RE: APPEAL BY FCC RECYCLING (UK) LTD PURSUANT TO REGULATION 31 OF THE ENVIRONMENTAL PERMITTING (ENGLAND AND WALES) REGULATIONS 2016 REGARDING DANESHILL SOIL TREATMENT FACILITY AT DANESHILL LANDFILL SITE AND 3C WASTE LIMITED PURSUANT TO REGULATION 31 OF THE ENVIRONMENTAL PERMITTING (ENGLAND AND WALES) REGULATIONS 2016 REGARDING MAW GREEN SOIL TREATMENT FACILITY AT MAW GREEN LANDFILL SITE

APPEAL REFERENCE APP/EPR/636 AND APP/EPR/651 (DANESHILL)

APPEAL REFERENCE APP/EPR/652 (MAW GREEN)

ENVIRONMENTAL PERMIT REFERENCE EPR/NP3538MF (DANESHILL)

ENVIRONMENTAL PERMIT REFERENCE EPR/BS7722ID (MAW GREEN)

ADDITIONAL REFERENCES SUPPORTING THE PROOF OF EVIDENCE OF SIMON JAMES COLE

Report Reference: 28480-HYD-XX-XX-RP-GE-0005

20 February 2024

Index

1. CIRIA, 2014. Asbestos in soil and made ground: a guide to understanding and managing risks

Full Reference: CIRIA, 2014. Asbestos in soil and made ground: a guide to understanding and managing risks, Nathanail, C P, Jones, A, Ogden, R, Robertson, A, C733, CIRIA, London

[Provided in full as a Core Document]

2. CIRIA, 2016. Asbestos in soil and made ground good practice guide

Full Reference: CIRIA, 2016. Asbestos in soil and made ground good practice guide, Studds, P, Bell, M, C765, CIRIA, London

[Provided in full as a Core Document]

3. CL:AIRE, 2014. SP1010 – Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination

Full Reference: CL:AIRE, 2014. SP1010 – Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination, Final Project Report (Revision 2), Defra Research Project SP1010, Contaminated Land: Applications in Real Environments (CL:AIRE), London, 24th September 2014

[Extract: pages 10-37]

SP1010 – Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination

Final Project Report (Revision 2)

FINAL

Contaminated Land: Applications in Real Environments (CL:AIRE)

24th September 2014

1. INTRODUCTION

This report presents a suggested methodology for the development of Category 4 Screening Levels (C4SLs). It constitutes the primary output of Defra research project SP1010, and it incorporates feedback from both the project's Steering Group and the wider contaminated land community, via meetings, workshops and correspondence.

The project's Steering Group comprised individuals from the following organisations:

- Department for Environment, Food and Rural Affairs (Defra)
- Department for Communities and Local Government (DCLG)
- Welsh Government (WG)
- Environment Agency (EA)
- Natural Resources Wales (NRW)
- Public Health England (PHE, formerly the Health Protection Agency)
- Food Standards Agency (FSA)
- Homes and Communities Agency (HCA)

Engagement with the wider contaminated land community primarily took the form of three stakeholder workshops, which took place at regular intervals during the project. Attendees at the stakeholder workshops included individuals and representatives from a variety of trade and professional organisations involved in the management of land contamination, as well as local authorities, learned societies and university departments. Individuals and organisations invited to send representatives to the workshops included the following:

Association of Geotechnical and Geoenvironmental Specialists (AGS)

British Geological Survey (BGS)

British Land Reclamation Society (BLRS)

British Property Federation

British Standards Institution (BSI) - EH/4 Soil Quality Committee

British Toxicology Society (BTS)

Chartered Institute of Environmental and Water Management (CIWEM)

Chartered Institute of Environmental Health (CIEH)

Chemical Industries Association (CIA)

City of London Law Society

Civil Engineering Contractors Association (CECA)

Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT)

Cranfield University

Energy Institute

Environmental Industries Commission (EIC) – Contaminated Land Working Group

Environmental Protection UK (EPUK) – Land Quality Group

Geological Society of London (GeolSoc)

Greater Manchester Contaminated Land Officers Group

Health and Safety Laboratory (HSL)

Home Builders Federation (HBF)

Institution of Civil Engineers (ICE)

Institution of Environmental Sciences (IES)

Local Authorities - East Midlands Region

Local Authorities - East of England Region

Local Authorities– London Region
Local Authorities - North East Region
Local Authorities - South Coast Region
Local Authorities - South East Region
Local Authorities - West Midlands Region
Local Authorities - West of England Region
Local Authorities– Yorkshire Region
National House Building Council (NHBC)
North-West Brownfield Remediation Forum (NWBRF)
Planning Officers Society
Register of Ground Engineering Professionals (RoGEP)
Royal Institution of Chartered Surveyors (RICS)
Royal Society of Chemistry (RSC) – Toxicology Group
Royal Town Planning Institute (RTPI)
Society for Environmental Geochemistry and Health (SEGH)
Society of Brownfield Risk Assessment (SoBRA)
Society of Chemical Industry (SCI)
Soil and Groundwater Technology Association (SAGTA)
Specialist in Land Condition (SiLC)
UK Contractors Group (UKCG)
UK Environmental Law Association (UKELA)
University of Nottingham
University of Reading
Waste and Resources Action Programme (WRAP)
Welsh Contaminated Land Working Group

An interim version of the methodology was published by Defra in February 2013 (Defra, 2013) and an initial final version, dated December 2014, was published in March, 2014, along with a Policy Companion Document and, more recently, two sets of peer review comments. This revised version of the final report corrects a number of minor errors which recently came to light, as detailed in the associated Erratum.

At the request of the Steering Group, this report stops short of providing "final C4SLs" for any substances but, instead, presents "provisional" values for certain test substances upon which finalised C4SLs could be based.

1.1 BACKGROUND AND OBJECTIVES

The overall objective of the C4SLs research project has been to assist the provision of technical guidance in support of Defra's revised Statutory Guidance (SG) for Part 2A of the Environmental Protection Act 1990 (Part 2A) (Defra, 2012a). Specifically, the project aimed to deliver:

- A methodology for deriving C4SLs for four generic land-uses comprising residential, commercial, allotments and public open space; and
- A demonstration of the methodology, via the derivation of C4SLs for six substances – arsenic, benzene, benzo(a)pyrene, cadmium, chromium (VI) and lead.

Part 2A was originally introduced to ensure that significant risks from land contamination to human health, property and the environment were identified and managed appropriately, with the revised SG being designed to address concerns

regarding the effectiveness and efficiency of its real-world application. Details of some of these concerns and the importance of striking the right balance between the benefits and impacts of regulatory action under Part 2A were provided in the consultation document issued by Defra in connection with the planned revisions to the SG in 2010 (Defra, 2010a). The resulting revisions to the SG were believed to address them, as described in the Ministerial foreword to the revised SG:

> *"It has been refined in order to give greater clarity to regulators as to how to decide when land is and is not actually contaminated land. It is shorter, simpler and more focused towards achieving optimum results in terms of dealing with sites most in need of remediation. Also included are various other improvements, reflecting the experience accumulated after eleven years of operating the regime and the progress in research and technology that we have seen in that time. They enable local authorities to take a more targeted approach which remains precautionary rather than a blanket approach which is over cautious."*

To help achieve a more targeted approach to identifying and managing contaminated land in relation to the risk (or possibility) of harm to human health, the revised SG presented a new four category system for considering land under Part 2A, ranging from Category 4, where there is no risk that land poses a significant possibility of significant harm (SPOSH), or the level of risk is low, to Category 1, where the risk that land poses a significant possibility of significant harm (SPOSH) is unacceptably high. More specific guidance on what type of land should be considered as Category 4 (Human Health) is provided in Paragraphs 4.21 and 4.22 of the revised SG, as follows:

"4.21 The local authority should consider that the following types of land should be placed into Category 4: Human Health:

(a) Land where no relevant contaminant linkage has been established.

(b) Land where there are only normal levels of contaminants in soil, as explained in Section 3 of this Guidance.

(c) Land that has been excluded from the need for further inspection and assessment because contaminant levels do not exceed relevant generic assessment criteria in accordance with Section 3 of this Guidance, or relevant technical tools or advice that may be developed in accordance with paragraph 3.30 of this Guidance.

(d) Land where estimated levels of exposure to contaminants in soil are likely to form only a small proportion of what a receptor might be exposed to anyway through other sources of environmental exposure (e.g. in relation to average estimated national levels of exposure to substances commonly found in the environment, to which receptors are likely to be exposed in the normal course of their lives).

4.22 The local authority may consider that land other than the types described in paragraph 4.21 should be placed into Category 4: Human Health if following a detailed quantitative risk assessment it is satisfied that the level of risk posed is sufficiently low."

The C4SLs are intended as "relevant technical tools" (in relation to Paragraph 4.21(c)) to help local authorities and others when deciding to stop further assessment of a site, on the grounds that it falls within Category 4 (Human Health).

The Impact Assessment (IA), which accompanied the revised SG (Defra, 2012b) provides further information on the nature and potential role of the C4SLs. Paragraph 47(h) of the IA states that:

"The new statutory guidance will bring about a situation where the current SGVs/GACs are replaced with more pragmatic (but still strongly precautionary) Category 4 screening levels (C4SLs) which will provide a higher simple test for deciding that land is suitable for use and definitely not contaminated land."

A key distinction between the Soil Guideline Values (SGVs) and the C4SLs is the level of risk that they describe. As described by the Environment Agency (2009a):

"SGVs are guidelines on the level of long-term human exposure to individual chemicals in soil that, unless stated otherwise, are tolerable or pose a minimal risk to human health."

The implication of Paragraph 47(h) of the IA (see above) is that minimal risk is well within Category 4 and that the C4SLs should describe a higher level of risk which, whilst not minimal, can still be considered low enough to allow a judgement to be made that land containing substances at, or below, the C4SLs would typically fall within Category 4. This reflects Paragraph 4.20 of the revised SG, which states:

"4.20 The local authority should not assume that land poses a significant possibility of significant harm if it considers that there is no risk or that the level of risk posed is low. For the purposes of this Guidance, such land is referred to as a "Category 4: Human Health" case. The authority may decide that the land is a Category 4: Human Health case as soon as it considers it has evidence to this effect, and this may happen at any stage during risk assessment including the early stages."

