	-	-	Human	It has been estimated that exposure to benzene concentrations of 160-480 mg/m3 (50-150 ppm) for 5 h causes headache, lassitude, and weakness.	Headache, lassitude, and weakness			
	Inhalation	Not applicable	-	-	NOAEL	80	mg/m ³	-	-	Human	It has been estimated that exposure to benzene concentrations of 80 mg/m3 (25 ppm) for 8 h is without clinical effect.	No adverse effects			
US ATSDR	Oral	MRL (acute)	-	-	-	-	-	-	-	-	No acute-duration oral MRL was derived due to a lack of appropriate data on the effects of acute oral exposure to benzene.	-	ATSDR, 2007. Benzene toxicological profile https://www.atsdr.cdc.gov/toxdocs/t2d0723.pdf	Oct-18	
	Oral	MRL (chronic)	0.0005	mg/kg/day	BMDD.25d	0.014	mg/kg bw/day	30	10 for human variability 3 for uncertainty in route-to-route extrapolation	Mice and rats	Based on statistically significantly decreased counts of B-lymphocytes in workers of shoe manufacturing industries in Tianjin, China using benchmark dose analysis. The BMDD.25d of 0.1 ppm was adjusted from the 8 hour TWAs to a continuous exposure concentration of 0.03 ppm using the default occupational minute volume. Toxicokinetic studies indicate that absorption of benzene at relatively low levels of exposure is approximately 50% of an inhaled dose and essentially 100% of an oral dose. Based on these assumptions, inhalation data can be used to estimate equivalent oral doses that would be expected to similarly affect the critical targets of benzene toxicity. Therefore, the point of departure for the chronic-duration inhalation was used as a basis for the oral MRL.	Hematotoxicity and immunotoxicity			
	Inhalation	MRL (acute)	0.029 (0.009)	mg/m3 (ppm)	LOAEL	10.2 (2.55 HEC)	ppm	300	10 for use of a LOAEL 3 for extrapolation from animals to humans using dosimetric conversion 10 for human variability	Mice	The acute-duration inhalation MRL of 0.009 ppm (0.029 mg/m3) was derived from a lowest-observed-adverse-effect level (LOAEL) value of 10.2 ppm for reduced lymphocyte proliferation following mitogen stimulation in mice. The concentration was adjusted for intermittent exposure by multiplying the LOAEL (10.2 ppm) by 1/24 to correct for less than a full day of exposure. The resulting adjusted LOAEL, 2.55 ppm, was then converted to a human equivalent concentration (HEC) LOAELHEC = LOAELADU = 2.55 ppm as the animal blood/gas partition coefficient is greater than the human blood/gas partition coefficient (therefore a default value of 1 was used)	Immunotoxicity			

