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Grenfell Investigation into Potential Land Contamination Impacts

Technical Note 8: COPC Toxicity

Royal Borough of Kensington and Chelsea

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		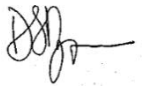	
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Table of Contents

Tables

1. Glossary of Key Terms

The following key acronyms and terms are used in this document:

Table TN08-01. Glossary of Key Terms

Term	Definition
HBGV	Health Based Guidance Value – used in this document to describe any value that represents an estimated dose in humans that is without appreciable risk over a lifetime. This may also be defined as being representative of minimal or tolerable risk. The term covers TDIs, RfDs, RfCs and MRLs.
SPOSH	Significant Possibility of Significant Harm: Under part 2A of the EPA 1990 land is determined as contaminated if it is deemed to be causing significant harm, or where there is a significant possibility of significant harm (SPOSH).
HCV	Health Criteria Value – This term was introduced by the EA SR2 guidance document where it was defined as *"A generic term used in this report to describe a benchmark level of exposure to a chemical derived from available toxicity data for the purposes of safeguarding human health (e.g. a tolerable daily intake)."* It is equivalent to an HBGV, but more specifically used within the context of the EA Contaminated Land Exposure Assessment (CLEA) methodology.
LLTC	Low Level of Toxicological Concern - A Low Level of Toxicological Concern represents an exposure equivalent to an intake of low concern but that definitely does not approach an intake level that could be defined as causing a SPOSH to human health. It is adopted in place of an HBGV/HCV in the derivation of C4SLs. The concept was introduced by Defra to assist specifically with assessment under Part 2A of the EPA.
TDI	Tolerable daily intake - defined as an estimate of the amount of a contaminant, expressed on a bodyweight basis [e.g. mg kg-1 bw day-1], that can be ingested daily over a lifetime without appreciable health risk.
MDI	Mean Daily Intake – the average "background intake" to which a population may be exposed. The MDI is given separately for oral and inhalation routes of exposure.
ID	Index Dose - describes an HCV, expressed as a daily dose, derived for a non-threshold carcinogen, which is expected to be associated with a minimal excess risk of cancer.
RfD	Reference Dose – Term used largely by the United States Environmental Protection Agency (USEPA) as an estimate of a daily **oral** exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime. May be used similarly to a TDI.
RfC	Reference Concentration - term also of USEPA origin, is equivalent to the RfD, but is based on **inhalation** and is defined as a concentration in air.
MRL	Minimal Risk Level – term used by the US Agency for Toxic Substances and Disease Registry (ATSDR). 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. It is equivalent to RfD (oral) and RfC (inhalation). MRL may be derived for acute, intermediate or chronic exposures should sufficient data be available.
NOAEL	No-observed adverse effect level - when assessing an endpoint displaying a threshold, this is the highest dose at which no adverse effects were seen in the toxicity study.
LOAEL	Lowest observed adverse effect level – the lowest dose at which adverse effects were seen.
ALARP	As Low As Reasonable Practicable – For non-threshold chemicals, the ALARP principal ensures that, irrespective of whether a HBGV is being breached or not, exposures must be kept as low as reasonably practicable.

2. Introduction

This technical note sets out AECOM's review of published, peer reviewed and authoritative evaluations of the toxicity of the fire effluents. The assessment is based on the final agreed specification for the Stage 1 assessment (AECOM Technical Note (TN) 1), and the scope of the evidence reviews as detailed in TN2.

The fire effluent COPC identified for this evidence review are taken from AECOM TN4, and are:

- Chlorinated and brominated dioxins and furans (PCDD/Fs and PBDD/Fs), and dioxin-like Polychlorinated Biphenyls (PCBs)
- Polycyclic aromatic hydrocarbons (PAHs)
- Benzene
- Non-dioxin-like PCBs
- Lead
- Cyanides
- Isocyanates
- Asbestos
- Synthetic Vitreous Fibres
- Organo-phosphorous flame-retardant compounds
- Brominated Flame Retardants

In carrying out this evidence review, preference has been given to a small number of authoritative bodies that publish toxicological evaluations of chemicals to streamline the review process. These are the principal organisations that publish toxicological evaluations either in the UK or globally:

- UK Department of Health Committees on Carcinogenicity (COC), Mutagenicity (COM), and Toxicity (COT) of chemicals in food, consumer products and the environment.
- The Food and Environment Research Agency (FERA)
- Chartered Institute of Environmental health (CIEH)
- Land Quality Management (LQM)[1]
- European Food Safety Authority (EFSA).
- Dutch National Institute for Public Health and the Environment (RIVM)
- US National Library of Medicine TOXNET hazardous substances data bank (HSDB)
- US Department of Health and Human Services Agency of Toxic Substances and Disease Registry (ATSDR).
- Department for Environment, Food and Rural Affairs (Defra)
- Environment Agency (EA).
- Public Health England (PHE).
- United Nations and World Health Organisation (WHO) International Programme on Chemical Safety (IPCS).
- World Health Organisation (WHO).
- United States Environmental Protection Agency (US EPA).

[1] Nathanail, C. P. et al., 2015. *The LQM/CIEH S4ULs for Human Health Risk Assessment (S4UL3092),* Nottingham: Land Quality Press. Copyright Land Quality Management Limited reproduced with permission; Publication Number S4UL3092. All rights reserved. For all future references to this document this copyright statement applies.

Preference has been given to the most recently published documents from any one or more of these organisations where relevant to a particular chemical or group of chemicals.

2.1 Objectives

The purpose of this Technical Note is to identify what toxicological information is available for each chemical, what health effects might arise from exposure to these chemicals, what levels of exposure are required before those health effects might occur (i.e. identification of health based guidance values (HBGVs), and whether any concentration guidelines or standards, generically referred to here as generic screening criteria (GSC), have been developed in relation to the protection of human health. It is not intended to provide detailed summaries for each chemical, or reproduce all relevant data contained within the reviewed literature – many of which are substantial documents running to many hundreds of pages.

2.2 Scope of Work

AECOM has completed an Evidence Review (ER) in accordance with the protocol described in 'Table TN02-04. COPC Toxicity' in TN2.

The key extracted information for each COPC is presented in Sections 2 to 13 of this TN.

The appendices to this report list the toxicological and information identified in the review:

- Appendix A - Web Search – details of the searches undertaken.
- Appendix B – Summary of Evidence Identified – high-level overview of information contained in the reference.
- Appendix C – Evidence Extraction – information summarised from each identified source.

2.3 Legislative context and guidance documentation

The context for this review is Part 2A of the Environmental Protection Act 1990 (Part 2A) (DEFRA, 2012); the statutory guidance for this regulatory regime defining "significant harm" to humans as:

- Death;
- Life-threatening diseases (e.g. cancers);
- Other diseases likely to have serious impacts on health;
- Serious injury;
- Birth defects; and
- Impairment of reproductive functions.

The statutory guidance goes on to state that Local Authorities may consider that other health effects constitute "significant harm" and gives examples of:

- physical injury;
- gastrointestinal disturbances;
- respiratory tract effects;
- cardio-vascular effects;
- central nervous system effects;
- skin ailments; and
- effects on organs such as the liver and kidneys.

In considering other health effects, the seriousness of the harm, including the impact on health and quality of life of any person suffering the harm, and the scale of the harm, should be considered in the context of the broad objectives of the Part 2A regime (DEFRA, 2012).

For land to be determined as Contaminated Land in accordance with Part 2A, it must be causing "significant harm" or there must be a significant possibility of significant harm (SPOSH) occurring.

Consideration should also be given to whether less significant harm might be a precursor of, or indicative of, or symptomatic of, a more serious form of harm, or that repeated episodes of minor harm might lead to more serious harm in the longer term (DEFRA, 2012).

2.4 Background to health based guidance values and generic screening criteria

2.4.1 Health Based Guidance Values and the Risk Assessment Process

Health based Guidance Values (HBGVs) are an important part of the risk assessment process for contaminated soils and are a critical component in the derivation of generic screening criteria (GSC) or site-specific assessment criteria.

The Defra C4SL report (DEFRA, 2014) defines an HBGV as *"the estimated dose in humans that is without appreciable risk over a lifetime"*. The Environment Agency adopted the term Health Criteria Value (HCV) to describe HBGVs within the context of the Contaminated Land Exposure Assessment (CLEA) guidance. The EA defined an HCV as "*A generic term used in this report to describe a benchmark level of exposure to a chemical derived from available toxicity data for the purposes of safeguarding human health (e.g. a tolerable daily intake)."* HCVs are established from a review of the evidence from occupational and environmental epidemiological studies, animal studies, and from scientific understanding of the mechanisms of absorption, transport, metabolism and toxicity of chemicals within the human body (EA, 2009b).

HCVs set levels of *minimal* or *tolerable* risk for long-term human exposure to chemicals in soil. They represent a baseline and health protective position to minimise risks of significant harm; they do not themselves necessarily represent thresholds above which an intake would be unacceptable, representing SPOSH in the context of Part 2A, but they can be a useful starting point for such an assessment (EA, 2009b). In the context of Part 2A, an assessor using HCVs can conclude that: human exposure below the HCV is unlikely to represent a SPOSH; human exposure above the HCV might represent SPOSH, with the significance linked to the margin of exceedance, the duration and frequency of exposure, and other factors that the enforcing authority may wish to take into account (EA, 2009b).

The methods used to derive HCVs are split into contaminants that display threshold toxicity and non-threshold carcinogenicity (EA 2009b).

For threshold toxicity, where there is some 'non-zero' amount of exposure (dose), prior to an adverse biological effect being produced, the HCV is derived by determining the critical 'no-observed adverse effect level' (NOAEL) or if this has not been identified the critical 'lowest-observed adverse effect level (LOAEL). In some cases it is possible to mathematically model the dose-response curve to estimate a benchmark dose level (BMDL) associated with a predetermined change in response rate e.g. 10% change in relative kidney weight. The BMDL is typically reported as the lower 95% confidence interval on dose giving a 10% (1 in 10) response ($BMDL_{10}$). NOAEL, LOAEL and BMDL are often termed the point of departure (POD). PODs are determined from pivotal toxicity studies (EA, 2009b).

When deriving HCVs for threshold critical toxicity, the most universally adopted chemical risk assessment programme is the tolerable daily intake (TDI). This is the amount of a contaminant expressed on a bodyweight basis that can be ingested daily over a lifetime without appreciable health risk (mg/kgBW/day) (EA, 2009b). To derive TDIs, uncertainty factors (UF) (i.e. differences between animal/human response or genetic profile, age, health status etc.) to a POD such as the NOAEL or LOAEL are also applied:

TDI = POD/UF

Where the difference in sensitivity of the test and target populations to a particular chemical is known and can be quantified or estimated a chemical-specific adjustment factor (CSAF) is applied (EA, 2009b).

The derivation of USEPA's reference dose (RfDs) and reference concentration (RfCs) and US ATSDR's Minimal risk levels (MRLs) largely follow the same principles as those used in the UK to derive threshold effect related TDIs (EA, 2009b). Differences in the choice of pivotal toxicology study and POD should be appreciated when comparing HCVs from different countries, as well as their conservatism, highlighted in their choice of uncertainty factors (DEFRA, 2014).

For non-threshold mechanism of toxicity there are two approaches to derive HCVs: Quantitative dose-response modelling and non-quantitative extrapolation (EA, 2009b).

Quantitative dose-response modelling also known as quantitative risk assessment (QRA) is used to derive an estimate of dose that corresponds to an excess lifetime cancer risk (ELCR). QRA is used by some public health organisations and non-UK regulatory bodies (e.g. the WHO drinking water guidelines working group and USEPA). However, in the UK, QRA has been used to derive HBGVs from human data, but not from animal study data. This is because the UK Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COC) does not recommend its use for routine risk assessment. COC considers that the models do not simulate the carcinogenic processes adequately and is critical of the precision of cancer risk erroneously implied (EA, 2009b).

Non-quantitative extrapolation approach involves assessment of all available carcinogenicity dose response data to identify an appropriate dose without discernible carcinogenic effect or the lowest dose tested if effects are apparent at all doses (EA, 2009b). HCVs derived using this approach have been called *'minimal risk levels'* by COC. COC define a 'minimal risk level' as "an estimate of daily human exposure to a chemical identified by expert judgement that is likely to be associated with a negligible risk of carcinogenic effect over a specified duration of exposure (usually a lifetime)". Minimal risk levels have been favoured over quantitative cancer risk estimates by COC, since they are considered to carry only a minimal cancer risk and thus fulfil their health protection goal without attempting to quantify the risk and imply a precision which may not be valid. In practice, the minimal risk level approach is similar to that for threshold chemicals, applying numerical (uncertainty) factors to a POD identified from the dose response data (EA, 2009b). Within the Category 4 Screening Level (C4SL) research (refer to Section 1.4.2 below), the uncertainty factors are termed 'suitable margins'.

Background exposure is also accounted for when deriving HCVs for substances with threshold toxicity. It is not taken into account for substances with non-threshold toxicity. Those exposed to chemical contaminants in soil may be subject to exposure from ambient levels of contaminants in food, drinking water and air. Therefore, the risk assessment needs to make allowance for these other exposures. A balanced consideration of background exposure should be achieved by estimating the mean daily intake (MDI) for the UK population of oral exposure (MDIoral) and inhalation exposure (MDIinh), reported in mass per day (e.g. µg/day) (EA, 2009b). To calculate human intakes for contaminant concentration data for water and air, default values for various physiological parameters (bodyweight, inhalation rate and drinking water consumption) must be used. The EA provide default values for an average UK adult. For children, correction factors that account for differences in dietary intakes and respiration rates are made (EA, 2009b).

2.4.2 Generic Screening Criteria (GSC)

GSC are concentrations of a chemical in soil (or other environmental media) which might be expected to result in long-term human exposure equal to the selected HBGV, based on various exposure assumptions and consideration of other background exposure routes (e.g. dietary intake). In a small number of cases, GSC may be based on HBGV/HCV that do not represent minimal risk, due to policy decision to avoid disproportionately enforcing stricter limits on contaminated land where lesser protection is afforded in other UK regulatory jurisdictions. This occurs for the inhalation HCV for benzo(a)pyrene, which has been set based on the UK Air Quality Guideline.

GSC are published by a variety of authoritative organisations, with the UK Environment Agency publishing Soil Guideline Values (SGVs) based on the CLEA methodology. In the UK, more recently published GSC derived in accordance with the principles of the EA CLEA methodology but also taking into account the Defra C4SL research publication) include the S4ULs, described in further detail below.

Soil Guideline Values

The EA SGVs provide guidelines on the level of long-term human exposure to individual chemicals in soil that, unless stated otherwise, are *tolerable* (where the HCV is a TDI) or pose a *minimal risk* (where the HCV is an index dose (ID)) to human health (EA, 2009c).

SGVs are generic screening values designed to be used for generic quantitative risk assessments (GQRA). They represent "trigger values" – indicators to a risk assessor that soil concentrations above this level may pose a SPOSH. SGVs do not of themselves represent the threshold at which there is a SPOSH. Nor do they automatically represent an unacceptable intake in the context of Part 2A (EA, 2009c).

SGVs are generically calculated based on a sandy loam soil with 6% soil organic matter and there are different SGVs according to land use (residential, allotments, commercial) because people use land differently as that impacts who and how people may be exposed to soil contamination (EA, 2009c). SGVs are available for the following chemicals: arsenic, selenium, cadmium, benzene, toluene, ethylbenzene and xylene, dioxins, furans and dioxin-like PCBs and phenol in soil (CL:AIRE, 2019).

For soil contaminants that display threshold toxicity the basic starting principle for establishing SGVs is to utilise the Average Daily Exposure (ADE). ADE to a contaminant in soil via ingestion, inhalation and absorption is estimated using the Contaminated Land Exposure Assessment (CLEA) model (EA, 2009c). The CLEA model provides generic estimates of adult and child exposures to soil contaminants for those potentially living, working and/or playing on contaminated sites over long time periods (EA, 2009c). SGVs are set such that the estimated Average Daily Exposure (ADE) to a chemical arising from its presence in soil at its SGV, when added to its background exposure (MDI), equals its TDI (i.e. ADE + MDI = TDI). For some contaminants the MDI may already occupy a high proportion of the TDI and so it would be impractical to propose SGVs on this basis. Therefore, Defra propose that the ADE should represent at least half of the TDI. The TDI that remains once MDI (background exposure) has been accounted for is termed the tolerable daily soil intake (TDSI). Thus ADE = TDSI at the SGV (EA, 2009c).

For non-threshold carcinogenic soil contaminants at low level of cancer risk the SGV is based on the HCV known as the index dose (ID). When sufficient human data are available for quantitative dose response modelling Defra recommend that the ID should be based on estimates of the dose corresponding to an excess lifetime cancer risk (ELCR) of 1 in 100,000 (as appropriate to represent minimal risk) (EA, 2009b). Where human data are available but are not suitable for quantitative modelling, it may be possible to propose an ID based on evaluation of the available data and identification of the dose associated with no discernible increase in cancer and the use of expert judgement to extrapolate this to the wider population (EA, 2009b).

Suitable for Use Levels (S4ULs)

In 2009, Land Quality Management (LQM) and Chartered Institute of Environmental Health (CIEH) published a set of over eighty generic assessment criteria (GAC) for common contaminants to inform site specific GQRAs. They were designed to provide GAC for the contaminants that did not have SGVs (Nathanail, et al., 2015). Similar to SGVs, they were intended to mark the concentration of a substance in soils at or below which human exposure can be considered to represent a 'tolerable' or 'minimal' level of risk.

In 2015, the 2009 LQM GAC were updated as S4ULs in line with the additional land uses and exposure assumptions presented in Defra's C4SL guidance, and for an extended range of 89 substances. As such, S4ULs have been derived for six generic land uses (including the two Public Open Space land uses defined in C4SL guidance). However, they are still based on HCVs that represent *minimal* or *tolerable* levels of risk to health as described in SR2 (EA, 2009b). Exceedance of an S4UL does not constitute evidence of a SPOSH, but should usually trigger further environmental risk assessment (Nathanail, et al., 2015).

Category 4 Screening Levels (C4SL)

Category 4 Screening Levels (C4SLs), were proposed by DEFRA as generic screening values, to be used in a similar manner to SGVs for generic quantitative risk assessments. They are intended as a "relevant technical tool" to help local authorities and others decide whether to stop further assessment of a site, on the grounds that the land falls within Category 4 (Human Health), as defined in Part 2A, where there is no risk that land poses a SPOSH (DEFRA, 2014).

Six substances (arsenic, benzene, benzo(a)pyrene, cadmium, chromium VI and lead) have been chosen for C4SLs because of their ubiquity in contaminated land risk assessment and because they cover a range of exposure pathways and toxicological effects (DEFRA, 2014b). C4SLs were derived for six different land uses. Three land uses similar to those used for SGVs: Residential (with and without home-grown produce), allotments and commercial, plus, additionally, two alternative types of Public Open Space (DEFRA, 2014b).

A key distinction between SGVs/S4ULs and C4SLs is the level of risk that they prescribe. Low level of toxicological concern (LLTC) is used in replacement of HCVs (used for SGVs and S4ULs). The purpose of a LLTC is to represent a *'low level of risk'* (that is more pragmatic but still a strongly precautionary level) that is above '*minimal risk'* described for SGVs. (DEFRA, 2014). The assessment is less conservative yet still protective of human health within the context of Part 2A.

Health based guidance values (HBGVs) are used for the derivation of LLTCs. This is because Defra recommend that a *minimal risk* HBGV for a chemical is understood before attempting to derive *a 'low level'* LLTC. HBGVs are defined as the estimated dose in humans that is without appreciable risk over a lifetime. Similar to HCVs, HBGVs are an umbrella term that encompasses TDI for threshold compounds and ID for non-threshold chemicals (DEFRA, 2014).

Defra (DEFRA, 2014) provide a flowchart that details how LLTCs should be derived. In summary, the first step is to choose a pivotal HBGV (similar to choosing a pivotal study for HCVs). Following that if there is adequate data from the chosen pivotal study, BMD modelling is carried out. BMD modelling is the preferred method of determining the POD for threshold chemicals, this is contrary to HCVs where NOAEL or LOAEL are generally applied. If robust data are available, a chemical specific adjustment factor (CSAF) is applied (in a similar way that CSAF is applied for the derivation of HCVs). If the available data are not considered to be robust then a default UF (usually 100) can be applied (DEFRA, 2014).

For non-threshold contaminants a chemical specific margin (CSM) should be defined based on a scientifically defensible rationale around the uncertainties in the toxicological data and with use of expert judgement. If data are not robust, a generic margin may be set. For the purpose of deriving LLTCs, a generic margin, that leads to a notional risk of 1 in 50,000, to represent a *low level of risk* is proposed (DEFRA, 2014). For human data, where data are not adequate for BMD modelling, an excess lifetime cancer risk (ELCR) can be defined. This is similar to SGVs ELCR. However, rather than 1 in 100,000 to represent '*minimal risk'* for SGVs, a risk estimate of 1 in 50,000 is proposed to represent *'low risk* for C4SLs (DEFRA, 2014).

Other GSC

GSC for soil published by other authoritative national and international organisations include GAC published in the UK by CL:AIRE/EIC/AGS, the USEPA Regional Screening Levels (RSLs), and the Dutch Intervention Values (DIV) published by RIVM.

These criteria are typically calculated using HBGVs (i.e. minimal or no appreciable risk) although the Dutch and US criteria make different assumptions for the relevant exposure pathways and parameterisation of those pathways.

3. Chlorinated Dioxins, Furans and Dioxin-like PCBs

3.1 Health Effects and Health Based Guidance Values

The most recent[2] and relevant authoritative reviews of this group of chemicals identified by the literature search have been published by the following:

- Risk for animal and human health related to the presence of dioxins and dioxin-like PCBs in feed and food. European Food Safety Authority (EFSA), June 2018 (as amended February 2019)
- Toxicological profile for Chlorinated Dibenzo-p-dioxins (CDDs). Agency of Toxic Substances and Disease Registry (ATSDR), 1998 (addendum November 2012)
- Dioxins: Toxicological Overview. Public Health England (PHE), 2008
- Supplementary information for the derivation of SGVs for dioxins, furans and dioxin-like PCBs. Environment Agency (EA), September 2009
- UK dietary exposure to PCDD/Fs, PCBs, PBDD/Fs, PBBs and PBDEs: comparison of results from 24-h duplicate diets and total diet studies. Food Addit Contam Part A. Chem Anal Control Expo Risk Assess. 34(1), pp. 65-77. Bramwell et al. 2017
- Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)

The primary source material used in this summary review is published by EFSA (EFSA, 2019), changes in conclusions compared to earlier studies including those of PHE and EA are referenced.

EFSA reviews the toxicity of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/F) and dioxin-like polychlorinated biphenyls (DL-PCBs). The PCDD/Fs are two groups of tricyclic planar compounds comprising 75 PCDDs and 135 PCDFs, of which only 17 of these congeners are considered relevant due to their relative persistence in humans and animals. The DL-PCBs are a subgroup of 12 PCBs that exhibit similar toxicological properties to tetrachlorodibenzo-p-dioxin (TCDD) and other PCDD/Fs. EFSA reviewed epidemiological evidence (evidence from human exposures) and evidence from animals (toxicological studies) and concluded that the most unequivocal toxicity outcome from human exposure studies was the adverse dermal health effect of chloracne. Children are reported to be particularly sensitive to this effect, however it is only associated with high exposures (defined by EFSA as resulting serum levels >20,000pg/g fat). EFSA concluded that this effect is not relevant to the derivation of a health-based guidance value for the general population. A health-based guidance value (HBGV) was determined based on adverse reproductive effects in male children (this being identified as the critical adverse health effect of relevance for humans). A no observed adverse effect level (NOAEL) in serum of 7pg/g fat was equated to a daily food intake of 0.5pg per kg bodyweight and a daily dietary intake of 0.25pg/kg bodyweight for breastfeeding mothers. Adjusting for uncertainties in the data, EFSA proposed a tolerable weekly intake (TWI) of 2pg per kg bodyweight, expressed as a WHO_{2005} TEQ[3] for the sum of all 17 PCDD/F congeners and 12 DL-PCBs. In evaluating current European-wide dietary exposure, EFSA concluded that typical dietary exposure exceeded the new TWI by factors of up to 5, and above average dietary intakes exceeded by factors of 3-15. EFSA identified 15 recommendations to improve the risk assessment and reduce the current uncertainties in the data, including a re-evaluation of the WHO_{2005} toxicity equivalence factors (TEFs) used to sum the toxicological risk from the 17 PCDD/F congeners and 12 DL-PCBs.

By comparison, the Environment Agency (EA, 2009) health criteria value (HCV) is a tolerable daily intake (TDI) of 2 pg per kilogram bodyweight per day for humans. This choice was based on the current advice of the UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT), and the WHO_{2005} TEFs were adopted for the summation of the 17 PCDD/F congeners and 12 DL-PCBs. The HCV was chosen on the basis of animal experiment data (rather than the human data used by EFSA) and was consistent with the views of EFSA and WHO at the time. The critical endpoint was essentially the same – adverse reproductive effects observed in male rats. The Environment Agency includes reference to the carcinogenicity of PCDD/Fs, noting that IARC and the UK Committee on Mutagenicity concluded that PCDD/Fs are not genotoxic

[2] Studies published after (and therefore not referenced in) the Environment Agency's Soil Guideline Value report, September 2009
[3] Toxicity equivalence quotient calculated in accordance with WHO 2005 methodology

carcinogens, however, IARC has classified 2,3,7,8-TCDD as a Group 1 carcinogen based on sufficient evidence in humans and experimental animals. The lack of data on all other PCDD/Fs results in them being assigned a Group 3 classification (not classifiable). Carcinogenicity is not however the critical adverse health effect according to the authors (i.e. carcinogenic risk at the exposure levels associated with adverse reproductive effects is not the driving factor). The US EPA published an upper bound lifetime cancer risk of 1 in 1000 per pg TCDD per kg bodyweight per day (USIM:CIDFS, 2003).

The more recent EFSA TWI is therefore 7 times lower than the HCV advocated by the Environment Agency in their review of authoritative evaluations and approximately 2860 times lower than exposures associated with acute health effects recorded in humans.

The toxicological overview by Public Health England is dated 2008 and is not referenced in the EA 2009 reports. The key references for the PHE review are IPCS EHC 88 (IPCS, 1989), (WHO, 1989) (ATSDR, 1998), (EA, 2009), (IARC, 1997), COC (1999 and 2001), and (WHO, 2002), which all pre-date the most recent EA and EFSA reviews.

The ATSDR supplement (ATSDR, 2012) is a non-peer reviewed supplement of the scientific data published in open peer reviewed literature since the publication of the toxicological profile for dioxins in 1998 by the IPCS (IPCS, 1989). The minimal risk level set in by IPCS in 1998 of 1pg/kgBW/day was based on neurobehavioural development changes in monkeys, and this is a different critical end-point to that chosen in more recent evaluations. ATSDR summarises the risk assessments conducted globally since 1998, including those from the UK, Japan, France, Germany, Netherlands, New Zealand, Australia and Canada – the majority choosing the TDIs recommended by WHO that vary from 1-4pg/kg BW/day. The US EPA evaluation dated February 2012 is also referenced. This evaluation adopts the human evidence chosen by EFSA in 2018 rather than the animal data adopted by earlier evaluations by ATSDR and WHO etc. The US EPA derived a non-cancer reference dose (RfD) of 0.7pg/kgBW/day for chronic oral exposure – lower that the earlier ATSDR and WHO evaluations, but higher than the later EFSA evaluation.

Table TN08-02. Published Health-based Guideline Values

Authoritative Body	Health-based Guideline Value
EFSA (2018)	Tolerable Weekly Intake (TWI) 2pg/kgBW/week (equivalent to 0.3pg/kgBW/day)
EA (2009)	Tolerable Daily Intake (TDI) 2pg/kgBW/day
ATSDR (1998)	Minimal risk level (MRL) 1pg/kgBW/day
JECFA (2001)	Provisional tolerable monthly intake (PTMI) 40-100pg/kgBW/month (equivalent to 1-3pg/kgBW/day)
WHO (1998)	Tolerable daily intake (TDI) 1-4pg/kgBW/day
US EPA (2012)	Reference dose (RfD) 0.7pg/kgBW/day

Source: (EFSA, 2019) (EA, 2009) (ATSDR, 2012)

The potency of the toxicological effects of individual dioxins, furans and dioxin-like PCBs varies over several orders of magnitude, and hence Toxic Equivalency Factors (TEFs) were proposed by the WHO *(Canady, et al., 2002)*. Use of the TEF concept rests on the assumption that chlorinated dioxins (PCDDs), furans (PCDFs) and coplanar PCBs have a common mechanism of action. The 29 dioxins, furans and dioxin-like PCBs listed in the table below have similar resistance to environmental and metabolic degradation and solubility in body fat, and they share a unique spectrum of toxic responses initiated by interaction with the aryl hydrocarbon receptor (AhR) receptor found in many tissues in the body. Many uncertainties exist in use of the TEF approach for human risk assessment, but pragmatically it is the most feasible approach available *(Canady, et al., 2002)*.

The TEF approach works by assigning a scaling factor for each dioxin like compound based on a comparison of its toxic potency to that of a designated index chemical. The index chemical is typically specified as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and is assigned a TEF of 1. 1,2,3,7,8-Pentachlorodibenzodioxin is of a similar potency. The other members of the subset are 10–10,000 times less toxic and are assigned TEFs accordingly.

PCBs exhibit different toxicological effects depending on the site of chlorine substitution on the phenyl rings. Congeners with no chlorine substitution in the ortho position (non-ortho) or those congeners having only ortho substitution in one position (mono-ortho) have toxicological activity similar to that of the PCDDs and PCDFs owing to their ability to adopt a similar planar structure and to bind strongly to the AhR. There are 209 PCB congeners in total and the non-ortho- and mono-ortho substituted PCBs in the environment and in foods generally comprise a small percentage of the total PCB contamination. The dioxin-like toxicity of the 12 dioxin-like PCBs is 10–100,000 times less than that of TCDD *(Canady, et al., 2002)*.

Concentration addition is used to estimate the overall toxicity of the dioxin and dioxin-like compounds. This is expressed as a toxic equivalent (TEQ) which is the sum of the product of TEFS and concentrations *(RIVM, 2014)*.

The recommended TEFs for dioxins, furans and dioxin-like compounds are listed in the EA SGV report *(EA, 2009)*, which refers to Van den Berg et al. (2006) as the source of the derived TEFs.

Table TN08-03. Recommended WHO_{2005} TEF values

Compound	WHO_{2005} TEF	Compound	WHO_{2005} TEF	Compound	WHO_{2005} TEF	Compound	WHO_{2005} TEF
PCDDs		**PCDFs**		**Dioxin-like PCBs (non-ortho)**		**Dioxin-like PCBs (mono-ortho)**	
2,3,7,8-TCDD	1	2,3,7,8-TCDF	0.1	3,3',4,4'-TCB (PCB-77)	0.0001	2,3,3'4,4'-PeCB (PCB-105)	0.00003
1,2,3,7,8-PeCDD	1	1,2,3,7,8-PeCDF	0.03	3,4,4',5-TCB (PCB-81)	0.0003	2,3,4,4',5-PeCB (PCB-114)	0.00003
1,2,3,4,7,8-HxCDD	0.1	2,3,4,7,8-PeCDF	0.3	3,3',4,4',5-PeCB (PCB-126)	0.1	2,3',4,4',5-PeCB (PCB-118)	0.00003
1,2,3,6,7,8-HxCDD	0.1	1,2,3,4,7,8-HxCDF	0.1	3,3'4,4'5,5'-HxCB (PCB-169)	0.03	2,3,4,4',5-PeCB (PCB-123)	0.00003
1,2,3,7,8,9-HxCDD	0.1	1,2,3,7,8,9-HxCDF	0.1			2,3,3',4,4',5-HxCB (PCB-156)	0.00003
1,2,3,4,6,7,8-HpCDD	0.01	1,2,3,6,7,8-HxCDF	0.1			2,3,3',4,4',5'-HxCB (PCB-157)	0.00003
OCDD	0.0003	2,3,4,6,7,8-HxCDF	0.1			2,3',4,4',5,5'-HxCB (PCB-167)	0.00003
		1,2,3,4,6,7,8-HpCDF	0.01			2,3,3',4,4',5,5'-HpCB (PCB-189)	0.00003
		1,2,3,4,7,8,9-HpCDF	0.01				
		OCDF	0.0003				

Source: (EA, 2009), Van den Berg et al. (2006)

Both EFSA and the EA have evaluated population dietary estimates for PCDD/Fs and dioxin-like PCBs. These are summarised below and provide one measure of context for the health-based guideline values in Table TN08-03.