C4SLs, therefore, should not be viewed as "SPOSH levels" and they should not be used as a legal trigger for the determination of land under Part 2A.

1.2 UK APPROACH TO CONTAMINATED LAND RISK ASSESSMENT

As outlined in the revised SG and Defra and the Environment Agency's CLR 11 document (Defra & EA, 2004), a "staged" or "tiered" approach is recommended for assessing risks from land contamination in the UK. After each tier of assessment, a decision is made as to whether further action is required, and whether this should entail further assessment (such as gathering more data or proceeding to the next tier) or risk mitigation (such as remediation or the implementation of risk control measures).

The revised SG and CLR 11 describe three tiers of assessment:

- **Preliminary Risk Assessment (PRA).** A primary objective of a PRA is to gather as much information as possible about a site so that a *conceptual model* can be developed that represents site characteristics and shows the possible relationships between contaminants, pathways and receptors. Any possible requirement for further assessment (e.g. intrusive investigation) or remediation can then be considered on the basis of the conceptual model.

- **Generic Quantitative Risk Assessment (GQRA).** In the event that the PRA indicates the existence of *plausibly significant contaminant linkages* (and remediation is not otherwise planned), GQRA is then carried out by comparison of measured concentrations (in, for example, soil, water or soil vapour) with generic screening values appropriate for the conceptual model and pollutant linkage(s) being assessed. In simple terms, provided the measured concentrations are below appropriate generic screening criteria, the risk from the pollutant linkages(s) being assessed are unlikely to be significant. Note that GQRA often involves the application of statistical methods to estimate a representative exposure concentration for comparison against the generic screening criteria.

- **Detailed Quantitative Risk Assessment (DQRA).** If contaminant levels exceed the generic screening criteria, or if use of generic screening criteria are not appropriate for a particular site, then DQRA may be carried out and site-specific assessment criteria (SSAC) developed. The outcome of the DQRA is a final assessment regarding which, if any, of the plausible contaminant linkages identified in the PRA and GQRA should be considered significant. If any pollution linkages are considered to be significant, then consideration of remedial options, or other corrective action can take place. In the event that no significant contaminant linkages (SCLs) are identified, then no further action is normally required.

The generic screening values referred to above usually take the form of risk-based Soil Guideline Values (SGVs) or other Generic Assessment Criteria (GACs) that are most typically derived using the Environment Agency's Contaminated Land Exposure Assessment (CLEA) model, as described in the Environment Agency's SR2, SR3 and SR7 reports (EA, 2009b & c; EA, 2008). It is anticipated that C4SLs will be used in a similar manner; as generic screening criteria that can be used within a GQRA, albeit describing a higher level of risk than the SGVs.

1.3 SUMMARY OF SUGGESTED APPROACH FOR DERIVING C4SLs

The suggested approach to the development of C4SLs described herein consists of the retention and use of the CLEA framework, modified according to considerations of the underlying science within the context of Defra's policy objectives relating to the revised SG (as outlined above). Within this context, it is suggested that the development of C4SLs may be achieved in one of three ways, namely:

- By modifying the toxicological parameters used within CLEA (while maintaining current exposure parameters);
- By modifying the exposure parameters embedded within CLEA (while maintaining current toxicological "minimal risk" interpretations); and
- By modifying both toxicological and exposure parameters.

There is also a suggested check on "other considerations" (e.g., background levels, epidemiological data, sources of uncertainty) within the approach, applicable to all three options.

1.4 REPORT FORMAT

The sections that follow describe the CLEA framework and the suggested modifications that could be made to it to derive C4SLs and incorporate feedback received from the Steering Group and stakeholders. They also discuss how "other considerations" should be factored into the overall C4SL methodology while a final section summarises relevant considerations regarding the potential use of C4SLs in assessing land contamination.

The report also presents details of sensitivity and probabilistic analyses that have been undertaken as part of the research, in order to help elucidate some of the uncertainty present in the exposure modelling. These are described in more detail in Appendices A and B, with other appendices comprising:

- Substance-specific reports, providing provisional C4SLs (pC4SLs) for arsenic, benzene, benzo(a)pyrene, cadmium, chromium (VI) and lead; and
- Review of the CIEH/CL:AIRE statistical guidance.

It is important to note that the methodology and provisional values presented herein represent the outcome of a research project and they do not, in any way, constitute formal guidance from Defra (or the consortium, or any other party). As indicated

above, further policy inputs are expected to be required in order to finalise the methodology and C4SLs (as noted in the text) and the report's findings have been designed more for discussion purposes than immediate application.

2. TOXICOLOGICAL ASSESSMENT

The toxicological assessment of contaminants is a key part of land contamination risk assessment. Such assessments are typically complex evaluations involving a significant amount of data, with different toxicity endpoints and study designs needing to be considered. As a consequence, toxicological assessments and reviews should only be performed by a suitably qualified individual who sufficiently understands the nature of toxicological data.

This section outlines the process of toxicological assessment for the purposes of land contamination risk assessment. It begins with a summary of the requirements of such assessments under Part 2A (in terms of the toxicological effects that are potentially relevant) and continues with a review of existing guidance to derive "minimal risk" Health Criteria Values (HCVs) under the CLEA framework (as outlined in SR2). It concludes with suggestions on how this framework could be adapted for the purpose of the development of C4SLs, presenting decisions on how such minimal risk values could be refined with further chemical-specific knowledge, to generate a new guidance value that can be regarded as meeting the requirements of the C4SLs.

Such an explicit deviation from the use of "minimal risk" levels is considered necessary in order that C4SLs can meet Defra's policy objectives outlined above. With this in mind, it should be noted that the adoption of "minimal risk" considerations is not a requirement of existing legislation or statutory guidance relating to the setting of screening criteria for use under Part 2A. Indeed, the potential usefulness of toxicological tools to derive substance-specific doses equivalent to different orders of risk, in relation to Part 2A, has been highlighted by the Royal Society of Chemistry (RSC, 2009).

It is suggested that a new term is defined for the toxicological guidance values associated with the derivation of C4SLs – a Low Level of Toxicological Concern (LLTC). An LLTC should represent an intake of low concern that remains suitably protective of health, and definitely does not approach an intake level that could be defined as SPOSH.

2.1 SIGNIFICANT HARM

When selecting critical study endpoints on which to base toxicological risk assessment for land contamination, it is important to consider whether such endpoints are relevant to assessing significant harm under Part 2A. The new Part 2A statutory guidance (April 2012) describes what types of harm to human health should be considered "significant" in relation to land contamination, as summarised in Table 2.1 below.

Table 2.1: Part 2A Statutory Guidance Definition of Harm to Human Health

	Part 2A Environmental Protection Act 1990 New Statutory Guidance 2012
Always considered as significant harm	Death
	Life threatening diseases (cancers)
	Serious injury caused by the chemical or biochemical properties of the substance, such as injury resulting from explosive or asphyxiating properties of gases
	Birth defects
	Impairment of reproductive functions
	Other diseases likely to have serious impacts on health
May or may not constitute significant harm	Physical injury
	Gastrointestinal disturbances
	Respiratory tract effects
	Cardiovascular effects
	Central nervous system effects
	Skin ailments
	Effects on organs such as kidney or liver
	Wide range of other health impacts

2.2 EXISTING GUIDANCE ON DERIVING HEALTH-BASED GUIDANCE VALUES

This section describes the current guidance for deriving Health-Based Guidance Values (HBGV) that are defined as the estimated dose in humans that is without appreciable risk over a lifetime. Examples of HBGVs include a tolerable daily intake (TDI) used for environmental contaminants or an acceptable daily intake (ADI) used for additives or residues in food.

Similarly, the term HCV has been used to describe the level of long-term human exposure to chemicals *in soil* that is tolerable or poses a minimal risk to health. It is an umbrella term that encompasses a TDI for thresholded compounds (i.e. compounds where there is a dose below which adverse effects are not discernible in experimental studies) and index dose (ID) for non-thresholded chemicals (i.e. chemicals where there is no dose under which effects do not occur in experimental studies). HCVs represent a baseline and health protective position to minimise risks of significant harm for all people exposed (including children); *they do not represent thresholds above which an intake would be unacceptable* (EA, 2009b; Defra, 2008).

The methods used to derive HBGVs differ depending on, amongst other things, whether or not a given chemical exhibits a threshold for its critical toxicological effects and the criteria that are applied by different worldwide authorities. The remainder of this section describes the derivation of HBGVs for both threshold and non-threshold chemicals.

2.2.1 SELECTION OF THE PIVOTAL STUDY AND IDENTIFICATION OF CRITICAL ENDPOINT

The first step in the derivation of a HBGV is the selection of the pivotal study and identification of the critical endpoint from an array of toxicity studies. This is done by reviewing all available toxicology data and identifying suitable Points of Departure (PODs) in the form of No Observed Adverse Effect Levels (NOAELs), Lowest Observed Adverse Effect Levels (LOAELs) or Benchmark Doses (BMDs). The NOAEL is the highest dose at which no adverse effects are seen in the toxicity study. If a NOAEL cannot be determined from the data, due to effects being seen at even the lowest dose tested, a LOAEL is determined i.e. the lowest dose at which some adverse effects are seen. A NOAEL (or LOAEL) is determined for all good quality studies and for all endpoints, and the study with the lowest (most sensitive) value is considered to be the pivotal study. If there is more than one good study for the most

sensitive effect, the highest NOAEL (or lowest LOAEL) is selected. Care should be taken in selecting the most sensitive NOAEL and it will depend on careful consideration of relevant studies, and factors such as dose-spacing and consistency between studies. This NOAEL (or LOAEL) represents the most sensitive endpoint of toxicity and can be used as a POD to form the basis of the HBGV derivation.

It should be noted that the magnitude of a NOAEL or LOAEL is highly dependent on the dosing regimen used and endpoints measured in the original toxicity study. As a consequence, the true "no effect level" could conceivably be higher or lower than the experimental NOAEL, depending on the sensitivity of the study and the choice of endpoint. Similarly, the true dose at which effects begin to occur could be lower than the experimental LOAEL. This makes a NOAEL or LOAEL a highly uncertain value in some studies.

As an alternative approach to qualifying hazard, a BMD may be derived. This is the dose that produces a predetermined change in response, the Benchmark Response (BMR), for a given toxicological effect. For risk assessment purposes, the 95% lower confidence limit of the BMD (BMDL) is often used as the POD.