	Inhalation	MRL (chronic)	0.00975 (0.003)	mg/m3 (ppm)	BMDD.25d	0.03	ppm	10	10 for human variability	Human	Based on statistically significantly decreased counts of B-lymphocytes in workers of shoe manufacturing industries in Tianjin, China using benchmark dose analysis. The BMDD.25d of 0.1 ppm was adjusted from the 8-hour TWA to a continuous exposure concentration of 0.03 ppm using the default occupational minute volume.	Immunotoxicity			
US EPA	Oral	10 day HA	0.0235	mg/kg		0.0235	mg/kg/day	100	10 for inter species variation and 10 for intraspecies variation	Inhalation study on rats	Sprague-Dawley rats inhaled benzene for 6hrs per day 4 days per week with review after second week.	Hematological impairment (including severe leukopenia)	Benzene Health Advisory, Office Of Drinking Water, US EPA	https://www.epa.gov/rodent/1981-1982-rodent-toxicology-studies-benzene	Oct-18
	Oral	RFD	0.004	mg/kg/day	BMDL	1.2	mg/kg/day	300	3 for effect level extrapolation 10 for intraspecies differences (human variability) 3 for subchronic-to-chronic extrapolation 3 for database deficiencies	Human	Decreased lymphocyte count in humans following occupational exposure. The BMDL was derived by route-to-route extrapolation with the assumptions that inhalation absorption was 50% and oral absorption was 100% in the dose range near the BMDL. BMDLAD ₀₁ = 8.2 mg/m ³ × 20 m ³ /day × 0.5 × 70 kg = 1.2 mg/kg/day.	Immunotoxicity	IRIS, 2000. Benzene.	https://cfpub.epa.gov/ncea/iris/docs/documents/document/subst/0171_summary.pdf	Dec-16
	Inhalation	RFI	0.02	mg/m ³	BMCL	8.2	mg/m ³	300	3 for effect level extrapolation 10 for intraspecies differences (human variability) 3 for subchronic-to-chronic extrapolation 3 for database deficiencies	Human	Decreased lymphocyte count in humans following occupational exposure	Immunotoxicity	IRIS, 2000. Benzene.	https://cfpub.epa.gov/ncea/iris/docs/documents/document/subst/0171_summary.pdf	Dec-16
	Inhalation	AEGL1 (10 min)	423	mg/m ³	-	-	-	-	-	-	"the airborne concentration... of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain nonsensory effects"	Notable discomfort, irritation, or certain nonsensory effects	Benzene. Interim acute exposure guideline levels for NAS/CDT subcommittee for AEGLs. (2009)	http://www.epa.gov/ogg/ogg/so/compiled_aeagl_update_Distc12014.pdf	Dec-16
	Inhalation	AEGL1 (30 min)	237	mg/m ³	-	-	-	-	-	-	"the airborne concentration... of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain nonsensory effects"	Notable discomfort, irritation, or certain nonsensory effects	Benzene. Interim acute exposure guideline levels for NAS/CDT subcommittee for AEGLs. (2009)	http://www.epa.gov/ogg/ogg/so/compiled_aeagl_update_Distc12014.pdf	Dec-16
New York State Department of Environmental Conservation and New York State Department of Health	Dermal	Not applicable	7.1	mg/cm2/day	NOEL	7.1	mg/cm2/day	-	-	Mouse	"Challenge" concentration from mouse ear swelling test (Gad et al, 1986) equivalent to 7.1mg/cm2/day. Challenge concentrations are generally assumed to be maximum non-irritating concentrations.	Skin irritation	New York State Brownfield Cleanup Program, Development of Soil Cleanup Objectives, Technical Support Document Prepared By: New York State Department of Environmental Conservation and New York State Department of Health September 2006. Appendix C.1. Method for Deriving Soil Cleanup Objectives (SCOs) for Soil Contaminants Based on Toxicity Data for Irritant Contact Dermatitis (Nonallergic Skin Irritation).	https://www.dec.ny.gov/chemical/194293.html	Dec-16
The National Institute for Occupational Safety and Health (NIOSH)	Inhalation	IDHL	1625 (500)	mg/m3 (ppm)	-	-	-	-	UF not described.	Human	The revised IDLH (immediately dangerous to life or health) for benzene is 500 ppm (1625 mg/m3) based on acute inhalation toxicity data in humans	-	NIOSH IDLH values May 1994	https://www.cdc.gov/niosh/idlh/71432.html	Dec-16
	Inhalation	PEL (8hr TWA)	3.25 (1)	mg/m3 (ppm)	-	-	-	-	-	-	Permissible exposure limit (PEL) - 8hr time weighted average (TWA) = 1ppm (3.25 mg/m3)	-	US Department of Health and Human Services (1988). Occupational Safety and Health Guideline for Benzene - Potential Human Carcinogen	https://www.cdc.gov/niosh/docs/91-123/pdfs/D049.pdf	
	Inhalation	PEL (15min STEL)	16.3 (5)	mg/m3 (ppm)	-	-	-	-	-	-	Permissible exposure limit (PEL) - 15min short term exposure limit (STEL)	-	NIOSH IDLH values May 1994	https://www.cdc.gov/niosh/idlh/71432.html	
	Inhalation	EEGL (1 hr)	163 (50)	mg/m3 (ppm)	-	-	-	-	-	-	Emergency Exposure Guideline Level (EEGL) - 1 hr	-			
	Inhalation	EEGL (4 hr)	6.5 (2)	mg/m3 (ppm)	-	-	-	-	-	-	Emergency Exposure Guideline Level (EEGL) - 4 hr	-			
Health and Safety Executive	Inhalation	8hr TWA WEL	3.25	mg/m3	-	-	-	-	-	-	8hr time weighted average (TWA) workplace exposure limit (WEL)	-	EH40/2005 Workplace exposure limits (Third edition, published 2018)	http://www.hse.gov.uk/pubns/fsn/fsn4404.pdf	Oct-18