Table TN08-04. Population Dietary Exposure to PCDD/Fs and DL-PCBs

Authoritative Body/Author	Estimated Dietary Intake (expressed as 2,3,7,8-TCDD TEQ WHO_{2005})
EFSA (2018)	Mean (upper bound) 0.39-2.57pg/kgBW/day (PCDD/Fs and DL-PCBs)

Authoritative Body/Author	Estimated Dietary Intake (expressed as 2,3,7,8-TCDD TEQ WHO_{2005})
EA (2009)	Average (upper bound) for UK adults 0.7pg/kgBW/day based on 2001 FSA Total Diet Study
Bramwell et al (2017)	Average (upper bound) for UK adults 0.52pg/kgBW/day based on 2012 FSA Total Diet Study and 0.27pg/kgBW/day based on a Duplicate Diet Study

Source: (EFSA, 2019) (EA, 2009) (Bramwell, et al., 2017)

Upper bound average population dietary exposures are above the revised EFSA TWI. EFSA calculated that dietary exposure (expressed as TEQ) is dominated by the dioxin-like PCBs (and specifically PCB-126) with dioxin-like PCBs contributing 63% to overall lower bound exposure, PCDDs contributing 14%, and PCDFs contributing 22.8%. The dominant dioxin is 1,2,3,7,8-PeCDD (7.4%); the dominant furan is 2,3,4,7,8-PeCDF (10.7%). The principal food groups contributing to exposure are all associated with high fat content (which is where PCDD/Fs bioaccumulate) – namely butter, cheese, livestock meat and fatty fish. Potatoes are also identified by EFSA as a main contributor for infant exposure, but this is likely due to the high consumption rate rather than high dioxin content within potatoes. Because of the low solubility of PCDD/Fs and dioxin-like PCBs, plant uptake of these chemicals is typically very low (EA, 2009).

3.2 Generic Screening Criteria

The Environment Agency published the following Soil Guideline Values (SGVs) for PCDD/Fs and dioxin-like PCBs which is applicable only to sites where there is no on-site source of dioxins (EA, 2009):

Table TN08-05. UK Soil Guideline Values

Land Use	Soil Guideline Value[4] (µg/kg dry weight TEQ)
Residential	8
Commercial	240
Allotment	8

Source: (EA, 2009)

USEPA RSLs for dioxins, (hexachlorodibenzo-p-dioxine, mixture and TCDD, 2,3,7,8), furans (dibenzofuran and furan) and dioxin-like PCBs are summarised in Table TN08-06 below.

Table TN08-06. USEPA RSLs for residential and commercial/industrial land use, values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial/industrial
Hexachlorodibenzo-p-dioxine, Mixture	USEPA RSL	0.0001	0.0001	0.00047
TCDD, 2,3,7,8,	USEPA RSL	0.0000048	0.0000048	0.000022
Dibenzofuran	USEPA RSL	73	73	1,000
Furan	USEPA RSL	73	73	1,000
Tetrachlorobiphenyl,	USEPA	380	380	16

[4] Based on the PCDD/F and DL-PCB profile for UK urban soils, the TDI of 2pg/kgBW/day, and the generic land use exposure scenarios incorporated into the CLEA model, as set out in the CLEA methodology (EA, 2009)

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial/industrial
3,3'4,4', PCB 77	RSL			
Tetrachlorobiphenyl, 3,4,4',5, PCB 81	USEPA RSL	0.012	0.012	0.048
Tetrachlorobiphenyl, 3,3',4,4',5, PCB 126	USEPA RSL	0.000036	0.000036	0.00015
Tetrachlorobiphenyl, 3,3',4,4',5,5', PCB 169	USEPA RSL	0.00012	0.00012	0.00051
Tetrachlorobiphenyl, 2,3,3',4,4', PCB 105	USEPA RSL	0.12	0.12	0.49
Tetrachlorobiphenyl, 2,3,4,4',5, PCB 114	USEPA RSL	0.12	0.12	0.5
Tetrachlorobiphenyl, 2,3'4,4'5, PCB 118	USEPA RSL	0.12	0.12	0.49
Tetrachlorobiphenyl, 2',3,4,4',5, PCB 123	USEPA RSL	0.12	0.12	0.49
Tetrachlorobiphenyl, 2,3,3',4,4',5, PCB 156	USEPA RSL	0.12	0.12	0.5
Tetrachlorobiphenyl, 2,3,3'4,4',5' PCB 157	USEPA RSL	0.12	0.12	0.5
Tetrachlorobiphenyl, 2,3',4,4',5,5' PCB 167	USEPA RSL	0.12	0.12	0.51
Tetrachlorobiphenyl, 2,3,3',4,4',5,5' PCB 189	USEPA RSL	0.13	0.13	0.52

(US_EPA, 2019)

4. Brominated Dioxins and Furans (and mixed halogenated dioxins and furans)

4.1 Health Effects and Health Based Guidance Values

No authoritative reviews were identified for brominated dioxins and furans, or mixed halogenated compounds with the exception of the reviews undertaken by COT in 2005 (COT, 2006) and 2010 (COT, 2010).

- COT statement on organic chlorinated and brominated contaminants in shellfish, farmed and wild fish [Online] Available at: https://cot.food.gov.uk/cotstatements/cotstatementsyrs/cotstatements2006/cotstatement2006fish surveys (COT, 2006).

- COT, 2010. COT statement on occurrence of mixed halogenated dioxins and biphenyls in UK food, COT Statement 2010/02, London: Committee on Toxicity in Food, Consumers Products and the Environment (COT, 2010).

The COT statement on organic chlorinated and brominated contaminants in shellfish, farmed and wild fish was utilised in their assessment on the occurrence of these compounds in food in the UK (COT, 2006). COT concluded that "there is increasing evidence that the brominated dioxins, furans and coplanar and mono-ortho polybrominated biphenyls are dioxin like in respect to their effects in vitro and in vivo mammalian test systems" (COT, 2006). COT agreed that in light of this evidence, and the absence of an alternative approach, it would be prudent to apply the 2005 WHO-TEFs for chlorinated dioxins and furans to the brominated and mixed halogenated dioxin and furans. COT also advocated combining the TEQs for the brominated contaminants with the WHO-TEQs for the chlorinated dioxins as this would provide an indication of the total intake of chemicals with dioxin-like properties.. In their 2010 review, COT reaffirmed the 2,3,7,8-TCDD TDI of 2pg/kgBW/day as the toxicological basis for the use of the 2005 WHO-TEFs (COT, 2010).

The FSA dietary data was for 19 selected mixed halogenated dioxin, furan and biphenyl congeners and therefore it was not possible to produce reliable dietary estimates for all congeners. The estimated high level adult dietary exposure was 1.4pg/kgBW/day (COT, 2010).

4.2 Generic Screening Criteria

No generic screening criteria have been identified from UK or international sources in this review.

5. Polycyclic aromatic hydrocarbons

5.1 Health Effects and Health Based Guidance Values

The most recent[5] and relevant authoritative reviews of this group of chemicals identified in the literature search are included in the following publications:

- The LQM/CIEH S4ULs for Human Health Risk Assessment. Nathanail, C P; McCaffrey, C; Gillett, A G; Ogden, R C; Nathanail, J F. 2015
- Compendium of Chemical Hazards. Polycyclic aromatic hydrocarbons (Benzo[a]pyrene): Toxicological Overview. Public Health England, PHE, 2018
- Toxicological Review of Benzo[a]pyrene. US EPA IRIS, 2017
- SP1010: Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination – Policy Companion Document. Department for Environment, Food and Rural Affairs, DEFRA, 2014
- Organic Environmental Contaminants in the 2012 Total Diet Study Samples: Report to the Food Standards Agency. FERA, 2012
- Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)

This summary therefore focuses on the report by (Nathanail, et al., 2015) with reference to changes in conclusions compared to those reported previously.

Polycyclic aromatic hydrocarbons (PCA) are described by (Nathanail, et al., 2015) as a group of congeners which contain at least 2 aromatic rings in their structure, generally occurring as complex mixtures. In the 1970s the US EPA adopted a list of 16 priority PAH (US EPA, 2017) which comprises:

Acenaphthene	**Benzo(a)pyrene**	**Chrysene**	**Fluorene**
Acenaphthylene	**Benzo(b)fluoranthene**	**Dibenz(ah)anthracene**	**Naphthalene**
Anthracene	**Benzo(k)fluoranthene**	**Indeno(123-cd)pyrene**	**Phenanthrene**
Benz(a)anthracene	**Benzo(ghi)perylene**	**Fluoranthene**	**Pyrene**

[5] Studies published after (and therefore not referenced in) (Nathanail, et al., 2015) The LQM/CIEH S4ULs for Human Health Risk Assessment.

The health effects associated with chronic exposure to PAHs were summarised by (PHE, 2018) as including increased risk of lung damage, breathing problems, skin irritation, weakened immune systems and heart disease. Cancer is another significant health effect resulting from chronic PAH exposure; however the risk posed by specific PAH congeners is variable. A summary of various studies examining the carcinogenicity and genotoxicity of the 16 PAHs was presented by (Nathanail, et al., 2015) and the most recent studies included are included below.

Table TN08-07. Carcinogenicity and genotoxicity of PAH

		Genotoxicity (SCF, 2002)				
		Genotoxic	Equivocal	Probably not genotoxic	Not genotoxic	Inadequate data
Carcinogenicity (IARC, 2010)	Group 1 – Carcinogenic to humans	Benzo(a)pyrene				
	Group 2A – Probably carcinogenic to humans	Dibenz(ah)anthracene				
	Group 2B – Possibly carcinogenic to humans	Benz(a)anthracene Benzo(b)fluoranthene Benzo(k)fluoranthene Chrysene Indeno(123-cd)pyrene		Naphthalene		
	Group 3 – Not classifiable	Benzo(ghi)perylene	Fluoranthene Phenanthrene		Anthracene Pyrene	Acenaphthene Fluorene
	Not evaluated					Acenaphthylene

(Nathanail, et al., 2015) reviewed previously derived health criteria values (HCVs) presented in a range of international studies, and these were used to determine values for TDI_{oral} or ID_{oral} and TDI_{inh} or ID_{inh} for each PAH. For the PAHs that have not been determined to be genotoxic, threshold TDI values were derived for the onset of health effects. In contrast, for the genotoxic PAHs, non-threshold ID values were derived. The Index Dose represents a dose that poses a minimal risk level from possible exposure from a particular source, with the additional requirement that exposure needs to be reduced to as low a level as reasonably practicable. Following the publication of the S4ULs by Nathanail et al. (2015), the US EPA published an update to their 2014 Toxicological Review of Benzo(a)pyrene (US EPA, 2017). The overall oral RfD and inhalation RfC of 3×10^{-4} mg/kg/d and 2×10^{-6} mg/m^3 respectively remained the same in the 2017 update, although it was noted that a reproductive RfC of 3×10^{-6} mg/m^3 had now been calculated. The carcinogenic risk slope factors for oral exposure of 1 per mg/kg/day was consistent with the 2014 version, as was the inhalation unit risk of 6×10^{-4} per µg/m^3. The 2014 version gave a dermal slope factor of 0.006 per µg/day which was based on an extrapolation of animal data considering several approaches for this. The 2017 version did not include a dermal slope factor, as the methodologies for interspecies extrapolation were still considered to be under development.

Background intake of PAHs has been analysed by a number of studies including ATSDR (1995) (ATSDR, 1995), SCF (2002) (WHO, 1998), JECFA (2005) and (EFSA, 2008), which are referenced and incorporated into the derivation of MDI values by (Nathanail, et al., 2015). It was reported that food is considered to be the major source of human PAH exposure, as PAHs can be formed during cooking or due to atmospheric deposition. Inhalation exposure to PAHs also occurs due to industrial sites, traffic emissions and domestic heating systems. PAH intake from the UK drinking water supply was assumed to be negligible. The MDI values given below by Nathanail et al. (2015) were based on the 2002 FSA Total Diet Study (TDS). More recently, an updated TDS was conducted for a number of contaminants including PAH (FERA, 2012). The results of the analysis of the 2012 TDS samples for PAH appear broadly consistent with the data generated for earlier studies and were all below the regulatory maximum levels. Therefore, the previously generated MDI values can be considered to still be applicable.

Table TN08-08. Derived Toxicological Values and Population Dietary Exposure to PAH

PAH Congener	Threshold effects Oral TDI (µg kg^{-1} bw day^{-1})	Non-threshold effects Oral ID (µg kg^{-1} bw day^{-1})	Threshold effects Inhalation TDI (µg kg^{-1} bw day^{-1})	Non-threshold effects Inhalation ID (µg kg^{-1} bw day^{-1})	Oral MDI – food (µg day^{-1})	Inhalation MDI (µg day^{-1})
Acenaphthene	60		60		0.98	0.025
Acenaphthylene	60		60		0.14	0.011
Anthracene	300		300		0.08	0.041
Benz(a)anthracene		0.155		0.0015	0.06	0.011
Benzo(a)pyrene		0.031		0.00030	0.11	0.006
Benzo(b)fluoranthene		0.039		0.00038	0.11	0.013
Benzo(ghi)perylene		3.44		0.033	0.06	0.010
Benzo(k)fluoranthene		1.03		0.010	0.09	0.007
Chrysene		0.31		0.0030	0.11	0.017
Dibenz(ah)anthracene		0.0031		0.00003	0.04	0.033
Fluoranthene	12.5		12.5		0.35	0.084
Fluorene	40		40		0.59	0.096
Indeno(123-cd)pyrene		0.443		0.0043	0.10	0.009
Naphthalene	20		0.86		7.0	2.8
Phenanthrene	12.5		12.5		1.54	0.518
Pyrene	30		30		0.35	0.065
Coal Tar (BaP as surrogate marker)		0.01		0.00030		

Source: (Nathanail, et al., 2015)*, oral MDI after FSA (2002)*

5.2 Generic Screening Criteria

The following S4ULs were derived for PAH (Nathanail, et al., 2015):

Table TN08-09. S4UL values for PAH for various end use scenarios

PAH Congener	Residential with home-grown produce (mg/kg)			Residential without home-grown produce (mg/kg)			Allotment (mg/kg)			Commercial (mg/kg)			Public Open Space – residential (mg/kg)			Public Open Space – park (mg/kg)		
	1% SOM	2.5% SOM	6% SOM	1% SOM	2.5% SOM	6% SOM	1% SOM	2.5%SOM	6% SOM	1% SOM	2.5% SOM	6% SOM	Sandy loam TOC >0.58 to <1.45%	Sandy loam TOC >1.45 to <3.48%	Sandy loam TOC >3.48%	Sandy loam TOC >0.58 to <1.45%	Sandy loam TOC >1.45 to <3.48%	Sandy loam TOC >3.48%
Acenaphthene	210	510	1100	3000	4700	6000	34	85	200	84000	97000	100000	15000	15000	15000	29000	30000	30000
Acenaphthylene	170	420	920	2900	4600	6000	28	69	160	83000	97000	100000	15000	15000	15000	29000	30000	30000
Anthracene	2400	5400	11000	31000	35000	37000	380	950	2200	520000	540000	540000	74000	74000	74000	150000	150000	150000
Benz(a)anthracene	7.2	11	13	11	14	15	2.9	6.5	13	170	170	180	29	29	29	49	56	66
Benzo(a)pyrene	2.2	2.7	3.0	3.2	3.2	3.2	0.97	2.0	3.5	35	35	36	5.7	5.7	5.7	11	12	14
Benzo(b)fluoranthene	2.6	3.3	3.7	3.9	4.0	4.0	0.99	2.1	3.9	44	44	45	7.1	7.2	7.2	13	15	18
Benzo(ghi)perylene	320	340	350	360	360	360	290	470	640	3900	4000	4000	640	640	640	1400	1500	1600
Benzo(k)fluoranthene	77	93	100	110	110	110	37	75	130	1200	1200	1200	190	190	190	370	410	470
Chrysene	15	22	27	30	31	32	4.1	9.4	19	350	350	350	57	57	57	93	110	130
Dibenz(ah)anthracene	0.24	0.28	0.3	0.31	0.32	0.32	0.14	0.27	0.43	3.5	3.6	3.6	0.57	0.57	0.58	1.1	1.3	1.4
Fluoranthene	280	560	890	1500	1600	1600	52	130	290	23000	23000	23000	3100	3100	3100	6300	6300	6400
Fluorene	170	400	860	2800	3800	4500	27	67	160	63000	68000	71000	9900	9900	9900	20000	20000	20000
Indeno(123-cd)pyrene	27	36	41	45	46	46	9.5	21	39	500	510	510	82	82	82	150	170	200
Naphthalene	2.3	5.6	13	2.3	5.6	13	4.1	10	24	190	460	1100	4900	4900	4900	1200	1900	1600
Phenanthrene	95	220	440	1300	1500	1500	15	38	90	22000	22000	23000	3100	3100	3100	6200	6200	6300
Pyrene	620	1200	2000	3700	3800	3800	110	270	620	54000	54000	54000	7400	7400	7400	15000	15000	15000
Coal Tar (BaP as surrogate marker for PAH mixture)	0.79	0.98	1.1	1.2	1.2	1.2	0.32	0.67	1.2	15	15	15	2.2	2.2	2.2	4.4	4.7	4.9

Source: (Nathanail, et al., 2015)

DEFRA has published the following C4SLs for benzo(a)pyrene. C4SLs are derived for a number of scenarios. The critical receptor is generally assumed to be a 0 to 6 year old child for residential and allotment land uses and a 16 to 65 year old adult for commercial land use. Public open space land uses are based on two scenarios: that of a green space close to housing that includes tracking back of soil (POS 1 or POS_{resi}) and a park-type scenario where the park is considered to be at a sufficient distance that there is negligible tracking back of soil (POS 2 or POS_{park}).

It is noted (DEFRA, 2014) that a surrogate marker approach is used in the derivation of the C4SLs for benzo(a)pyrene. This allows the toxicity of a mixture of PAH to be estimated using toxicity data for a PAH mixture of known composition. In this approach, exposure to the surrogate marker is assumed to represent exposure to all PAH in the mixture. Three main assumptions within the surrogate marker approach were listed: the surrogate marker must be present in all samples, the profile of different PAH relative to the surrogate marker should be similar in all samples, and the PAH profile in the samples should be similar to that used in the pivotal toxicity study on which the health values were based. The rationale for the adoption of this approach is supported by (HPA, 2009) (PHE, 2017). This is not the approach adopted by other countries, such as the USA, as explained in the HPA notes.

The health-based guidelines developed by the C4SL project (termed a "Level of Low Toxicological Concern" (LLTC) are:

- Oral: 0.042µg/kgBW/day; based on a margin of 5000 from a benchmark dose (BMD10) of 0.21mg/kgBW/day. This $LLTC_{oral}$ is 4.2 times higher than the Index Dose used in the derivation of the S4ULs but based on the same Culp et al coal tar toxicological study.
- Inhalation: 0.3ng/kgBW/day; based on an excess lifetime cancer risk of 1 in 10,000 and an air concentration of $1ng/m^3$. This $LLTC_{inhal}$ is the same as the Index Dose used in the derivation of the S4ULs and is based on an evaluation of UK and European air quality targets for benzo(a)pyrene.

Table TN08-10. C4SLs for benzo(a)pyrene

Land Use	C4SL (mg/kg) 6% SOM
Residential with home-grown produce	5.0
Residential without home-grown produce	5.3
Allotment	5.7
Commercial	77
Public open space 1	10
Public open space 2	21

Source: (DEFRA, 2014)

No update on estimates for background exposure were made as part of the C4SL project because benzo(a)pyrene is a genotoxic carcinogen and therefore ALARP applies (HSE, n.d.).

USEPA RSLs for PAHs are listed in the table below.

Table TN08-11. USEPA RSLs for residential and commercial industrial land use, values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial industrial
Acenaphthene	USEPA RSL	3,600	3,600	45,000
Anthracene	USEPA RSL	18,000	18,000	230,000
Benz(a)anthracene	USEPA RSL	1.1	1.1	21

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial industrial
Benzo(a)pyrene	USEPA RSL	0.11	0.11	2.1
Benzo(b)fluoranthene	USEPA RSL	1.1	1.1	21
Benzo(i)fluoranthene	USEPA RSL	0.42	0.42	1.8
Benzo(k)fluoranthene	USEPA RSL	11	11	210
Chrysene	USEPA RSL	110	110	2,100
Dibenz(ah)anthracene	USEPA RSL	11	11	2.1
Fluoranthene	USEPA RSL	2,400	2,400	30,000
Fluorene	USEPA RSL	2,400	2,400	30,000
Indeno(123-cd)pyrene	USEPA RSL	1.1	1.1	21
Naphthalene	USEPA RSL	3.8	3.8	17
Chrysene	USEPA RSL	110	110	2,100
Pyrene	USEPA RSL	1,800	1,800	23,000

(US_EPA, 2019)

6. Benzene

6.1 Health Effects and Health Based Guidance Values

The most recent[6] and relevant authoritative reviews for benzene identified in the literature search are included in the following publications:

- The LQM/CIEH S4ULs for Human Health Risk Assessment. Nathanail, C P; McCaffrey, C; Gillett, A G; Ogden, R C; Nathanail, J F. 2015
- Soil Guideline Values for benzene in soil. Science Report SC050021/benzene. Environmental Agency, EA, 2009
- EA, 2009
- SP1010: Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination – Policy Companion Document. Department for Environment, Food and Rural Affairs, DEFRA, 2014
- Benzene. Agency for Toxic Substances and Disease Registry, ATSDR, 2015
- ATSDR, 2015

Benzene: Toxicological overview. Public Health England, PHE, 2007. Benzene is an aromatic hydrocarbon that is naturally present in crude petroleum and coal tar and is used in the manufacture of other chemicals and products. Information reported by (PHE, 2007) indicates that the primary exposure pathway is through inhalation of benzene, and that acute exposure can lead to throat, nose, eye and skin irritation and breathing difficulties. Chronic exposure to benzene can have genotoxic and carcinogenic effects. The most significant chronic effects of benzene exposure are on the blood and

[6] Studies published after (and therefore not referenced in) Nathanail et al. (2015) The LQM/CIEH S4ULs for Human Health Risk Assessment.

immune system (EA, 2009), and are reported by PHE as decreases in white blood cell counts and myeloid and non-lymphocytic leukaemia. Benzene is classified by the (US EPA, 2002) , ACGIH (2014) and (IARC, 1987) as a known human carcinogen (TOXNET, 2014).

Nathanail et al. (2015) reviewed previously derived health criteria values presented in a range of international studies, and these were used to determine values for ID_{oral} and ID_{inh} for benzene. Index Doses are used as benzene exhibits non-threshold effects. The derived Index Doses for benzene were as follows: ID_{oral} = 0.29 µg kg^{-1} bw day^{-1} and ID_{inh} = 1.4 µg kg^{-1} bw day^{-1}. These values are consistent with those given by the EA (2009).

Background intake of benzene was examined by the EA (2009) (EA, 2009) and (DEFRA, 2014). Nathanail et al. (2015) reported that the EA determined that MDI_{oral} < 3 µg day^{-1} and MDI_{inh} = 200 µg day^{-1}. However, these exposures were not considered in the derivation of S4ULs, as the health criteria for benzene are Index Doses.

6.2 Generic Screening Criteria

The following S4UL values were derived for benzene (Nathanail, et al., 2015). It should be noted that in accordance with UK policy, because benzene is a non-threshold substance background exposures are not considered in the derivation of the S4ULs.

Table TN08-12. S4ULs for Benzene

Land Use	S4ULs for Benzene (mg/kg DW)		
	1% SOM	2.5% SOM	6% SOM
Residential with home-grown produce	0.087	0.17	0.37
Residential without home-grown produce	0.38	0.70	1.4
Allotment	0.017	0.034	0.075
Commercial	27	47	90
Public open space 1 (POS_{resi})	72	72	73
Public open space 2 (POS_{park})	90	100	110

Source: Nathanail et al. (2015)

Source: (Nathanail, et al., 2015)

The EA published the following Soil Guideline Values for benzene (EA, 2009), based on a sandy loam soil and 6% SOM:

Table TN08-13. Soil Guideline Values for Benzene

Land use	Soil Guideline Value (mg/kg DW)
Residential	0.33
Allotment	0.07
Commercial	95

Source: EA (2009)

NOTE: SGVs are based on superseded exposure assumptions which were set by the C4SL project.

The following C4SLs for benzene were derived by DEFRA. As for PAH, C4SLs were developed using a different toxicological approach to that used for the S4ULs. The health-based guidelines developed by the C4SL project (termed a “Level of Low Toxicological Concern” (LLTC) are:

- Oral: 0.57µg/kgBW/day; based on an excess lifetime cancer risk of 1 in 50,000 for leukemia (2-20 times higher than the risk used to set drinking water standards by WHO and EU respectively. This $LLTC_{oral}$ is 2 times higher than the Index Dose used in the derivation of the S4ULs.

- Inhalation: 1.4µg/kgBW/day; based on the UK air quality standard of 5µ/m^3. This is associate with an estimated excess lifetime cancer risk of 1 in 34,000. The $LLTC_{inhal}$ is the same value as the ID_{inhal} used in the derivation of the S4ULs.

Table TN08-14. C4SLs for Benzene

Land Use	C4SL (mg/kg DW) 6%
Residential with home-grown produce	0.87
Residential without home-grown produce	3.3
Allotment	0.18
Commercial	98
Public open space 1	140
Public open space 2	230

Source: (DEFRA, 2014)

No update on estimates for background exposure were made as part of the C4SL project because benzene is a genotoxic carcinogen and therefore ALARP applies (HSE, n.d.).

7. Non dioxin-like PCBs

7.1 Health Effects and Health Based Guidance Values

The main references identified by the literature search are:

- HSDB: Polychlorinated biphenyls. Hazardous Substances Data Bank, HSDB, 2017
- Opinion of the scientific panel on contaminants in the food chain on a request for the Commission related to the presence of non dioxin-like polychlorinated biphenyls (PCB) in feed and food. EFSA, 2005

PCBs were manufactured as complex mixtures of congeners by the progressive chlorination of batches of biphenyl until a target percentage of chlorine by weight was achieved. Of the 209 congeners that are theoretically possible, only about 130 have been identified in commercial products that were marketed (WHO, 2016).

To standardize the identification of the individual PCB congeners, a numbering system was developed by Ballschmiter & Zell (1980), following the International Union of Pure and Applied Chemistry (IUPAC) rules for characterization. In this scheme, a number, called the “BZ number”, is attributed to each individual congener. This number correlates the structural arrangement of the PCB congener and ascending order of number of chlorine substitutions within each sequential homologue. Thus, congeners are numbered from PCB 1 to PCB 209, a useful shorthand nomenclature. However, it is important to note that it obscures the chemical identity of the congener and does not strictly follow the IUPAC rules (WHO, 2016).

PCBs exhibit different toxicological effects depending on the site of the chlorine substitution on the phenyl rings. As addressed in section 3, the 12 dioxin-like PCBs have congeners with only one or no chlorine atoms in the ortho position (having a similar planar structure, and therefore toxicological activity, to PCDDs and PCDFs). The other 197 remaining congeners are referred to as Non-Dioxin like PCBs (NDL-PCBs) (WHO, 2016).

NDL-PCBs do not show dioxin-like toxicity and have a slightly different toxicological profile, in particular with respect to effects on the developing nervous system and neurotransmitter function (feed & food) compared to dioxin like PCBs. However, chloracne and other skin & eye irritations were listed by many studies referenced within (TOXNET, 2017) as being the key acute symptoms of PCB exposure. The liver is considered to be one of the most important target organs for NDL-PCB toxicity, along with immunological and neurological symptoms that have also been reported (TOXNET, 2017).

PCBs (unspecified) were classified by (IARC, 1987), the (US EPA, 1994) and the DHHS & NTP (2009) as probable human carcinogens. Results of genotoxicity studies indicate that PCBs (unspecified) are not mutagenic, although some less chlorinated congeners can cause DNA damage, probably resulting from the formation of reactive oxygen species.

Benchmark doses were calculated by (EFSA, 2005) based on studies on human developmental neurotoxicity and immunotoxicity after perinatal exposure to total DL and NDL-PCB. The study determined a 95% lower confidence limit of benchmark dose (BMDL) of approximately 1 µg PCB/g lipid which was four times higher than the contemporary median concentration in human milk. The study focused on the toxicology of NDL-PCB congeners, concluding that the most sensitive effects seen in animal studies were liver and thyroid toxicity. In 90-day rat studies, the NOAELs for these effects for PCB 28, 128, and 153 were in the range of 30-40 µg/kg bw per day. Due to the lipophilic nature of NDL-PCBs which results in a tendency to accumulate in the body, an evaluation based on Body Burden (BB) calculations was considered instead of a consideration of the external dose alone. Applying a BB approach to the rat studies mentioned above the estimated NOAEL body burdens were determined to be 400, 800, and 1,200 µg/kg bw per day for PCB 28, 128, and 153 respectively. An overall conservative NOAEL BB of 500 µg/kg bw per day for all individual NDL-PCB and for the sum of NDL-PCB occurring in human tissues was derived. Older exposure limits were listed by NIOSH (1985). A 10-hour TWA exposure limit of 1 µg/m3 was recommended by NIOSH. 8-hour TWA permissible exposure limits (PEL) of 1 mg/m3 for chlorodiphenyl products containing 42% chlorine and 0.5 mg/m3 for chlorodiphenyl products containing 54% chlorine were given by OSHA and based on TLVs given by ACGIH (1968). The ACGIH Short Term Exposure Limits (STEL) for chlorodiphenyls are 2 mg/m3 and 1 mg/m3 for 42% and 54% chlorine products, respectively.

International bodies have identified seven PCBs that can be used to characterize the presence of PCB contamination. Six of the seven are NDL-PCBs (PCB 28, PCB 52, PCB 101, PCB 138, PCB 153 and PCB 180), and one is a DL-PCB (PCB 118). These seven PCBs are often called “indicator PCBs (WHO, 2016). These seven PCBs were selected as indicators as they make up ~20% by weight of PCBs in commercial mixtures and they have a wide chlorination range. Plus, they are more likely to be found in environmental samples and at higher concentrations than the group of twelve speciated PCBs adopted by the WHO.

The current RIVM intervention value for PCBs (sum 7) is 1 mg/kg dw and dates back to the 1990s. RIVM proposed new intervention values for individual PCBs in 2001, however, the proposed values were never formalized (RIVM, 2014).

7.2 Generic Screening Criteria

In 2014 RIVM proposed risk-based standards for PCBs in soil for the sum of 7 indicator PCBs (RIVM, 2014). The human risk values are based on maximal permissible risk (MPR) and derived using the human risk assessment model CSOIL and presented for a variety of different exposure scenarios. The value for the sum of 7 indicator species was derived using a mixture based on Arochlor[7] standard 1254 (normalised to make up for 100% of the mixture). A second mix was used representing the distribution of individual PCB congeners in the generic background concentration in Dutch soil (AW2000). However, comparing the resulting sum of 7 PCB human reference values for Arochlor and AW2000, very little variation was observed. Therefore, preference was given to the sum value based on Arochlor mixture, due to familiarity by users and international acceptance (RIVM, 2014).