The concept of the benchmark dose is illustrated below in Figure 2.1.

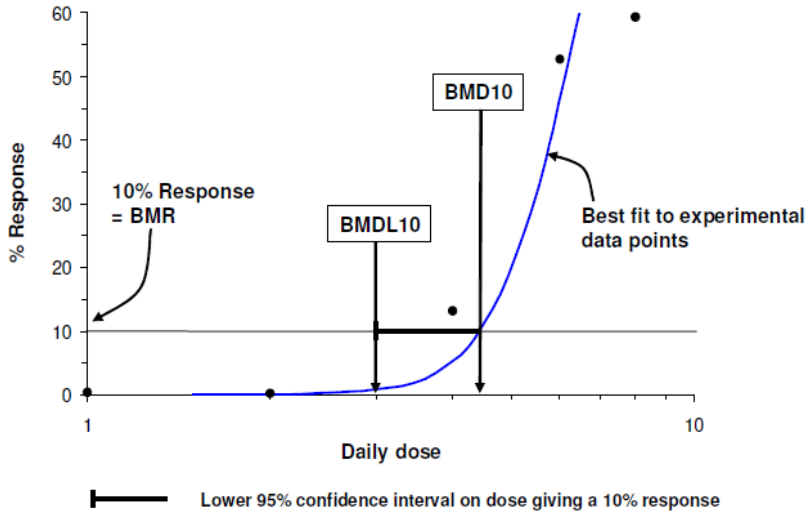

Figure 2.1: Hypothetical dose-response curve to illustrate the concepts of BMR, BMD and BMDL, for a 10% incidence response above control (taken from EFSA 2005)

The use of the BMD is beneficial as it is based on all available data of the dose response, and is on the scale of observable effects, rather than being based on one uncertain data point e.g. a NOAEL (EFSA, 2005, 2009a). However, there may be some endpoints not amenable for BMD modeling (e.g. in a study where no response is seen at any dose) for which a NOAEL approach should still be used (USEPA, 2012).

BMD modelling is being used more widely for dose-response modelling (USEPA, 1995 & 1996). In the EU, EFSA (2005) recommended the use of BMD modelling for genotoxic carcinogens, as well as other toxicity endpoints, as the method of choice to derive a quantitative POD. A citation from EFSA (2005) indicates the main scientific rationale as to why a BMD is considered a better choice than a NOAEL for quantitative risk assessment, as follows:

> *"…..the Scientific Committee concludes that the BMD approach is a scientifically more advanced method to the NO(A)EL …..it makes extended*

use of available dose-response data and it provides a quantification of the uncertainties in the dose-response data."

The UK COC also recommends the use of the BMD approach for the interpretation of carcinogenicity dose-response data (COC, 2012). The BMD refers to central estimates for continuous and dichotomous endpoints, based on a predefined level of response above background (the BMR). For dichotomous endpoints e.g. incidence data such as carcinogenic endpoints, an incidence of 10% is commonly used largely due to the 10% response being at or near the limit of sensitivity in most cancer bioassays (Benford *et al*., 2010). A default BMR of 5% is recommended by EFSA for continuous data e.g. an increase in kidney/liver enzymes (EFSA, 2009). A lower BMR for either dichotomous or quantal data could be used if the study has greater sensitivity or is considered biologically relevant (eg. for lead, a BMR of 1% has been selected by EFSA, 2010 and for arsenic a BMR of 0.5% has been able to be calculated for lung carcinogenicity effects (WHO, 2011)). It is also possible to calculate a higher BMR value that represents an incidence rate of effect higher than 10%. A quantitative selection for the incidence rate that can be determined from the sensitivity and quality of the dataset is a scientific judgment based on the data. To date, toxicology data for only a few land contaminants have been interpreted using BMD modeling, and this approach has not formed the basis of any published HCVs (although the HPA's Contaminated Land Information Sheet publication on benzo[a]pyrene/PAHs adopts this approach).

2.2.2 DEALING WITH UNCERTAINTY

In order to derive a HBGV for a given substance, the selected POD is divided by a measure of uncertainty in order to derive an estimated intake for humans that is judged to be protective of public health. The Uncertainty Factors (UFs) or margin (i.e. the difference between the POD and exposure intake) selected depend upon the quality and type of toxicity study, the species used in the pivotal study and the nature of the critical endpoint. The incorporated uncertainty aims to account for potential differences in the human response to the chemical compared to the species used in the toxicity study, and also variability in human responses due to age, genetic factors and health status.

Threshold chemicals

For all thresholded chemicals, an UF approach is recommended (COT, 2007). The recent COC (2012) guidance also advocates the use of such an approach, which has not changed from the COC guidance of 2004 on which SR2 is based. The choice of UFs depends on the quality of the animal data and the uncertainties in the evaluation of the toxicological data (COT, 2007; COC, 2012).

When basing a HBGV on a NOAEL from a chronic animal study, a default UF of 100 is typically used, consisting of a factor of 10 for interspecies variability (4 for toxicokinetics[1] and 2.5 for toxicodynamics[2]) and 10 to account for intraspecies differences (3.2 for toxicokinetics and 3.2 for toxicodynamics) (EFSA, 2012a; IPCS, 2005). Put another way, the first factor of 10 is assumed to move the dose response curve in the test species to an exposure value for the average human (taking account of the fact that the true no effect level in average humans could actually be 10-fold less than the animal NOAEL, given toxicokinetic and toxicodynamic differences); and the second factor of 10 is assumed to move an exposure value in the average human to a value that will cover the whole population, including sensitive sub-groups (Walton *et al*., 2001).

[1] Toxicokinetics - the rates that chemicals pass into, through and out of the body's organs.

[2] Toxicodynamics - the interactions the chemicals have with molecules, cells and organs of the body.

In many cases, the use of default UFs that are generic and not chemical- or species-specific will result in conservative HBGVs being derived, as the underlying data supporting them are generic and show wide variability. Default UFs may not take into consideration the sensitivity of the animal used in the toxicity study, the number of doses used, the interval between doses, the number of animals per dose group and the choice of toxicological endpoint (Health Council of the Netherlands, 2008). An alternative approach may therefore be to define chemical specific adjustment factors (CSAFs) on a case by case basis, making each uncertainty and its associated factor transparent. For example, the CSAF will replace the default UF if suitable data are available showing differences in target organ exposure in animals and humans, therefore enabling the toxicokinetic factor to be amended (IPCS, 2005). As indicated above, evidence suggests that a distinction should be made between toxicokinetic and toxicodynamic components, as both can contribute to species differences, although variations between animals and humans are often due to absorption, distribution, metabolism and excretion (toxicokinetic factors) (Health Council of the Netherlands, 2008).

SR2 already supports the use of CSAFs for thresholded substances and states the following in relation to this issue:

Box 2.4 Uncertainty factors

Uncertainty factor is the generic term used in the UK for the numerical factors applied to toxicity data (points of departure) to take into account the uncertainty in extrapolating the data to derive HCVs for humans. Various terms are used by different organisations to denote such factors, including **safety factor**, **variability factor**, **assessment factor** and others. These terms are generally interchangeable. In some cases, however, it may not be uncertainty that dictates the application of the factor, but rather evidence that humans or a human subpopulation are more sensitive than the subjects (either animal or human) of the critical study. Similarly, there may be evidence of decreased sensitivity of the target population relative to the test population, in which case a smaller than usual factor may be applied. 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** is applied (see IPCS, 2005).

Moreover, for non-genotoxic carcinogens, the COC also advocates that default factors could be replaced in part or in full by CSAFs if the available data provide adequate information on interspecies or human variability (COC, 2012; Meek *et al.*, 2002).

Non-threshold chemicals

Some chemicals exhibit an effect that does not have an observable threshold (i.e. there is no dose under which effects do not occur in experimental studies). This is often a cancer related effect but may also include other endpoints (e.g. neurobehavioural toxicity for lead also shows no threshold in human epidemiological studies). Specifically, 'genotoxic carcinogens' that are seen to damage DNA in genotoxicity assays are chemicals that are considered to have no threshold dose. For these substances, all doses however small, may carry a risk of effect, even at the level of minimal risk described in SR2.

The principle of "As Low As Reasonably Practicable" (ALARP) automatically applies to the regulation and management of non-threshold chemicals in the UK. It is important to note that ALARP remains the overriding principle even when a margin of exposure or minimal risk level suggests there is unlikely to be a concern for human health (COC 2012; EA 2009b). What is considered practicable is a remediation/risk management decision.

SR2 is based on guidance from the COC in 2004. This has now been superseded as of October 2012, as the Committee on Carcinogenicity (COC) published a new guidance document (G06) for the risk assessment of chemical carcinogens (COC, 2012). However, the basic principles for defining 'minimal risk' as described in SR2

remain valid and hence that document can still be referred to for 'minimal risk' guidance. For circumstances where exposure to non-thresholded chemicals is unavoidable, COC (2012) states:

> 'For carcinogens which do not show a threshold for effect, exposure should be as low as reasonably practicable (ALARP). In addition, the Committee recommends that the Margin of Exposure (MOE) approach be adopted as a tool *to indicate the level of concern* in situations where exposure is unavoidable. When it is necessary to set a standard or guideline value for a genotoxic contaminant, identification of a minimal risk level may be appropriate.'
>
> It continues: 'The derivation of a minimal risk level for a genotoxic and carcinogenic contaminant or impurity involves assessment of all available dose-response data for carcinogenicity to determine an appropriate point of departure and *use of expert judgement to identify a suitable margin between this point of departure and a level of exposure which would result in a minimal risk*. One proposal is that a suitable margin might be 10,000 (Gaylor, 1994; Gold *et al*, 2003), which parallels the margin of exposure approach, where an MOE of 10,000 is considered to be *unlikely to be of concern* when based on a BMDL10 from an animal study. For a genotoxic and carcinogenic contaminant or impurity, a comparison of the minimal risk level with estimated exposure can be informative to risk managers.'