Contaminant	Benzene
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$$AGAC_{oral} = \frac{BW \cdot ARfD_{oral}}{MS_{ing} \cdot RBA_{ing}} \times 1000 \text{ g.kg}^{-1}$$

AGAC_{oral} - child receptor				
Parameter	Symbol	Units	Value	Justification
Body weight	BW	kg	10	Based on CLEA body weight of 9.8 kg for 1-2 year old female children and common practice when assessing risks to children
Mass of soil ingested in acute event	MS _{ing}	g	5	Value acknowledged by EA as not unreasonable for assessing children with pica behaviour, and within range of measured short term soil ingestion rates for children. Value used by HPA and by RIVM
Relative bioavailability oral (soil:tox)	RBA _{oral}	-	1	Assumes that absolute bioavailability of contaminant in soil = absolute bioavailability of contaminant in form used to derive ARfD
Acute oral reference dose	ARfD _{oral}	mg.kg(bw) ⁻¹	0.0235	USEPA Health advisory for 10 day drinking water standard
Acute Generic Assessment Criteria - Child	AGAC _{oral} - child	mg.kg ⁻¹	4.70E+01	

AGAC_{oral} - adult receptor				
Parameter	Symbol	Units	Value	Justification
Body weight	BW	kg	70	Based on CLEA body weight for a female worker
Mass of soil ingested in acute event	MS _{ing}	g	0.4	Factor of two applied to value selected for non-contact intensive activities and of same order as values of 330 – 480 mg/d commonly used in risk assessments for construction workers
Relative bioavailability oral (soil:tox)	RBA _{oral}	-	1	Assumes that absolute bioavailability of contaminant in soil = absolute bioavailability of contaminant in form used to derive ARfD Default - assume 1
Acute oral reference dose	ARfD _{oral}	mg.kg(bw) ⁻¹	0.0235	USEPA Health advisory for 10 day drinking water standard
Acute Generic Assessment Criteria - Child	AGAC _{oral} - adult	mg.kg ⁻¹	4.11E+03	

Key
Non contaminant specific input
Contaminant Specific Input
Calculated AGAC

Contaminant	Benzene
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$$AGAC_{dermal} = \frac{ARfD_{dermal}}{ABS_d \times AF} \times 10^6 \text{ mg.kg}^{-1}$$

AGAC _{dermal} - child receptor				
Parameter	Symbol	Units	Value	Justification
Dermal adherence factor	AF	mg.cm ⁻²	5	Conservative estimate based on the 95th percentile mass of soil on skin for children playing in wet soil of 3.2mg/cm ² and 0.4 mg soil/cm ² for dry soil (USEPA 2004)
Dermal absorption fraction	ABS _d	-	0.1	CLEA default for VOCs- Updated technical background to the CLEA model,
Acute dermal reference dose	ARfD _{dermal}	mg.cm ⁻²	7.1	New York State Brownfield Cleanup Program Development of Soil Cleanup
Acute Generic Assessment Criteria - Child	AGAC _{dermal} - child	mg.kg ⁻¹	1.42E+07	

AGAC _{dermal} - adult receptor				
Parameter	Symbol	Units	Value	Justification
Dermal adherence factor	AF	mg.cm ⁻²	0.9	Based on 95th percentile soil adherence for Utility workers USEPA 2004
Dermal absorption fraction	ABS _d	-	0.1	CLEA default for VOCs- Updated technical background to the CLEA model,
Acute dermal reference dose	ARfD _{dermal}	mg.cm ⁻²	7.1	New York State Brownfield Cleanup Program Development of Soil Cleanup
Acute Generic Assessment Criteria - Adult	AGAC _{dermal} - adult	mg.kg ⁻¹	7.89E+07	