Table TN08-15. Proposed maximal values (human) based on the reference values for ‘residential’ and ‘industry’ for PCBs (sum7), based on mixtures of Arochlor1254.

Mixture	Proposed maximal Value Residential (mg/kg dw)	Proposed maximal value Industrial (mg/kg dw)
PCBs (sum7) based on Arochlor 1254	0.39	15
(RIVM, 2014)		

[7] 'Aroclor' is a trademarked name for commercial mixtures of PCBs used by Monsanto, and some toxicological studies refer to the effects of Aroclor. Other commercial mixtures of PCBs are marketed under names such as Clophen, Phenochlor, Kanechlor, Pyralene, Fenclor, Delor amongst others.

As different mixtures of PCBs might occur in practice, RIMV also proposed reference values for individual PCB congeners based on human risk assessment (RIVM, 2014).

Although, no formal proposal for an intervention value for the sum of PCBs was given in the 2014 RIVM report it was noted that the contribution of the individual PCB congeners in the Arochlor mixture could be used to derive a value for the sum PCBs based on the proposed intervention values from 2001. This value would be 0.78 mg/kg dw for soil (RIVM, 2014).

SGVs, C4SLs or S4ULs have not been published for the majority of PCBs, excepting the DL-PCBs which are included in Section 2.

8. Cyanide

8.1 Health Effects and Health Based Guidance Values

The main references identified by the literature search are:

- Contaminants in soil: collation of toxicological data and intake values for humans. inorganic cyanide. Environmental Agency, EA, 2002
- Cyanide, free CASRN 57-12-5. US EPA, 2010
- (PHE, 2016). Public Health England, PHE, 2016
- Toxicological profile for Cyanide. Agency for Toxic Substances and Disease Registry, ATSDR, 2006
- Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)

Cyanide toxicity results from inhibition of cytochrome oxidase, which limits the absorption of oxygen at the cellular level and therefore deprives the body of oxygen. In addition, chronic cyanide exposure may lead to excess thiocyanate in the body, which can affect the ability of the thyroid gland to accumulate iodine (EA, 2002). Chronic exposure can also lead to neurological effects and neuropathies. However, hydrogen cyanide has no mutagenic properties and is not considered to be a carcinogen (PHE, 2016). According to the (EA, 2002), the average fatal oral dose for humans is 1.52 mg CN kg^{-1} bw and the lowest reported fatal oral dose for humans is 0.56 mg kg^{-1} bw. The average fatal dose for dermal exposure has been estimated to be 100 mg CN kg^{-1} bw.

The discussion of TDIs below focuses on findings of the (EA, 2002). It was noted that this report draws on reviews of toxicity published by Way (1984), Shifrin et al (1996), the World Health Organization WHO (1996), ATSDR (1997) and the US EPA (1984, 1985, 2001). The TDI, MRL and RfD/C derived in these previous studies were used in by the EA to determine their TDI values. The JECFA (1981) TDI was 0.05 mg/kg bw. ATSDR gave an MRL of 50 µg kg^{-1} bw day^{-1} for oral exposure of up to 1 year, i.e. exposure of intermediate duration. A chronic MRL was not derived. The (WHO, 2004) gave a TDI of 1.2 µg/kg bw. When allocating 20% of the TDI to exposure from drinking water, a guideline value of 70 µg/L was derived. The (US EPA, 2010) gave an oral RfD for HCN and its sodium, potassium, calcium, silver and zinc salts of 20 µg CN kg^{-1} bw day^{-1}. The EPA assigned a medium degree of confidence to this value. An inhalation RfC for HCN of 3 µg/m^3 was also determined.

Based on the WHO and EPA findings, the EA derived the following TDIs for cyanide:

Table TN08-16. TDIs for Cyanide

Exposure Type	Derived TDI (µg CN kg^{-1} bw day^{-1})
Oral (TDI_{oral})	12
Inhalation (TDI_{inh})	0.9

Source: (EA, 2002)

(ATSDR, 2006) noted in their review that cyanides are a diverse family of compounds, the most common forms found in the environment being sodium and potassium cyanide and hydrogen cyanide. Numerous plants contain cyanide glycosides, including apricot and apple seeds and cassava root. No dietary intake estimates were possible based on the data available to ATSDR. An intermediate (exposure duration between 15 days and 1 year) minimal risk level (MRL) of 0.05mgCN^- /kgBW/day was developed for oral exposure based on a NOAEL of 4.5mg CN^-/kgBW/day for an animal study using sodium cyanide and the critical effect of reproductive toxicity. No acute or chronic oral MRLs were developed, nor were any inhalation MRLs due to the severity of acute exposure effects and lack of evidence for chronic exposure.

The US EPA re-evaluated free cyanide, hydrogen cyanide, calcium, sodium, potassium and potassium silver cyanides, and cyanogen in 2010 (US EPA, 2010). A summary of the health-based guideline values derived by the US EPA is provided below:

Table TN08-17. US EPA Health-based Guidelines for Cyanides

Compound	Health-based Guideline	Critical Toxic Effect
Calcium cyanide	Oral RfD 0.001mg/kgBW/day	Reproductive; calculated from BMD for CN^- adjusting for molecular weight
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable
Free cyanide (CN^-)	Oral RfD 0.00063mg/kgBW/day	Reproductive; $BMDL_{1SD}$ 1.9mg/kgBW/day
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable
Sodium cyanide	Oral RfD 0.001mg/kgBW/day	Reproductive; calculated from BMD for CN^- adjusting for molecular weight
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable
Potassium cyanide	Oral RfD 0.002mg/kgBW/day	Reproductive; calculated from BMD for CN^- adjusting for molecular weight
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable
Potassium silver cyanide	Oral RfD 0.005mg/kgBW/day	Reproductive; calculated from BMD for CN^- adjusting for molecular weight
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable
Cyanogen (CN_2)	Oral RfD 0.005mg/kgBW/day	Reproductive; calculated from BMD for CN^- adjusting for molecular weight
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable
Hydrogen cyanide (HCN)	Oral RfD 0.0007mg/kgBW/day	Reproductive; calculated from BMD for CN^- adjusting for molecular weight
	Inhalation RfC not evaluated (insufficient evidence)	Not applicable

Source: (US EPA, 2010)

PHE reviewed the toxicity of hydrogen cyanide (HCN) in 2016 (PHE, 2016). Unlike the majority of the other chemicals reviewed the primary health concern in relation to exposure to HCN is acute toxicity; ranging from non-specific central nervous system effects to rapid loss of consciousness and death. However, no long-term health effects are anticipated from single brief exposures to low concentrations of HCN from which the individual recovers quickly. Chronic exposure to HCN has bene associated with a range of adverse health effects including non-specific neurological effects, and effects on the thyroid, skin and gastrointestinal tract. Thiocyanate is produced in the detoxification of cyanide in the body and it is thought that it is this rather than the cyanide that causes the adverse effects in the thyroid. The UK workplace exposure limit for HCN is defined as a short-term exposure limit (STEL) of 11mg/m3 (averaged over a 15-period). PHE reports that there is limited data on the

ingestion of HCN. One study in animals produced a NOAEL of 10.8mg/kgBW/day based on the absence of treatment related toxicological effects at the lowest dose administered [note this is higher than the NOAEL referenced by ATSDR for intermediate exposure].

8.2 Generic Screening Criteria

Although there are no UK SGVs, C4SLs or S4ULs for cyanide species the following Dutch Intervention Values and USEPA RSLs based on 1% SOM are available (values in mg/kg):

Table TN08-18. Dutch Intervention Values and USEPA RSLs (values in mg/kg)

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Allotment	Commercial industrial
Cyanide (free)	Dutch Intervention Value	20	20	20	150
Cyanide Total	USEPA RSL	78	78	-	1200
Hydrogen Cyanide	USEPA RSL	23	23	-	13

(US_EPA, 2019)

9. Asbestos fibres

9.1 Health Effects and Health Based Guidance Values

The main references for asbestos identified by the literature search are:

- HSDB: Asbestos. HSDB, 2018
- Asbestos: Toxicological Overview, Public Health England, PHE, 2017 and 2007
- Statement on the relative vulnerability of children to asbestos compared to adults. Committee on Carcinogenicity, 2013
- Current Intelligence Bulletin 62: Asbestos Fibres and Other Elongate Mineral Particles: State of the Science and Roadmap for Research, Revised Edition. NIOSH, 2011
- Toxicological profile for Asbestos. Agency for Toxic Substances and Disease Registry, ATSDR, 2003

The adverse health effects associated with asbestos are relatively well understood and are based on epidemiological studies on cohorts of workers in the asbestos manufacturing and mining industries. Whilst the literature is in general agreement over the adverse health effects of exposure to asbestos (asbestosis, pleural plaques, lung cancer and mesotheliomas), there is far less agreement on the relative potency of different asbestos types (chrysotile, amosite, crocidolite, tremolite, anthophyllite and actinolite) and different fibre sizes. (PHE, 2017) refers to asbestos as a Category 1 carcinogen. The primary route of exposure for asbestos is inhalation, and to a much lesser extent ingestion. Dermal exposure is not thought to result in systemic circulation even though asbestos fibres can penetrate the skin (PHE, 2017)).

Published soil-based and air-based criteria are summarised below – the variability in the values a reflection of the differing approaches taken by different organisations.

Table TN08-19. Summary of Asbestos Air Quality Guidelines (f/m3)

Organisation	All asbestos types/type not specified	Amosite	Chrysotile/amosite mixtures	Chrysotile

Organisation	All asbestos types/type not specified	Amosite	Chrysotile/amosite mixtures	Chrysotile
World Health Organisation	100-1000			
US EPA (1988)	80			
US EPA (2008)	200			
US EPA (2014)		120		
Dutch National Institute for Public Health and Environment (RIVM)		1000		10000
Health Effects Institute (HEI)	50			
Health Council of the Netherlands (HCN)		30	130	280

Source: Baker et al, Discussion Paper on Guideline Values for Airborne Concentrations of Asbestos Fibres in Ambient Air: Implications for Quantitative Risk Assessment, SoBRA, 2017

9.2 Generic Screening Criteria

Risk-based asbestos in soil guideline values have been derived by Dutch Authorities (RIVM and VROM) and latterly utilised by other countries such as Australia and Belgium. The values developed by the Dutch are summarised below:

Table TN08-20. Dutch Asbestos in Soil Criteria

Criterion	Assessment Stage	Applicability
0.01% by weight	Tier 1	To be compared to the total concentration of serpentine asbestos (chrysotile) + 10 x concentration of amphibole asbestos (amosite and crocidolite) as an average concentration across an area of 1000m2. Designed to be protective of human health under all normal land-uses.
0.1%	Tier 2	To be compared to the concentration of serpentine asbestos (chrysotile) + 10 x concentration of amphibole asbestos (amosite and crocidolite) for non-friable asbestos e.g. fragments of asbestos cement
0.01% by weight	Tier 2	As above but for friable asbestos e.g. asbestos insulation materials
0.001% by weight	Tier 3	To be compared to counted respirable asbestos fibres only, and to be compared to the concentration of serpentine asbestos (chrysotile) + 10 x concentration of amphibole asbestos (amosite and crocidolite)

Source: VROM Soil Remediation Circular, 2013

There are no UK-specific soil guideline values for asbestos in soil which can be used as a suitable threshold screening criteria. In air, the only values are those specified in the Control of Asbestos Regulations 2012 – which stipulates a 4-h time-weighted average control limit of 0.1f/ml (100,000 f/m^3), a short-term exposure limit of 0.6f/ml over a 10-minute period, and a clearance indicator level of 0.01f/ml (10,000f/m^3). These values are equally applicable to all types and forms of asbestos.

Asbestos is found in the environment either as a result of natural occurrence in rock or because of the historic widespread use in construction and other industries. PHE estimate that background levels of asbestos in air in the UK may be below 0.0001f/ml in rural and urban areas, and below 0.001f/ml in buildings (PHE, 2017). Asbestos has also been found to be present in UK drinking water supplies, with PHE estimating that levels in drinking water could range from non-detect to 1 million fibres per litre of water (PHE, 2017).

10. Synthetic vitreous fibres

10.1 Health Effects and Health Based Guidance Values

The main references for synthetic vitreous fibres identified by the literature search are:

- Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibres: The Influence of Fibre Length. Agency for Toxic Substances and Disease Registry, ATSDR, 2003
- Toxicological profile for Synthetic Vitreous Fibers. Agency for Toxic Substances and Disease Registry. ATSDR, 2004
- HSDB: Synthetic vitreous fibres. HSDB, 2005 (ATSDR, 2004) identifies three categories of SVF – only two of which are relevant to this study; glass fibres (including glass wool and continuous filament glass), and mineral wool (containing either stone wool or slag wool). The third category is refractory ceramic fibres – these being used to insulate industrial furnaces. ATSDR also includes a more detailed categorisation developed by WHO/IARC.

(ATSDR, 2004) concluded in its review of toxicological and epidemiological evidence for adverse health effects associated with synthetic vitreous fibres that occupational exposure was not associated with increased lung problems. Short-term exposure could however cause reversible skin, eye, nose, throat and lung irritation. No clear association between exposure to SVF and cancer in humans was found, and whilst animal studies have shown that repeated exposure to high levels of refractory ceramic fibres can cause scarring of the lung tissue, this did not occur with exposure to glass fibres.

Reference is made by (ATSDR, 2004) to the OSHA PEL of 5mg/m^3 for inert or nuisance dust and a voluntary limit for fibreglass and mineral wool of 1f/cm^3. The UK Workplace Exposure Limit for Machine-made mineral fibres (MMMF) is 2f/cm^3 and 5 mg/m^3 (HSE (2018). The HSE definition of M (ATSDR, 2019)MMF is “man-made vitreous(silicate) fibres with random orientation with alkaline oxide and alkaline earth oxide content greater than 18%” and specifically excludes refractory ceramic fibres.

ATSDR derived a minimal risk level (MRL) for chronic exposure to SVFs of 0.03 f/cm^3 based on animal study data for refractory ceramic fibres. No MRL was derived for glass fibres, with ATSDR noting that any use of the MRL in assessing likely health hazards from insulation wools should acknowledge the evidence that many of the insulation wools are markedly less durable and less potent than the refractory ceramic fibres on which the MRL was based.

Expected levels of airborne SVFs in outdoor and indoor air is expected to be <0.0001f/cm^3. Higher levels (>1f/cm3) have been observed in homes where insulation is being installed (ATSDR, 2004).

11. Lead

11.1 Health Effects and Health Based Guidance Values

The main references identified by the literature search are:

- SP1010: Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination – Policy Companion Document. DEFRA, 2014
- Toxicological profile for Lead. ATSDR, 2019
- HSDB: Lead, elemental. HSDB, 2016
- Compendium of Chemical Hazards. Lead: Toxicological Overview. PHE, 2016 and 2017
- Scientific Opinion on Lead in Food. EFSA, 2012 and 2013
- Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)

The toxicological information for lead published by (PHE, 2016, 2017) and (ATSDR, 2019) indicates that the effects of lead are typically observed following lower level chronic exposure. Chronic plumbism can affect nearly every system in the body, however developmental neurotoxicity in children, elevation of blood pressure and nephrotoxicity are considered to be the most sensitive lead toxicity endpoints, having no apparent threshold. It was also reported that the US EPA IRIS program

and IARC have classified lead and lead compounds as probable human carcinogens (TOXNET, 2016; ATSDR, 2019). (EFSA, 2013) reported that the toxicological effects of lead on children are also of particular concern, as lead is absorbed more than in adults and accumulates in the body over time.

The scientific opinion of the CONTAM panel (EFSA, 2013) concluded that, due to a lack of evidence for a threshold for critical toxicological effects, the previous PTWI of 25 µg/kg bw (JECFA, 1986) was no longer appropriate, and no new PTWI was derived. (ATSDR, 2019) did not derive MRLs for lead exposure, as for the most studied toxicological endpoints, serious adverse effects occur at the lowest blood lead concentrations studied (≤5 µg/dL). However, ATSDR listed existing regulations and guidelines applicable to lead, the most recent of which are summarised below.

Table TN08-21. Regulations and guidelines applicable to lead

Authoritative Body	Health-based Guideline Value
ATSDR (2019)	MRL not derived, no threshold observed
US EPA NAAQS (2017)	0.15 µg/m^3 in air
WHO (2010) air quality guidelines	No data
US EPA (2018) drinking water standards and health advisories	No data
WHO (2017) drinking water quality provisional guideline value	0.01 mg/L[8]
EFSA (2013)	PTWI not derived, no threshold observed $BMDL_{01}$ of 1.2µg/dl calculated based on a 1 IQ point drop in children

Source: (ATSDR, 2019)

Background exposure to lead occurs primarily via ingestion of lead-containing food and water. (EFSA, 2013) reported that adult average lead dietary exposure ranges from 0.36 to 1.24 µg/kg bw per day, up to 2.43 µg/kg bw per day, and in children average exposure is greater, ranging from 0.80 to 3.10 µg/kg bw per day and up to 5.51 µg/kg bw per day.

The authors of the C4SL report (DEFRA, 2014) noted that the US EPA and the Canadian Council of Ministers of the Environment (CCME) were drafting reports on the toxicity at the time of the C4SL report, and that the UK Committee on Toxicity of chemicals in food, consumer products and the environment (COT) were also in the process of assessing lead intake in infant diets. The previous health-based guideline for lead in the UK was 10µg/dl (as a blood lead level) and the basis for the now withdrawn Defra/Environment Agency Soil Guideline Value of 450mg/kg for residential areas. The blood lead level chosen by DEFRA to be protective (LLTC) of neurobehavioural effects in children was 3.5 µg/dl. This was calculated to be associated with an equivalent dose (LLTC) of 1.4µg/kg/BW/day in children and between 0.63 and 1.3 µg/kgBW/day in adults. For comparison the authors calculated that the UK and EU drinking water standard of 10µg/l equates to an intake of 0.29µg/kgBW/day for adults and 0.67µg/kgBW/day for children. The LLTC applies to all exposure routes; no inhalation or dermal LLTC were developed.

COT (COT, 2013) used the EFSA $BMDL_{01}$ of 1.2µg/dl in their evaluation of the potential risk from infant dietary exposure to lead and equated this to a dietary exposure of 0.5µg/kgBW/day. COT estimated the following for background exposures in the UK:

Table TN08-22. COT estimates of background exposure to lead in the UK for infants

Exposure Route	Estimated Median Dose
Soils and dust	0.36µg/kgBW/day for rural soils 0.59µg/kgBW/day for semi-urban soils 1.7µg/kgBW/day for urban soils
Air	0.00031-0.0023 µg/kgBW/day

[8] *WHO (2017) based their provisional drinking water guideline on treatability performance and analytical achievability and is not a health-based guideline; WHO's position is that concentrations of lead in drinking water should be as low as reasonably practical.*

Exposure Route	Estimated Median Dose
Diet	Up to 0.4 µg/kgBW/day based on mean dietary exposure from FSA (2006) Total Diet Study
Drinking Water	0.044-0.086 µg/kgBW/day

Source: (COT, 2013)

11.2 Generic Screening Criteria

The following C4SLs for lead were derived by DEFRA using the C4SL methodology for 6% SOM.

Table TN08-23. C4SLs for Lead

Land Use	Soil Guideline Value (mg/kg)
Residential with home-grown produce	200
Residential without home-grown produce	310
Allotment	80
Commercial	2300
Public open space 1	630
Public open space 2	1300

Source: (DEFRA, 2014)

USEPA Regional Screening Levels (RSLs) for lead are presented in the table below.

Table TN08-24. USEPA RSLs for Lead compounds, values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial industrial
Lead Phosphate	USEPA RSL	82	82	380
Lead acetate	USEPA RSL	64	64	270
Lead and compounds	USEPA RSL	400	400	800

(US_EPA, 2019)

12. Isocyanates

12.1 Health Effects and Health Based Guidance Values

The most recent and relevant authoritative reviews of this group of chemicals is from the following sources, although it is notable that data for each chemical in the group is not presented in each of the reference below:

- Toxicological profile for hexamethylene diisocyanate (HDI) (ATSDR, 1998)
- Toxic substances portal - toluene diisocyanate methylenediphenyl diisocyanate (ATSDR, n.d. (b))
- UK National Poisons Information Service. (INCHEM, 1997)
- US National Library of Medicine. (TOXNET, 2018)
- Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)

The data reviewed presents both acute and chronic adverse health effects attributed to exposure to isocyanates.

The isocyanates identified as COPC are:

- Isocyanic acid
- Methyl isocyanate
- Ethyl isocyanate
- Propyl isocyanate
- Phenyl isocyanate
- Hexamethylene diisocyanate
- Toluene-2,4-diisocyanate
- Toluene-2,6-diisocyanate
- Methylene diphenyl diisocyanate
- Isophorone diisocyanate

Of these, a commercially available laboratory analytical method has not been identified for phenyl isocyanate and isophorone diisocyanate.

Acute effects typically relate to irritation of the eyes, nose and throat (ATSDR, 1998), skin, mucous membranes, gastrointestinal problems. respiratory system effects and lacrimation. Contact dermatitis is also reported for some compounds (including phenyl isocyanate). Prolonged exposure may lead to permanent eye damage. Immunological effects are reported for Toluene-2,6-diisocyanate and Toluene-2,4-diisocyanate (TOXNET, 2012).

Chronic effects are primarily reported to be asthma and reduce pulmonary function due to sensitisation from exposure to isocyanates, typically taking months to years to develop (ATSDR, 1998). The INCHEM (1997) reports that toluene-2,4-diisocyanate may result in chronic recurrent episodes of influenza-like illness, associated with pneumonitis or active interstitial lung disease. Skin sensitisation on repeated exposure to toluene diisocyanates may occur as well as ocular: corneal oedema and conjuctival infection (INCHEM, 1997).

Where listed, with the exception of toluene diisocyanates, other isocyanate compounds are not classifiable for their carcinogenicity in humans (Group 3). Toluene diisocyanates are reported to be possibly carcinogenic to humans (Group 2b) (TOXNET, 2012). An excess of lung cancer was seen in some workers at a polyurethane foam manufacturing plant. However, it is not known if exposure to Toluene diisocyanates was the cause. A study in animals orally exposed to Toluene diisocyanates reported increases in tumors in the pancreas, mammary gland, and liver.

The main health effects, relating to toluene diisocyanates and methylenediphenyl diisocyanate retardants and associated exposure doses are summarised below.

Table TN08-25. Summary of ATSDR Reported Effects for toluene diisocyanate and methylenediphenyl diisocyanate

Compound	MRL*	Observed Effect Level
Toluene diisocyanate		
Inhalation	Acute – $1x10^{-15}$ ppm Intermediate – None determinable Chronic – $3x10^{-6}$ ppm	Acute LOAEL – 0.00125 ppm Intermediate - none Chronic mean daily exposure level – 0.0012 ppm
Oral	Acute – none required Intermediate – none required Chronic – none required	Acute LOAEL – n/a Intermediate – n/a Chronic mean daily exposure level – n/a
Methylenediphenyl diisocyanate		

Compound	MRL*	Observed Effect Level
Inhalation	Acute – none determinable Intermediate – None determinable Chronic – 0.001 mg/m^3	Acute LOAEL – none Intermediate - none Chronic BMCL^{HEC} – 0.039 mg/m^3
Oral	Acute – none required Intermediate – none required Chronic – none required	Acute LOAEL – n/a Intermediate – n/a Chronic mean daily exposure level – n/a

(ATSDR, n.d. (b))

*Minimal risk levels (MRLs) are based on noncancer health effects only and so do not consider cancer effects.

12.2 Generic Screening Criteria

There are no UK guideline values for diisocyanates. However, USEPA RSLs are displayed in the table below.

Table TN08-26. USEPA RSLs for diisocyanates, values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial industrial
1,6-Hexamethylene diisocyanate	USEPA RSL	3.1	3.1	13
4,4-Methylene diphenyl diisocyanate	USEPA RSL	850,000	850,000	3,600,000
2,4-Toluene diisocyanate	USEPA RSL	6.4	6.4	27
2,6-Toluene diisocyanate	USEPA RSL	5.3	5.3	22

(US_EPA, 2019)

13. Organo-phosphate ester flame retardants

13.1 Health Effects and Health Based Guidance Values

The most recent and relevant authoritative reviews of this group of chemicals are from the following sources:

- ATSDR, 2012. Toxicological profile for Phosphate Ester Flame Retardants, Atlanta: U.S. Department of Health and Human Services, Public Health Service (ATSDR, 2012)Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)
- US National Library of Medicine. (TOXNET, 2018)

The data reviewed presents both acute and chronic adverse health effects attributed to exposure to phosphorus compounds.

Acute effects from phosphorus compounds are varied dependent on the specific compound. The Compound dependent acute effects include irritation of the respiratory tract, skin (potentially harmful if absorbed), eyes (References listed in Appendix B should be consulted for compound specific information). Some compounds (including triethyl phosphate) report cholinergic symptoms (ATSDR, 2012) and the potential for muscle weakness and pulmonary oedema. The US Library of Medicine (TOXNET, 2018) that there have been a few cases of allergic reactions to consumer products that contain triphenyl phosphate (TPP), but a study that examined several hundred people exposed to

plastics and glues that contained TPP or TCP did not find any allergic reactions. In addition, there was no evidence of toxic effects in workers following exposure to low air levels of triphenyl phosphate over time (TOXNET, 2018).

Chronic effects on humans are not reported in the references reviewed in this Technical Note. In animal studies, which may not be directly translated to human chronic toxicological exposure, for triphenyl phosphate and Triethyl phosphate. (OECD, n.d.), exposure resulted in decreased body weight and increased liver weight (TOXNET, 2018).

Where information is stated in the reviewed documents listed in Table B, phosphorus compounds are not reported to be genotoxic.

The references reviewed state that the majority of phosphorus compounds are not classifiable as human carcinogens. Is it stated that the potential for trioctyl phosphate to cause systemic toxicity, cancer or reproductive and developmental toxicity has not been assessed in humans (TOXNET, 2015).

According to the Agency for Toxic Substances and Disease Registry symptoms (ATSDR, 2012) there is not enough information available to determine with certainty whether or not phosphate ester flame retardants produce cancer in humans. Studies of workers employed in the manufacture of TDCP and TCP did not find significant associations between exposure and cancer. No information was available regarding the carcinogenic potential of the other phosphate esters to humans.

A summary of the MRLs are based on noncancerous health effects only and do not consider carcinogenic effects; data for inhalation was not considered reliable. The main health effects, relating to phosphorus ester flame retardants and associated exposure doses are summarised below.

Table TN08-27. Summary of ATSDR Reported Oral Health Effects for Phosphorus Ester Flame Retardants

Compound	MRL (mg/kg/day)	Observed Effect Level (mg/kg/day)
Tris(2-chloroethyl) phosphate (TCEP)	Acute – none determinable Intermediate – 0.6 Chronic – 0.2	Acute $BMDL_{10}$ - no data Intermediate $BMDL_{10}$ - 60.76 (reproduction) Chronic $BMDL_{10}$ - 23.44 (renal)
Tributyl phosphate (TnBP)	Acute – 1.1 Intermediate – 0.08 Chronic – 0.08	Acute $BMDL_{1SD}$ - 111.47 (reproduction) Intermediate $BMDL_{10}$ - 8.03 (urinary bladder hyperplasia) Chronic $BMDL_{10}$ - 8.03 (urinary bladder hyperplasia)
Tris(2-butoxyethyl) phosphate (TBEP)	Acute – 4.8 Intermediate – 0.09 Chronic – none determinable	Acute $BMDL_{10}$ - 477.25 (reproduction) Intermediate $BMDL_{10}$ - 8.88 (hepatocyte vacuolization) Chronic $BMDL_{10}$: no data
Tris(1,3-dichloro-2-propyl) phosphate (TDCP)	Acute – none determinable Intermediate – 0.05 Chronic – 0.02	Acute $BMDL_{10}$ - no data Intermediate $BMDL_{10}$ - 4.69 (renal) Chronic $BMDL_{10}$ - 1.94 (renal tubule hyperplasia)
Tricresyl phosphate (TCP)	Acute – none determinable Intermediate – 0.04 Chronic – 0.02	Acute $BMDL_{10}$ - none Intermediate $BMDL_{10}$ - 3.72 (reproductive - ovarian lesions) Chronic $BMDL_{10}$ – 2.12 3.72 (reproductive - ovarian lesions)
Triisobutyl phosphate (TiBP)	Acute – none determinable Intermediate – none determinable Chronic – none determinable	Acute $BMDL_{10}$ - none Intermediate $BMDL_{10}$ - none Chronic $BMDL_{10}$ – 2.12 3.72 none
Tri-(2-chloroisopropyl) phosphate (TCPP)	Acute – none determinable Intermediate – none determinable Chronic – none determinable	Acute $BMDL_{10}$ - none Intermediate $BMDL_{10}$ - none Chronic $BMDL_{10}$ – 2.12 3.72 none

13.2 Generic Screening Criteria

There are no UK soil guidance values though there are values provided by the USEPA (RSLs).

Table TN08-28. USEPA RSLs for residential and commercial industrial land use values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Allotment	Commercial industrial	Public Open Space (Resi)	Public Open Space (Parks)
Tributyl phosphate	USEPA RSL	60	60	-	110260	-	-
Tris(2-chloroethyl) phosphate	USEPA RSL	27	27	-	110	-	-
Tris(2-ethylhexyl) phosphate	USEPA RSL	170	170	-	720	-	-
Trimethyl phosphate	USEPA RSL	27	27	-	110	-	-

(US_EPA, 2019)

14. Brominated flame retardants

The most recent and relevant reviews identified by the literature search are:

- ATSDR Toxicological Profile for polybrominated diphenyl ethers (ATSDR, 2017)
- COT Statement on tetrabromobisphenol A - a review of toxicological data, COT Statement 2004/02, London: Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT, 2004)
- COT Statement on polybrominated biphenyls (PBBs) in the infant diet, COT Statement 2015/03, London: Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT, 2015)
- COT Statement on the potential risks from hexabromocyclododecanes (HBCDDs) in the infant diet, COT Statement 2015/02, London.: Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT, 2015)
- COT Statement on the potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet, COT Statement 2015/01, London: Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT, 2015)
- Public Health England's toxicological overview (PHE, 2009)
- US EPA IRIS toxicological reviews for decabromodiphenyl ether, hexabromodiphenyl ether, pentabromodiphenyl ether and tetrabromodiphenyl ether (US EPA, 2008), (US EPA, 2008), (US EPA, 2008), (US EPA, 2008)
- EFSA Scientific Opinion on PBDEs in Food (EFSA, 2011).
- Regional Screening Level (RSL) Summary Table (TR-1E-06, HQ-1) April 2019. (US_EPA, 2019)

The brominated flame retardants identified as chemicals of potential concern (COPC) for this study in AECOM Technical Note 4 are:

- Polybrominated Diphenyl Ethers (PBDEs) [those being analysed for in soil samples are 2,2',4,4',6-pentabromodiphenyl ether, 2,2',3,4,4',5'hexabromodiphenyl ether, 2,2',4,4',5,5'hexabromodiphenyl ether, 2,2',4,4',5,6'-hexabromodiphenyl ether, 2,2',4,4',5,6'-hexabromodiphenyl ether, 2,4,4'-tribromodiphenyl ether, 2,4,4'-tribromodiphenyl ether, 2,2',4,4'-tetrabromodiphenyl ether, 2,3',4,4'-tetrabromodiphenyl ether, 2,2',3,4,4'-pentabromodiphenyl ether, 2,2',4,4',5-pentabromodiphenyl ether, 2,2',3,4,4',5',6-heptabromodiphenyl ether)]

- Polybrominated biphenyls (PBBs) [those being analysed for in soil samples are hexabromobiphenyl (2,2',4,4',5,5'-) (PBB 153), 4,4'-dibromobiphenyl (PBB 15), 2,2',5-tribromobiphenyl (PBB 18), 2,2'-dibromobiphenyl (PBB 4), tetrabromobiphenyl (3,3',5,5'-) (PBB 80)]
- Tetrabromobisphenol A
- Hexabromocyclododecane
- Polybrominated diphenyl ethanes (no commercially available laboratory test method identified and therefore not included further in this review)

14.1 Polybrominated Diphenyl Ethers (PBDEs)

14.1.1 Health Effects and Health Based Guidance Values

There are 209 congeners (individual compounds) that make up PBDEs. The three important commercial mixtures identified by ATSDR are penta-, octa-, and deca-BDEs. Adverse health effects attributed to oral exposure to PBDEs include neurobehavioral and reproductive effects, immune system, thyroid and liver effects. Liver tumours have been seen in animals exposed to very large amounts of decaBDE, and lower-brominated BDEs have not been tested for cancer. IARC therefore classifies PBDEs as Group 3 (not classifiable) due to the inadequacy of the evidence. Similarly, the US EPA assigns Group D (not classifiable) to all BDEs except deca BDE for which it states that there is suggestive evidence of carcinogenic potential.