The usual way of implementing a 'margin of exposure' approach is to divide the POD by an exposure intake value estimated using a model of the exposure scenario (e.g. that would mean to use CLEA in 'forward mode' to derive an average daily exposure (ADE) for each site assessed and compare with the POD to arrive at an MOE). One would then decide in the context of risk management as to whether the MoE was 'acceptable' or 'unacceptable'. The exposure used to calculate the MOE for a genotoxic carcinogen should be chosen carefully, and adequately justified. MoE approaches to risk characterisation are being used more widely and in particular, for the risk characterisation of genotoxic carcinogens in foods (EFSA, 2005; IPCS-WHO, 2009; EFSA, 2009a & USEPA, 1995). A joint EFSA, ILSI and WHO workshop was held in 2005, and a comprehensive list of the advantages and limitations of adopting an MOE approach was produced afterwards (EFSA, 2005).

EFSA (2005 & 2012b) have indicated that for genotoxic and carcinogenic contaminants, in general, an MOE of ≥10,000 is of low public health concern when based on a $BMDL_{10}$ from an animal study. The exact recommendations from the EFSA statement in 2012 are as follows:

> *'In the 2005 opinion, the Scientific Committee gave some guidance on how to interpret the MOE. It was stated that "The Scientific Committee is of the view that in general a margin of exposure of 10,000 or higher, if it is based on the BMDL10 from an animal carcinogenicity study, and taking into account overall uncertainties in the interpretation, would be of low concern from a public health point of view and might be reasonably considered as a low priority for risk management actions. However, such a judgment is ultimately a matter for the risk managers. Moreover an MOE of that magnitude should not preclude the application of risk management measures to reduce human exposure".*
>
> *The Scientific Committee is aware that the magnitude of an MOE only indicates a level of concern and does not quantify risk. Moreover, the implications of any MOE need to be considered case-by-case, looking at both its magnitude and the uncertainties regarding its derivation. The Scientific Committee reiterates that an MOE of 10,000 or higher is considered of low concern from a public health point of view with respect to the carcinogenic effect. As a small MOE represents a higher risk than a larger MOE, it follows that a very high MOE would be very unlikely to be of safety concern.*
>
> *However, there is at present no international consensus on banding of MOEs and corresponding descriptive terminology. When using the MOE approach for assessing impurities, EFSA Scientific Committee and Panels should describe the derivation of the MOE, its magnitude, and the associated uncertainties regarding its derivation. They should also give their view on whether the MOE is of high concern, low concern, or unlikely to be of safety concern. It will then be*

the role of the risk managers to decide whether the substance containing the impurities should be authorised.'

The UK Committee on Carcinogenicity (2007) have agreed MOE bandings for genotoxic carcinogens, for use in risk management and communication, as follows:

Table 2.2: MOE bands (as agreed by COC, 2007)

MOE band	Interpretation
< 10,000	May be a concern
10,000 – 1,000,000	Unlikely to be a concern
>1,000,000	Highly unlikely to be a concern

An MOE of 10,000 represents a default 100-fold difference between the point of departure and human exposures to allow for general differences between species and for human variability and an additional 100-fold difference has been suggested to allow for the additional uncertainties due to using a BMDL and due to the inter-individual variability in carcinogenic processes. Therefore, a MOE of 10,000 or higher when used with a $BMDL_{10}$ would be unlikely to be a concern from a public health point of view, whereas a MOE of less than 10,000 indicates that exposure 'may be of concern' (EFSA, 2005). Proposals on interpreting the magnitude of the MOE were adopted and expanded by COC and a system for banding MOE values was proposed, as above. There is no precedent set for what margin may constitute *low concern*. One suggestion proposed here for the first time, is that a generic margin of 5000 could constitute 'low concern' when using a $BMDL_{10}$. This would lead to a notional risk level of 1 in 50,000, as compared to the risk level of 1 in 100,000 used currently to represent minimal risk in contaminated land risk assessment and the derivation of SGVs. However, the choice of margin and level of concern is not a purely scientific matter, but a matter of risk management that must be agreed by a broad range of stakeholders and policy makers. Other margins could constitute low concern when used with other BMDs relating to lower BMRs (see Table 5.5).

However, it should be noted that, whilst the MOE is a usefully flexible approach for risk characterisation, the MOE approach does not lead to a HBGV as needed for input into the CLEA model . The conceptual difference between the use of guideline values versus the margin of exposure approaches in risk characterisation is well described in Figure 2 of the IGHRC CR9 (2003). In general, hazard assessment often leads to a health based guidance value such as a TDI, or in this case an LLTC. Risk characterisation is then conducted by comparing the standard with the estimated exposure. Alternatively, a comparison between the hazard assessment (i.e. the point of departure) and the exposure assessment can be made, leading to a ratio (the MOE), which can be interpreted in terms of potential risk of adverse effects. Notwithstanding this, a 'margin' approach, which parallels the MOE approach, can be implemented when setting guideline values, as described below (Section 2.2.4).

2.2.3 HCVs FOR THRESHOLD SUBSTANCES

As mentioned above, according to SR2, HCVs for threshold substances are typically referred to as TDI values in the UK. A TDI is defined as 'the estimated amount of a chemical (expressed on a body weight basis) that can be ingested daily over a lifetime without appreciable risk to health' and it is typically calculated by dividing a POD by a UF. For inhalation exposure, a tolerable concentration in air (TCA) can instead be defined, as the estimated amount of a chemical (expressed as an atmospheric concentration) that can be inhaled over a lifetime without appreciable risk. The TDIs and TCAs used in the UK are equivalent to many of the toxicological criteria used in other countries, such as JECFA's provisional maximum tolerable daily intakes

(PMTDI) and USEPA's Reference Doses (RfDs), Reference Concentrations (RfCs) and US ATSDR's Minimal risk Levels (MRLs). All of these criteria take data from a pivotal toxicology study and incorporate a value (an uncertainty or assessment factor) to account for uncertainties in the data. Differences in the choice of pivotal toxicology study and POD should be appreciated when comparing HCVs from different jurisdictions as well as their conservatism, highlighted in their choice of uncertainty factors (EA, 2009b).

2.2.4 HCVs FOR NON-THRESHOLD SUBSTANCES

According to SR2, HCVs for non-threshold effects (i.e. those chemicals whose toxic effects do not exhibit a threshold) should take the form of an ID. An ID is defined as 'a daily dose, derived for a non-threshold carcinogen, which is expected to be associated with a minimum excess risk of cancer'. IDs can be derived using two approaches, referred to in SR2 as "quantitative dose-response modeling" and "non-quantitative extrapolation". The selection of which approach to use is largely dependent on the extent and quality of data available (EA, 2009b).

Non-quantitative extrapolation has been used in SR2 to set IDs for non-threshold carcinogens using an approach which is similar to that used for threshold chemicals (i.e. a POD divided by a default UF). The POD, in the form of a BMD, is identified from relevant carcinogenicity data as the dose where effects may be observed.. As with threshold effects, the consideration of uncertainty needs to account for potential inter and intraspecies differences. However, additional factors are also included to reflect the additional uncertainties for substances that are genotoxic and carcinogenic; due to human variability in cell cycle control and DNA repair,for example, as well as the uncertainties surrounding using a reference point that is not equivalent to a NO(A)EL.

The EFSA Scientific Committee considered the application of additional measures of uncertainty to allow for the severity of an effect. Whilst this is not routinely used, it should be considered on a case by case basis as there are some examples where the toxicological effects are judged to be irreversible or particularly severe (EFSA, 2012a). The Guidelines for Drinking Water Quality (WHO, 2011), suggested that additional uncertainty may be needed for endpoints such as foetal malformations, or carcinogenicity with a non-thresholded mode of action.

For deriving guideline values for non-thresholded carcinogens, there is now strong support in COC (2012) for adopting an approach that parallels the 'margin of exposure' approach described above in section 2.2.2. The 'margin' applied to the POD is a value derived to represent a specified level of concern and is arrived at by reviewing the toxicological evidence, reviewing the uncertainties in the data (similar in approach to that above for thresholded chemicals) using expert judgment (the basis for which should be well documented) and also with good knowledge of the exposure model context and uncertainties within the exposure parameters.

The default margin of 10,000 between human exposure and a $BMDL_{10}$ from an animal study is considered to be 'unlikely to be a concern' (COC, 2007 & 2012), and echoes the way of defining minimal risk as per SR2 (EA, 2009b), DEFRA (2008) and COC (2004). Using a $BMDL_{10}$ for non-threshold carcinogenic effects divided by a default UF of 10,000 has been equated to a minimal risk level of 1 in 100,000 (EA, 2009b). If scientific evidence is available to refine the degree of uncertainty required in a chemical specific manner, lower margins than 10,000 may describe 'low' concern scenarios (EFSA 2012b).

In *quantitative dose-response modeling*, numerical approaches are used to derive an estimate of dose that corresponds to an excess lifetime cancer risk (ELCR) (EA, 2009b; DEFRA, 2008). Although this approach is used in some parts of the world (e.g. by USEPA, WHO) with data obtained from high dose animal studies, the Committee on Carcinogenicity does not recommend its use for routine risk assessment, as the models used to extrapolate data do not adequately simulate carcinogenic processes and can lead to highly variable outcomes (COC, 2004; COC, 2012). As a consequence, it is only recommended for use in the UK where there are human data,

and even then, if BMD modeling can be carried out against the dataset this should be done in preference over using an ELCR. Defra has considered that an ELCR of 1 in 100,000 (10^{-5}) based on suitable human cancer data is appropriate to represent "minimal risk" (EA, 2009b; DEFRA, 2008). Given that C4SLs are designed to represent risks which are 'low', consideration could be given to defining an ELCR that represents a 'low level of concern' in the derivation of toxicological criteria using this approach.

For non-thresholded chemicals, as explained above, the concept of ALARP automatically applies in the UK, as per the guidance in SR2, which states "The ALARP principle ensures that, irrespective of whether a health-based guideline is being breached or not, exposures are kept 'as low as reasonably practicable". What is considered practicable is a risk management decision.

2.2.5 LIFE-TIME AVERAGING

CLEA currently does not allow the user to select an averaging time greater than exposure duration but the user is able to select the age classes considered in the ADE calculations and thus can base the ADE calculations on exposure over a lifetime. As indicated in Section 3.5.1.2, averaging exposure over a lifetime can have a large influence on the ADE estimates derived by CLEA and, therefore, any guideline values derived.