Key
Non contaminant specific input
Contaminant Specific Input
Calculated AGAC

Contaminant	Benzene
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$$C_{vap} = \frac{K_{aw}}{K_{sw}} \times C_{soil}$$

$$K_{eq} = \frac{K_{aw}}{K_{sw}} \times \frac{\theta_a}{\rho_{soil}}$$

$$D_{eff} = \frac{D_a \theta_a^{3.33}}{\theta_t^2}$$

$$K_{sw} = \frac{\theta_w + (K_{oc} \cdot f_{oc} \cdot \rho_{soil}) + (K_{aw} \cdot \theta_a)}{\rho_{soil}}$$

$$AGAC_{soil} = \frac{ARfC_{inh} \times W \times h \times u}{\left[\frac{K_{aw}}{K_{sw}} \times Q \times \theta_a \times ExC \times 10^3 \right] + \left(\frac{A \times 10 \times \rho_{soil}}{\frac{\theta_a}{K_{eq} \times k_g} + \sqrt{\frac{\pi \times t}{D_{eff} \times K_{eq}}}} \right)}$$

AGAC _{inhal} - child receptor - vapour				
Parameter	Symbol	Units	Value	Justification
Surface area of which emissions occur	A	m ²	3.98E+01	Comprised of total emitting area from trial pit and stockpile as set out below: Trial pit: Assumes excavation is 0.6m wide (2ft JCB bucket), 3.0m long (ground investigation trial pit) and 3.0m deep. Includes emissions from side walls of pit with contamination present from ground surface Stockpile: Assumes regular cone of 4.0m diameter and 1.7m height
Height of mixing zone box	h	m	1.00E+00	Assumed height of child
Wind speed through box	u	m.s ⁻¹	1.00E+00	Minimum value given in US EPA offices of Air and Radiation Research and Development (1997)
Width of box perpendicular to wind direction over which emissions occur	W	m	5.00E+00	Based on 4m wide diameter stockpile + 1m for trial pit
Excavation rate	Q	m ³ .s ⁻¹	3.00E-03	Assume that a 1.8m ² x 3m deep TP excavated in 30 mins
Air filled porosity of soil	θ _a	-	2.00E-01	CLEA default value for sandy loam soil
Water filled porosity	θ _w	-	3.30E-01	CLEA default value for sandy loam soil
Total porosity of soil	θ _T	-	5.30E-01	CLEA default value for sandy loam soil
Density of soil	ρ _{soil}	g.cm ⁻³	1.21E+00	CLEA default value for sandy loam soil
Fraction of organic carbon in soil	f _{oc}	-	5.30E-03	Assumption of 1% SOM
Soil gas to atmosphere exchange constant	ExC	-	3.30E-01	USEPA 1997 suggested value for dry sandy soil
Gas-phase mass transfer coefficient	k _g	cm.s ⁻¹	1.50E-01	USEPA 1997 suggested value
Time since start of excavation of soil of interest	t	s	6.00E+01	USEPA 1997 suggested value
Diffusivity in air	D _a	cm ² .s ⁻¹	8.77E-02	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Air water partition coefficient at ambient temperature	K _{aw}	-	1.16E-01	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Organic carbon partition coefficient	K _{oc}	cm ³ .g ⁻¹	1.83E+00	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007

Contaminant	Benzene			
Acute inhalation reference concentration	ARFC _{air}	mg.m ⁻³	9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)
Effective diffusivity	Deff	cm ² .s ⁻¹	1.47E-03	
Total soil-water partition coefficient	Ksw	cm ³ .g ⁻¹	3.02E-01	
Weight fraction of VOC in airspace	Keq	-	6.36E-02	
Volatilisation part of AGAC equation	-	g.s ⁻¹	7.62E-02	
Diffusion part of AGAC equation	-	g.s ⁻¹	3.34E-01	
Acute Generic Assessment Criteria - Child	AGAC _{inhal} - child	mg.kg ⁻¹	1.19E+02	