EFSA (EFSA, 2011) determined that the critical adverse health effect from PBDEs is neurodevelopment, deriving a benchmark doses (BMDL10) for a number of individual congeners (summarised below). Due to the uncertainties in the database, EFSA chose not to derive health-based guidance values.

Table TN08-29. EFSA Benchmark Doses for PBDE Congeners

PBDE Congener	Animal BMDL10 (µg/kgBW/day)	Human body-burden at BMDL10 (µg/kgBW/day)
TetraBDE (BDE-47)	309	232
PentaBDE (BDE-99)	12	9
HexaBDE (BDE-153)	83	62
DecaBDE (BDE-209)	1700	N/A

ATSDR derived minimal risk levels (MRLs) for PBDEs are listed below. Please note that although ATSDR findings are included the USA have far higher BDE47 and BDE99 (penta commercial product) usage, and therefore exposure than in the UK.

Table TN08-30. ATSDR Minimal Risk Levels for PBDEs for Lower Brominated PBDEs (mono-nona)

MRL type	MRL	Toxicological Basis
Inhalation:		
Acute	Not derived	Insufficient evidence
Intermediate	0.006mg/m3	Animal study - NOAEL of 1.1mg/m3 based on thyroid effects from exposure to octaBDE
Chronic	Not derived	Insufficient evidence
Oral:		
Acute	0.06µg/kgBW/day	Animal study - LOAEL of 6µg/kgBW/day based on endocrine, reproductive and neurobehavioural effects from exposure to pentaBDE
Intermediate	0.003µg/kgBW/day	Animal study - LOAEL of 1µg/kgBW/day based on reproductive

MRL type	MRL	Toxicological Basis
		effects from exposure to tetraBDE
Chronic	Not derived	Insufficient evidence

Table TN08-31. ATSDR Minimal Risk Levels for PBDEs for DecaBDE

MRL type	MRL	Toxicological Basis
Inhalation	None derived	Insufficient evidence
Oral:		
Acute	10µg/kgBW/day	Animal study - NOAEL of 1340µg/kgBW/day based on neurobehavioural effects
Intermediate	0.2µg/kgBW/day	Animal study - LOAEL of 50µg/kgBW/day based on blood glucose effects that might be associated with pancreatic toxicity. The relevance to humans is uncertain
Chronic	Not derived	Insufficient evidence

In deriving the health-based guidelines, ATSDR acknowledges that the MRLs are based on the toxicity studies of commercial mixtures or individual congeners, and not on environmental mixtures which may have a higher or lower toxicity or potency compared to the test compound.

Reported mean upper bound concentrations in food surveyed by EFSA ranged from 0.00004-0.00398µg/g for individual congeners, with estimated average dietary intakes varying from 0.00042-0.0138 µg/kgBW/day for individual congeners. Exposure from PBDEs in house dust and cars is also highlighted as an important additional source of exposure, particularly for young children (estimated to be 0.0005-0.08µg/kgBW/day).

ATSDR references US EPA studies on population exposure, with estimated exposures of 0.0472 µg/kgBW/day for 1-5yr olds, 0.013 µg/kgBW/day for 6-11 yr olds, 0.0083 µg/kgBW/day for 12-19 yr olds and 0.0071 µg/kgBW/day for adults. The higher dose for young children was attributed to higher soil/dust ingestion, and it was estimated that 90% of the reported exposures resulted from house dust inhalation or dermal exposure.

PHE's review of PBDE's was undertaken in 2009, and the primary reference sources were an earlier ATSDR Toxicological Profile for PBBs and PBDE's in 2004, and earlier evaluations by IPCS, IARC, the European Chemicals Bureau and the UK Committee on Toxicity of Chemicals in Food Consumer Products and the Environment.

US EPA IRIS evaluations for p-bromodiphenyl ether, decabromodiphenyl ether, dibromodiphenyl ether, hexabromodiphenyl ether, nonabromodiphenyl ether, octabromodiphenyl ether, pentadiphenyl ether, tetrabromodiphenyl ether and tribromodipehnylether are summarised below:

Table TN08-32. US EPA IRIS Entries for PDBEs

Compound	Date of Evaluation by US EPA	Health-based Guideline	Critical Toxic Effect
p-bromodiphenyl ether	1990	Not evaluated (insufficient evidence)	Not applicable
decabromodiphenyl ether (BDE209)	2008	Oral RfD 7 µg/kgBW/day	Neurobehavioral effects; NOAEL 2,220 µg/kgBW/day
		Inhalation RfC not derived (insufficient evidence)	Not applicable
		Suggestive evidence of carcinogenic potential via oral	Liver tumours

Compound	Date of Evaluation by US EPA	Health-based Guideline	Critical Toxic Effect
		route – slope factor 0.7 per µg/kgBW/day	
dibromodiphenyl ether	1990	Not evaluated (insufficient evidence)	Not applicable
hexabromodiphenyl ether (BDE153)	2008	Oral RfD 0.2 µg/kgBW/day Inhalation RfC not derived (insufficient evidence)	Neurobehavioral effects; NOAEL 450 µg/kgBW/day Not applicable
nonabromodiphenyl ether	1990	Not evaluated (insufficient evidence)	Not applicable
octabromodiphenyl ether	1987	Oral RfD 3 µg/kgBW/day Inhalation RfC not derived (insufficient evidence)	Liver histopathology; NOAEL 2,510 µg/kgBW/day Not applicable
pentabromodiphenyl ether (BDE99)	2008	Oral RfD 0.1 µg/kgBW/day Inhalation RfC not derived (insufficient evidence)	Neurobehavioral effects; BMDL(1SD) 290 µg/kgBW/day Not applicable
tetrabromodiphenyl ether (BDE47)	2008	Oral RfD 0.1 µg/kgBW/day Inhalation RfC not derived (insufficient evidence)	Neurobehavioral effects; BMDL(1SD) 350 µg/kgBW/day Not applicable
tribromodiphenylether (BDE28)	1990	Not evaluated (insufficient evidence)	Not applicable

COT evaluated the potential risks from PBDEs in the diet of UK infants in 2015 (COT, 2015). COT concluded that there was insufficient evidence to establish health-based guidelines. The benchmark doses derived by EFSA were used for the evaluation with the exception that COT extended the body burden calculations to include BDE-209 (BB $BMDL_{10}$ of 425 µg/kgBW/day).

COT, in their statement, include data on concentrations of PBDEs in air, household dust and dietary foods. The subsequent estimations of environmental and dietary exposures are dominated by BDE-209, with mean dust exposure of 2.7 µg/kgBW/day for BDE-209, mean air exposure of up to 0.000012 µg/kgBW/day for the sum of BDEs 28, 47, 49, 66, 99, 100, 153 and 154, and mean dietary exposure up to 0.00671 µg/kgBW/day.

14.1.2 Generic Screening Criteria

The USEPA RSLs for BDE-47, BDE-99 and BDE-153 are displayed in Table TN08-33 below.

Table TN08-33. USEPA RSLs for residential and commercial industrial land use, values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial industrial
Tetrabromodiphenyl ether, 2,2'4,4' (BDE 47)	USEPA RSL	6.3	6.3	82
Pentabromodiphenyl ether, 2,2',4,4',5 (BDE 99)	USEPA RSL	6.3	6.3	82

Hexabromodiphenyl ether, 2,2',4,4',5,5' (BDE 153)	USEPA RSL	13	13	160

(US_EPA, 2019)

14.2 Polybrominated Biphenyls (PBBs)

14.2.1 Health Effects and Health Based Guidance Values

The primary sources of information for polybrominated biphenyl compounds (PBBs) identified by the literature search are the published peer review undertaken by ATSDR (ATSDR, 2004) and the scientific opinion published by EFSA (EFSA, 2010). Adverse health effects attributed to oral exposure to PBBs include skin disorders (such as acne and hair loss), body weight loss, nervous system effects, and liver, kidney, thyroid and immune system injury. Birth defects have also been seen in animals exposed to PBBs. The National Toxicology Program of the US Department of Health and Human Studies has determined that PBBs may reasonably be anticipated to be carcinogenic based on the evidence of liver tumours in animals. The US EPA has not classified PBBs are carcinogens, but IARC has also determined that PBBs are possibly carcinogenic to humans (Group 2B). Dermal and ocular effects have bene noted from dermal studies, and no significant adverse effects have been noted in the limited number of inhalation studies reviewed. A summary of the main health effects and associated exposure doses identified by ATSDR are summarised below:

Table TN08-34. Summary of ATSDR Reported Health Effects for PBBs

Health Effect	Study type	Observed Effect Level
Thyroid	Acute (oral) – animal	NOAEL 1mg/kgBW/day LOAEL 3mg/kgBW/day
	Intermediate (oral) – animal	LOAEL 0.05mg/kgBW/day
Hepatic (liver)	Acute (oral) - animal	NOAEL 1mg/kgBW/day LOAEL 3mg/kgBW/day
	Intermediate (oral) – animal	LOAEL 0.05mg/kgBW/day
Immune System	No reliable data	
Neurological effects	Intermediate (oral) - animal	LOAEL 0.2mg/kgBW/day
Dermal and ocular (skin and eyes)	Acute (dermal) – animal	Direct exposure likely to be irritating to eyes and skin
Reproductive effects	Intermediate (oral) - animal	LOAEL 0.012mg/kgBW/day
Developmental effects	Intermediate (oral) - animal	LOAEL 0.012mg/kgBW/day
Cancer	Oral - animal	Sufficient evidence in animals for liver tumours but insufficient evidence in humans. Insufficient evidence about which constituents of the PBB mixtures are carcinogenic, or what the carcinogenic mechanism is.

The ASTDR concluded that there was insufficient evidence to derive inhalation guidelines (minimal risk levels (MRL)) for PBBs. For oral exposure a MRL of 0.01mg/kgBW/day was set for acute exposure (less than 14 days exposure) based on thyroid effects. Intermediate and chronic exposure MRLs were not derived because of the seriousness of the developmental and neurological effects seen at the lowest test dose in experiments (0.012mg/kgBW/day) (i.e. the lack of evidence for doses associated with either less serious or no adverse effects).

The European Food Safety Authority published its scientific opinion on PBBs in food in 2010 (EFSA, 2010). EFSA's CONTAM Panel selected liver carcinogenic effects as the critical effect; proposing a no observed effect level (NOEL) 0.15mg/kgBW/day. However, since the toxicity study was based on a

technical mixture of PBB not found in food, it was concluded that it was inappropriate to use this NOEL to derive a health-based guidance value. Highest reported European dietary exposure was approximately 5-6 orders of magnitude lower than this NOEL (0.15-1.4ng/kgBW/day) and therefore the risk from exposure through diet was considered of no concern.

COT evaluated the potential risks from PBBs in the diet of UK infants in 2015 (COT, 2015). COT concluded that the key toxicological effect is liver carcinogenicity and that planar and non-planar PBBs need separate consideration with planar PBBs expected to behave as per dioxins. It was also considered that the 2005 WHO-TEFs for PCBs could be conservatively assigned to PBBs in order to compare EQ values to the TDI for dioxins. Insufficient toxicological data was available for non-planar PBBs, and insufficient data was available for the occurrence of PBBs in the environment and foodstuffs for a meaningful assessment to be made.

14.2.2 Generic Screening Criteria

The USEPA RSL for PBBs- is displayed in Table TN08-35 below.

Table TN08-35. USEPA RSLs for residential and commercial industrial land use, values in mg/kg

Chemical	Source	Residential with Plant uptake	Residential without plant uptake	Commercial industrial
Polybrominated Biphenyls	USEPA RSL	0.018	0.018	0.077

(US_EPA, 2019)

14.3 Tetrabromobisphenol A

14.3.1 Health Effects and Health Based Guidance Values

The only authoritative reviews identified by the literature search are those undertaken by EFSA in 2011 (EFSA, 2011 (amended in December 2013)) and COT in 2004 (COT, 2004).

EFSA developed a benchmark dose (BMDL10) of 16mg/kgBW/day based on the main toxicological effect - changes in thyroid hormone homeostasis. TBBPA was not found to be genotoxic. Due to limitations and uncertainties in the database, EFSA did not derive a health-based guideline value.

EFSA based its estimates on dietary exposure on information covering the period 2003-2010 from four countries (UK, Ireland, Norway and Spain). All analytical results were returned below the limit of quantification (typically <1ng/g). Therefore, a meaningful estimate of dietary exposure is not possible.

COT in 2004 advocated a TDI of 1mg/kgBW/day based on a NOAEL of 1000mg/kgBW/day from an animal reproductive toxicity study and noted that TBBPA has a low acute toxicity.

There are no UK or other international guideline values identified in this study.

14.3.2 Generic Screening Criteria

No GSCs for tetrabromobisphenol A were identified in this study.

14.4 Hexabromocyclododecane

14.4.1 Health Effects and Health Based Guidance Values

Two reviews have been identified by the literature search, one by EFSA in 2011 and one by the US EPA at the same time. The IRIS assessment programme for this compound has however been discontinued. A Preliminary Assessment Materials Report was undertaken in 2011 and released in 2014. The EPA website states that the Preliminary Assessment Material Report contains the draft literature searches and associated search strategies, evidence tables, and exposure response arrays for the purpose of obtaining input from stakeholders and the public prior to developing the draft IRIS

assessment. The report therefore does not include the EPA's evaluation of the data obtained from the literature searches and has not been reviewed further for the purposes of this Evidence Review.

EFSA reviewed the presence of HBCDDs in food in 2011 (EFSA, 2011). The EFSA review focused on three stereo isomers of HBCDD (α, β and γ) and identified that the main adverse health effects were associated with the liver, thyroid, reproduction, nervous system and immune system. HBCDDs were not found to be genotoxic, and the critical endpoint was identified as neurobehavioral effects; a body burden benchmark dose ($BMDL_{10}$) of 0.79mg/kgBW/day established from the toxicological evidence. However, EFSA concluded that it was inappropriate to derive a health-based guideline since the elimination of HBCDDs in animals and humans is expected to differ.

EFSA identified that mean dietary exposure to HBCDD across European countries varied from 0.15-1.85ng/kgBW/day for children from the ages of 3-10 years old and was approximately half that for adults (0.09-0.99ng/kgBW/day).

In addition, COT evaluated the potential risks from HBCDDs in infant diets in 2015 (COT, 2015). COT utilises the EFSA (2011) toxicological evaluation, quoting a $BMDL_{10}$ of 0.93mg/kgBW/day and a human body burden equivalent of 3µg/kgBW/day for neurodevelopmental effects. It is noted that the $BMDL_{10}$ values are not the same as those quoted in the EFSA Journal article referenced above. EFSA used an 85% oral absorption correction for rodents to convert the $BMDL_{10}$ of 0.93mg/kgBW/day to body burden equivalent of 0.79mg/kgBW/day.

COT presents concentrations of HBCDDs in air, dust and food; indoor air concentrations ranging from 67-1300pg/m^3 in homes in Birmingham, dust concentrations in the same homes ranging from 140-140000ug/kg, and food concentrations measured in the 2012 FSA Total Diet Study mostly being lower than the limits of detection (<0.01-<0.1µg/kg). No data for soil and drinking water was available. Average exposure for infants to HBCDD in dust was estimated to be104ng/kgBW/day, and in food 3.24-3.81ng/kgBW/day.

There are no UK or other international guideline values identified in this study.

14.4.2 Generic Screening Criteria

No GSCs for tetrabromobisphenol A were identified in this study.

15. Conclusions

Based on the evidence reviewed the following categories of health-based guideline value (HBGV) availability have been determined:

COPC with published UK-specific health-based guideline values or values adopted by UK authorities are:

- PAHs
- Benzene
- Lead
- PBDEs
- PBBs
- TBBPA
- HBCDD
- Dioxins, furans and dioxin-like biphenyls (chlorinated, brominated and mixed halogenated)

COPC with published peer reviewed international health-based guidelines are:

- Asbestos
- Non-dioxin-like PCBs
- Cyanide
- Synthetic vitreous fibres (machine-made mineral fibres)
- Isocyanates (data limited to inhalation HBGVs) Toluene-2,4-diisocyanate and Toluene-2,6-diisocyanate, hexamethylene diisocyanate and methylene diphenyl diisocyanate
- Phosphate esters (organo-phosphate flame retardants) – not including those with available test methods for soil (see below)

COPC where no published peer reviewed health-based guidelines have been found are:

- Isocyanic acid
- Methyl isocyanate
- Ethyl isocyanate
- Propyl isocyanate
- Phenyl isocyanate
- Isophorone diisocyanate
- tris(1-chloro-2-propyl) phosphate (TCPP)
- Tris(2-ethylhexyl) phosphate

Appendix B provides a detailed summary of the available information for each compound based on the evidence review undertaken which is too extensive to summarise here.

Where no health-based guidance values or generic screening criteria have been identified further toxicological assessment would be required to develop appropriate thresholds. This could involve derivation of an HCV in accordance with the EA SR2 guidance.

The level of evidence identified for each compound or group of compounds is variable. All are associated with adverse health effects that could meet the Part 2A definition of “significant harm” should viable exposure scenarios exist.

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Appendix TN08-A: Web Search

Search number	Date of Search	Search tool/origin or other tracing information	Hyperlink to origin (URL)	Benzene	PAHs	PCDD/Fs	PBDD/Fs	PCBs	PBBs	Phosphate Esters	PBDEs	HBCCDs	TBBPA	Isocyanates	Asbestos	SVFs	Cyanides	Lead
1		ATSDR ToxFAQs	https://www.atsdr.cdc.gov/substances/indexAZ.asp	X	X	X		X	X	X	X			X	X	X	X	X
2		ATSDR ToxGuide	https://www.atsdr.cdc.gov/substances/indexAZ.asp	X	X	X		X	X	X	X			X	X	X	X	X
3		ATSDR Tox. Profile	https://www.atsdr.cdc.gov/substances/indexAZ.asp	X	X	X		X	X	X	X			X	X	X	X	X
4		EFSA Scientific Opinions	http://www.efsa.europa.eu/en/publications?f%5B0%5D=im_field_subject%3A61826		X				X		X	X	X				X	
5		COT Statements	https://cot.food.gov.uk/committee/committee-on-toxicity/cotstatements			X	X		X		X	X	X					X
6		COC Statements	https://www.gov.uk/government/collections/coc-guidance-statements and https://webarchive.nationalarchives.gov.uk/*/http:/www.iacoc.org.uk/															
7		COM Statements	https://www.gov.uk/government/organisations/committee-on-mutagenicity-of-chemicals-in-food-consumer-products-and-the-environment															
8		US EPA IRIS Tox. Review	https://cfpub.epa.gov/ncea/iris_drafts/atoz.cfm?list_type=alpha or	X	X		X	X			X	X		X	X		X	X
9		WHO EHCs	http://www.inchem.org/	X		X		X	X	X			X	X				X

Search number	Date of Search	Search tool/origin or other tracing information	Hyperlink to origin (URL)	Benzene	PAHs	PCDD/Fs	PBDD/Fs	PCBs	PBBs	Phosphate Esters	PBDEs	HBCCDs	TBBPA	Isocyanates	Asbestos	SVFs	Cyanides	Lead
10		LQM S4ULs	Hard copy only	X	X													
11		Defra SP1010 (C4SLs)	http://sciencesearch.defra.gov.uk/Default.aspx?Location=None&Module=FilterSearchNewLook&Completed=0	X	X													X
12		EA SGV reports	https://www.claire.co.uk/useful-government-legislation-and-guidance-by-country/209-assessing-risks-to-human-health-info-ra2-2	X		X												X
13		TOXNET	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	X	X	X	X	X	X	X	X			X	X	X	X	X
14		PHE CoCH	https://www.gov.uk/government/collections/chemical-hazards-compendium	X	X	X					X			X	X		X	X

Appendix TN08-B: Summary of Evidence Identified

Evidence Number	Evidence Reference	Evidence hyperlink (if available)	Evidence Type	Corresponding search number	Brief summary of evidence available from source	
1	Benzene Agency for Toxic Substances and Disease Registry. 2007	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=14	Report	2+3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
2	Toxicological profile for Naphthalene, 1-Methylnapthalene, 2-Methylnapthalene. 2005. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/toxprofiles/tp67.pdf	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
3	Toxicological profile for Polycyclic Aromatic Hydrocarbons (PAHs). 1995. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=25	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
4	Toxicological profile for Chlorinated Dibenzo-p-dioxins (CDDs). 1998. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=63	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
5	Toxicological profile for Chlorodibenzofurans (CDFs). 1994. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=194	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
6	Toxicological profile for Polychlorinated Biphenyls (PCBs). 2000. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=26	Report	3	Toxicity review	Y
					Population exposure review	N

					HBGV developed	Y
					Soil-based GVs developed	N
7	Toxicological profile for Polybrominated Biphenyls (PBBs). 2004. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=94	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
8	Toxicological profile for Phosphate Ester Flame Retardants. 2012. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=239	Report	2+3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
9	Toxicological profile for Polybrominated Diphenyl Ethers (PBDEs). 2017. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=183	Report	2+3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
10	Toxicological profile for Hexamethylene Diisocyanate (HDI). 1998. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=170	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
11	Toxicological profile for Toulene Diisocyanate Methylenediphenyl Diisocyanate. 2018. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=245	Report	2+3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
12	Toxicological profile for Asbestos. 2001. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=4	Report	3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
13	Toxicological profile for Synthetic Vitreous Fibers. 2004. Agency for Toxic Substances and Disease	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid	Report	3	Toxicity review	Y

	Registry	=185			Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
14	Toxicological profile for Cyanide. 2006. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=19	Report	2+3	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
15	Toxicological profile for Lead. 2019. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=22	Report	2	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
16	Polycyclic Aromatic Hydrocarbons in Food Scientific Opinion of the Panel on Contaminants in the Food Chain. 2008. Jan Alexander, Diane Benford, Andrew Cockburn, Jean-Pierre Cravedi, Eugenia Dogliotti, Alessandro Di Domenico, María Luisa Fernández-Cruz, Johanna Fink-Gremmels, Peter Fürst, Corrado Galli, Philippe Grandjean, Jadwiga Gzyl, Gerhard Heinemeyer, Niklas Johansson, Antonio Mutti, Josef Schlatter, Rolaf van Leeuwen, Carlos Van Peteghem, Philippe Verger.	http://www.efsa.europa.eu/sites/default/files/scientific_output/files/main_documents/724.pdf	Report	4	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
17	Risk for animal and human health related to the presence of dioxins and dioxin□like PCBs in feed and food. 2018. EFSA Panel on Contaminants in the Food Chain (CONTAM) Helle Katrine Knutsen Jan Alexander Lars Barregård Margherita Bignami Beat Brüschweiler Sandra Ceccatelli Bruce Cottrill Michael Dinovi Lutz Edler Bettina Grasl□Kraupp Christer Hogstrand Carlo Stefano Nebbia Isabelle P Oswald Annette Petersen Martin Rose Alain□Claude Roudot Tanja Schwerdtle Christiane Vleminckx Günter Vollmer Heather Wallace Peter Fürst Helen Håkansson Thorhallur Halldorsson Anne□Katrine Lundebye Raimo Pohjanvirta Lars Rylander Andrew Smith Henk van Loveren Ine Waalkens□Berendsen Marco Zeilmaker Marco Binaglia José Ángel Gómez Ruiz Zsuzsanna Horváth Eugen Christoph	https://efsa.onlinelibrary.wiley.com/doi/10.2903/j.efsa.2018.5333	Report	4	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N

	Laura Ciccolallo Luisa Ramos Bordajandi Hans Steinkellner Laurentius (Ron) Hoogenboom				
18	Opinion of the scientific panel on contaminants in the food chain on a request for the Commission related to the presence of non dioxin-like polychlorinated biphenyls (PCB) in feed and food. 2005. Jan Alexander, Herman Autrup, Denis Bard, Angelo Carere, Lucio Guido Costa, Jean□Pierre Cravedi, Alessandro Di Domenico, Roberto Fanelli, Johanna Fink□Gremmels, John Gilbert, Philippe Grandjean, Niklas Johansson, Agneta Oskarsson, Andrew Renwick, Jirí Ruprich, Josef Schlatter, Greet Schoeters, Dieter Schrenk, Rolaf van Leeuwen, Philippe Verger	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2005.284	Report	4	Toxicity review: Y Population exposure review: Y HBGV developed: Y Soil-based GVs developed: N
19	Scientific Opinion on Polybrominated Biphenyls (PBBs) in Food. 2010. European Food Safety Authority (EFSA), Parma, Italy	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2010.1789	Report	4	Toxicity review: Y Population exposure review: Y HBGV developed: N Soil-based GVs developed: N
20	Scientific Opinion on Tetrabromobisphenol A (TBBPA) and its derivatives in food. 2011. European Food Safety Authority (EFSA), Parma, Italy	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2011.2477	Report	4	Toxicity review: Y Population exposure review: Y HBGV developed: Y Soil-based GVs developed: N
21	Scientific Opinion on Polybrominated Diphenyl Ethers (PBDEs) in Food. 2011. European Food Safety Authority (EFSA), Parma, Italy	https://efsa.onlinelibrary.wiley.com/doi/pdf/10.2903/j.efsa.2011.2156	Report	4	Toxicity review: Y Population exposure review: N HBGV developed: N Soil-based GVs developed: N
22	Scientific Opinion on Hexabromocyclododecanes (HBCDDs) in Food. 2011. European Food Safety Authority (EFSA), Parma, Italy	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2011.2296	Report	4	Toxicity review: Y Population exposure review: N HBGV developed: Y Soil-based GVs developed: N
23	Evaluation of the health risks related to the presence of cyanogenic glycosides in foods other than raw apricot kernels. 2019.EFSA Panel on Contaminants in the Food Chain (CONTAM),	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2019.5662	Report	4	Toxicity review: Y Population exposure review: N HBGV developed: N

	Dieter Schrenk, Margherita Bignami, Laurent Bodin, James Kevin Chipman, Jesus del Mazo, Bettina Grasl-Kraupp, Christer Hogstrand, Laurentius (Ron) Hoogenboom, Jean-Charles Leblanc, Carlo Stefano Nebbia, Elsa Nielsen, Evangelia Ntzani, Annette Petersen, Salomon Sand, Christiane Vleminckx, Heather Wallace, Diane Benford, Leon Brimer, Francesca Romana Mancini, Manfred Metzler, Barbara Viviani, Andrea Altieri, Davide Arcella, Hans Steinkellner and Tanja Schwerdtle				Soil-based GVs developed	N
24	Scientific Opinion on Lead in Food. 2013. European Food Safety Authority (EFSA), Parma, Italy	https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2010.1570	Report	4	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
25	COT STATEMENT ON OCCURRENCE OF MIXED HALOGENATED DIOXINS AND BIPHENYLS IN UK FOOD. 2010. Committee on toxicity of chemicals in food, consumer products and the environment	https://cot.food.gov.uk/sites/default/files/cot/cotstatementhalogenatedioxins201002.pdf	Report	5	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
26	Statement on polybrominated biphenyls (PBBs) in the infant diet. 2015. Committee on toxicity of chemicals in food, consumer products and the environment	https://cot.food.gov.uk/sites/default/files/pbbstatementfinal.pdf	Report	5	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
27	Statement on the potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet. 2015. Committee on toxicity of chemicals in food, consumer products and the environment	https://cot.food.gov.uk/sites/default/files/PBDEstatementfinal.pdf	Report	5	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
28	COT statement on tetrabromobisphenol A - Review of toxicological data. 2004. Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment	https://cot.food.gov.uk/sites/default/files/cot/cotstatements04tbbpa.pdf	Report	5	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
29	Statement on the potential risks from	https://cot.food.gov.uk/sites/def	Report	5	Toxicity review	Y

	Hexabromocyclododecanes (HBCDDs) in the infant diet. 2015. Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment	ault/files/HBCDDsstatementfinal.pdf			Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
30	Statement on the potential risks from lead in the infant diet. 2013. Committee on toxicity of chemicals in food, consumer products and the environment	https://cot.food.gov.uk/sites/default/files/cot/cotstatlead.pdf	Report	5	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
31	Toxicological review of benzene. 2002. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0276tr.pdf	Report	8	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
32	Acenaphthylene; CASRN 208-96-8. 1991. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0443_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
33	Anthracene CASRN 120-12-7. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0434_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
34	Benz[a]anthracene CASRN 56-55-3. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0454_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
35	Benzo[b]fluoranthene CASRN 205-99-2. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0453_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N

36	Benzo[g,h,i]perylene CASRN 191-24-2. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0461_sum8mary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
37	Chrysene CASRN 218-01-9. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0455_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
38	Dibenz[a,h]anthracene CASRN 53-70-3. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0456_summary.pdf#namedest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
39	Fluoranthene CASRN 206-44-0. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0444_summary.pdf#namedest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
40	Fluorene CASRN 86-73-7. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0435_summary.pdf#namedest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
41	Indeno[1,2,3-cd]pyrene CASRN 193-39-5. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0457_summary.pdf#namedest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
42	Naphthalene CASRN 91-20-3. 1998. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0436_summary.pdf#namedest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y

					Soil-based GVs developed	N
43	Pyrene CASRN 129-00-0.1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0445_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
44	Brominated dibenzofurans CASRN NA. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0514_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
45	2,3,7,8-Tetrachlorodibenzo-p-dioxin CASRN 1746-01-6. 2012. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/1024_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
46	Aroclor 1016 CASRN 12674-11-2. 1993. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0649_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
47	**Aroclor 1248 CASRN 12672-29-6. 1994.** USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0649_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
48	Aroclor 1254 CASRN 11097-69-1. 1994. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0389_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
49	Polychlorinated biphenyls (PCBs) CASRN 1336-36-3. 1994 USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0294_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N

					HBGV developed	Y
					Soil-based GVs developed	N
50	2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl ether (BDE-209) CASRN 1163-19-5. 2008. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0035_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
51	Hexabromodiphenyl ether CASRN 36483-60-0. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0494_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
52	2,2',4,4',5,5'-Hexabromodiphenyl ether (BDE-153) CASRN 68631-49-2. 2008. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/1009_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
53	p,p'-Dibromodiphenyl ether. CASRN 2050-47-7. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0491_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
54	Octabromodiphenyl ether CASRN 32536-52-0. 1987. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0180_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
55	Pentabromodiphenyl ether CASRN 32534-81-9. 1987. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0184_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
56	2,2',4,4',5-Pentabromodiphenyl ether (BDE-99) CASRN 60348-60-9. 2008. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/sub	Report	8	Toxicity review	N

		st/1008_summary.pdf#nameddest=rfd			Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
57	2,2',4,4'-Tetrabromodiphenyl ether (BDE-47) CASRN 5436-43-1. 2008. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/1010_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
58	Tetrabromodiphenyl ether CASRN 40088-47-9. 1990. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0493_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
59	1,6-Hexamethylene diisocyanate CASRN 822-06-0. 1994. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0638_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
60	Methyl isocyanate CASRN 624-83-9. ND. USEPA	https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?&substance_nmbr=527	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
61	Methylene Diphenyl Diisocyanate (monomeric methylenediphenyl diisocyanate) and polymeric methylenediphenyl diisocyanate (PMDI) CASRN 101-68-8, 9016-87-9. 1998. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0529_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
62	2,4-/2,6-Toluene diisocyanate mixture (TDI) CASRN 26471-62-5. 1995. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0503_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N