Lifetime averaging as a concept arises from Haber's rule in the context of acute inhalation toxicity and is described as the concentration/dose x time of exposure = toxic effect (C x t = k). The USEPA (and others) assume that the lifetime cumulative dose (LCD) is appropriate for cancer risk assessment. When assessing less than lifetime exposure periods, it is assumed that a high dose over a shorter periods is equivalent to a low dose over a longer (lifetime) period. However, for shorter exposure periods a dose rate correction factor may be needed to correct for dose-related toxic effects and it is important that toxicokinetic factors are also taken into account (Felter *et al.*, 2011). Other authors have suggested that the risk attributable to early-life exposure often appears modest compared with the risk from lifetime exposure, but it can be about 10-fold higher than the risk from an exposure of similar duration occurring later in life (Ginsberg, 2003).

A key consideration in regards to lifetime averaging is whether there are differences in susceptibility to the chemical between children and adults. As mentioned in Section 2.2.2, the default UF of 10 for intraspecies differences already allows for variation within the human population, including specific subgroups such as children (COT, 2007). The US Food Quality Protection Act (USA, 1996) proposed the need for additional UFs to calculate HBGVs of pesticides for infants and children. Such a need is based on whether the 10-fold intraspecies UF is sufficiently protective of pregnant women, embryo/foetuses, infants and children. It has been proposed that elimination/clearance of some xenobiotics is higher in children than in adults hence in that instance children could be less sensitive as they could have lower body burden than adults for the same daily intake, when expressed on a body weight basis, and in fact, the higher elimination of the chemical may in part compensate for increased organ sensitivities during child development (Renwick, 1998). Therefore it has been suggested that an additional UF to account for infants and children is not required in relation to age-related toxicokinetics (Renwick, 1998; Renwick *et al*., 2000). Moreover, Renwick *et al*. (2003) also suggested that additional UFs would not be required if age-related differences are tested for in animal toxicology studies. The scientific evidence for making these arguments in risk assessment is not extensive however.

The current understanding of the biological processes of carcinogenesis is that young animals or children are more susceptible to many carcinogens compared to mature animals or adults (McConnell, 1992; Anderson *et al*., 2000; Birnbaum and Fenton, 2003; Ginsberg, 2003; Miller *et al*., 2002; Scheuplein *et al*., 2002). Studies in rodents being exposed to chemicals with a mutagenic mode of action suggest a decline in cancer risk with age at exposure, as the earliest two or three postnatal weeks in

rodents appear to be most susceptible (USEPA, 2005 a & b). This is due to a variety of biological mechanisms:

- There can be differences in the capacity to metabolize and eliminate chemicals, resulting in different internal doses of the active agent(s), depending on whether the parent compound or metabolite is the active agent.
- More frequent cell division during development can result in enhanced expression of mutations due to the reduced time available for DNA repair (Slikker *et al*., 2004).
- More frequent cell division during development can result in clonal expansion of cells with mutations from prior unrepaired DNA damage (Slikker *et al*., 2004).
- Key DNA repair enzymes are sometimes lacking in embryonic cells, such as brain cells.
- Some components of the immune system are not fully functional during development (Holladay and Smialowicz, 2000; Holsapple *et al*., 2003).
- Hormonal systems operate at different levels during different lifestages.
- Induction of developmental abnormalities can result in a predisposition to carcinogenic effects later in life (Anderson *et al*., 2000; Birnbaum and Fenton, 2003; Fenton and Davis, 2002).

Understanding the mode of action of the compound where a key event is likely to occur in children, as well as understanding the toxicokinetics in different life stages that may predict a sufficiently large internal dose in children, are critical in the understanding of whether children are in fact more susceptible than adults. For example, pro-carcinogens may require metabolic activation by hepatic enzymes (cytochrome P450) to exert their carcinogenic effect. The expression and activity of some cytochrome P450 isoforms in some cases has been shown to be lower in neonates and children compared to adults (Faustmann *et al.*, 2000). Therefore, in terms of pro-carcinogens, children may effectively be protected against carcinogenic metabolites due to their lower metabolic capacity. Conversely, if the parent compound exerts the toxicological effects then a reduced metabolism and elimination could result in higher body burden. Moreover, exposures to chemicals acting through a mutagenic, as well as through other modes of action could result in a greater susceptibility for the development of tumours when the exposures occur in early life stages (USEPA 2005 a & b). The COC have recently discussed the US EPA document on life stage sensitivity to carcinogens (July 2006; http://www.iacoc.org.uk/meetings/Minutes13.07.2006.htm) and concluded that at this time "there was insufficient evidence at this stage to adopt adjustment factors for genotoxic carcinogens for different life stages".

The decision to perform lifetime averaging when using CLEA is therefore not trivial, and it should be taken at the toxicology-exposure interface, with the question being considered on a chemical-by-chemical basis, where evidence permits. If there is evidence to suggest that a child could be more susceptible than an adult to a chemical's toxic effect, based on the mode of action of the chemical for the critical toxicity endpoint and child specific toxicokinetic/toxicodynamic factors, then averaging exposure over a lifetime would not be considered appropriate. Where there is an absence of evidence either way regarding the mode of action and the sensitivity of children, a precautionary position could be adopted i.e. that a child *could* be more sensitive and therefore lifetime averaging is not applied, or alternatively, lifetime averaging is adopted as there is no evidence to suggest children are more sensitive than adults. Within CLEA, the current position is the former conservative position for most chemicals, with the exception of cadmium where lifetime averaging was considered to be appropriate.

It should also be noted that the fact that children often have higher exposure to soil than adults, due to their assumed behaviour and lower body weight, is accounted for in the parameters and modeling of the CLEA model.

2.2.6 USE OF DEFAULT VALUES FOR PHYSIOLOGICAL PARAMETERS

During the derivation of toxicological criteria, it is sometimes necessary to calculate human dose estimates from chemical concentrations in water or air (e.g. drinking water standards and air quality standards/objectives). Default values for physiological parameters such as body weight, inhalation rate and drinking water consumption are used for this purpose. The body weight parameter used for derivation of a HCV in the UK is based on a 70 kg adult drinking 2 litres per day (EA, 2009b). This correlates with new guidance recently published by EFSA who stated that a body weight of 70 kg should be used as a default for the European adult population. Moreover, a 2L default value for chronic daily total liquid intake was also recommended (EFSA, 2012a).

The inhalation rate is also based on a 70 kg adult breathing 20 cubic metres of air per day (EA, 2009b).

There are deviations from these values in other parts of the world. For example, other authoritative bodies such as the World Health Organisation (WHO) use a default body weight of 60 kg (WHO, 2011).

2.3 DEFINITION OF A LOW LEVEL OF TOXICOLOGICAL CONCERN (LLTC)

As indicated above, for the purposes of defining a C4SL, it is suggested that a new term is defined – a Low Level of Toxicological Concern (LLTC) – which would correspond to a pragmatic intake level that remains sufficiently protective of health but represents a level of concern that is low. The units of the LLTC will be the same as those of the HCVs - mg kg^{-1} bw day^{-1} (unless judged otherwise) and they will be used to provide information on the toxicological aspects of a substance, as part of a range of factors to be considered in deriving a C4SL.

It could be argued that it might be simple and effective to adopt a policy decision to derive LLTCs and simply multiply the minimal risk HCVs by a factor of, say, 10. The advantage of this approach is that it would, in theory, be easy to implement, as risk assessors would not have to review the toxicology data and simply multiply the existing HCVs/GACs by a fold factor (assuming linearity and that all substances are the same). However, significant differences between substances exist in reality and there are serious downsides with this approach. If a generic fold increase were employed the resulting modified HCV for one substance may still lie within a low risk/low level of concern range but for another substance it may represent a level of concern that could be SPOSH i.e. if the dose-effects curve is steep. Also, if a small uncertainty factor was used in the derivation of the HCV e.g. 10, then applying a generic fold increase to the HCV of 10 would result in the LLTC being the same as the POD with no aspect of uncertainty being accounted for. Also, in setting the HCV, the most sensitive effect has been looked at quantitatively. Multiplying the HCV by a fold factor may then encroach on a different health effect where the dose-response curves overlap. Hence, there could be a risk of significant harm occurring, if a generic and purely numerical approach to raising the HCV to an LLTC were taken. The same would be true if increases in exposure were advocated without knowing where those exposures lie on the toxicological dose-response curve. Hence interpretation of dose response information is critical, especially when going above minimal risk. Therefore, a scientific approach to define LLTCs is recommended as described in Section 2.4 below.

2.4 SUGGESTED FRAMEWORK FOR DEFINING A LOW LEVEL OF TOXICOLOGICAL CONCERN (LLTC)

A framework for evaluating chemical-specific toxicology data for the purposes of C4SL derivation is presented in the form of a flowchart in Figure 2.2. The remainder of this section is structured to guide the reader through the flowchart by referring to, and providing further information on, its numbered elements. It is recommended that a suitably qualified individual who sufficiently understands the nature of toxicological data, collates the evidence and produces a document for each substance being considered, that works through the steps of the framework for each route of exposure.