AGAC _{inhal} - adult receptor - vapour				
Parameter	Symbol	Units	Value	Justification
Surface area of which emissions occur	A	m ²	3.98E+01	Assuming the excavation is 600mm wide (2ft JCB bucket) and 3m long (GI trial pit)
Height of mixing zone box	h	m	2.00E+00	Assumed height of adult
Wind speed through box	u	m.s ⁻¹	1.00E+00	Minimum value given in US EPA offices of Air and Radiation Research and Development (1997)
Width of box perpendicular to wind direction over which emission occurs	W	m	5.00E+00	Based on 4m wide diameter stockpile + 1m for trial pit
Excavation rate	Q	m ³ .s ⁻¹	3.00E-03	Assume that a 1.8m ² x 3m deep TP excavated in 30 mins
Air filled porosity of soil	θa	-	2.00E-01	CLEA default value for sandy loam soil
Water filled porosity	θw	-	3.30E-01	CLEA default value for sandy loam soil
Total porosity of soil	θT	-	5.30E-01	CLEA default value for sandy loam soil
Density of soil	psoil	g.cm ⁻³	1.21E+00	CLEA default value for sandy loam soil
Fraction of organic carbon in soil	foc	-	5.30E-03	Assumption of 1% SOM
Soil gas to atmosphere exchange constant	ExC	-	3.30E-01	USEPA 1997 suggested value for dry sandy soil
Gas-phase mass transfer coefficient	kg	cm.s ⁻¹	1.50E-01	USEPA 1997 suggested value
Time since start of excavation of soil of interest	t	s	6.00E+01	USEPA 1997 suggested value
Diffusivity in air	Da	cm ² .s ⁻¹	8.77E-02	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Air water partition coefficient at ambient temperature	Kaw	-	1.16E-01	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Organic carbon partition coefficient	Koc	cm ³ .g ⁻¹	1.83E+00	Compilation of data for priority organic pollutants for derivation of soil guideline values: Environment Agency 2007
Acute inhalation reference concentration	ARFC _{air}	mg.m ⁻³	9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)
Effective diffusivity	Deff	cm ² .s ⁻¹	1.47E-03	
Total soil-water partition coefficient	Ksw	cm ³ .g ⁻¹	3.02E-01	
Weight fraction of VOC in airspace	Keq	-	6.36E-02	
Volatilisation part of AGAC equation	-	g.s ⁻¹	7.62E-02	
Diffusion part of AGAC equation	-	g.s ⁻¹	3.34E-01	

Calculation sheet for AGAC for inhalation exposure - vapour

Contaminant	Benzene			
Acute Generic Assessment Criteria - Adult	AGAC _{inhal} - adult	mg.kg ⁻¹	2.38E+02	

Key
Non contaminant specific input
Contaminant Specific Input
Calculated value
Calculated AGAC

Contaminant	Benzene
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$$AGAC_{inh_dust} = \frac{ARfC_{inh} \times W \times h \times u \times (M/2)^{1.4}}{k \times Q \times \rho_{soil} \times 0.0016 \times (u/2.2)^{1.3}}$$

AGAC _{inhal} - child receptor - dust				
Parameter	Symbol	Units	Value	Justification
Height of mixing zone box	h	m	1	Assumed height of child
Wind speed through box	u	m.s ⁻¹	1	Minimum value given in US EPA offices of Air and Radiation Research and Development (1997)
Width of mixing zone box perpendicular to wind	W	m	5	Based on 4m wide diameter stockpile + 1m for trial pit
Excavation rate	Q	m ³ .s ⁻¹	3.00E-03	Assumed that a 3m deep trial pit is excavated, with a surface area of 1.8m2 in 30min
Density of soil	ρ _{soil}	g.cm ⁻³	1.21	CLEA default value for sandy loam soil
Moisture content of soil	M	%	4.8	Highest moisture content in AP42 - Note equations are only up to 1 to 4.8% from AP42
Particle size multiplier	k	-	0.35	Value in AP42 for dust particles less than 10 μm in diameter
Acute inhalation reference concentration	ARfC _{air}	mg.m ⁻³	9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)
Acute Generic Assessment Criteria - Child	AGAC _{inhal} - child	mg.kg ⁻¹	2.28E+08	