63	Asbestos CASRN 1332-21-4. 1988. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0371_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
64	Barium cyanide CASRN 542-62-1. ND.USEPA	https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?&substance_nmbr=9	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
65	Calcium cyanide CASRN 592-01-8. 2010. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
66	Chlorine cyanide CASRN 506-77-4. 1987. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0024_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
67	Copper cyanide CASRN 544-92-3. 1988. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0029_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
68	Cyanide, free CASRN 57-12-5. 2010. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
69	Hydrogen Cyanide and Cyanide Salts CASRN Various. 2010. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y

					Soil-based GVs developed	N
70	Potassium cyanide CASRN 151-50-8. 2010. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
71	Potassium silver cyanide CASRN 506-61-6. 2010. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
72	Silver cyanide CASRN 506-64-9. 1987. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0100_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
73	Sodium cyanide CASRN 143-33-9. 2010. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
74	Zinc cyanide CASRN 557-21-1. 1987. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0127_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
75	Lead and compounds (inorganic) CASRN 7439-92-1. 2004. USEPA	https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0277_summary.pdf#nameddest=rfd	Report	8	Toxicity review	N
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
76	International Agency for Research on Cancer (IARC) - Summaries & Evaluations: Benzene (Group 1). 1987. IARC.	http://www.inchem.org/documents/iarc/suppl7/benzene.html	Report	9	Toxicity review	Y
					Population exposure review	Y

					HBGV developed	N
					Soil-based GVs developed	N
77	Environmental Health Criteria 150: Benzene. 1993. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc150.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
78	Benzene: Poisons Information Monogaph 63. 1993. IPCS.	http://www.inchem.org/documents/pims/chemical/pim063.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
79	Benzene. 2016. ILO & WHO.	http://www.inchem.org/documents/icsc/icsc/eics0015.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
80	Environmental Health Criteria 202: Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons. 1998. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc202.htm#SectionNumber:1.1	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
81	WHO Food Additives Series 48 Safety evaluation of certain food additives and contaminants: Polychlorinated dibenzodioxins, polychlorinated dibenzofurans and coplanar polychlorinated biphenyls. 2002. Canady *et al.*	http://www.inchem.org/documents/jecfa/jecmono/v48je20.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
82	Environmental Health Criteria 88: Polychlorinated dibenzo-para-dioxins and dibenzofurans. 1989. IPCS.	http://www.inchem.org/documents/ehc/ehc/ehc88.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
83	IARC Summaries & Evaluations: Polychlorinated dibenzo-para-dioxins.1997. IARC.	http://www.inchem.org/documents/iarc/vol69/dioxin.html	Report	9	Toxicity review	Y

					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
84	Environmental Health Criteria 140: Polychlorinated biphenyls and terphenyls (Second Edition). 1993. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc140.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
85	Polychlorinated Biphenyls (PCBs) and Polychlorinated Terphenyls (PCTs): Health and Safety Guide. 1992. WHO.	http://www.inchem.org/documents/hsg/hsg/hsg68.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
86	Concise International Chemical Assessment Document 55 Polychlorinated Biphenyls: Human Health Aspects. 2003. WHO.	http://www.inchem.org/documents/cicads/cicads/cicad55.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
87	Environmental Health Criteria 152: Polybrominated Biphenyls. 1994. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc152.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
88	International Agency for Research on Cancer (IARC) - Summaries & Evaluations: Polybrominated Biphenyls (Group 2B). 1987. IARC.	http://www.inchem.org/documents/iarc/suppl7/polybrominated biphenyls.html	Report	9	Toxicity review	N
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
89	SIDS Initial Assessment Report for SIAM 15: Triphenyl phosphate. 2002. OECD SIDS.	http://www.inchem.org/documents/sids/sids/115866.pdf	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N

90	Environmental Health Criteria 111: Triphenyl phosphate. 1991. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc111.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
91	Triphenyl phosphate. 2000. IPCS.	http://www.inchem.org/documents/icsc/icsc/eics1062.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
92	SIDS Initial Assessment Profile: Triethyl phosphate. OECD SIDS.	http://www.inchem.org/documents/sids/sids/78400.pdf	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
93	Environmental Health Criteria 162: Brominated diphenyl ethers. 1994. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc162.htm#PartNumber:8	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
94	Environmental Health Criteria 172: Tetrabromobisphenol A and derivatives. 1995. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc172.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
95	Concise International Chemical Assessment Document 27: Diphenylmethane diisocyanate 2000. WHO.	http://www.inchem.org/documents/cicads/cicads/cicad27.htm	Report	9	Toxicity review	N
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
96	Toluene-2,4-diisocyanate. 1997. IPCS.	http://www.inchem.org/documents/pims/chemical/pim534.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N

					Soil-based GVs developed	N
97	Environmental Health Criteria 75: Toluene diisocyanates. 1987. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc75.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
98	Monograph for UKPID: toluene diisocyanate. UK National Poisons Information Service.	http://www.inchem.org/documents/ukpids/ukpids/ukpid30.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
99	Hexamethylene Diisocyanate: SIDS Initial Assessment Report for 12th SIAM. 2001. OCED SIDS.	http://www.inchem.org/documents/sids/sids/822060.pdf	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
100	Environmental Health Criteria 203: Chrysotile Asbestos. 1998. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc203.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
101	Environmental Health Criteria 53: Asbestos and other Natural Mineral Fibres. 1986. WHO.	http://www.inchem.org/documents/ehc/ehc/ehc53.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
102	IARC Summaries & Evaluations: Asbestos (Actinolite, amosite, anthophyllite, chrysotile, crocidolite, tremolite) (Group 1). 1998. IARC.	http://www.inchem.org/documents/iarc/suppl7/asbestos.html	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
103	Concise International Chemical Assessment Document 61 Hydrogen Cyanide and Cyanides: Human Health Aspects. 2004. WHO.	http://www.inchem.org/documents/cicads/cicads/cicad61.htm	Report	9	Toxicity review	Y
					Population exposure review	N

					HBGV developed	Y
					Soil-based GVs developed	N
104	Cyanides: International Programme on Chemical Safety Poisons Information Monograph. 1991. IPCS.	http://www.inchem.org/documents/pims/chemical/pimg003.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
105	IPCS/CEC Evaluation of Antidotes Series: Volume 2 Antidotes For Poisoning By Cyanide. 1993. IPCS/CEC.	http://www.inchem.org/documents/antidote/antidote/ant02.htm	Report	9	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
106	Monograph for UKPID: Lead. 1996. National Poisons Information Service.	http://www.inchem.org/documents/ukpids/ukpids/ukpid25.htm	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	N
					Soil-based GVs developed	N
107	Lead and Lead Compounds: Summary of Data Reported and Evaluation. 1980. IARC.	http://www.inchem.org/documents/iarc/vol23/lead.html	Report	9	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
108	Benzene; The LQM/CIEH S4ULs for Human Health Risk Assessment. 2015. Nathanail, C P; McCaffrey, C; Gillett, A G; Ogden, R C; Nathanail, J F	Hard copy	Book	10	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
109	Polycyclic Aromatic Hydrocarbons; The LQM/CIEH S4ULs for Human Health Risk Assessment. 2015. Nathanail, C P; McCaffrey, C; Gillett, A G; Ogden, R C; Nathanail, J F	Hard copy	Book	10	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
110	SP1010: Development of Category 4 Screening Levels for Assessment of Land Affected by	http://randd.defra.gov.uk/Default.aspx?Module=More&Locatio	Report	11	Toxicity review	Y

	Contamination – Policy Companion Document. 2014. DEFRA.	n=None&ProjectID=18341			Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
111	SP1010: Appendix D Provisional C4SLs for benzene.	http://randd.defra.gov.uk/Default.aspx?Module=More&Location=None&ProjectID=18341	Report	11	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
112	SP1010: Appendix E Provisional C4SLs for benzo(a)pyrene as a surrogate marker for PAHs	http://randd.defra.gov.uk/Default.aspx?Module=More&Location=None&ProjectID=18341	Report	11	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
113	SP1010: Appendix H Provisional C4SLs for lead.	http://randd.defra.gov.uk/Default.aspx?Module=More&Location=None&ProjectID=18341	Report	11	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
114	Soil Guideline Values for benzene in soil. Science Report SC050021 / benzene SGV. 2009. Environment Agency.	https://webarchive.nationalarchives.gov.uk/20140328153731/http://www.environment-agency.gov.uk/static/documents/Research/SCHO0309BPQI-e-e.pdf	Report	12	Toxicity review	N
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	Y
115	Supplementary information for the derivation of SGV for benzene. Better Regulation Science Programme Science report: SC050021. 2009. Environment Agency.	https://webarchive.nationalarchives.gov.uk/20140328153756/http://www.environment-agency.gov.uk/static/documents/Research/SCHO0309BPQC-e-e.pdf	Report	12	Toxicity review	N
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	Y
116	Soil Guideline Values for dioxins, furans and dioxin-like PCBs in soil. Science Report SC050021 / Dioxins SGV. 2009. Environment Agency.	https://webarchive.nationalarchives.gov.uk/20140328153735/http://www.environment-agency.gov.uk/static/documents/Research/SCHO0909BQYQ-e-e.pdf	Report	12	Toxicity review	N
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	Y

117	Supplementary information for the derivation of SGVs for dioxins, furans and dioxin-like PCBs Better Regulation Science Programme Science report: SC050021/Technical Review dioxins, furans and dioxin-like PCBs. 2009. Environment Agency.	https://webarchive.nationalarchives.gov.uk/20140328153845/http://www.environment-agency.gov.uk/static/documents/Research/SCHO0909BQYS-e-e.pdf	Report	12	Toxicity review	N
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	Y
118	Soil Guideline Values for lead. These have now been withdrawn.	http://randd.defra.gov.uk/Default.aspx?Module=More&Location=None&ProjectID=18341	Report	12	Toxicity review	N
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	Y
119	HSDB: 2,3,7,8-tetrachlorodibenzo-p-dioxin	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
120	HSDB: Pentachlorodibenzo-p-dioxin.	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
121	HSDB: Hexachlorodibenzo-p-dioxin	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
122	HSDB:Octachlorodibenzo-p-dioxin	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
123	HSDB: 2,3,7,8-Tetrachlorodibenzofuran.	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N

					Soil-based GVs developed	N
124	HSDB: Lead, elemental	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
125	HSDB: Lead, compounds	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
126	HSDB: PAH	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
127	HSDB: Napthalene	https://toxnet.nlm.nih.gov/cgi-bin/sis/search2	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
128	HSDB: Acenaphthylene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
129	HSDB: Acenaphthene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
130	HSDB: Fluorene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y

					HBGV developed	N
					Soil-based GVs developed	N
131	HSDB: Phenanthrene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
132	HSDB: Anthracene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
133	HSDB: Fluoranthene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
134	HSDB: Pyrene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
135	HSDB: Benz(a)anthracene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
136	HSDB: Chrysene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
137	HSDB: Benzo(a)pyrene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y

					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
138	HSDB: Indeno(1,2,3-cd)pyrene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
139	HSDB: Dibenz(a,h)anthracene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
140	HSDB: Benzo(g,h,i)perylene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
141	HSDB: Benzo(b)fluoranthene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
142	HSDB: Benzo(k)fluoranthene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
143	HSDB: Methyl isocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N

144	HSDB: Ethyl isocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
145	HSDB: Phenyl isocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
146	HSDB: Hexamethylene diisocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
147	HSDB: Toluene-2,4-diisocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
148	HSDB: Toluene-2, 6-diisocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
149	HSDB: 4,4'-methylene diphenyl diisocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
150	HSDB: Isophorone diisocyanate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N

					Soil-based GVs developed	N
151	HSDB: Hydrogen cyanide	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
152	HSDB: Tri-(2-chloroisopropyl) phosphate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
153	HSDB: Triphenyl phosphate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
154	HSDB: Tris(2-butoxyethyl) phosphate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
155	HSDB: Triethyl phosphate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
156	HSDB: Trioctyl phosphate	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
157	HSDB: Benzene	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y

					HBGV developed	N
					Soil-based GVs developed	N
158	HSDB: Polybrominated biphenyls	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
159	HSDB: Hexabromodiphenyl	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
160	HSDB: 2,2',6,6'-tetrabromobisphenol A	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
161	HSDB: Polybrominated diphenyl ethers	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
162	HSDB: Decabromo diphenyl ether	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
163	HSDB: Octabromodiphenyl ethers	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
164	HSDB: Hexabromocyclododecane	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y

					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
165	HSDB: 1,2,5,6,9,10-hexabromocyclododecane	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
166	HSDB: Asbestos	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
167	HSDB: Chrysotile asbestos	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
168	HSDB: Amosite asbestos	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
169	HSDB: Tremolite asbestos	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
170	HSDB: Synthetic vitreous fibres	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N

171	HSDB: Polychlorinated biphenyls	https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm	Report	13	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
172	Benzene: Toxicological overview. 2007. Public Health England.	https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/337516/hpa_benzene_toxicological_overview_v2.pdf	Report	14	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
173	Compendium of Chemical Hazards. Polycyclic aromatic hydrocarbons (Benzo[a]pyrene): Toxicological Overview. 2018. Public Health England.	https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/737017/PAH_TO_PHE_240818.pdf	Report	14	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
174	Dioxins: Toxicological Overview. 2008. Public Health England.	https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/339481/Dioxins_Toxicological_Overview_phe_v1.pdf	Report	14	Toxicity review	Y
					Population exposure review	N
					HBGV developed	Y
					Soil-based GVs developed	N
175	Asbestos: Toxicological Overview. 2007. Public Health England.	https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/627190/Asbestos_toxicological_overview.pdf	Report	14	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	N
					Soil-based GVs developed	N
176	Hydrogen Cyanide: Toxicological Overview. 2016. Public Health England.	https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/500821/Hydrogen_Cyanide_PHE_TO_120216.pdf	Report	14	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y
					Soil-based GVs developed	N
177	Compendium of Chemical Hazards. Lead: Toxicological Overview. 2017. Public Health England.	https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/653725/Lead_toxicological_overview.pdf	Report	14	Toxicity review	Y
					Population exposure review	Y
					HBGV developed	Y

Soil-based GVs developed	N

Appendix TN08-C: Evidence Extraction

Evidence Number (from Table B)	Detailed summary of evidence

1 Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute MRL-intermediate MRL - chronic	None derived due to a lack of appropriate data None derived 0.0005 mg/kg/day	
Inhalation	MRL- acute MRL-intermediate MRL - chronic	0.009 ppm 0.006 ppm 0.003 ppm	LOAEL value of 10.2 ppm for reduced lymphocyte proliferation following mitogen stimulation in mice LOAEL value of 10 ppm for significantly delayed splenic lymphocyte reaction to foreign antigens evaluated in in vitro mixed lymphocyte reaction following the exposure of male C57Bl/6 mice to benzene vapours 6 hours/day, 5 days/week for 20 exposure days
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	
Background exposure (diet, air, drinking water etc)			

2 Napthalene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute	0.6 mg/kg/day	Rat developmental toxicity study involving exposure of Sprague-Dawley rats to gavage doses of 50,150, or 450 mg/kg/day naphthalene on gestation days 6-15
	MRL-intermediate	0.6 mg/kg/day	Three intermediate-duration oral toxicity studies in laboratory animals
	MRL - chronic	None derived	No appropriate studies
Inhalation	MRL- acute	None derived	Inadequate data
	MRL-intermediate	None derived	Inadequate data
	MRL - chronic	0.0007 ppm	Two chronic inhalation toxicity and carcinogenicity studies with mice and rats
Dermal	MRL- acute	None derived	
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

3 PAHs

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute	None derived	Inadequate data
	MRL-intermediate		
	Acenapthene	0.6 mg/kg/day	Minimal LOAEL of 175 mg/kg/day
	Fluoranthene	0.4 mg/kg/day	Minimal LOAEL of 125 mg/kg/day
	Fluorene	0.4 mg/kg/day	Minimal LOAEL of 125 mg/kg/day
	Anthracene	10 mg/kg/day	NOAEL of 1,000 mg/kg/day
	MRL - chronic	None derived	Inadequate data
Inhalation	MRL- acute	None derived	No adequate dose-response data for PAHs that identify threshold levels for non-cancer health effects are available in humans or animals for any duration of exposure.
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Dermal	MRL- acute	None derived	Lack of appropriate methodology for the development of dermal MRLs
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

4 Chlorinated dibenzo-p-dioxins(2,3,7,8-TCDD)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute	2×10-4 µg/kg/day	NOAEL of 0.005 µg/kg and a LOAEL of 0.01 µg/kg for immunological effects in female mice
	MRL-intermediate	2×10-5 µg/kg/day	NOAEL of 0.0007 µg/kg/day for immunological effects in Hartley guinea pigs fed 2,3,7,8-TCDD in the diet for 90 days
	MRL - chronic	1×10-6 µg/kg/day	LOAEL of 0.00012 µg/kg/day for developmental toxicity in rhesus monkeys
Inhalation	MRL- acute	None derived	
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Dermal	MRL- acute	None derived	
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

5 Chlorodibenzofurans (CDFs) (2,3,4,7,8-pentaCDF)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute	0.001 µg/kg/day	LOAEL for mild thymic lymphoid hypoplasia identified in groups of 6 male Hartley guinea pigs
	MRL-intermediate	0.00003 µg/kg/day	LOAEL for hepatic effects (increased serum bilirubin, decreased serum triglycerides) identified in groups of six male and six female 1va:SIVSO (SD) rats.
	MRL - chronic	None derived	No studies located
Inhalation	MRL- acute	None derived	Inhalation exposure to CDFs because human and animal data for all durations are lacking.
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Dermal	MRL- acute	None derived	
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

6 Polychlorinated Biphenyls (PCBs)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute MRL-intermediate MRL - chronic	None derived 0.03 µg/kg/day 0.02 µg/kg/day	Limited information LOAEL of 0.0075 mg/kg/day for neurobehavioral alterations in infant monkeys LOAEL of 0.005 mg/kg/day for immunological effects in adult monkeys that were evaluated after 23 and 55 months of exposure to Aroclor 1254
Inhalation	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Lack of adequate human and animal data
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	
Background exposure (diet, air, drinking water etc)			

7 Polybrominated Biphenyls (PBBs)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute MRL-intermediate MRL - chronic	0.01 mg/kg/day None derived None derived	NOAEL of 1 mg/kg/day for decreased serum levels of thyroid T4 hormone identified in groups of 8–11 male rats Serious developmental and reproductive effects were observed in monkeys that had been exposed to PBBs for durations that spanned the intermediate and chronic categories at the lowest dose tested in the database
Inhalation	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Human and animal data for all durations are either insufficient or lacking.
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	
Background exposure (diet, air, drinking water etc)			

8 Phosphate Ester Flame Retardants

41/96

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute		
	TCEP	None derived	Inadequate data
	TnBP	1.1 mg/kg/day	Reduced body weight gain in pregnant rats
	TBEP	4.8 mg/kg/day	Reduced body weight gain in pregnant rats
	TDCP	Not derived	Insufficient information
	TCP	Not derived	Insufficient information
	TPP	Not derived	Insufficient information
	TiBP	Not derived	Insufficient information
	TCPP	None derived	Insufficient information
	MRL-intermediate		
	TCEP	0.6 mg/kg/day	Necrosis of hippocampal neurons in female rats.
	TnBP	0.08 mg/kg/day	Urinary bladder lesions in male rats.
	TBEP	0.09 mg/kg/day	Hepatocyte vacuolization in male rats
	TDCP	0.05 mg/kg/day	Increased absolute kidney weight in rats
	TCP	0.04 mg/kg/day	Ovarian lesions in rats
	TPP	None derived	Insufficient information
	TiBP	None derived	Insufficient information
	TCPP	None derived	Insufficient information
	MRL – chronic		
	TCEP	0.2 mg/kg/day	Renal tubule lesions in female rats
	TnBP	0.08 mg/kg/day	Urinary bladder from rats
	TBEP	None derived	Insufficient information
	TDCP	0.02 mg/kg/day	Renal tubule hyperplasia in male rats
	TCP	0.02 mg/kg/day	Ovarian lesions in rats
	TPP	None derived	Insufficient information
	TiBP	None derived	Insufficient information
	TCPP	None derived	Insufficient information
Inhalation	MRL- acute	None derived	Inadequate data
	MRL-intermediate		
	MRL - chronic		
Dermal	MRL- acute	None derived	Inadequate data
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

9 Polybrominated Diphenyl Ethers (PBDEs)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute Lower brominated BDEs Decabromodiphenyl ether MRL-intermediate Lower brominated BDEs Decabromodiphenyl ether MRL - chronic Lower brominated BDEs Decabromodiphenyl ether	 0.00006 mg/kg/day 0.01 mg/kg/day 0.000003 mg/kg/day 0.0002 mg/kg/day None derived None derived	 Endocrine effects in rats Neurobehavioral effects in mice Reduction in testosterone in rats Glucose in adult rats Insufficient data Insufficient data
Inhalation	MRL- acute Lower brominated BDEs Decabromodiphenyl ether MRL-intermediate Lower brominated BDEs Decabromodiphenyl ether MRL - chronic Lower brominated BDEs Decabromodiphenyl ether	 None derived None derived 0.006 mg/m3 None derived None derived None derived	 Insufficient information Insufficient information NOAEL of 1.1 mg/m3 for changes in thyroid hormones in rats Insufficient information Insufficient information Insufficient information
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	
Background exposure (diet, air, drinking water etc)			

10 Hexamethylene Diisocyanate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute	None derived	Insufficient data
	MRL-intermediate	None derived	Insufficient data
	MRL - chronic	None derived	Insufficient data
Inhalation	MRL- acute	None derived	Insufficient data
	MRL-intermediate	3.0x 10-5 ppm	NOAEL of 0.005 ppm administered to rats
	MRL - chronic	1.0x10-5 ppm	Nasal cavity epithelial hyperplasia in female rats (minimal LOAEL)
Dermal	MRL- acute	None derived	
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

11 Toluene Diisocyanate Methylenediphenyl Diisocyanate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Oral exposure to Toluene diisocyanate and methylenediphenyl diisocyanate is unlikely as both are rapidly hydrolysed in water
Inhalation	MRL- acute TDI MRL-intermediate TDI MRL – chronic TDI MDI	 1x10-5 ppm None derived 3x10-6 ppm 0.001 mg/m3	 Limited number of human studies Insufficient information Study of asthma symptoms in workers Hyperplasia in rats
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Insufficient information
Background exposure (diet, air, drinking water etc)			

12 Asbestos

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Insufficient information
Inhalation	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Insufficient information
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Insufficient information
Background exposure (diet, air, drinking water etc)			

13 Synthetic Vitreous Fibers

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute	None derived	Insufficient information
	MRL-intermediate	None derived	Insufficient information
	MRL - chronic	None derived	Insufficient information
Inhalation	MRL- acute	None derived	Insufficient information
	MRL-intermediate		
	Refractory ceramic fibers	0.03 WHO fibers/cc	Chronic exposure
	MRL – chronic		
	Refractory ceramic fibers	0.03 WHO fibers/cc	Pulmonary inflammation in rats
Dermal	MRL- acute	None derived	Insufficient information
	MRL-intermediate	None derived	
	MRL - chronic	None derived	
Background exposure (diet, air, drinking water etc)			

14 Cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL- acute MRL-intermediate MRL - chronic	None derived 0.05 mg CN– /kg/day None derived	No suitable data Studies on rat, pigs and mice No suitable data
Inhalation	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	Available data indicated serious adverse effects occurring even at the lowest reported exposure levels
Dermal	MRL- acute MRL-intermediate MRL - chronic	None derived None derived None derived	
Background exposure (diet, air, drinking water etc)			

15 Lead

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	MRL	None derived	Lowest blood levels (≤5 µg/dL) are associated with serious adverse effects (e.g., declining cognitive function in children)
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

16 PAHs

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	TEF	None derived	Not considered to be scientifically valid because of the lack of data from oral carcinogenicity studies for different PAHs, their different modes of action and the evidence of poor predictively of the carcinogenic potency of PAH mixtures based on the currently proposed TEF values
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)	Proposed low concentrations Benzo[a]anthracene 0.2 µg/kg wet weight Chrysene/Triphenylene 0.8 µg/kg wet weight Benzo[b]fluoranthene 0.6 µg/kg wet weight Benzo[j]fluoranthene 0.6 µg/kg wet weight Benzo[k]fluoranthene 0.2 µg/kg wet weight Benzo[a]pyrene 0.1 µg/kg wet weight Indeno[1,2,3-cd]pyrene 0.2 µg/kg wet weight Benzo[ghi]perylene 0.3 µg/kg wet weight		

17 Dioxins and Dioxin-like PCBs

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Tolerable weekly intake (TWI) Dietary intake No observed adverse effect level (NOAEL)	2pg per kg bodyweight 0.25 pg TEQ/kg bw/day 7 pg WHO-TEQ/g fat	Regularly exceeded by factors of up to 5 in typical dietary exposure
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

18 Non dioxin-like PCB

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Benchmark dose (BMDL) NOAEL (PCB 28, 128, and 153)	1 µg PCB/g lipid 30-40 µg/kg bw per day	 Liver and thyroid toxicity in animals
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

19 Polybrominated Biphenyls (PBBs)	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	No observed effect level (NOEL)	0.15mg/kgBW/day	Liver carcinogenic effects. Considered too conservative to base health based guidance value for PBBs
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			
20 Tetrabromobisphenol A (TBBPA)	Identified adverse health effects – acute	Main toxicological effect - changes in thyroid hormone homeostasis.		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	Benchmark dose (BMDL10)	16mg/kgBW/day	
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

21 Polybrominated Diphenyl Ethers (PBDEs)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Mean upper bound concentrations Human body-burden at BMDL10 TetraBDE (BDE-47) PentaBDE (BDE-99) HexaBDE (BDE-153) DecaBDE (BDE-209)	0.04-3.98ng/g 232 µg/kgBW/day 9 µg/kgBW/day 62 µg/kgBW/day N/A	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

22 Hexabromocyclododecanes (HBCDDs)	Identified adverse health effects – acute	Main adverse health effects were associated with the liver, thyroid, reproduction, nervous system and immune system		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	Body burden benchmark dose (BMDL10) Mean dietary exposure	0.79mg/kgBW/day 0.15-1.85ng/kgBW/day for children 0.09-0.99ng/kgBW/day	
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			
23 Cyanide	Identified adverse health effects – acute	The limited data from animal and human studies do not allow the derivation of a chronic health-based guidance value (HBGV) for cyanide (CN)		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

24 Lead

Identified adverse health effects – acute	EFSA (2013) reported that the toxicological effects of lead on children are also of particular concern, as lead is absorbed more than in adults and accumulates in the body over time. The scientific opinion of the CONTAM panel (EFSA, 2013) concluded that, due to a lack of evidence for a threshold for critical toxicological effects, the previous PTWI of 25 µg/kg bw (JECFA, 1986) was no longer appropriate, and no new PTWI was derived.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

25 Mixed halogenated dioxins and biphenyls

Identified adverse health effects – acute	COT concluded that the 2005 WHO-TEFs for chlorinated dioxins and furans could also be used for brominated and mixed halogenated dioxin and furans. It was also advocated that combining the TEQs of the chlorinated and brominated compounds would provide a more protective assessment of potential health risk.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	TDI	2pg/kgBW/day	Used as toxicological basis for 2005 WHO-TEFs.
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

26 Polybrominated Biphenyls	Identified adverse health effects – acute	COT concluded that the key toxicological effect is liver carcinogenicity and that planar and non-planar PBBs need separate consideration with planar PBBs expected to behave as per dioxins. It was also considered that the 2005 WHO-TEFs for PCBs could be conservatively assigned to PBBs in order to compare EQ values to the TDI for dioxins. Insufficient toxicological data was available for non-planar PBBs, and insufficient data was available for the occurrence of PBBs in the environment and foodstuffs for a meaningful assessment to be made		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			
27 Polybrominated Diphenyl Ethers (PBDEs)	Identified adverse health effects – acute	COT concluded that there was insufficient evidence to establish health-based guidelines.		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	BB BMDL10 Mean dietary exposure	425µg/kgBW/day 6.71ng/kgBW/day	
	Inhalation	BDE-209	2.7µg/kgBW/day	
	Dermal	Mean air exposure	0.012ng/kgBW/day	
	Background exposure (diet, air, drinking water etc)			

28 Tetrabromobisphenol A	Identified adverse health effects – acute	TBBPA has a low acute toxicity		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	TDI	1mg/kgBW/day	NOAEL of 1000mg/kgBW/day from an animal reproductive toxicity study
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			
29 Hexabromocyclododecane	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	BMDL10 human body burden equivalent food concentrations dust concentrations Dust exposure for infants Food exposure for infants	0.93mg/kgBW/day 3µg/kgBW/day <0.01-<0.1µg/kg 140-140000ug/kg 104ng/kgBW/day 3.24-3.81ng/kgBW/day	
	Inhalation	indoor air concentrations	67-1300pg/m3	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

30 Lead

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	BMDL01 Dietary exposure	1.2µg/dl 0.5µg/kgBW/day	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

Exposure Route	Estimated Median Dose
Soils and dust	0.36µg/kgBW/day for rural soils 0.59µg/kgBW/day for semi-urban soils 1.7µg/kgBW/day for urban soils
Air	0.00031-0.0023 µg/kgBW/day
Diet	Up to 0.4 µg/kgBW/day based on mean dietary exposure from FSA (2006) Total Diet Study
Drinking Water	0.044-0.086 µg/kgBW/day

31 Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	1 × 10-3 mg/kg/day 4 × 10-3 mg/kg/day	LOAEL of 7.6 ppm BMCL
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

32 Acenaphylene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	not evaluated	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

33 Anthracene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	3E-1 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

34 Benz[a]anthracene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
35 Benzo[b]fluoranthene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

36 Benzo[g,h,i]perylene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
37 Chrysene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

38 Dibenz[a,h]anthracene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
39 Fluoranthene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	4E-2 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

40 Fluorene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	4E-2 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

41 Indeno[1,2,3-cd]pyrene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	not evaluated	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

42 Naphthalene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	2E-2 mg/kg/day	
	Inhalation	RfC	3E-3 mg/m3	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
43 Pyrene	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	3E-2 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

44 Brominated dibenzofurans	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
45 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	7E-10 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

46 Aroclor 1016

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	7E-5 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

47 Aroclor 1248

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	unverifiable	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

48 Aroclor 1254	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	2E-5 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
49 Polychlorinated biphenyls (PCBs)	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	unverifiable	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

50 2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	7E-3 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
51 Hexabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

52 2,2',4,4',5,5'-Hexabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	2E-4 mg/kg/day	
	Inhalation	RfC	no information available	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
53 p,p'-Dibromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

54 Octabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	3E-3 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
55Pentabromodipheny l ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	2E-3 mg/kg/day	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

56 2,2',4,4',5-Pentabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	1E-4 mg/kg/day	
	Inhalation	RfC	insufficient information	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
57 2,2',4,4'-Tetrabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	1E-4 mg/kg/day	
	Inhalation	RfC	insufficient information	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

58 Tetrabromodiphenyl ether	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
59 1,6-Hexamethylene diisocyanate	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	1E-5 mg/cu.m	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

60 Methyl isocyanate	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	insufficient information	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
61 Methylene Diphenyl Diisocyanate (monomeric methylenediphenyl diisocyanate) and polymeric methylenediphenyl diisocyanate (PMDI)	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	not evaluated	
	Inhalation	RfC	6E-4 mg/cu.m	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