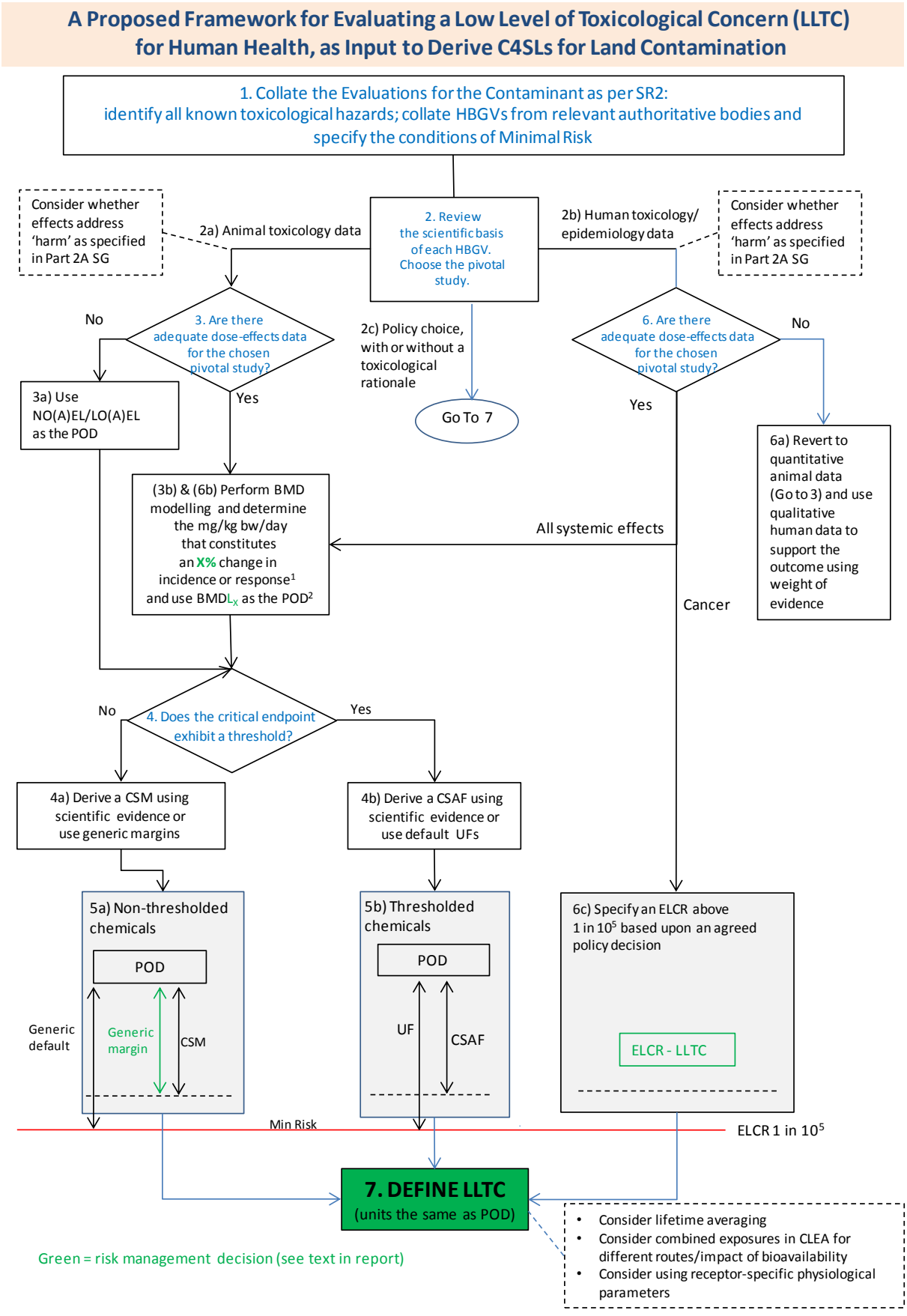

Figure 2.2: Toxicological Framework for Defining LLTCs

2.4.1 FLOWCHART ELEMENT 1: COLLATE THE EVALUATIONS FOR THE CONTAMINANT AS PER SR2: IDENTIFY ALL KNOWN TOXICOLOGICAL HAZARDS; COLLATE HBGVS FROM RELEVANT AUTHORITATIVE BODIES AND SPECIFY THE CONDITIONS OF MINIMAL RISK

The general principles described in the section above, together with the detailed methods published in SR2 and the COC guidance (2012) form the basis of defining a minimal risk HBGV that is unlikely to represent a health concern. Since the purpose of deriving an LLTC is to underpin the definition of C4SLs representing a low level of risk (at a "more pragmatic but still strongly precautionary" level above minimal risk), it is recommended that, for any substance, the minimal risk HBGV position is understood and mapped first, before attempting to derive an LLTC. This is the purpose of flowchart element 1.

It is simplest to collate a record of the information initially in spreadsheet form (for example by following the Human Toxicological Data Sheet (HTDS) template used in Appendices C to H) to provide an overview of the various existing HBGVs derived for each substance and note the underpinning basis for each HBGV. A repository of the original publically available reports, reviews and relevant data from authoritative bodies should be gathered in a data repository file electronically, as a record of all relevant publically available information for each substance. All of the identified human health hazards by the oral, inhalation and dermal routes should be noted, and where possible a POD determined from the pivotal study for the endpoint and exposure route. All of the authoritative evaluations of the substance, by worldwide organisations (as mentioned in SR2) are tabulated in descending order of the HBGV derived (as in section II of the HTDS). It should be noted that the HBGVs have not necessarily been calculated for the purposes of assessing land contamination and that they may have been derived in the context of specific accompanying exposure scenarios.

2.4.2 FLOWCHART ELEMENT 2: REVIEW THE SCIENTIFIC BASIS OF EACH HBGV. CHOOSE THE PIVOTAL STUDY.

Flowchart element 2 requires a suitably qualified individual who sufficiently understands the nature of toxicological data to review the scientific basis of all existing HBGVs and choose the pivotal toxicology study for the LLTC calculation. This should be a study that has been reviewed and recommended as good quality by an authoritative body.

Three possible options are provided for the type of pivotal study that could be chosen at this point, i.e. in the form of: 1) animal toxicology data; 2) human toxicology/epidemiology data; and 3) a policy choice (i.e. based on an existing guideline from another regime, with or without a toxicological rationale). Good quality human data should predominate as the pivotal study over animal data evaluations where both exist. Each of the three options is described in more detail below.

2a) Animal Toxicology Data

Many *in vivo* toxicological studies are available to study the effects of chemicals, including acute, sub-acute, sub-chronic and chronic toxicity tests, as well as one- and two-generational reproductive studies. For the purposes of deriving HBGVs, data from chronic toxicity tests, carcinogenicity tests, as well as reproductive studies are predominantly used, if available, as these better simulate the chronic exposure of humans to contaminants in soil. In general, *in vivo* studies should be performed in accordance with internationally accepted guidelines (e.g. OECD guidelines).

Chronic toxicity studies are used to characterise the profile of the chemical in a mammalian species (usually rodents), and to determine the dose-response relationships, following prolonged and repeated exposure to defined doses of chemical. Carcinogenicity studies are carried out to observe test animals for the majority of their life span for the development of neoplastic lesions during or after exposure to a chemical via various routes of exposure.

One-generation studies are designed to evaluate the reproductive and developmental effects that may occur following pre- and postnatal chemical exposure, as well as to assess systemic toxicity in pregnant and lactating females, and young and adult offspring. Pups are assessed for reproductive and developmental effects, developmental neurotoxicity and developmental immunotoxicity (OECD, 2012).

Two-generation studies are designed to provide general information on the effects of a chemical on the integrity and performance of male and female reproductive systems, as well as on the growth and development of offspring. Data from such a study should provide an estimation of the no-effect level and an understanding of the adverse effects on reproduction, parturition, lactation, postnatal development, growth and sexual development (OECD, 2001).

2b) Human Toxicology/Epidemiology Data

It is clearly not ethical to perform toxicology studies in humans. Therefore, much of the human dose-response data comes from epidemiology studies carried out following unavoidable chemical exposure, where humans have suffered adverse effects. Such studies are often in worker populations, where exposure to a substance has occurred within a given exposure scenario, and in population studies where people were exposed to chemicals inadvertently or in an unregulated context. It can be difficult to gain good quantitative dose-effects information from human data, but evidence of effects in man can corroborate the findings from animal studies in a weight-of-evidence approach. The most useful epidemiological data for the purposes of setting an LLTC are obtained from observational studies, such as cohort and case-control studies, in an occupational setting.

A cohort study looks at the effects that arise following exposure to a chemical. Subjects are defined according to their exposure status and followed over a period of time to assess the prevalence of health outcomes. In contrast, case-control studies select subjects on the basis of their disease status. Their potential chemical exposures are then compared with a control, non diseased group. Data from both types of study may be used as the basis of an LLTC, although in most cases, cohort studies are most relevant. If epidemiology or other human data are available, they will often take precedence over animal data, although this is largely dependent on the quality of the human data (EA, 2009b).

2c) Policy choice, with or without a toxicological rationale

Where there is not a toxicological basis on which to base the derivation of an LLTC, in the absence of evidence, a value could be based on policy decisions alone. For instance, where there are insufficient scientifically robust toxicity data to derive a POD. In such cases it would be a policy decision if and whether to go forward with stating an LLTC for the substance.

A policy driven approach may also be used in cases where the C4SL that would reflect low risk is considered unachievable in practical terms, or if it would disproportionately target exposures from soil compared with other media such as water or air. In such cases, a toxicologically-based LLTC could be derived which would then be over-ridden by a policy based LLTC that would be recommended centrally by UK government. It is advisable that the scientific evaluation is performed and communicated, such that there is transparency in providing information of the level of toxicological concern the policy-based LLTC represents.

2.4.3 FLOWCHART ELEMENT 3: ARE THERE ADEQUATE DOSE-EFFECTS DATA FOR THE CHOSEN PIVOTAL STUDY – ANIMAL DATA?

This element of the flowchart relates to the use of animal toxicology data to derive an LLTC. More specifically, it requires a suitably qualified individual who sufficiently understands the nature of toxicological data to consider whether there are adequate data from the chosen pivotal study to perform BMD modelling.

If the answer is “no”, then a NO(A)EL/LO(A)EL should be used as the POD (3a). In this case, the process would be the same as described in SR2 and COC guidance (2012).

If the answer is “yes”, then BMD modelling should be performed (3b) in order to provide a more quantitative interpretation of the data. A chemical-specific decision regarding what % increased incidence of effect i.e. the BMR is necessary.

(3a) If the answer is “no”, then the assessor should use a NOAEL/LOAEL as the POD. In this case, the process would be the same as described in SR2 (EA, 2009b) and COC guidance (2012), as the information provided in the study would be considered too weak to draw good quantitative conclusions about the dose response, or to provide robust scientific evidence of the level of risk/concern at doses higher than a single POD. Depending upon the substance and the nature of the data in the pivotal toxicology study, it may be possible to use a NO(A)EL to define minimal risk, and a LO(A)EL to define the LLTC. However, this would need to be judged on a substance by substance basis, looking at the dosing regimen used in the study. One could also consider using an value in between the NO(A)EL and the LO(A)EL (e.g. the median point).

(3b) If the answer is “yes”, then BMD modelling should be performed. As explained above, BMD modelling provides a more quantitative way of interpreting toxicology data, such that incremental increases in exposure can be aligned to an increase or decrease in continuous data as well as to an increased incidence of an effect. Therefore, if data are available, that are suitable for BMD modelling, then such modelling should indeed be carried out in order to provide a more quantitative interpretation of the data. If BMD modelling has been performed under the auspices of an authoritative body, this should be used in preference to an evaluation from the open peer review literature or performed afresh.