AGAC _{inhal} - adult receptor - dust				
Parameter	Symbol	Units	Value	Justification
Height of mixing zone box	h	m	2	Assumed height of adult
Wind speed through box	u	m.s ⁻¹	1	Minimum value given in US EPA offices of Air and Radiation Research and Development (1997)
Width of mixing zone box perpendicular to wind	W	m	5	Based on 4m wide diameter stockpile + 1m for trial pit
Excavation rate	Q	m ³ .s ⁻¹	3.00E-03	Assumed that a 3m deep trial pit is excavated, with a surface area of 1.8m2 in 30min
Density of soil	ρ _{soil}	g.cm ⁻³	1.21	CLEA default value for sandy loam soil
Moisture content of soil	M	%	4.8	Highest moisture content in AP42 - Note equations are only up to 1 to 4.8% from AP42
Particle size multiplier	k	-	0.35	Value in AP42 for dust particles less than 10 μm in diameter
Acute inhalation reference concentration	ARfC _{air}	mg.m ⁻³	9.75	HSE, 2018. EH40/2005 Workplace Exposure Limits (third edition)
Acute Generic Assessment Criteria - Adult	AGAC _{inhal} - adult	mg.kg ⁻¹	4.55E+08	

Key
Non contaminant specific input
Contaminant Specific Input
Calculated value
Calculated AGAC

H200 Unstable explosive
H201 Explosive; mass explosion hazard
H202 Explosive; severe projection hazard
H203 Explosive; fire, blast or projection hazard
H204 Fire or projection hazard
H205 May mass explode in fire
H220 Extremely flammable gas
H221 Flammable gas
H222 Extremely flammable aerosol
H223 Flammable aerosol
H224 Extremely flammable liquid and vapour
H225 Highly flammable liquid and vapour
H226 Flammable liquid and vapour
H227 Combustible liquid
H228 Flammable solid
H229 Pressurized; may burst if heated
H230 May react explosively even in the absence of air
H231 May react explosively even in the absence of air at elevated pressure and/or temperature
H240 Heating may cause an explosion
H241 Heating may cause a fire or explosion
H242 Heating may cause a fire
H250 Catches fire spontaneously if exposed to air
H251 Self-heating; may catch fire
H252 Self-heating in large quantities; may catch fire
H260 In contact with water releases flammable gases which may ignite spontaneously
H261 In contact with water releases flammable gas
H270 May cause or intensify fire; oxidizer
H271 May cause fire or explosion; strong oxidizer
H272 May intensify fire; oxidizer
H280 Contains gas under pressure; may explode if heated
H281 Contains refrigerated gas; may cause cryogenic burns or injury
H290 May be corrosive to metals
H300 Fatal if swallowed
H301 Toxic if swallowed
H302 Harmful if swallowed
H303 May be harmful if swallowed
H304 May be fatal if swallowed and enters airways
H305 May be harmful if swallowed and enters airways
H310 Fatal in contact with skin
H311 Toxic in contact with skin
H312 Harmful in contact with skin
H313 May be harmful in contact with skin
H314 Causes severe skin burns and eye damage
H315 Causes skin irritation
H316 Causes mild skin irritation
H317 May cause an allergic skin reaction
H318 Causes serious eye damage
H319 Causes serious eye irritation
H320 Causes eye irritation
H330 Fatal if inhaled

- H331 Toxic if inhaled
- H332 Harmful if inhaled
- H333 May be harmful if inhaled
- H334 May cause allergy or asthma symptoms or breathing difficulties if inhaled
- H335 May cause respiratory irritation
- H336 May cause drowsiness or dizziness
- H340 May cause genetic defects
- H341 Suspected of causing genetic defects
- H350 May cause cancer
- H351 Suspected of causing cancer
- H360 May damage fertility or the unborn child
- H361 Suspected of damaging fertility or the unborn child
- H361d Suspected of damaging the unborn child
- H362 May cause harm to breast-fed children
- H370 Causes damage to organs
- H371 May cause damage to organs
- H372 Causes damage to organs through prolonged or repeated exposure
- H373 May cause damage to organs through prolonged or repeated exposure

Environmental hazards

- H400 Very toxic to aquatic life
- H401 Toxic to aquatic life
- H402 Harmful to aquatic life
- H410 Very toxic to aquatic life with long lasting effects
- H411 Toxic to aquatic life with long lasting effects
- H412 Harmful to aquatic life with long lasting effects
- H413 May cause long lasting harmful effects to aquatic life
- H420 Harms public health and the environment by destroying ozone in the upper atmosphere
- H420: Harms public health and the environment by destroying ozone in the upper atmosphere