62 2,4-/2,6-Toluene diisocyanate mixture (TDI)

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	not evaluated	
Inhalation	RfC	7E-5 mg/cu.m	
Dermal			
Background exposure (diet, air, drinking water etc)			

63 Asbestos

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	not evaluated	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

64 Barium cyanide	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	insufficient information	
	Inhalation	RfC	not evaluated	
	Dermal			
	Background exposure (diet, air, drinking water etc)			
65, 68, 69, 70, 71 and 73 Hydrogen Cyanide and Cyanide Salts	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	RfD	6E-4 mg/kg/day	
	Inhalation	RfC	8E-4 mg/m3	
	Dermal			
	Background exposure (diet, air, drinking water etc)			

66 Chlorine cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	5E-2 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

67 Copper cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	5E-3 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

72 Silver cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	1E-1 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

74 Zinc cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	5E-2 mg/kg/day	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

75 Lead

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	RfD	insufficient information	
Inhalation	RfC	not evaluated	
Dermal			
Background exposure (diet, air, drinking water etc)			

76 Benzene

Identified adverse health effects – acute	Relationship between exposure to benzene and the occurrence of various types of leukaemia Benzene is carcinogenic to humans (Group 1)		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Standardized mortality ratio (SMR) Standardized mortality ratio (SMR)	194 394	Four human cases of benzene exposed workers (acute nonlymphocytic leukaemia and myelogenous leukaemia) Eight human cases of benzene exposed workers (only myelogenous leukaemia)
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

77 Benzene

Identified adverse health effects – acute	Benzene is a moderate eye irritant and is irritating to rabbit skin after multiple applications of the undiluted chemical.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	Between 3000 and 8100 mg/kg	In rats
Inhalation	LC50	44 000 mg/m3	In rats
Dermal			
Background exposure (diet, air, drinking water etc)			

78 Benzene

Identified adverse health effects – acute	Acute exposure to high concentrations of benzene in air results in neurological toxicity and may sensitize the myocardium to endogenous catecholamines. Acute ingestion of benzene causes gastrointestinal and neurological toxicity.		
Identified adverse health effects – intermediate/chronic	Chronic exposure to benzene results primarily in haematotoxicity, including aplastic anemia, pancytopenia, or any combination of anaemia, leukopenia, and thrombocytopenia. Chronic benzene exposure is associated with an increased risk of leukemia		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

79 Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal	TLV EU-OEL	0.5 ppm (TWA) 2.5 ppm (STEL) 3.25 mg/m^3	
Background exposure (diet, air, drinking water etc)			

80 PAHs

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation	LC50 Naphthalene Three-ring PAH Four, five and six ring PAH LC50 Naphthalene Three-ring PAH Four and five ring PAH	 100-2300 µg/litre < 1 and 260 µg/litre 0.2-1200 µg/litre 110 to > 10 000 µg/litre 30-4000 µg/litre 0.7-26 µg/litre	In invertebrates In fish
Dermal			
Background exposure (diet, air, drinking water etc)			

81 Polychlorinated dibenzodioxins, polychlorinated dibenzofurans and coplanar polychlorinated biphenyls

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Toxic equivalency factor TCDD 1,2,3,7,8-PeCDD 1,2,3,4,7,8-HxCDD 1,2,3,6,7,8-HxCDD 1,2,3,6,7,9-HxCDD 1,2,3,4,6,7,8-HpCDD OCDD 2,3,7,8-TCDF 1,2,3,7,8-PeCDF 2,3,4,7,8-PeCDF 1,2,3,4,7,8-HxCDF 1,2,3,6,7,8-HxCDF 1,2,3,7,8,9-HxCDF 2,3,4,6,7,8-HxCDF 1,2,3,4,6,7,8-HpCDF 1,2,3,4,7,8,9-HpCDF OCDF 3,3´,4,4´-TCB 3,4,4´,5-TCB 3,3´,4,4´,5-PeCB 3,3´,4,4´,5,5´-HxCB 2,3,3´,4,4´-PeCB 2,3,4,4´,5-PeCB 2,3´,4,4´,5-PeCB 2,3´,4,4´,5´-PeCB 2,3,3´,4,4´,5-HxCB 2,3,3´,4,4´,5´-HxCB 2,3´,4,4´,5,5´-HxCB 2,3,3´,4,4´,5,5´-HpCB	 1 1 0.1 0.1 0.1 0.01 0.0001 0.1 0.05 0.5 0.1 0.1 0.1 0.1 0.01 0.01 0.0001 0.0001 0.0001 0.1 0.01 0.0001 0.0005 0.0001 0.0001 0.0005 0.0005 0.0001 0.0001	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

82 Polychlorinated dibenzo-para-dioxins and dibenzofurans

Identified adverse health effects – acute	For occupational and accidental exposures to PCDDs and PCDFs, in spite of many clinical and follow-up studies, no clear-cut persistent systemic effects have been delineated except for chloracne. Other effects have been noted, but, apart from chloracne and perhaps minor functional disorders, none has been persistent.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

83 Polychlorinated dibenzo-para-dioxins.

Identified adverse health effects – acute	Human exposure to 2,3,7,8-TCDD or other PCDD congeners due to industrial or accidental exposure has been associated with chloracne and alterations in liver enzyme levels in both children and adults. Changes in the immune system and glucose metabolism have also been observed in adults. Infants exposed to PCDDs and PCDFs through breast milk exhibit alterations in thyroid hormone levels and possible neurobehavioural and neurological deficits.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)	Mean background levels of 2,3,7,8-tetrachlorodibenzo-para-dioxin (2,3,7,8-TCDD) in human tissues today are in the range of 2-3 ng/kg fat.		

84 Polychlorinated biphenyls and terphenyls

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50 Aroclor NOEL Aroclor 1254 Aroclor 1260	>750 to 4000 mg/kg body weight 1.3-2.5 g/kg body weight 4-11 g/kg body weight 0.32 mg/kg body weight 7.5 mg/kg body weight	Mink Young Rats Adult Rats Rats
Inhalation	LC50	0.008 to >100 mg/litre	Fish
Dermal	LD50 Aroclor 1260 PCB mixture	 >1.26 to <2 g/kg body weight 0.79 to <3.17 g/kg body weigth	 Rabbits Rabbits
Background exposure (diet, air, drinking water etc)			

85 Polychlorinated Biphenyls (PCBs) and Polychlorinated Terphenyls (PCTs)

Identified adverse health effects – acute	Same information as given above.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

86 Polychlorinated Biphenyls

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)	In 1978, the estimated dietary intake of PCBs by adults in the USA was 0.027 µg/kg body weight per day, but it declined to 0.0005 µg/kg body weight per day in 1982–1984 and <0.001 µg/kg body weight per day for the period 1986–1991		

87 Polybrominated Biphenyls

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	NOEL LD50 Carcinogenic response	0.15 mg/kg body weight per day >1 g/kg body weight 0.5 mg/kg body weight daily for 6 months	Rats and male mice Rats, rabbits and quails Rats and mice
Inhalation			
Dermal	LD50	>1 g/kg body weight	Rats, rabbits and quails
Background exposure (diet, air, drinking water etc)			

88 Polybrominated Biphenyls

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Of the 91 workers potentially exposed on a 'routine' basis, none died during the study period; among the 237 'non-routinely' exposed, two deaths were observed, with 6.4 expected, one of which was due to cancer of the large intestine		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

89 Triphenyl phosphate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50 NOEL	3000 to above 20 000 mg/kg bw 161 mgkg bw/day	Rats, mice, rabbits and guinea pigs Rats
Inhalation			
Dermal	LD50	>7900 mg/kg bw	Rabbits
Background exposure (diet, air, drinking water etc)			

90 Triphenyl phosphate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	>6.4 g/kg >2.0 g/kg	Rats Chickens
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

91 Triphenyl phosphate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	The substance may have effects on the peripheral nervous system. This may result in impaired functions.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	TLV	3 mg/m3 as TWA	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

92 Triethyl phosphate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	NOEL Subacute toxicity Teratogenicity Fertility effects	 1000 mg/kg bw 625 mg/kg bw/day 335 mg/kg bw	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

93 Brominated diphenyl ethers

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	A morbidity study of extruder personnel blending polybutyl-eneterephthalate containing DeBDE, with consequently potential exposure to PBDD and PBDF for 13 years, did not reveal any deleterious effects, even though 2,3,7,8-TeBDF and -TeBDD were detected in the blood. Results of immunological studies showed that the immune system of the exposed persons was not adversely affected in 13 years.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	>2000 mg/kg bw	Rats
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

94 Tetrabromobisphenol A and derivatives

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	>5 g/kg bw 10 g/kg bw	Rat Mouse
Inhalation	LC50	0.5 mg/litres	Mouse, rat and guinea pig
Dermal	LD50	>2 g/kg bw	Rabbit
Background exposure (diet, air, drinking water etc)			

95 Diphenylmethane diisocyanate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation	NOAEL	2.2 mg/m3 9 mg/m3 4 mg/m3	Increase in lung sizes in male rats Development toxicity in rats Maternal and fetal toxicity in rats
Dermal			
Background exposure (diet, air, drinking water etc)			

96 Toluene-2,4-diisocyanate

Identified adverse health effects – acute	Toluene-2-4-diisocyanate is highly volatile and is an irritant to the skin and mucous membranes. The most common and most serious effects occur in the lungs. Sudden acute bronchospasm attacks, pulmonary oedema, and respiratory distress may be life-threatening and may occur days, or even months, after the first exposure. Symptoms may also be delayed. An additional risk is CNS-depression that can occur after exposure to high levels of toluene-2-4-diisocyanate. Toluene-2-4-diisocyanate can also irritate the skin, eyes, nose, and throat.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

97 Toluene diisocyanates

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	3.06 to 4.13 g/kg bw	Rabbits
Inhalation	LC50 Irritation of eyes, nose and respiratory tract	70 to 356 mg/m3 0.35 mg/m3	Rabbits Humans
Dermal	LD50	10 g/kg bw	Rabbits
Background exposure (diet, air, drinking water etc)			

98 Toluene diisocyanate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation	8 hour TWA 4 15 minute periods a day TWA	0.005 ppm 0.02 ppm	ACGIH
Dermal			
Background exposure (diet, air, drinking water etc)			

99 Hexamethylene Diisocyanate

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50 Maternal effects Developmental toxicity	746 – 959 mg/kg bw ≥ 0.052 ppm 0.308 ppm	Rat Rat Rat
Inhalation	LC50 NOEL	0.124 mg/l 0.005 ppm	Rat Rabbit
Dermal	LD50	599 mg/kg bw	Rabbit
Background exposure (diet, air, drinking water etc)			

100 Chrysotile Asbestos	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation	Asbestotic changes	5 to 20 f/ml	Prolonged exposure to humans
	Dermal			
	Background exposure (diet, air, drinking water etc)			
101 Asbestos and other Natural Mineral Fibres	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic	Epidemiological studies, mainly on occupational groups, have established that all types of asbestos fibres are associated with diffuse pulmonary fibrosis (asbestosis), bronchial carcinoma, and primary malignant tumours of the pleura and peritoneum (mesothelioma).		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

102 Asbestos (Actinolite, amosite, anthophyllite, chrysotile, crocidolite, tremolite)	Identified adverse health effects – acute	Numerous reports from several countries have described cases or series of pleural and peritoneal mesotheliomas in relation to occupational exposure to various types and mixtures of asbestos (including talc containing asbestos), although occupational exposures have not been identified in all cases.		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			
103 Hydrogen Cyanide and Cyanides	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral	Lowest reported lethal dose Average adsorbed dose at death	0.54 mg/kg bw 1.4 mg/kg bw	Human
	Inhalation	NOAEL	4.5 mg/kg bw per day	Rats
	Dermal			
	Background exposure (diet, air, drinking water etc)			

104 Cyanides

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Histotoxic anoxia in the brain and heart may cause early coma, respiratory failure and cardiovascular collapse. Reduced oxygen utilization and lactate acidosis cause severe metabolic effects.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

105 Cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation	Fatal in minutes Fatal	50 ml 250g	Human
Dermal			
Background exposure (diet, air, drinking water etc)			

106 Lead

Identified adverse health effects – acute	• Ingestion: Gastrointestinal colic (nausea, vomiting, anorexia, and abdominal pain). Malaise, convulsions, coma, encephalopathy, hepatic and renal damage, anaemia, hypertension and bradycardia. Neurological effects include headache, insomnia, drowsiness and more rarely convulsions. • Inhalation: lead does not cause any local irritation but may cause the same effects as for ingestion if enough is inhaled. Organic lead compounds can cause severe toxicity by inhalation, onset can be delayed for acute mania, convulsions, delirium, fever and coma. • Dermal: Inorganic lead salts may cause mild local irritation but systemic toxicity from this route has not been described. Acute poisoning may occur from dermal exposure to organic lead compounds, with delayed onset of effects as for ingestion. • Ocular: irritation and inflammation have been observed for some lead compounds.		
Identified adverse health effects – intermediate/chronic	• Ingestion/dermal: nausea, vomiting, abdominal pain, metallic taste, anorexia and general feeling of malaise or fatigue. Longer exposures may lead to joint pain, progressive fatigue and anaemia. Altered renal and hepatic function. Motor weakness may progress to paralysis of the extensor muscles of the wrist (wrist drop) and less often the ankles (foot drop). Adults may have a bluish gingival "lead-line". Encephalopathy (vomiting, confusion, ataxia, apathy, bizarre behaviour and coma and convulsions due to cerebral oedema) rarely occurs in adults except from exposure to organic lead. In children lead may also be drawn to areas of the skeleton that grow most rapidly and in some cases hypermineralisation of the bones may occur. • Chronic low level exposures in children are linked with decreased intelligence and behavioural and learning disorders. There is evidence to suggest that growth may be inhibited. Thyroid and adrenal functions may also be inhibited. • Organic lead: asthenia, weakness, fatigue, pallor, headache, nausea, vomiting, diarrhoea, anorexia and weight loss. Ataxia, tremor, hypotonia, bradycardia & hypotension and hypothermia may also develop. In more severe poisoning disorientation, hallucinations, facial contortions and episodes of intense hyperactivity may occur. In severe cases maniacal behaviour and convulsions may develop which may lead to coma and death. • Hypertension.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

107 Lead

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	There is sufficient evidence that lead subacetate is carcinogenic to mice and rats and that lead acetate and lead phosphate are carcinogenic to rats. In the absence of adequate human data, it is reasonable, for practical purposes, to regard these compounds as if they presented a carcinogenic risk to humans.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Chromosomal aberrations	100-1000 mg/l blood lead level	Peripheral lymphocytes of lead-exposed populations
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

108 Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	ID MPR	0.29 µg/kg bw per day 3.3 µg/kg bw per day	
Inhalation	ID MPR	1.4 µg/kg bw per day 0.33 µg/kg bw per day	
Dermal			
Background exposure (diet, air, drinking water etc)			

109 PAHs

Identified adverse health effects – acute

Identified adverse health effects – intermediate/chronic

Health-Based Guideline Values (HBGV)

Oral/Inhalation

PAH Congener	Oral TDI (µg kg^{-1} bw day^{-1})	Oral ID (µg kg^{-1} bw day^{-1})	Inhalation TDI (µg kg^{-1} bw day^{-1})	Inhalation ID (µg kg^{-1} bw day^{-1})	Oral MDI – food (µg day^{-1})	Inhalation MDI (µg day^{-1})
Acenapthene	60		60		0.98	0.025
Acenapthylene	60		60		0.14	0.011
Anthracene	300		300		0.08	0.041
Benz(a)anthracene		0.155		0.0015	0.06	0.011
Benzo(a)pyrene		0.031		0.00030	0.11	0.006
Benzo(b)fluoranthene		0.039		0.00038	0.11	0.013
Benzo(ghi)perylene		3.44		0.033	0.06	0.010
Benzo(k)fluoranthene		1.03		0.010	0.09	0.007
Chrysene		0.31		0.0030	0.11	0.017
Dibenz(ah)anthracene		0.0031		0.00003	0.04	0.033
Fluoranthene	12.5		12.5		0.35	0.084
Fluorene	40		40		0.59	0.096
Indeno(123-cd)pyrene		0.443		0.0043	0.10	0.009
Napthalene	20		0.86		7.0	2.8
Phenanthrene	12.5		12.5		1.54	0.518
Pyrene	30		30		0.35	0.065
Coal Tar (BaP as surrogate marker)		0.01		0.00030		

Background exposure (diet, air, drinking water etc)

110. Benzene

Identified adverse health effects – acute	This is the C4SL main document that provides the steps for deriving C4SL but not the actual levels and it is not specific to benzene. The section below (111) includes the C4SLs for benzene and is very comprehensive.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

111. Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Inhalation: leukaemia.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral/ inhalation/ dermal	Option 1: pC4SL use of minimal risk HCVs with changes to exposure parameters (as summarised in Section 3.5.7 of the main report) (6% SOM).	0.29 µg/kg bw per day (HCV oral) 0.42 mgkg – residential with home grown produce 1.4 mg/kg – residential without home grown produce 0.09 Allotments 90 mg/kg commercial 72 mg/kg POS resi 110 mg/kg POSpark	A range of pC4SLs have been derived for benzene using three alternative sets of deterministic exposure parameters and varying concentrations of SOM
	Option 2: pC4SL use of LLTCs (low level of toxicological concern) with no change to exposure parameters (i.e. as defined in SR3). (6% SOM).	0.57 ug/kgbw per day (LLTC oral) 0.69 mg/kg Residential with home grown produce 2.3 mg/kg Residentiall without home grown produce 0.15 mg/kg Allotments 100 mg/kg Commercial	
	Option 3a: pC4SL use of LLTCs with changes to exposure parameters (6% SOM)	0.57 ug/kg bw per day (LLTC oral) 0.87 mg/kg Residential with home grown produce 3.3 mg/kg Residential without home grown produce 0.18 mg/kg Allotments 98 mg/kg Commercial 140 mg/kg POSresi 230 mg/kg POSpark	
	Option 3b: pC4SL use of LLTCs with changes to exposure parameters (1% SOM)	0.57 ug/kg bw per day (LLTC oral) 0.2 mg/kg Residential with home grown produce 0.89 mg/kg Residential without home grown produce 0.039 mg/kg Allotments 27 mg/kg Commerical 140 mg/kg POSresi 190 mg/kg POSpark	

112. Benzo(a)pyrene as a surrogate marker for PAHs

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Critical toxic endpoint for all toxicity studies in animal toxicology data is: Carcinogenicity, including tumours of he liver, forestomach, lunch, gastrointestinal tract, oesophagus, larynx or tongue.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral/ inhalation/ dermal	Option 1: pC4SL use of minimal risk HCVs with changes to exposure parameters (as summarised in Section 3.5.7 of the main report) (6% SOM). Option 2: pC4SL use of LLTCs (low level of toxicological concern) with no change to exposure parameters (i.e. as defined in SR3). (6% SOM). Option 3: pC4SL use of LLTCs with changes to exposure parameters (6% SOM)	0.02 ug/kg bw day (HCV oral) 2.4 mg/kg Residential with consumption of home grown produce 2.5 mg/kg Residential without consumption of home grown produce 2.7 mg/kg Allotments 36 mg/kg Commercial 4.9 mg/kg POS resi 10 mg/kg POS park 0.042 ug/kg bw day (LLTC oral) 3.2 mg/kg Residential with consumption of home grown produce 3.4 Residential without consumption of home grown produce 5.1 Allotments 77 Commercial 0.042 ug/kg bw day (LLTC oral) 5 mg/kg Residential with consumption of home grown produce 5.3 mg/kg Residential without consumption of home grown produce 5.7 mg/kg Allotments 77 Commercial 10 POSresi 21 POSpark	A range of pC4SLs have been derived for BaP – as a surrogate marker for genotoxic PAHs using three alternative sets of deterministic exposure parameters.
Inhalation	HCV or LLTC (µg/kg bw day)	7e-5 µg/kg (HCV) with exposure changes only; 3e-4 – 6.6e-4 µg/kg (LLTC) exposure parameters as SR3; 3e-4 – 6.6e-4 µg/kg (LLTC) with changes in exposure and LLTC	
Dermal			
Background exposure (diet, air			

113. Lead

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Causal relationships for: Nervous system effects, cardiovascular effects, haematological effects, reproductive effects and likely causal relationships for renal effects, cancer and immune system effects (USEPA 2012).		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral/ inhalation/ dermal	Option 1: pC4SL use of LLTCs (low level of toxicological concern) with no change to exposure parameters (i.e. as defined in SR3).	LLTC1: Intake leading to blood lead concentration of 1.6 ug/l. **Method 1:** 0.6 ug/kg bw day 82 mg/kg residential with home grown produce. 130 mg/kg residential without home grown produce. 30 mg/kg allotments **Method 2:** 0.29 ug/kg bw day 1100 mg/kg commercial **Method 3:** 0.57 ug/kg bw day 2200 mg/kg commercial LLTC2: Intake leading to blood lead concentration of 3.5 ug/l. **Method 1:** 1.4 ug/kg bw day 190 mg/kg residential with home grown produce. 310 mg/kg residential without home grown produce. 70 mg/kg allotments **Method 2:** 0.63 ug/kg bw day 2300 mg/kg commercial **Method 3:** 1.3 ug/kg bw day 4800 mg/kg commerical	**Method 1.** IEUBK used to estimate the intake that would lead to the proposed alternative LLTC where a child is the critical receptor i.e. for the residential, allotments and POS land uses. **Method 2.** USEPA adult Lead (Pb) methodology (ALM) used as the second option to estimate the intake that would lead to the proposed alternative LLTC where an adult is the critical receptor i.e. for commercial land use. **Method 3**. The Carlisle and Wade method used as one option to estimate the intake that would lead to the proposed alternative LLTC where an adult is the critical receptor i.e. for the commercial land use. **Note:** The LLTC of 5 ug/l is based on CDC's target blood lead concentration in children for all exposure to lead and

114 Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	The blood and immune system are the main targets of chronic exposure to benzene. The critical effect of both oral and inhalation exposure is considered to be the potential for leukemia (Environment Agency, 2009).		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral/ inhalation/ dermal (SGV)	SGV	0.33 mg/kg DW Residential. 0.07 mg/kg DW Allotment. 95 mg/kg DW Commercial. DW = Dry weight.	Based on percentage of exposure at the SGV attributable to each individual relevant pathway for each land use. Based on sandy loam soil as defined in EA 2009 and 6% SOM. At a lower SOM SGV may not be sufficiently protective. Generic Assessment Criteria (GAC) for benzene will vary according to SOM for all land uses. SGVs assume that free phase contamination is not present. SGVs based on a subsurface soil to indoor air correction factor of 10.
Inhalation	Health criteria value (HCV)	1.4 ug/kg bw day	Based on background inhalation of 200 ug/day in ambient air for an adult weighing 70 kg and inhaling 20 m^3/day.
Dermal			
Background exposure (diet, air, drinking water etc)	HCV 0.29 ug/kg bw day (Oral) based on WHO guideline for drinking water quality for benzene.		

115 Benzene

Identified adverse health effects – acute	This is the supplementary information on plant uptake and phyto toxicity that goes will the SGV for Benzene (section 114 - above). As such there are no SGVs included in this technical note.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

116 Dioxins (PCDD), Furans (PCDFs) and dioxin like PCBs (PCBs)

WHO recommended toxic equivalency factors (TEFs)

PCDDs:
2,3,7,8-TCDD 1
1,2,3,7,8-PeCDD 1
1,2,3,4,7,8-HxCDD 0.1
1,2,3,6,7,8-HxCDD 0.1
1,2,3,7,8,9-HxCDD 0.1
1,2,3,7,8,9-HpCDD 0.01
OCDD 0.0003

PCDFs:
2,3,7,8-TCDF 0.1
1,2,3,7,8-PeCDF 0.03
2,3,4,7,8-PeCDF 0.3
1,2,3,4,7,8-HxCDF 0.1
1,2,3,7,8,9-HxCDF 0.1
1,2,3,6,7,8-HxCDF 0.1
2,3,4,6,7,8-HxCDF 0.1
1,2,3,4,6,7,8-HpCDF 0.01
1,2,3,4,7,8,9-HpCDF 0.01
OCDF 0.0003

PCBs:
non-ortho
PCB-77 0.0001
PCB-81 0.0003
PCB-126 0.1
PCB-169 0.03

mono-ortho
PCB-105 0.00003
PB-114 0.00003
PCB-118 0.00003
PCB-123 0.00003
PCB-156 0.00003
PCB-157 0.00003
PCB-167 0.00003
PCB-189 0.00003

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Dioxin 2,3,7,8 – TCDD is considered the most toxic congener and is a proven carcinogen although there is no convincing evidence that it possesses genotoxic potential. The other non-cancer health effects induced by dioxins and dioxin like compounds (in human or animal studies) are chloracne, immune system suppression, and reproductive and developmental toxicity (Environment Agency 2009).		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral Note: The toxicity of compounds showing dioxin-line modes of action is considered additive, but the potency of such chemicals varies over orders of magnitude. Therefore when assessing PCDDs, PCDFs and dioxin like PCBs, the toxic equivalence (WHO-TEQ) of an individual compound relative to 2,3,7,8-TCDD (most toxic congener) is estimated by multiplying its estimated or measured exposure dose by the TEF value (see left hand column). The overall toxicity of a mixture of PCDDs and dioxin like compounds is then assessed as the sum of the who-TEQ exposures for the individual compounds present.	SGV (sum of PCDDs, PCDFs and dioxin like PCBs) Tolerable daily intake (TDIoral) Mean daily intake (MDIoral)	8 ug/kg DW Residential 8 ug/kg DW Allotment 240 ug/kg DW Commercial 1pg WHO-TEQ kg/bw/day 49pg WHO-TEQ/day	The SGVs apply to the sum of the soil concentrations for the individual congeners/ compounds list in the left hand column. The SGVs are derived assuming a generic profile or congener pattern that was based on the results of the UK Soil and Herbage Survey. The TDIoral is based on the low dose effects of 2,3,7,8-TCDD on sperm production and morphology in the offspring of treated rats (Environment agency 2009). The TDI oral applies to the sum of Who-toxic equivalences (TEQ) exposure estimates for all PCDDS and dioxin like compounds listed in the left hand column. The adult oral mean daily intake (MDIoral) from food and water combined is approx. 4 pg WHO –TEQ for the sum of PCDDs and dioxin like compounds.
Inhalation	Mean daily intake (MDIinh)	0.2pg TEQ /day.	No authoritative assessments of the health risks posed by inhalation or dermal exposures to PCDDs or dioxin-like compounds have been identified.
Dermal			
Background exposure (diet, air, drinking water etc)			

117Dioxins (PCDD), Furans (PCDFs) and dioxin like PCBs (PCBs)	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

118 Lead	Identified adverse health effects – acute			
	Identified adverse health effects – intermediate/chronic	Causal relationships for: Nervous system effects, cardiovascular effects, haematological effects, reproductive effects and likely causal relationships for renal effects, cancer and immune system effects (USEPA 2012).		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral Note: SGVs for lead have been withdrawn. Previously the value of 10 ug/L blood was selected by the EA as the HCV. In 2009 the EA withdrew the 2002 SGV report to be re-evaluated in the new CLEA framework. In 2011 the EA withdrew the published toxicology report for lead in light of new scientific evidence indicating that significant health effects could be observed at levels <10 ug/L blood.	LLTC1: Intake leading to blood lead concentration of 10 ug/l.	450 mg/kg Residential with home grown produce. 450 mg/kg Residential without home grown produce. 450 mg/kg Allotments. 750 mg/kg Commercial.	
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

119 2,3,7,8 – tetrachlorodibenzo-p-dioxin (TCDD)	Identified adverse health effects – acute	The onset of symptoms after acute exposure to TCDD-containing substances may include skin, eye, and respiratory tract irritation; headache; dizziness; and nausea. The most commonly reported symptom related to TCDD exposure in man has been chloracne. The acneform lesions of the skin may develop a few weeks after the exposure and may persist for over a year following the cessation of exposure. Thyroid disease and appendicitis have been associated with the chloracne. Other skin problems which have been reported include hyperpigmentation, hirsutism, increased skin fragility, and vesicular eruptions on exposed areas of the skin. Dosage estimate: To produce chloracne 0.13-0.31 ug 2,3,7,8-tetrachlorodibenzodioxin/kg (using EPA toxicity equivalence factors). Based on a Spanish family's olive oil supply becoming contaminated (1991). This is the first incident in which human toxicity is related primarily to ingestion of polychlorinated dibenzo-p-dioxins and for which estimates of dosage can be made. Hepatic dysfunction, perpheral neuropathy, fat metabolism disorders, elevated serum cholesterol, and porphyria cutanea tarda are the other findings associated most frequently with TCDD exposure in industrial settings. Effects of acute massive exposure in workers exposed to 2,3,7,8-TCDD in an industrial accident in Germany included bronchitis and laryngitis a few days after exposure, and hemorrhagic pleuritis 11 months after exposure.		
	Identified adverse health effects – intermediate/chronic	**Carcinogenicity:** 2,3,7,8-tetrachlorodibenzo-p-dioxin is carcinogenic to humans (Group 1). Cancers of gastrointestinal sites and of the lymphatic and hematopoietic tissue (Seveso incident, Italy, 1976). Increased risks of Hodgkins lymphoma, myeloid leukemia, and thyroid cancer were also reported among children who were 0-19 years old at the time of the Seveso accident. However, the differences in relative risks (RRs) for these cancer types between the Seveso residents and the control population did not reach statistical significance.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

120 Pentachlorodibenzo-p-dioxin.

Identified adverse health effects – acute	The most prominent acute manifestation of toxicity that has been shown in humans is chloracne. Data are not yet available to determine the amount, route, or duration of exposure that is necessary to cause chloracne. Dosage estimate: To produce chloracne 0.13-0.31 ug 2,3,7,8-tetrachlorodibenzodioxin/kg (using EPA toxicity equivalence factors). Based on a Spanish family's olive oil supply becoming contaminated (1991). This is the first incident in which human toxicity is related primarily to ingestion of polychlorinated dibenzo-p-dioxins and for which estimates of dosage can be made. Due to their lipophilicity, chlorinated dibenzo-p-dioxins (CDDs) can concentrate in human breast milk and can be transferred to infants through nursing. Other less consistency reported effects from dioxin exposure in humans include aesthenia, headaches, and pain in the extremities, peripheral neuropathy, ulcers, altered liver function, enzyme induction, altered lipid metabolism, and abnormal urinary porphyrin patterns. Immune system dysfunction and altered T-cell subsets have been reported by some investigators but have not been found by others.		
Identified adverse health effects – intermediate/chronic	B2; probable human carcinogen. BASIS: Hepatic tumours in mice and rats by gavage.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

121 Hexachlorodibenzo-p-dioxin.

Identified adverse health effects – acute	The most prominent acute manifestation of toxicity that has been shown in humans is chloracne. Data are not yet available to determine the (precise) amount, route, or duration of exposure that is necessary to cause chloracne. Dosage estimate: To produce chloracne 0.13-0.31 ug 2,3,7,8-tetrachlorodibenzodioxin/kg (using EPA toxicity equivalence factors). Based on a Spanish family's olive oil supply becoming contaminated (1991). This is the first incident in which human toxicity is related primarily to ingestion of polychlorinated dibenzo-p-dioxins and for which estimates of dosage can be made. Due to their lipophilicity, chlorinated dibenzo-p-dioxins (CDDs) can concentrate in human breast milk and can be transferred to infants through nursing. Other less consistently reported effects from dioxin exposure in humans include /asthenia/, headaches, and pain in the extremities, peripheral neuropathy, ulcers, altered liver function, enzyme induction, altered lipid metabolism, and abnormal urinary porphyrin patterns. Immune system dysfunction and altered T-cell subsets have been reported by some investigators but have not been found by others.		
Identified adverse health effects – intermediate/chronic	B2; probable human carcinogen. BASIS: Hepatic tumours in mice and rats by gavage.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

122 Octachlorodibenzo-p-dioxin

Identified adverse health effects – acute	The most prominent acute manifestation of toxicity that has been shown in humans is chloracne. Data are not yet available to determine the (precise) amount, route, or duration of exposure that is necessary to cause chloracne. Dosage estimate: To produce chloracne 0.13-0.31 ug 2,3,7,8-tetrachlorodibenzodioxin/kg (using EPA toxicity equivalence factors). Based on a Spanish family's olive oil supply becoming contaminated (1991). This is the first incident in which human toxicity is related primarily to ingestion of polychlorinated dibenzo-p-dioxins and for which estimates of dosage can be made. Due to their lipophilicity, chlorinated dibenzo-p-dioxins (CDDs) can concentrate in human breast milk and can be transferred to infants through nursing. Other less consistently reported effects from dioxin exposure in humans include asthenia/, headaches, and pain in the extremities, peripheral neuropathy, ulcers, altered liver function, enzyme induction, altered lipid metabolism, and abnormal urinary porphyrin patterns. Immune system dysfunction and altered T-cell subsets have been reported by some investigators but have not been found by others.		
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)	Based on monitoring of Canadian drinking water, the avg daily intake of Octachlorodibenzo-p-dioxin has been estimated to be 0.0001 ng/adult/day. Food: the daily intake of all polychlorinated dibenzodioxin isomers has been estimated to be 0.1397 ng/adult/day, the majority of which is octachlorodibenzo-p-dioxin. Based upon a Japanese market basket survey, the avg daily intake for **octachlorodibenzo-p-dioxin** has been estimated as 0.690 ng/adult/day. Based on food samples collected in Madrid, Spain during April 1995, the avg daily intake of **octachlorodibenzo-p-dioxin** has been estimated as 16,144.96 pg/person/day.		