Benchmark dose software (BMDS) is freely available from the USEPA, as well as PROAST software developed by the Netherlands National Institute for Public Health and the Environment (RIVM) (EFSA, 2011; USEPA, 2012). Additional commercially available resources include the Excel-based Wizard and DRAGON software products developed by ICF international (USEPA, 2012). Whilst it is mathematically straightforward to use the software, accompanying technical guidance should be closely followed and care taken in modelling the data appropriately and transparently. The output is a curve from which various BMRs and their associated BMDs can be calculated as options from which to choose the POD for an LLTC calculation.

As discussed in Section 2.2.1, a decision is necessary as to what % increased incidence of effect (i.e. the BMR and associated BMD or BMDL) is considered appropriate to represent low concern for each substance. The shape of the dose-response curve may influence this choice, and advice should be sought from a person who understands the nature of the toxicology data and health effect of pivotal concern.

For the purposes of LLTC derivation, it may be considered pragmatic and precautionary from a risk management perspective to use the same BMR as used in minimal risk calculations (i.e. in most cases 10% BMR is proposed for carcinogenicity studies and 5% as a default BMR for continuous data, although this could be smaller for incidence data in epidemiology studies with large populations (EFSA 2009)). This would mean, in scientific terms, that when the BMDL (representing the lower 95th percentile confidence limit of the BMD) defines minimal risk, the BMD of the same BMR would be used as the POD for LLTC derivation wherever possible, unless there are justifiable reasons to choose otherwise. Maximally for an LLTC, based upon a widely held view in stakeholder feedback, it is also suggested that the BMR chosen should not be above 10% incidence for any effect that is chosen as a measure of low concern. However, the final choice of what level of BMR represents 'low concern' for the purposes of deriving a C4SL is a risk management choice.

2.4.4 FLOWCHART ELEMENT 4: DOES THE CRITICAL ENDPOINT EXHIBIT A THRESHOLD?

If the answer is “no”, i.e. for non-thresholded chemicals, then the assessor should look to use either a generic margin or a chemical-specific margin (CSM) if robust data are available.

If the answer is “yes” i.e. for thresholded chemicals, then the assessor should look to derive a chemical-specific adjustment factor (CSAF), if robust data are available.

The identification of whether the chemical in question exhibits a threshold for the critical toxicity endpoint is a key decision in the framework and should be made by a suitably qualified individual who understands the nature of the toxicology data.

(4a) If the answer is “no”, i.e. for non-thresholded chemicals, then a chemical-specific margin should be defined based on a scientifically defensible rationale around the uncertainties in the toxicological data and with the use of expert judgement.

A margin that would constitute ‘low concern’ for the C4SL policy objectives may be derived either generically (e.g. a set default margin to be applied to all genotoxic carcinogens) or in a chemical specific way using scientific information in the assessment of uncertainty that is specific for the chemical being evaluated in each case.**Generic margins** to be applied to all non-thresholded genotoxic carcinogens, are dependent upon the BMR and BMD(L) chosen for the POD and should be chosen on the basis of scientific knowledge. As mentioned above in Section 2.2.2, the COC (2012) propose that a suitable margin might be 10,000 as applied to a $BMDL_{10}$, for minimal risk or is ‘unlikely to be of concern’ (COC 2012). The EFSA Scientific Committee (2005) also considered this generic figure of 10,000 for a MOE with a $BMDL_{10}$ from an animal study (which parallels the COC-proposed margin approach) (EFSA 2005). Similarly, SR2 mentioned the application of a factor of 10,000 to a $BMDL_{10}$ as representing minimal risk (EA, 2009b).

A different margin representing ‘low concern’ may be chosen to apply to a BMD_{10} or $BMDL_{10}$ from animal data. For the purpose of deriving LLTCs, a generic margin of 5,000 is proposed, when a BMD_{10} is used as the POD. This leads to a notional risk level of 1 in 50,000. Other margins would need to be chosen and developed for use with BMRs lower than a 10% increased incidence of effect in order to achieve a similar notional risk level across different substance (see table 5.5), or a transparent explanation given if the resulting risk level is different across different substances.Alternatively, a **Chemical Specific Margin** (CSM) may be based on a scientifically defensible rationale around the uncertainties in the toxicological data and with the use of expert judgement. EFSA (2005) suggest the following uncertainties be considered in setting a margin of exposure.

- Intraspecies differences (human variability factors) – range 1-10
- Interspecies differences (animal to human factors) – range 1-10
- Additional uncertainties – range 1-100

Such an approach could be adopted in setting a CSM. Differences in fate and behaviour between animals and human could be amended if there are toxicokinetics/dynamic data that show there is <10-fold difference between animals and humans. Similarly, toxicokinetic/dynamic data may indicate that there is <10-fold difference between individuals. The factor of 100 covers additional uncertainties including inter-individual variability in cell cycle control and DNA repair as well as the uncertainties surrounding the use of a point of departure that does not represent a no effect level. Quality of the database/study should also be considered. Again such factors could be amended as appropriate. This approach to set a CSM would have to be carried out on a chemical specific basis. In practical terms, there is currently no guidance on how the 100-fold factor for additional uncertainties would be modified if

one had data on DNA repair or cell cycle control etc. though in qualitative ways these aspects can vary between individuals and should be accounted for, therefore it should be regarded that this application of a 100-fold assessment factor is a pragmatically applied tool to represent such uncertainty at this time.

An example of a breakdown of factors that can be used to account for specified uncertainties in a dataset, that have been used in UK Government chemical risk assessment, are shown in Table 2.3, as presented by the Interdepartmental Group on Health Risks from Chemicals (IGHRC, CR9, 2003). As shown in the table, various factors in considering the toxicology data could be amended and used to derive CSMs.

Table 2.3: Example of default factors used in UK Government risk assessment (IGHRC, 2003)

Chemical sector	Animal to human factor	Human variability factor	Quality or quantity of data factor	Severity of effect factor
Food additives and contaminants	10	10	2-10	2-10
Agricultural pesticides	10	10	2-10	2-10
Veterinary products	10	10	2-5	2-10
Air pollutants	10	10	-	-
Consumer products	10	10	2 or greater	2 or greater
Drinking water contaminants	1-10	1-10	1-10	1-10
Soil contaminants	1-10	1-10	1-10	1-10
Human medicines	1-10	1-10	1-100	-

If robust data are not available on which to make an informed decision on how to derive a CSM, then a default generic margin should be used.

(4b) If the answer is “yes” i.e. for thresholded chemicals, then the assessor should look to derive a CSAF if robust data are available. As described above, chemical specific toxicokinetic or toxicodynamic data may be used, if available, to help identify more specifically the differences in sensitivity between humans and the animals used in the toxicity study, and between different human populations (i.e. adults and children). Hence more specific factors for toxicokinetics and toxicodynamics could be used rather than the default factors of 10 (IPCS, 2005).

This is not a new concept as it was described in SR2 (EA, 2009b) as a potential methodology for deriving HCVs and has also been used by other authoritative bodies. For example, the European Food Safety Authority (EFSA) used a CSAF of 3.9 to a $BMDL_5$ to derive a urinary cadmium concentration (see Appendix F). The EA suggests that where differences in sensitivity of the test and target population to a chemical are known and can be quantified or estimated, then a CSAF may be applied i.e. humans may be more or less sensitive than the test population hence a larger or smaller factor may be applied (EA, 2009b).

If there is no additional information available that could be used, or if the available data are not considered to be robust and scientifically defensible, then default UFs should be used. For thresholded systemic toxicity, such a default factor is usually 100. There may be some cases where the UF needs to be higher than the default, if a special consideration needs to be taken into account, e.g. for sensitive subgroups.

For both threshold and non-threshold chemicals, factors for all of the individual uncertainties are simply multiplied together to contribute to an overall value for a CSM (for non-threshold chemicals) or a CSAF (for threshold chemicals), that is then applied to the POD.

2.4.5 FLOWCHART ELEMENT 5: CALCULATING THE LLTC

For non-thresholded chemicals, the LLTC is calculated by dividing the POD by the margin (either a generic margin or a CSM)

POD/margin = LLTC (units as per POD)

For thresholded chemicals, the POD is divided by a CSAF (or default UF);

POD/(CSAF or default UF) = LLTC (units as per POD)

Flowchart element 5 requires the derivation of the LLTC by performing the calculation shown above using the POD and the appropriate measure of uncertainty in the form of a margin or CSAF.

These calculations yield a fixed value based upon the uncertainties in the toxicology data for the pivotal study on which the POD is based.

2.4.6 FLOWCHART ELEMENT 6: ARE THERE ADEQUATE DOSE-EFFECTS DATA FOR THE CHOSEN PIVOTAL STUDY – HUMAN DATA?

If the answer is "no", then the assessor should revert if possible to quantitative data from animal studies. If the answer is "yes" then BMD modelling can be performed on the human data or an excess lifetime cancer risk (ELCR) can be defined.

This element of the flowchart relates to the use of human toxicological/epidemiological data to derive an LLTC. More specifically, it requires a suitably qualified individual who understands the nature of the toxicology or epidemiology data to assess whether there are adequate quantitative data from the chosen pivotal human study. If "no", then the assessor should revert to quantitative data from animal studies (6a). If the answer is "yes" then BMD modelling can be performed on the human data (6b) or an excess lifetime cancer risk (ELCR) can be defined (6c). If both have been performed, the BMD modelling route should carry more weight over an ELCR calculation, the latter of which is only a rough estimation of risk. However, worldwide authoritative bodies do use the concept of ELCR and it is useful as a comparator alongside the BMD approach.

(6b) In circumstances where there are good dose-effects relationships in human epidemiology data, they can be modelled using BMD approaches, as with animal data (see above). In such cases, as with animal data, a CSAF or margin may also be derived, which conceivably may be lower as interspecies differences do not need to be accounted for, and an LLTC may be derived. Good human data tend to carry more weight than animal data, where both are available.