123 2,3,7,8-Tetrachlorodibenzofuran.	Identified adverse health effects – acute	Following rice poisoning incident: Yusho incident 1968 and Yi-Chang incident 1979 over 10 and 9 months respectively, the following health effects were observed: Characteristic skin changes included marked enlargement, elevation and keratotic plugging of follicular orifices, comedo formation, acneform eruptions, hyperpigmentation, hyperkeratosis, and deformed nails. Eye discharge and other severe ocular effects including meibomian gland changes (enlargement, inflammation, hypersecretion of cheese-like material) and dark-coloured pigmentation of the conjunctivae and eyelids. Immune effects: frequent or more severe skin and respiratory infections and lowered resistance to illness. Irregular menstrual cycles and abnormal basal body temperature patterns. In babies born to mothers exposed: hyperpigmentation of skin, nails, gingivae, conjunctivitis, acne. Perinatal death due to pneumonia, bronchitis and prematurity and developmental (growth and neurobehavioural) impacts. Two studies of nursing infants suggest that ingestion of breast milk with a higher dioxin-furan toxic equivalence (TEQ) value may alter thyroid function.		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

124 Lead (elemental)

Identified adverse health effects – acute Exposure via: Ingestion and inhalation of dust and fume.	Lead affects the developing nervous system of children, and no safe blood lead level (BLL) in children has been identified. Elevated BLLs in childhood are associated with hyperactivity, attention problems, conduct problems, and impairment in cognition. Young children are at higher risk for environmental lead exposure from putting their hands or contaminated objects in their mouth. Lead has a negative effect on the central nervous system (CNS), However, knowledge of the CNS effects at low exposure is insufficient. Studies to date suggest that the primary anatomical site for lead effect on the brain is the endothelial cell of the blood-brain barrier. Although changes in microvascular morphology are not evident with low level exposure, there may be lead-induced biochemical and functional changes that permit lead entry to the brain. Experimental studies suggest that the immature endothelial cells forming capillaries of the developing brain are less resistant to the effects of lead than are capillaries from mature brains and permit fluid and cations to reach newly formed components of the brain, particularly neurons and astrocytes . The uptake of lead by the fetal brain during gestation is greater than after birth.		
Identified adverse health effects – intermediate/chronic Exposure via: Ingestion and inhalation of dust and fume.	Carcinogenicity: B2; probable human carcinogen. BASIS: Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumours with dietary and subcutaneous exposure to several soluble lead salts. Short term studies show that lead affects gene expression. Human evidence is inadequate (USEPA 2009). GENOTOXICITY**:** Lead has not been shown to be a mutagen on human cells except at levels thought to be toxic to cells. However, lead exposure has been associated with chromosomal changes, and ... a relationship /was shown/ between sister chromatid exchanges and lead exposure in workers. In a number of DNA structure and function assays, lead has been shown to affect the molecular processes associated with the regulation of gene expression. Other Chronic effects: Negative effects on memory, learning, and reaction times. Arterial hypertension is one of the physical complications of chronic lead exposure.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

125 Lead (compounds)

Identified adverse health effects – acute Exposure via: Ingestion and inhalation of dust and fume.	Lead compounds affect many biochemical processes including hemopoietic, neurological, metabolic and behavioral, with the developing human generally being more sensitive than the adult. Children who are exposed to lead in the womb may have a lower IQ, behavioural problems, nerve damage or delayed growth. Children are particularly sensitive to the effects of lead on the nervous system as their brains are still developing. Children exposed to lead during the first few years of life may have a lower IQ, behavioural problems or nerve damage. Increased lead exposure is also associated with neuropsychiatric disorders such as attention deficit hyperactivity disorder and antisocial behaviour. The effects of lead on the hemopoietic system result in decreased hemoglobin synthesis, and anemia has been observed in children at blood concentrations above 1.92 umol/L (40 ug/dL). The reproductive effects of lead in the male are limited to sperm morphology and count. In the female, some adverse pregnancy outcomes have been attributed to lead.		
Identified adverse health effects – intermediate/chronic Exposure via: Ingestion and inhalation of dust and fume.	Carcinogenicity: B2; probable human carcinogen. BASIS: Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumour sites. Short term studies show that lead affects gene expression. Human evidence is inadequate (USEPA 2009). Persons occupationally exposed to lead compounds are burdened with significantly higher 10-year risk of fatal cardiovascular incident than individuals from the same population not exposed to lead. Chronic exposure to high levels of lead can result in sclerotic changes and interstitial fibrosis, resulting in decreased kidney function. Long term exposure in the workplace to lead has been found to impair psychological and neurobehavioral functions and cause peripheral neuropathy.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)	An average daily intake of 231 ug lead for Finnish adult males and 178 ug lead for adult females was reported; this is consistent with a British study reporting 274 ug lead/day for young adults and with a Japanese study reporting 299 ug lead/day for adult males doing medium work (USEPA 1980). In 1990 it was estimated that North Americans ingested approximately 50 ug/day of lead through food, beverages and dust, with 30-50% of this amount via food and beverages (USEPA 2006).		

126 PAHs

Identified adverse health effects – acute Exposure via inhalation, ingestion and dermal contact	Skin dermatitis, regressive verrucae, chronic dermatitis & keratosis. Respiratory diseases (emphysema), impaired immune system function, decreased fertility, and adverse birth outcomes (reduced birth weight, altered growth and development. Cardiovascular disease. Effect on male reproductive health. Lungs - patch opacities, prominent bronchiovascular markings, and pleural effusions, asthma. Other symptoms included bloody vomit, breathing problems, chest pains, chest irritation, throat irritation, and cough.		
Identified adverse health effects – intermediate/chronic Exposure via inhalation, ingestion and dermal contact	Carcinogenicity: Cancers include lung, skin, bladder, mouth & throat, kidney, breast and blood cancers. Tumour location may be influenced by the means of exposure. The International Agency for Research on Cancer has classified several PAH mixtures as carcinogenic to humans, including exposure to PAHs in soot, coal pitch, or emissions from coal gasification, coke production, iron and steel founding, coal-tar distillation, or household combustion of coal or wood. The U.S. EPA IRIS program classified coke oven emissions as a human carcinogen. The U.S. National Toxicology Program 14th Report on Carcinogens classified coal tars, coal tar pitches, coke-oven emissions, and soots as known to be human carcinogens. (SRC) GENOTOXICITY : The formation of diol epoxides that covalently bind to DNA appears to be the primary mechanism of action for both genotoxicity and carcinogenicity of several of the unsubstituted PAHs that are genotoxins (benzo[a]pyrene, benz[a]anthracene, dibenz[a,h]anthracene, chrysene, benzo[b]fluoranthene, benzo[j]fluoranthene. There was insufficient evidence to draw meaningful conclusions regarding the genotoxic potential of benzo[g,h,i]perylene, although some evidence does exist. With regard to the unsubstituted PAHs that either lack a bay region configuration (acenaphthene, acenaphthylene, anthracene, fluorene, and pyrene) or appear to have a weakly reactive bay region (phenanthrene), there is no compelling evidence to suggest that they interact with or damage DNA. The five PAHs that appear to be exceptions to the bay region diol epoxide hypothesis are fluoranthene, benzo[k]fluoranthene, benzo[j]fluoranthene, and indeno[1,2,3-cd]pyrene (no bay region), and benzo[e]pyrene (two bay regions). The evidence does suggest, however, that fluoranthene possesses genotoxic properties while benzo[e]pyrene is either weakly mutagenic or nonmutagenic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

127 Napthalene

Identified adverse health effects – acute Exposure via inhalation, ingestion and dermal contact	General exposure: nausea, vomiting, diarrhe, blood in urine and jaundice. Hemolytic anaemia, agitation, lethargy, and seizures, haemotoxicity, renal failure and blockade, liver necrosis, peripheral neuropathy. Dermal contact: dermatitis, eye irritation. On ingestion: Confusion, altered sensorium, listlessness and lethargy, and vertigo. Muscle twitching, convulsions, decreased responses to painful stimuli, coma (prior to death). The lethal oral doses determined in cases of accidental poisoning are 5-15 g for adults and 2 g within two days for a six-year old child.		
Identified adverse health effects – intermediate/chronic Exposure via inhalation, ingestion and dermal contact	Carcinogenicity: The International Agency for Research on Cancer (IARC) and the Environmental Protection Agency have determined that naphthalene is a possible human carcinogen. The Department of Health and Human Services (DHHS), National Toxicology Program (NTP) listed naphthalene as "reasonably anticipated to be a human carcinogen" in the Report on Carcinogens.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

128 Acenaphthylene

Identified adverse health effects – acute	Data on the potential for acenaphthylene to produce toxic effects in humans were not available. A few animal toxicity studies of acenaphthylene are available in the published scientific literature, but the studies are inadequate to identify potential non-cancer health effects. Data on the potential for acenaphthylene to cause birth defects or reproductive effects in laboratory animals were not available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: The U.S. EPA IRIS program determined that data are inadequate for an assessment of the human carcinogenic potential of acenaphthylene based on no human data and inadequate data from laboratory animal studies. The potential for acenaphthylene to cause cancer in humans has not been assessed by the International Agency for Research on Cancer or the U.S. National Toxicology Program 14th Report on Carcinogens.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

129 Acenaphthene

Identified adverse health effects – acute	Data on the potential for acenaphthene to produce toxic effects in humans were not available. Liver toxicity was observed in laboratory animals fed high doses of acenaphthene over time. Data on the potential for acenaphthene to cause infertility, abortion, or birth defects in laboratory animals were not available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: The International Agency for Research on Cancer has determined that acenaphthene is not classifiable as to its carcinogenicity to humans due to lack of human data and inadequate studies in laboratory animals. The potential for acenaphthene to cause cancer in humans has not been assessed by the U.S. EPA IRIS program or the U.S. National Toxicology Program 13th Report on Carcinogens. (SRC) Class D.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

130 Fluorene

Identified adverse health effects – acute	Data on the potential for fluorene to produce toxic effects in humans were not available. Evidence of slight anemia and mild liver, kidney, and spleen changes were observed in laboratory animals following repeated oral exposure to high doses of fluorene. Decreased anxiety-like behaviours were observed in laboratory animals that repeated breathed air containing low levels of fluorene. No changes in motor activity or learning and memory were observed. Data on the potential for fluorene to cause birth defects or reproductive effects in laboratory animals were not available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Group 3: The agent is not classifiable as to its carcinogenicity to humans. IARC. D; not classifiable as to human carcinogenicity. US EPA.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

131 Phenanthrene

Identified adverse health effects – acute	Data on the potential for phenanthrene alone to produce toxic effects in humans were not available. Increased blood uric acid levels were associated with phenanthrene exposure in coke oven workers exposed to a mixture of PAHs. This indicates a potential risk for kidney stones or gout. Dermal/ inhalation: Based on data for other PAHs, irritation to skin, nose, and throat may occur after touching or inhaling phenanthrene. Increased sensitivity to sunlight and/or skin allergic reactions may occur following skin exposure. Animal effects: Enlarged heart (cardiac hypertrophy) in young rats following repeated injections of phenanthrene in the first 3 weeks after birth. Cardiac hypertrophy is a risk factor for heart disease. In zebrafish embryos exposed to phenanthrene abnormal heart development, irregular heartbeat and abnormal spine and eye development were observed. Other available animal studies are inadequate to identify potential non-cancer health effects.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Group 3: The agent is not classifiable as to its carcinogenicity to humans. IARC.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

132 Anthracene

Identified adverse health effects – acute	Anthracene is a mild skin irritant in laboratory animals, and increases the risk of skin damage ("sunburn") caused by ultraviolet radiation (photosensitizer). Data on the potential for anthracene to cause infertility, abortion, or birth defects are not available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Group 3: The agent is not classifiable as to its carcinogenicity to humans. IARC. D; not classifiable as to human carcinogenicity. US EPA. A single study detailing anecdotal evidence of a potential link between prolonged use of anthracene-containing oral laxatives and increased incidence of black pigments in the lining of the colon and rectum (melanosis). This condition has been associated with increased risk of colorectal tumours. However, the available data are not adequate to support a direct link between anthracene exposure and melanosis. Animal effects: No toxic effects were observed in laboratory animals following long term oral exposure to low-to-high doses of anthracene.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

133 Fluoranthene

Identified adverse health effects – acute	There is no human toxicity effect data for fluoranthene alone. Studies in humans have shown immunotoxic reactions within coke oven workers exposed to fluoranthene as a part of a mixture of PAHs, including perylene, pyrene, benzo(a)pyrene, chrysene, benz(a)anthracene, dibenz(a,h)anthracene and benzo(g,h,i)perylene. However, the contribution of fluoranthene to the immunotoxicity is unknown. Animal effects: Temporary incoordination, weakness, and decreased response to stimuli following a single high-dose oral exposure. Liver and kidney toxicity and evidence of anemia were observed in laboratory animals repeatedly given high oral doses of fluoranthene. Eye injury was observed in laboratory animals following direct contact. Increased fetal deaths were reported in laboratory animals following injections of fluoranthene during pregnancy. Data on the potential for fluoranthene to cause birth defects or reproductive effects in laboratory animals following oral, inhalation, or dermal exposure were not available.		
Identified adverse health effects – intermediate/chronic	Concinogenicity: Group 3: The agent is not classifiable as to its carcinogenicity to humans. IARC. D; not classifiable as to human carcinogenicity. US EPA.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

134 Pyrene

Identified adverse health effects – acute	Exposure to the sun may provoke an irritating effect of pyrene on skin and lead to chronic skin discoloration. There is no human toxicity effect data for pyrene alone. Studies in humans have shown immunotoxic reactions within coke oven workers exposed to pyrene as a part of a mixture of PAHs, including perylene, fluoranthene, benzo(a)pyrene, chrysene, benz(a)anthracene, dibenz(a,h)anthracene and benzo(g,h,i)perylene. However, the contribution of pyrene to the immunotoxicity is unknown. Animal effects: Kidney damage was observed in mice following exposure to high oral doses of pyrene. Toxicity was not observed in any other major organ system. Dermal exposure to pyrene resulted in changes in blood, weight loss, skin irritation, skin discoloration, and sensitivity to sunlight. Data on the potential for pyrene to cause infertility, abortion, or birth defects in laboratory animals were not available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Group 3: The agent is not classifiable as to its carcinogenicity to humans. IARC.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

135 Benz(a)anthracene

Identified adverse health effects – acute	Data on the potential for benz(a)anthracene alone to produce toxic effects in humans were not available. There is no human toxicity effect data for benz(a)anthracene alone. Studies in humans have shown immunotoxic reactions within coke oven workers exposed to benz(a)anthracene as a part of a mixture of PAHs, including perylene, fluoranthene, benzo(a)pyrene, chrysene, pyrene, dibenz(a,h)anthracene and benzo(g,h,i)perylene. However, the contribution of benz(a)anthracene to the immunotoxicity is unknown. Animal Effects: A few laboratory animal toxicity studies of benz(a)anthracene are available in the published scientific literature, but the studies are inadequate to identify potential non-cancer health effects. Data on the potential for benz(a)anthracene to cause infertility, abortion, or birth defects in laboratory animals were not available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: B2; probable human carcinogen. IARC: Group 2B: The agent is possibly carcinogenic to humans. ACGIH: A2; Suspected human carcinogen.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

136 Chrysene

Identified adverse health effects – acute	Data on the potential for chrysene alone to produce toxic effects in humans were not available. There is no human toxicity effect data for chrysene alone. Studies in humans have shown immunotoxic reactions within coke oven workers exposed to chrysene as a part of a mixture of PAHs, including perylene, fluoranthene, benzo(a)pyrene, benz(a)anthracene, pyrene, dibenz(a,h)anthracene and benzo(g,h,i)perylene. Additionally, several studies have shown increased mortality due to lung cancer in workers exposed to mixtures of PAHs, including chrysene, benzo(a)pyrene, benz(a)anthracene, benzo(b)fluoranthene, and dibenz(a,h)anthracene, such as coke oven emissions, roofing-tar emissions, and cigarette smoke. However, the contribution of chrysene to the immunotoxicity and carcinogenicity of these mixtures cannot be determined. Animal effects: Increased liver weight was observed in some laboratory animals exposed to moderate doses for a short period. Injection studies in laboratory animals do not indicate that exposure to chrysene affects the immune system.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: B2; probable human carcinogen. IARC: Group 3: The agent is not classifiable as to its carcinogenicity to humans. ACGIH: A3: Confirmed animal carcinogen with unknown relevance to humans.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

137 Benzo(a)pyrene

Identified adverse health effects – acute	Data on the potential for benzo(a)pyrene alone to produce toxic effects in humans were not available. However, several toxic effects have been associated with occupational exposure to PAH mixtures containing benzo(a)pyrene. Non-cancer health effects potentially associated with benzo(a)pyrene exposure in PAH mixtures include skin dermatitis, respiratory diseases such as emphysema, impaired immune system function, decreased fertility, and adverse birth outcomes (e.g., reduced birth weight, altered growth and development). Animal effects: oral and/or inhalation exposure effects include reduced resistance to disease, reproductive effects (reduced fertility and damage to reproductive organs), and developmental effects (decreased survival, decreased birth weight, skeletal defects, and altered nervous system, reproductive system, and heart development). It has been well demonstrated that polycyclic aromatic hydrocarbons (PAHs) can cause reproductive toxicity, and shorter telomere length in sperm may be one of the factors causing male infertility. Exposure to cigarette smoke containing PAH during pregnancy is deleterious to fetal development as reflected by reduced neonatal weight at birth.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: Carcinogenic to humans. ACGIH: A2; Suspected human carcinogen. IARC: Benzo[a]pyrene is carcinogenic to humans (Group 1). Carcinogenic effects have been associated with occupational exposure to PAH mixtures containing benzo(a)pyrene. Lung and skin cancer were the primary cancers observed, but there is also evidence of increased risk for bladder, mouth and throat, and blood cancers. However, the contribution of benzo(a)pyrene to the toxic effects of PAH mixtures cannot be determined. Genotoxicity: Benzo(a)pyrene is genotoxic		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

138 Indeno(1,2,3-cd)pyrene	Identified adverse health effects – acute	Data on the potential for indeno(1,2,3-cd)pyrene alone to produce toxic effects in humans were not available. No data regarding the potential for non-cancer toxic effects in laboratory animals were available. Data on the potential for indeno(1,2,3-cd)pyrene to cause infertility, abortion, or birth defects in laboratory animals were not available.		
	Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: B2; probable human carcinogen. IARC: Group 2B: The agent is possibly carcinogenic to humans.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			
139 Dibenz(a,h)anthracene	Identified adverse health effects – acute	Blood chemistry effects were noted among coke oven workers inhaling a high concentration of a mixture polyaromatic hydrocarbons (PAHs) which included dibenz(a,h)anthracene.		
	Identified adverse health effects – intermediate/chronic	Carcinogenicity: IARC: Group 2A: The agent is probably carcinogenic to humans. US EPA: B2; probable human carcinogen.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

140
Benzo(g,h,i)perylene

Identified adverse health effects – acute	Data on the potential for benzo(ghi)perylene alone to produce toxic effects in humans were not available. Studies in humans have shown changes in immune system components in the blood in coke oven workers exposed to a mixture of PAHs, including fluoranthene, perylene, pyrene, benzo(a)pyrene, chrysene, benz(a)anthracene, dibenz(a,h)anthracene, and benzo(ghi)perylene. However, the contribution of benzo(ghi)perylene to the toxic effects of this mixture cannot be determined. No data regarding the potential for non-cancer toxic effects in laboratory animals were available.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: D; not classifiable as to human carcinogenicity. IARC: Group 3: The agent is not classifiable as to its carcinogenicity to humans. Genotoxicity: Benzo(g,h,i) is genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

141
Benzo(b)fluoranthene

Identified adverse health effects – acute	Data on the potential for benzo(b)fluoranthene alone to produce toxic effects in humans were not available. Several studies have shown increased mortality due to lung cancer in workers exposed to mixtures of PAHs, including benzo(a)pyrene, chrysene, benz(a)anthracene, benzo(b)fluoranthene, and dibenz(a,h)anthracene, such as coke oven emissions, roofing-tar emissions, and cigarette smoke. However, the contribution of benzo(b)fluoranthene to the carcinogenicity of the mixtures cannot be determined. Animal effects: Impaired sperm production in male offspring of laboratory mice exposed to low levels of benzo(b)fluoranthene during pregnancy. No additional data on the potential for benzo(b)fluoranthene to cause general toxicity, birth defects, or reproductive effects in laboratory animal were available. Dermal contact may cause irritation or skin allergy, which is greatly aggravated by sunlight on contaminated skin. Fume exposure may cause irritation and a reaction greatly aggravated by sunlight during or shortly following exposure. Direct contact or "fumes" can cause irritation of the nose, throat, and bronchial tubes; skin irritation, redness, and possible swelling.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: B2; probable human carcinogen. ACGIH: A2; Suspected human carcinogen. IARC: Group 2B: The agent is possibly carcinogenic to humans. Genotoxicity: Benzo(b)fluoranthene is genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

142
Benzo(k)fluoranthene

Identified adverse health effects – acute	Data on the potential for benzo(k)fluoranthene to produce toxic effects in humans were not available. Animal toxicity studies are inadequate to identify the potential for benzo(k)fluoranthene to cause general toxicity, birth defects, or reproductive effects.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: US EPA: B2; probable human carcinogen. IARC: Group 2B: The agent is possibly carcinogenic to humans. Genotoxicity: Benzo(k)fluoranthene is genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

143 Methyl isocyanate

Identified adverse health effects – acute	Acute symptoms include skin and eye injuries (can cause permanent eye damage), asthma, chest pain, pulmonary edema and damage to alveoli, dyspnea, respiratory failure, and death. Known to be highly reactive and acutely toxic to human beings. Irritation of the eyes, nose and throat and lacrimation from 2ppm. Unbearable exposure at 21ppm. Symptoms of overexposure include coughing, mucous secretions, chest pain, dyspnea, asthma, eye and skin injury. Inhalation: Cough. Laboured breathing. Shortness of breath. Sore throat. Vomiting; Dermal: May be absorbed. Redness. Pain. Burning sensation. Loss of vision; Ingestion--Abdominal pain. Burning sensation. Shock or collapse.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Potentially an increased risk of cancer. Not listed by IARC. Genotoxicity: Potentially genotoxic. Repeated or prolonged contact may cause skin sensitization. Causes toxicity to human reproduction or development. Cognitive impairment. Neurological and psychiatric symptoms.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

144 Ethyl isocyanate

Identified adverse health effects – acute	Not specific to Ethyl-isocyanate: Isocyanates are irritating to the skin and the mucous membranes, the skin conditions ranging from localized itching to more or less widespread eczema. The commonest and most serious troubles are those affecting the respiratory systems. Isocyanates produce lung disease in manufacture of plastics and chemical industry. Acute effects include airway irritation, cough and dyspnea; chronic effects include asthma, and reduced pulmonary function. Occupational asthma and bronchial hyperresponsiveness. Methyl isocyanate: cough, dyspnea, chest pain and an increase in secretions.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: not listed by IARC Genotoxicity: Lack on information (methyl isocyanate may be genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

145 phenyl isocyanate

Identified adverse health effects – acute	Phenyl isocyanate is a potent chemical sensitizer. Not specific to phenyl isocyanate: Exposure to isocyanates irritates, mucous membranes, eyes (lachrymator) and respiratory tract. Dermal/ inhalation/ oral: Contact dermatitis (rash, itching, hives and swelling of the extremities). Isocyanate induced hypersensitivity pneumonitis symptoms are flu like which includes shortness of breath, nonproductive cough, fever, chills sweats and malaise and nausea. Irreversible decline in pulmonary function and interstitial fibrosis have been observed. At high doses isocyanates affect the mucous membranes of the respiratory tract and may lead to fatal pulmonary edema or chromic catarrh.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: not listed by IARC Genotoxicity: Potentially genotoxic – limited information.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

146 Hexamethylene diisocyanate

Identified adverse health effects – acute	Oral exposure – irritation of the gastrointestinal tract. Exposure to low & often even unmeasurable isocyanate concentrations results in sensitization of organisms either by local injury accompanied by the formation of heterogeneous protein to which the organism becomes allergic or by secondary bacterial. At high doses the isocyanates affect the mucous membranes of the respiratory tract and may lead to fatal pulmonary edema or chronic catarrh. Most of the early reports of respiratory illness in workers exposed to diisocyanates described bronchial asthma or chronic bronchitis often considered to involve evidence of sensitization. Bronchospasm. Alveolitis. Isocyanate-induced hypersensitivity pneumonitis (HP). Symptoms are flu-like, including shortness of breath, non-productive cough, fever, chills, sweats, malaise, and nausea. Haematological – effect on cell counts.		
Identified adverse health effects – intermediate/chronic	After the onset of HP, prolonged and/or repeated exposures may lead to an irreversible decline in pulmonary function and lung compliance and to the development of diffuse interstitial fibrosis.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Occupational exposure: Threshold Limit Values (ACGIH) Excursion limit Recommendation	0.005 ppm	8 hr time weighted average May exceed 5x the TLV-TWA for no more than 30 minutes of work a day and under no circumstances should they exceed 5x the TLV-TWA.
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

147 Toluene-2,4-diisocyanate

Identified adverse health effects – acute	Exposure to isocyanates is irritating to the skin, mucous membranes, eyes, and respiratory tract (coughing, wheezing, shortness of breath, tightness in the chest, and nocturnal awakening) and cause gastrointestinal tract symptoms. The most common adverse health outcome associated with isocyanate exposure is asthma due to sensitization; less prevalent are contact dermatitis (both irritant and allergic forms) and hypersensitivity pneumonitis (HP). Contact dermatitis can result in symptoms such as rash, itching, hives, and swelling of the extremities. The initial symptoms associated with isocyanate-induced hypersensitivity pneumonitis (HP) are flu-like, including shortness of breath, non-productive cough, fever, chills, sweats, malaise, and nausea. At high doses the isocyanates affect the mucous membranes of the respiratory tract and may lead to fatal pulmonary edema or chronic catarrh. Exposure to low & often even unmeasurable isocyanate concentrations results in sensitization of organisms either by local injury accompanied by the formation of heterogeneous protein to which the organism becomes allergic or by secondary bacterial. Inhalation: Organic isocyanates are particularly poisonous when breathed. Furthermore the toxicity varies according to physical & chemical nature of isocyanate. Among most poisonous are toluene 2,4- & 2,6-diisocyanates. Diisocyanates are potent pulmonary sensitizers which cause bronchospasm, even in patients without prior airway hyper-reactivity. At high doses, toluene diisocyanate may act directly on bronchial mucosa by interfering with cholinergic and adrenergic mechanisms. Immunological effects.		
Identified adverse health effects – intermediate/chronic	After the onset of HP, prolonged and/or repeated exposures may lead to an irreversible decline in pulmonary function and lung compliance and to the development of diffuse interstitial fibrosis. Respiratory sensitization usually takes a few months to several years of exposure to develop. Carcinogenicity: IARC: Toluene diisocyanates are possibly carcinogenic to humans (Group 2B). Toluene Diisocyanates: Reasonably anticipated to be human carcinogens.U.S. Department of Health & Human Services/National Toxicology Program. ACGIH: A4: Not classifiable as a human carcinogen. /Toluene-2,4- or 2,6-diisocyanate (or as a mixture). Genotoxicity: Potentially genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

148 Toluene-2, 6-diisocyanate

Identified adverse health effects – acute	Exposure to isocyanates is irritating to the skin, mucous membranes, eyes, and respiratory tract (coughing, wheezing, shortness of breath, tightness in the chest, and nocturnal awakening) and cause gastrointestinal tract symptoms. The most common adverse health outcome associated with isocyanate exposure is asthma due to sensitization; less prevalent are contact dermatitis (both irritant and allergic forms) and hypersensitivity pneumonitis (HP). Contact dermatitis can result in symptoms such as rash, itching, hives, and swelling of the extremities. The initial symptoms associated with isocyanate-induced hypersensitivity pneumonitis (HP) are flu-like, including shortness of breath, non-productive cough, fever, chills, sweats, malaise, and nausea. At high doses the isocyanates affect the mucous membranes of the respiratory tract and may lead to fatal pulmonary edema or chronic catarrh. Exposure to low & often even unmeasurable isocyanate concentrations results in sensitization of organisms either by local injury accompanied by the formation of heterogeneous protein to which the organism becomes allergic or by secondary bacterial. Inhalation: Organic isocyanates are particularly poisonous when breathed. Furthermore the toxicity varies according to physical & chemical nature of isocyanate. Among most poisonous are toluene 2,4- & 2,6-diisocyanates. Diisocyanates are potent pulmonary sensitizers which cause bronchospasm, even in patients without prior airway hyper-reactivity. At high doses, toluene diisocyanate may act directly on bronchial mucosa by interfering with cholinergic and adrenergic mechanisms. Immunological effects.		
Identified adverse health effects – intermediate/chronic	After the onset of HP, prolonged and/or repeated exposures may lead to an irreversible decline in pulmonary function and lung compliance and to the development of diffuse interstitial fibrosis. Respiratory sensitization usually takes a few months to several years of exposure to develop. Carcinogenicity: IARC: Toluene diisocyanates are possibly carcinogenic to humans (Group 2B). Toluene Diisocyanates: Reasonably anticipated to be human carcinogens.U.S. Department of Health & Human Services/National Toxicology Program. ACGIH: A4: Not classifiable as a human carcinogen. /Toluene-2,4- or 2,6-diisocyanate (or as a mixture). Genotoxicity: Potentially genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

149 4,4'-methylene diphenyl diisocyanate

Identified adverse health effects – acute	Methylenediphenyl diisocyanate is irritating to skin, eyes, and respiratory passages. Nose and throat irritation has also been observed. A few cases of alveolitis have been reported in workers exposed to methylenediphenyl diisocyanate vapours. Contact allergy and allergic contact eczema have been reported in workers exposed to methylenediphenyl diisocyanate. Cases of asthmatic breathing have been observed in workers. Lung fibrosis is also been observed in workers exposed to methylenediphenyl diisocyanate. Children living in proximity of an accidental methylenediphenyl diisocyanate release had signs of sore throat, dizziness, nausea and breathing difficulties methylenediphenyl diisocyanate is an allergic sensitizer. Workers in occupational settings have the potential for inhalation or skin contact with particles of methylenediphenyl diisocyanate. 4,4-Methylenediphenyl diisocyanate is irritating to the skin, eyes and respiratory tract and induces asthma in humans. Asthmatic breathing, retrosternal soreness, constriction of the chest, cough, retrobulbar pain, depression, headache, nasal discharge, and insomnia. Exposure to methylenediphenyl diisocyanate was more commonly associated with isocyanate-induced alveolitis (restrictive reduction in lung function, interstitial fibrosis, cell changes) than exposure to toluene diisocyanate or HDI. Strong mucous membrane irritation causes eye, pulmonary and gastrointestinal tract symptoms. Potent pulmonary sensitizers which causes bronchospasm, even in patients without prior airway hyper-reactivity. Diisocyanate compounds act either as inducers of nonspecific bronchial hyperreactivity or as direct pharmacologic agonists.		
Identified adverse health effects – intermediate/chronic	Respiratory sensitization usually takes a few months to several years of exposure to develop. Long-term exposure tends to restrict pulmonary function and cause decrease in CO single breath transfer factor. Carcinogenicity: IARC: not classifiable as to its carcinogenicity in humans (Group 3). US EPA: Not classifiable (Group D). Genotoxicity: Potentially genotoxic		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Threshold limit values (ACGIH) Recommended NIOSH Recommended exposure limit	0.005 ppm 0.05 mg/cu m(0.005ppm)	8 hr time weighted average May exceed 5x the TLV-TWA for no more than 30 minutes of work a day and under no circumstances should they exceed 5x the TLV-TWA. 10 hour time weighted Average:
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

150 Isophorone diisocyanate

Identified adverse health effects – acute	Occupational asthma has been associated with isophorone diisocyanate exposure in the workplace and may occur through inhalation and dermal contact with this compound at workplaces where isophorone diisocyanate is produced or used. Cross sensitivity can occur between isophorone diamine and isophorone diisocyanate which are chemically related in workers. Human volunteers exposed to aerosol of isophorone diisocyanate exhibited irritation of the mucous membranes of the eyes and nose; as concentrations increased strong irritation of the mucous membranes of the eyes and breathing passages was observed. Strong mucous membrane irritation causes eye, pulmonary and gastrointestinal tract symptoms. Contact dermatitis, both irritant and allergic forms, have been observed in individuals exposed to isocyanates. Isocyanate induced asthma and sensitization will exhibit traditional symptoms of acute airway obstruction, coughing, wheezing, shortness of breath, tightness of the chest and nocturnal awakening. Asthma reactions may result. Isocyanate induced hypersensitivity pneumonitis symptoms are flu like including shortness of breath, non-productive cough, fevers, chills, sweats, malaise and nausea. Irreversible decrease in pulmonary function and the development of interstitial fibrosis may result. Exposure to high doses of isocyanates may produce fatal pulmonary edema or catarrh. The initial symptoms associated with isocyanate-induced hypersensitivity pneumonitis (HP) are flu-like, including shortness of breath, non-productive cough, fever, chills, sweats, malaise, and nausea.		
Identified adverse health effects – intermediate/chronic	Respiratory sensitization usually takes a few months to several years of exposure to develop. After the onset of hypersensitivity pneumonitis, prolonged and/or repeated exposures may lead to an irreversible decline in pulmonary function and lung compliance and to development of diffuse interstitial fibrosis. Carcinogenicity: IARC: not listed.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

151 Hydrogen cyanide

Identified adverse health effects – acute	Hydrogen cyanide poisoning is fatal following high inhalation, oral, or dermal exposures due to severe depression of the central nervous system. Neurological effects observed following non-fatal exposures include light-headedness, dizziness, headache, confusion, weakness, and unconsciousness. Permanent nervous system effects may occur, including psychiatric conditions and Parkinson's-like symptoms. Upper respiratory irritation, cough, altered sense of smell, nasal congestion, nose bleed, coughing up blood, and shortness of have been also reported in workers exposed to non-fatal amounts of hydrogen cyanide. Altered thyroid hormone levels, nerve damage, headache, dizziness, weakness, irritability, weight loss, decreased appetite, and gastrointestinal complains have been reported with low-level exposure overtime. A high incidence of thyroid disease has been observed in newborns of mothers living in the tropics and who have ingested a large amount of cassava root, which contains cyanide and thiocyanate, during pregnancy. No data on potential reproductive effects in humans after exposure to hydrogen cyanide were available. In laboratory animals, damage to male reproductive organs, birth defects, and delayed development were observed following exposure to cyanide in the diet. The primary targets of cyanide toxicity in humans are the cardiovascular, respiratory, and central nervous systems. Sequelae after severe acute intoxications may include neuropsychiatric manifestations and Parkinson type disease. Cyanide from tobacco smoke has been implicated as a contributing factor in tobacco alcohol amblyopia.. Cyanides are weakly irritating to the skin and eye. Typical signs of acute cyanide poisoning include tachypnea, headache, vertigo, lack of motor coordination, weak pulse, cardiac arrhythmias, vomiting, stupor, convulsions, and coma. Only occasionally has reference been made to irritation of eye, conjunctivitis, or superficial keratitis developing after chronic exposure to hydrogen cyanide gas.		
Identified adverse health effects – intermediate/chronic	The endocrine system is a potential target for long term toxicity, as a function of continued exposure to thiocyanate, which prevents the uptake of iodine in the thyroid and acts as a goitrogenic agent. Long term exposure to lower concentrations of cyanide in occupational settings can result in a variety of symptoms related to central nervous system effects. Chronically exposed workers may complain of headache, eye irritation, easy fatigue, chest discomfort, palpitations, loss of appetite, and nosebleeds. Carcinogenicity: Not listed by IARC. Genotoxicity: No information.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Adult lethal dose	50 mg	
Inhalation	Average fatal concentration for humans	546 ppm hydrogen cyanide	After 10 minute exposure
Dermal	Average LD50 Fatal concentrations	100 mg/kg bw 7000 to 12,000 mg/cu m	Estimated for humans. After 5 minutes exposure of workers

152 Tri-(2-chloroisopropyl) phosphate	Identified adverse health effects – acute	Respiratory tract irritation. Harmful if absorbed through skin. Causes skin irritation. Causes eye irritation. Animal effects: depression and intermittent muscle spasms. Higher dose levels induced spasms, salivation, ataxia and spasmodic jumping. Increased or decreased activity, oral, nasal, perianal and ocular discharge, hunching, rough coat, aggression, diarrhea, dehydration, decreased body temperature, alopecia, emaciation, decreased excreta, anorexia, and sporadic twisting and teeth chattering.		
	Identified adverse health effects – intermediate/chronic	Genotoxicity: does not appear to be genotoxic.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

153 Triphenyl phosphate	Identified adverse health effects – acute	Data on the potential for triphenyl phosphate to cause toxic effects in humans are limited. In the only available study, there was no evidence of toxic effects in workers following exposure to low air levels of triphenyl phosphate over time. It causes a delayed peripheral neuritis involving motor neurons, resulting in a flaccid paralysis, particularly of the distal muscles. No sensory disturbances. Potential symptoms of overexposure are minor changes in blood enzymes. Animal Effects: No toxic effects were noted in laboratory animals that breathed high air levels of triphenyl phosphate. At extremely high doses it resulted in liver, kidney and central nervous system congestion and hemorrhage. Some animals died.		
	Identified adverse health effects – intermediate/chronic	Decreased body weight and elevated liver weight were the only effects observed in laboratory animals exposed to low oral doses over time. Carcinogenicity: Not assessed by IARC or US National Toxicology Program. ACGIH: A4; Not classifiable as a human carcinogen.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

154 Tris(2-butoxyethyl) phosphate	Identified adverse health effects – acute	Dermal: Direct contact with tri(2-butoxyethyl) phosphate can produce mild irritation to skin or eyes. Allergic skin reactions to tri(2-butoxyethyl) phosphate were not found in a study with human volunteers. A repeat human insult patch test indicated no skin sensitization and minimal skin irritation. Oral: Swallowing a large amount of tri(2-butoxyethyl) phosphate can cause nervous system tissue damage and death. Animal effects: Microscopic changes to the liver and nervous system tissues were found in laboratory animals repeatedly given high doses of tri(2-butoxyethyl) phosphate by mouth or in food. No abortions or birth defects in offspring were found after high doses of tri(2-butoxyethyl) phosphate were given by mouth to pregnant laboratory animals. Pregnant animals given the high doses could not control muscle movements and had decreased body weight gain.		
	Identified adverse health effects – intermediate/chronic	Carcinogenicity: Not assessed by IARC or US National Toxicology Program.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

155 Triethyl phosphate

Identified adverse health effects – acute	Tributyl phosphate is primarily an irritant. Large doses might produce muscle weakness, pulmonary edema, and cholinergic symptoms. The most likely route of significant occupational exposure is dermal. Vapors may cause headache, irritation to the eyes and mucous membranes of the nose. Hot vapour is more irritating than cold. Animal effects: This agent has caused pulmonary edema in animals. Skin irritation and dermatitis may be seen. Muscle twitching and weakness have been reported in animals.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Not listed by IARC.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD (human)	0.5-5 g/kg, between 1 oz & 1 pint (or 1 lb) for 70 kg person (150 lb)	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

156 Trioctyl phosphate

Identified adverse health effects – acute	Dermal: A skin and eye irritant. However, in a study on human volunteers, no skin irritation was reported. Animal effects: High daily doses of trioctyl phosphate decreased body weight and microscopic changes to the liver, thyroid, and stomach were also observed.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Not been assessed by the EPA IRIS program, the International Agency for Research on Cancer, or the U.S. National Toxicology Program. (SRC)		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

157 Benzene

Identified adverse health effects – acute	Dermal/ inhalation: Direct exposure of the eyes, skin, or lungs to benzene can cause tissue injury and irritation. Inhalation: drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness; death at very high levels. Acute hemorrhagic pneumonitis. Ingestion: vomiting, stomach irritation, dizziness, sleepiness, convulsions, rapid heart rate, coma, and death at very high levels. Acute deaths from benzene exposure at high concentrations have been due to ventricular fibrillation caused by exertion and release of epinephrine. Occasionally hemorrhages in retina & in conjunctiva are found in systemic poisoning by benzene. In rare instances neuroretinal edema & papilledema have been described accompanying retinal hemorrhages. Acute granular tracheitis, laryngitis, bronchitis, and massive hemorrhages of the lungs.		
Identified adverse health effects – intermediate/chronic	With long-term exposure, the major effect of benzene is on the blood. Benzene causes harmful effects on the bone marrow in animals and humans. It can cause a decrease in red blood cells leading to anemia. Significant reductions in WBC, RBC, and platelet counts. Benzene can also affect the immune system, increasing the chance for infection. Some women who breathed high levels of benzene for many months had irregular menstrual periods and a decrease in the size of their ovaries. Chronic benzene toxicity is expressed as bone marrow depression resulting in leucopenia, anemia, or thrombocytopenia (leukemogenic action). With continued exposure the disease progresses to pancytopenia resulting from bone marrow aplasia. In some studies, low birth weights, delayed bone formation, and bone marrow damage were found in offspring of animals who breathed benzene regularly during pregnancy. Carcinogenicity: Benzene causes cancer in humans. Exposure over a long time can cause leukemia. IARC: Group 1 The chemical is carcinogenic to humans. ACGIH: A1; Confirmed human carcinogen. US EPA: Cancer Classification: Carcinogenic to Humans Genotoxicity: Benzene is genotoxic in humans: a significantly increased frequency of chromatid and isochromatid breaks in the cultured lymphocytes of exposed workers has been reported, as well as a significant increase of peripheral blood lymphocyte chromosomal aberrations.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

158 Polybrominated biphenyls

Identified adverse health effects – acute	Neurological symptoms. E.g. prose recall, short-term memory, concentration, and cognitive flexibility. Plus, joint pain, headache, muscle pain, dizziness, backache, joint stiffness, blurred vision, somnolence, nervousness, rash of various kinds, abdominal pain, irritability and diarrhoea. Reported symptoms of overexposure include impaired immune system; hypothyroidism; neurological effects, headache, joint stiffness, memory loss; chloracne-like lesions. Effects for PCBs: Dermal: chloracne, simple erythematous eruptions with pruritus, acute eczematous contact dermatitis, burning sensation and edema of the face and hands, thickening of the skin, pigmentation of skin and nails, excessive eye discharge, swelling of eyelids, and distinctive hair follicles. Liver effects: in persons with systemic intoxication, usual signs and symptoms are nausea, vomiting, weight loss, jaundice, edema, and abdominal pain. Neurologic effects - headache, dizziness, depression, and nervousness may occur. Muscle and joint pain have been observed. Gastrointestinal effects - severe abdominal pain, nausea, vomiting, and diarrhea have been reported following acute and chronic exposures. Fetotoxic effects - Fetotoxic effects have been reported following maternal exposure in both humans and experimental animals.		
Identified adverse health effects – intermediate/chronic	For PCBs: Irritant of the eyes and mucous membranes and is toxic to the liver. Systemic effects are: abdominal pain, anorexia, nausea, chloracne, edema or burning sensation of the face and hands, and eczematous rash on the legs and hands. Metabolic and immune effects. Carcinogenicity: Polybrominated Biphenyls reasonably anticipated to be human carcinogens. DHHS/NTP. IARC: 2A Polybrominated biphenyls are probably carcinogenic to humans.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

159
Hexabromodiphenyl

Identified adverse health effects – acute	Effects for PCBs (see 125)		
Identified adverse health effects – intermediate/chronic	Effects for PCBs (see 125) Carcinogenicity: IARC: 2A Polybrominated biphenyls are probably carcinogenic to humans.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

160 2,2',6,6'-tetrabromobisphenol A	Identified adverse health effects – acute	Data on the potential toxic effects of 2,2',6,6'-tetrabromobiphenol A in humans is limited. Exposure to 2,2',6,6'-tetrabromobiphenol A may result in changes to thyroid hormones, but data are too limited to know for sure. Animal effects: High concentrations decreased activity, squinting, slight changes in breathing, and skin redness occurred. Mild changes in blood components and biochemistry were the only effects observed in adult animals exposed to high oral doses for shorter durations.		
	Identified adverse health effects – intermediate/chronic	Lesions in the uterus, ovary, liver, kidney, and stomach were observed in laboratory animals following lifetime oral exposure. Kidney lesions, altered behaviour and learning, impaired hearing, and altered hormone levels and delayed sexual development were observed in animals exposed to 2,2',6,6'-tetrabromobiphenol A during the first couple weeks of life. Carcinogenicity: Increased incidence of tumours in the testes, uterus, liver, and intestine were observed in laboratory animals following lifetime dietary exposure to 2,2',6,6'-tetrabromobiphenol A. IARC 2A: 2,2',6,6'-tetrabromobiphenol A is probably carcinogenic to humans based on sufficient evidence in animals. The potential for 2,2',6,6'-tetrabromobiphenol A to cause cancer in humans has not been assessed by the U.S. EPA IRIS program or the U.S. National Toxicology Program 14th Report on Carcinogens. (SRC)		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

161 Polybrominated diphenyl ethers	Identified adverse health effects – acute	Dermal: Skin irritation: very slight or mild erythema. Fetal neurodevelopmental effects in relation to cord blood polybrominated diphenyl ethers concentrations. In utero exposure to low doses of polybrominated diphenyl ether may result in lower birth weight and shorter birth length.		
	Identified adverse health effects – intermediate/chronic	Workers exposed to decabromodiphenyl ether and polybrominated biphenyls experienced higher than normal prevalence of primary hypothyroidism and a significant reduction in sensory and fibula motor velocities; no other dermatological or neurological effects were seen. It could not be determined whether the effects were caused by decabromodiphenyl ether and polybrominated biphenyls. Effects on menstrual cycle characteristics – but results are not conclusive. Potential reproductive effects. Endocrine effects - in vivo metabolism of polybrominated diphenyl ethers may produce more potent pseudoestrogens. Genotoxicity: Not believed to be genotoxic.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

162 Decabromodiphenyl ether

Identified adverse health effects – acute	Dermal: Very slight or mild erythema (redness of skin) in humans. Thyroid hormone changes have been reported in some workers exposed decabromodiphenyl (along with other chemicals). Animal effects: Liver, thyroid, spleen, and pancreas damage occurred in laboratory animals repeatedly fed low-to-high doses of decabromodiphenyl ether. Altered behavior was observed in young laboratory mice after a single low oral dose of decabromodiphenyl ether shortly after birth.		
Identified adverse health effects – intermediate/chronic	Carcinogenicity: Liver and thyroid tumours occurred in laboratory animals fed very high doses of decabromodiphenyl ether for their lifetime. U.S. EPA IRIS program (Class C): Decabromodiphenyl ether is a potential human carcinogen. IARC (Group 3): Decabromobiphenyl ether is not classifiable as to its carcinogenicity to humans. The potential for decabromobiphenyl ether to cause cancer in humans has not been assessed by the U.S. National Toxicology Program 13th Report on Carcinogens		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

163 Octabromodiphenyl ethers

Identified adverse health effects – acute	Effects on menstrual cycle characteristics – but results are not conclusive. Animal Effects: Liver is the key target organ in animals. Thyroid effects. Developmental/fetal effects.		
Identified adverse health effects – intermediate/chronic	No chronic or carcinogenicity studies in animals are available. Genotoxicity: Not believed to be genotoxic in animals.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50 (rat)	>28,000 mg/kg	
Inhalation			
Dermal	LD50 (rabbits)	>2,000 mg/kg	Applied neat under occlusive wraps for 24 hours.
Background exposure (diet, air, drinking water etc)			

164 Hexabromocyclododecane	Identified adverse health effects – acute	No health effect information in humans are available. Animal Effects of hexabromocyclododecane: Mild eye irritation occurred in laboratory animals with direct exposure. Liver and thyroid effects occurred in laboratory animals repeatedly fed high-to-very-high doses. Increased weight gain was observed in laboratory animals fed low doses with a high-fat diet, leading to obesity. Decreased fertility and increased pup mortality in laboratory animals fed very high levels for two generations.		
	Identified adverse health effects – intermediate/chronic	Carcinogenicity: There is limited information from only one long-term toxicity/carcinogenicity study in mice, indicating that the incidence of altered foci in the liver of males was increased, as was the incidence of liver carcinoma in females, but without a dose-relationship. The potential for HBCDD to cause cancer in humans has not been assessed by the U.S. EPA IRIS program, the International Agency for Research on Cancer, or the U.S. National Toxicology Program 13th Report on Carcinogens Genotoxicity: The available studies indicate that hexabromocyclododecane is genotoxic.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

165 1,2,5,6,9,10-hexabromocyclododecane	Identified adverse health effects – acute	Human data not found. Animal effects: Minimally irritating to the skin of rabbits. Acute oral toxicity study in rats the following non-lethal toxicity signs were observed during the 14-day observation time. Females: diarrhea in 1 of 5, hypoactivity in 1 of 5. Males: hypoactivity in 3 of 5, corneal opacity in 3 of 5 and ptosis in 3 of 5. Inhalation study: 90 minutes after initiation of exposure to the end of the 4 hours exposure period the rats exhibited slight dyspnea (shortness of breath). Developmental effects oral (10,000 ppm): weak hypothyroidism evident with increases in thyroid weight, thyroid follicular cell hypertrophy and serum concentrations of thyroid-stimulating hormone as well as decreases of serum T(3) concentrations in offspring at weaning. Studies suggest the chemical can interfere with thyroid hormone action in target organs, including the developing brain. Adult stage (1000 ppm): Increased thyroid weights and decreased serum T(3) concentrations.		
	Identified adverse health effects – intermediate/chronic			
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

166 Asbestos

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	Asbestos-related diseases may take many years to develop following exposure. Build-up of scar-tissue in the lungs has been reported in workers breathing in high levels of asbestos fibres over time. Asbestosis, lung cancer, mesothelioma and pleural disease can be caused by asbestos exposure but there are differences in the degree of disease, depending on the type of asbestos fibre. The data reported over the last several decades consistently support the conclusions that exposure to fibres longer than 10 um and perhaps 20 um are required to significantly increase the risk of developing asbestos-related disease in humans and that there is very little, if any, risk associated with exposure to fibres shorter than 5 um. Asbestosis is a form of pulmonary fibrosis with characteristically diffuse collagen foci and the presence of asbestos fibres, either free or coated with proteinaceous material (asbestos bodies). Malignant mesothelioma is a rare form of cancer that develops mainly in the pleural mesothelium, the protective lining that covers the lungs, diaphragm, and interior of the chest wall. Unlike lung cancer, mesothelioma is not associated with smoking history. Respiratory exposure to high levels of asbestos in the workplace has been associated with pain in the chest, pleural frictional rubbing, rales (wheezing sound in the lower pulmonary region), cyanosis (low oxygen content of blood), loss of weight, clubbing of the fingers and formation of asbestos warts on the hands. The cardinal symptom of asbestosis is dyspnea, which may have a variable but progressive course. Dyspnea on climbing two flights of stairs is characteristic; however,by the time dyspnea on exertion develops, the disease has already reached a progressive stage. Cough and sputum are common, and a pleuritic chest pain or chest tightness may occur. These symptoms, however, may also herald concomitant disease such as lung cancer or pleural effusion. Carcinogenicity: Asbestos is a human carcinogen. Lung cancer and mesothelioma, which is cancer of the tissue surrounding the lung or stomach, have been reported following lifetime exposure in some workers. Asbestos exposure may also increase the chances of developing cancer in other parts of the body. Cancers of ovary, and possibly other organs have been described. IARC (Group 1): All forms of asbestos should be regarded as carcinogenic to humans. The U.S. EPA IRIS program (Class A): Asbestos is a human carcinogen. ACGIH: A1: Confirmed human carcinogen. The U.S. National Toxicology Program 14th Report on Carcinogens has determined that asbestos and all commercial forms of asbestos are known to be human carcinogens. These assessments are based on sufficient evidence of carcinogenicity from studies in humans. Genotoxicity: Studies of exposed asbestos workers, residentially exposed Turkish villagers, mesothelioma patients, and lung cancer patients suggest that asbestos is genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis

167 Chrysotile asbestos

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	See 131 for asbestos related health effects. Most airborne chrysotile fibres are considered respirable because their fibre diameters are less than 3 um. Analyses of human lungs of workers exposed to chrysotile asbestos indicate much greater retention of tremolite, an amphibole asbestos commonly associated with commercial chrysotile in small proportions, than of chrysotile. Carcinogenicity: In humans commercial grades of chrysotile have been associated with an increased risk of lung cancer in epidemiological studies of exposed workers. Mesothelioma and digestive-tract cancer were observed in workers occupationally exposed to chrysotile. An excess of laryngeal cancer was reported in studies of chrysotile miners. IARC (Group 1): All forms of asbestos should be regarded as carcinogenic to humans. The U.S. EPA IRIS program (Class A): Asbestos is a human carcinogen. ACGIH: A1: Confirmed human carcinogen. The U.S. National Toxicology Program 14th Report on Carcinogens has determined that asbestos and all commercial forms of asbestos are known to be human carcinogens. These assessments are based on sufficient evidence of carcinogenicity from studies in humans. Genotoxicity: Cytogenetic study of workers in the chrysotile asbestos industry demonstrated that chrysotile is genotoxic.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

168 Amosite asbestos

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	See 131 for asbestos related health effects. All fibrous forms of asbestos are hazardous, and all can cause cancer, but amphibole forms of asbestos (anthophyllite, amosite, crocidolite, tremolite, and actinolite) are considered to be somewhat more hazardous to health than chrysotile asbestos which is the predominant commercial form. Carcinogenicity: Occupational exposure to amosite increases the risk of lung cancer. Mesothelioma and digestive-tract cancer were observed in workers occupationally exposed to amosite. Co-exposure to asbestos and tobacco smoking increased the risk of lung cancer in a synergistic manner. IARC (Group 1): All forms of asbestos should be regarded as carcinogenic to humans. The U.S. EPA IRIS program (Class A): Asbestos is a human carcinogen. ACGIH: A1: Confirmed human carcinogen. The U.S. National Toxicology Program 14th Report on Carcinogens has determined that asbestos and all commercial forms of asbestos are known to be human carcinogens. These assessments are based on sufficient evidence of carcinogenicity from studies in humans.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

169 Tremolite asbestos

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic	See 131 for asbestos related health effects. All fibrous forms of asbestos are hazardous, and all can cause cancer, but amphibole forms of asbestos (anthophyllite, amosite, crocidolite, tremolite, and actinolite) are considered to be somewhat more hazardous to health than chrysotile asbestos which is the predominant commercial form. Carcinogenicity: Occupational exposure to amosite increases the risk of lung cancer. Mesothelioma and digestive-tract cancer were observed in workers occupationally exposed to amosite. Co-exposure to asbestos and tobacco smoking increased the risk of lung cancer in a synergistic manner. Cancers of the digestive tract (stomach, colon, rectum) were also linked to asbestos exposure. IARC (Group 1): All forms of asbestos should be regarded as carcinogenic to humans. The U.S. EPA IRIS program (Class A): Asbestos is a human carcinogen. ACGIH: A1: Confirmed human carcinogen. The U.S. National Toxicology Program 14th Report on Carcinogens has determined that asbestos and all commercial forms of asbestos are known to be human carcinogens. These assessments are based on sufficient evidence of carcinogenicity from studies in humans. Genotoxicity: In genotoxicity assay, an increase in the frequency of micronuclei in binucleated cells was observed, where crocidolite was most genotoxic, followed by tremolite, and amosite the least.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

170 Synthetic vitreous fibres	Identified adverse health effects – acute	Dermal: Irritant contact dermatitis. The irritant dermatitis induced by man-made mineral fibres (MMMF) may be complicated by an urticarial and eczematous reaction that sometimes mimics an allergic response, both clinically and histologically. In addition, allergic reactions to resins used in MMMF production occasionally occur. Dermatitis can be transient. Eye irritation, itching, pain, or a foreign body sensation typically develops in patients with ocular exposures. Inhalation: Respiratory exposures generally present with irritation, burning, or pain in the throat or nose, chest discomfort, coughing, or wheezing.		
	Identified adverse health effects – intermediate/chronic	Working with some substances in this group may in time lead to (slightly) increased blood levels of some heavy metal-compounds, depending on the substance and amount used. Lung: small nodular opacities in lower lung fields and multiple cystic lesions and low attenuation areas in upper lung fields, mild interstitial fibrosis and mononuclear cell infiltration in alveolar walls without birefringent substances. Health effects include: Emphysema and sarcoidosis. Carcinogenicity: IARC (Group 2B): Special-purpose glass fibres such as E-glass and '475' glass fibres are possibly carcinogenic to humans. Refractory ceramic fibres are possibly carcinogenic to humans. IARC (Group 3): Insulation glass wool, continuous glass filament, rock (stone) wool and slag wool are not classifiable as to their carcinogenicity to humans. ACGIH: Refactory ceramic fibres A2; Suspected human carcinogen. Glass wool fibres, rock wool fibres, slag wool fibres, ACGIH: Special purpose glass fibres A3; Confirmed animal carcinogen with unknown relevance to humans. ACGIH: Continuous filament glass fibres A4; Not classifiable as a human carcinogen.		
	Health-Based Guideline Values (HBGV)	Type	Value	Basis
	Oral			
	Inhalation			
	Dermal			
	Background exposure (diet, air, drinking water etc)			

171 Polychlorinated biphenyls

Identified adverse health effects – acute	Chloracne and other dermal alterations are well known markers of exposure to PCBs and structurally related halogenated aromatic hydrocarbons. In general, chloracne appears in individuals with serum PCB levels 10-20 times higher than those of the general population, but there is great variability among individuals. Dermal: skin rashes, pigmentation disturbances of skin and nails, erythema and thickening of the skin, and burning sensations. The primary ocular effects reported by workers exposed to airborne PCBs were eye irritation, tearing, and burning. Inhalation: may produce irritation to nose, throat, and lungs. The vapors can cause coughing and/or difficulty in breathing. Liver damage and damage to CNS in high doses. Acute and long-term exposures to PCBs have been reported to cause neurological and unspecific psychological or psychosomatic effects, such as headache, dizziness, nausea, depression, sleep and memory disturbances, nervousness, fatigue, and impotence. The liver is considered to be one of the most important target organs for PCB toxicity. Acute exposures to PCBs cause alterations in liver enzyme activities.		
Identified adverse health effects – intermediate/chronic	Acne; hyperpigmentation of skin; hyperactive meibomian glands; conjunctivitis; edema of eyelids; subcutaneous edema; keratin cysts in hair follicles; hyperplasia of hair follicle epithelium; hepatic hypertrophy; decreased number of red blood cells; decreased hemoglobin; serum hyperlipidemia; leucocytosis. Irregular menstrual cycles, early abortions and the birth of small, hyperpigmented, and hyperkeratotic infants have been observed. Effects on developing foetus and child development. CARCINOGENICITY IARC (1987): Group 2A: The agent is probably carcinogenic to humans USEPA (2006): Group B2 Probable Human Carcinogen DHHS/NTP (2009): reasonably anticipated to be human carcinogens.		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

172 Benzene

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Fatal	125 mg/kg	
Inhalation	NOEL Fatal	80 mg/m3 60800 – 64000 mg/m3	Exposure for 480 minutes Exposure for 5 -10 minutes
Dermal			
Background exposure (diet, air, drinking water etc)			

173 PAHs

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Decreased growth rate	1,100 mg/kg bw/day	Rats
Inhalation	NOEL	44.8 mg/m3	Hamsters
Dermal			
Background exposure (diet, air, drinking water etc)			

174 Dioxins

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	0.6 µg/ kg bw 5051 µg/ kg bw 43 µg/ kg bw	Guinea pigs Hamster Rats
Inhalation			
Dermal	LD50	275 µg/ kg 0.1 µg/ kg	Rabbits Mice
Background exposure (diet, air, drinking water etc)			

175 Asbestos

Identified adverse health effects – acute	Acute exposure to asbestos fibres does not produce immediate acute effects other than some irritancy of skin, eyes and lungs with high concentrations. Temporary breathing difficulties have been reported in individuals exposed to high concentrations of asbestos dust. Short-term high level exposure to asbestos has been associated with lung cancer, mesothelioma and pleural disorders such as pleural plaques although risks are likely to be very low. Asbestos fibres may penetrate the skin and cause benign lesions around the implanted fibres, such as warts and corns, known as asbestos corns. Approximately 60 % of workers installing amosite insulation in the past reported lesions on the hands within 10 days		
Identified adverse health effects – intermediate/chronic	Most health effects caused by asbestos occur after a latent period following exposure. Asbestos is carcinogenic by inhalation, and does not produce acute effects, but lung toxicity (the target organ) may be manifest after many years. Clinical manifestations often occur approximately 30 years after the first exposure. However, the risks of serious long-term health effects from a single exposure are judged to be very low		
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral			
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			

176 Hydrogen Cyanide

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	LD50	3 – 4 mg/kg 2 – 3 mg/kg	Rat Rabbit
Inhalation	Fatal	300 mg/m3	Immediate death
Dermal	LD50	7 – 10 mg/m3	Rabbit
Background exposure (diet, air, drinking water etc)			

177 Lead

Identified adverse health effects – acute			
Identified adverse health effects – intermediate/chronic			
Health-Based Guideline Values (HBGV)	Type	Value	Basis
Oral	Developmental neurotoxicity Gastrointestinal symptoms (children) Acute interstitial nephrtitis Hypertension Encephalopathy (children) Encephalopathy (adults) Gastrointestinal symptoms (adults)	2 µg/dL BLL 40-60 µg/dL BLL 40-80 µg/dL BLL 48-120 µg/dL BLL 80-100 µg/dL BLL 100-120 µg/dL BLL 100-400 µg/dL BLL	
Inhalation			
Dermal			
Background exposure (diet, air, drinking water etc)			