(6c) As indicated above, quantitative dose-response modelling of cancer data involves the concept of ELCR, defined as:

> *'Potential carcinogenic effects that are characterized by estimating the probability of cancer incidence in a population of individuals for a specific lifetime from projected intakes (and exposures) and chemical-specific dose-response data (i.e., slope factors). By multiplying the intake by the slope factor, the ELCR result is a probability.'*

From such quantitative risk estimations, relevant guidance has stated that an ELCR of 1 in 100,000 (10^{-5}) should constitute minimal risk (EA, 2009a; DEFRA, 2008). However, it is also considered in previous guidance that ELCR calculations are approximations of risk (i.e. what could be considered a rough estimate rather than an accurate prediction of risk). For the purposes of C4SL derivation, a risk estimate of 1

in 10,000 – 1 in 50,000 could be specified as 'low risk' and this would be a generic level used for all human carcinogens, irrespective of mode of action.

2.4.7 FLOWCHART ELEMENT 7: DERIVE LLTC

The definition of the LLTC has been described previously. Overall, there are 3 routes to deriving an LLTC:

- For thresholded chemicals: derivation of a human intake using POD divided by CSAFs (or default UFs). The POD can be derived from animal or human data.
- For non-thresholded chemicals: derivation of a human intake using POD divided by a recommended CSM (or default generic margin). The POD can be derived from animal or human data.
- For human carcinogens (with quantitative data): Recommendation of an intake dose based on human data that equals a specified ELCR that is considered low risk.

2.4.8 CALCULATION OF A CHILD-SPECIFIC LLTC

The use of default values for physiological parameters when deriving HBGV (in units of µg kg $bw^{-1}.day^{-1}$) from drinking water guidelines or air quality standards/objectives has been discussed in Section 2.2.6. Typically default values based on adult exposure are used, but this can introduce unnecessary conservatism where the HBGVs are compared to exposure estimates for children. For example, inhalation HBGVs ($HBGV_{inh}$) for volatile contaminants are recommended as intake values (µg kg^{-1} bw day^{-1}) for use in CLEA, and have often been based on airborne contaminant concentrations (mg m^{-3}) such as reference concentrations taken from toxicology studies (e.g. USEPA RfCs) or Air Quality Objectives AQOs/Standards. These RfCs and AQOs/Standards are generally recommended for long-term or lifetime exposure with minimal risk. The conversion from an airborne concentration to a $HBGV_{inh}$ is based on adult receptor characteristics (i.e. daily inhalation rate of 20 m^3 and 70 kg body weight) whereas the calculation of exposure for the residential land-use scenario is for a 0-6 year old child (with the default lower inhalation rate and significantly lower body weight). This approach is considered to introduce an unnecessary level of conservatism as a child's exposure relative to body weight is approximately 2-3 times higher than that for an adult. A similar situation can arise where ingestion HBGVs are based on drinking water guidelines.

For the purposes of generating the C4SL it is therefore proposed that receptor-specific LLTCs are derived where they are based on airborne contaminant concentrations such as RfCs and Air Quality Standards/Objectives (mg m^{-3}) or drinking water guidelines (mg L^{-1}). However, it is not considered appropriate to derive receptor-specific LLTCs where there is uncertainty over the how the media concentration has been derived, i.e. a media concentration may be derived from a toxicologically-derived intake value but it may not be transparent as to whether this is based on child or adult physiological characteristics and consumption or respiration rates.

Physiological parameter values and respiration rates should be based on those recommended for the relevant age class(es) for derivation of the C4SL (see Section 3) and default water consumption rates of 1 $L.day^{-1}$ for children and 2 $L.day^{-1}$ for adults.

It is recommended that adult receptor characteristics are assumed for derivation of LLTC for commercial land-use or where lifetime averaging has been assumed.

2.4.9 OTHER TOXICOLOGICAL CONSIDERATIONS

In addition to the provision of an LLTC, the toxicological evaluation should also identify whether lifetime averaging should be performed during the exposure modelling. The

same considerations that apply to lifetime averaging discussed in Section 2.2.5 for derivation of the HCVs also apply to the derivation of LLTCs.

Further consideration must also be given with regard to whether modelled exposure via different routes need to be combined for the C4SL derivation. In simple terms, if the critical effect is systemic and can be induced following absorption into the body via any route – oral, inhalation or dermal - then exposure needs to be combined, on the assumption that a person can be exposed concomitantly. If the critical effect is local (e.g. site of contact carcinogenicity), then exposure from different routes does not need to be combined.

If local effects (e.g. skin allergy, skin cancer, lung irritation etc) are of potential concern (e.g. chromium VI allergy), this should be considered during the setting of the C4SLs. In all cases of HCVs derived to date by the Environment Agency, they have been protective of any local effects occurring and it is not expected that the modest increase represented by LLTCs would lead to any significantly increased risk of harm via the local route.

It should be noted that HCVs and LLTCs have been developed for chronic exposure scenarios and are not applicable to high dose acute exposure situations.

2.4.10 COMPARISON OF LLTCs WITH HCVs

The overall LLTC derivation methodology described above contains several elements which are similar to or, conversely, differ from, current approaches to deriving “minimal risk” HCVs. Key aspects of the similarities and differences between the approaches are summarised below:

Table 2.4: Key aspects of the derivation of LLTCs and HCVs.

Aspect	HCV	LLTC
Database	Expert body evaluation from authoritative sources as listed in SR2	Expert body evaluation from authoritative sources as listed in SR2
Pivotal study	Most appropriate study as chosen by a suitably qualified individual who understand the nature of the data as described in SR2	Most appropriate study as chosen by a suitably qualified individual who understand the nature of the data as described in SR2
Critical effect	Most sensitive effect	Most sensitive effect. Care must be taken to ensure that an LLTC derived using this data does not overlap the next most sensitive effect.
POD	NO(A)EL/LO(A)EL/BMDL*	BMD*/NO(A)EL/LO(A)EL
BMR	Not used in any HCVs to date 10% (animal carcinogenicity studies); <10% could be used if data sensitivity allows.	10% (animal carcinogenicity studies); <10% BMR could be used if data sensitivity allows. Maximally a BMR of 10%.
Uncertainty evaluation - threshold chemicals	Default generic UF/CSAF	CSAF/default generic UF
Uncertainty evaluation - non-threshold chemicals (animal data)	Default 10,000	CSM or generic 5,000
Uncertainty evaluation - non-threshold chemicals (human data)	Not used in any HCVs to date	CSM or generic margin to complement choice of BMR to achieve a notional ELCR between 1 in 10,000 – 1 in 50,000
ELCR	1 in 100,000	1 in 10,000 - 1 in 50,000
Policy-driven approach where necessary, if appropriate and scientifically justified	Applicable	Applicable

* SR2 states that a BMD approach could be taken to deriving an HCV but in practice it has never been adopted. In principle, a BMDL of the lowest response seen in the study would be the minimal risk POD. For an LLTC derivation, BMD modelling is suggested as the preferred approach, if data allow.

It is important to reiterate that, although the above table summarises the generalised LLTC derivation methodology (versus that used for deriving HCVs), deviations may be appropriate for certain substances, as long as the rationale for doing so is transparent and scientifically justifiable.

4. CL:AIRE, 2016. CAR-SOIL™ Control of Asbestos Regulations 2012 Interpretation for Managing and Working with Asbestos in Soil and Construction and Demolition Materials Industry Guidance

Full Reference: CL:AIRE, 2016. CAR-SOIL™ Control of Asbestos Regulations 2012 Interpretation for Managing and Working with Asbestos in Soil and Construction and Demolition Materials Industry Guidance, CL:AIRE, in association with Joint Industry Working Group Asbestos in Soil and Construction and Demolition Materials, July 2016.

[Provided in full as a Core Document]

5. CL:AIRE, 2017a. Decision Support Tool for the Categorisation of Work Activities Involving Asbestos in Soil and Construction & Demolition Materials in accordance with the Control of Asbestos Regulations 2012

Full Reference: CL:AIRE, 2017a. Decision Support Tool for the Categorisation of Work Activities Involving Asbestos in Soil and Construction & Demolition Materials in accordance with the Control of Asbestos Regulations 2012: v2.1 March 2017. Access to Excel™ spreadsheet at https://www.claire.co.uk/projects-and-initiatives/asbestos-in-soil?start=4

[An Excel™-based spreadsheet tool available at the web link above]

6. CL:AIRE, 2017b. Decision Support Tool for the Qualitative Risk Ranking of Work Activities and Receptors Involved in or Exposed to Asbestos in Soil and Construction & Demolition Materials

Full Reference: CL:AIRE, 2017b. Decision Support Tool for the Qualitative Risk Ranking of Work Activities and Receptors Involved in or Exposed to Asbestos in Soil and Construction & Demolition Materials : v2.1 March 2017. Access to Excel™ spreadsheet at https://www.claire.co.uk/projects-and-initiatives/asbestos-in-soil?start=4

[An Excel™-based spreadsheet tool available at the web link above]

7. Darnton, 2023. Quantitative assessment of mesothelioma and lung cancer risk based on Phase Contrast Microscopy (PCM) estimates of fibre exposure: an update of 2000 asbestos cohort data

Full Reference: Darnton, 2023. Quantitative assessment of mesothelioma and lung cancer risk based on Phase Contrast Microscopy (PCM) estimates of fibre exposure: an update of 2000 asbestos cohort data, Lucy. Darnton, Environmental Research, 230, 2023.

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Quantitative assessment of mesothelioma and lung cancer risk based on Phase Contrast Microscopy (PCM) estimates of fibre exposure: an update of 2000 asbestos cohort data

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A B S T R A C T

An earlier meta-analysis of mortality studies of asbestos-exposed worker populations, quantified excess mesothelioma and lung cancer risks in relation to cumulative exposure to the three main commercial asbestos types. The aim of this paper was to update these analyses incorporating new data based on increased follow-up of studies previously included, as well as studies of worker populations exposed predominantly to single fibre types published since the original analysis.

Mesothelioma as a percentage of expected mortality due to all causes of death, percentage excess lung cancer and mean cumulative exposure were abstracted from available mortality studies of workers exposed predominantly to single asbestos types. Average excess mesothelioma and lung cancer per unit of cumulative exposure were summarised for groupings of studies by fibre type; models for pleural and peritoneal mesothelioma risk and lung cancer risk in terms of cumulative exposure for the different fibre types were fitted using Poisson regression.

The average mesothelioma risks (per cent of total expected mortality) per unit cumulative exposure (f/cc.yr), R_M, were 0.51 for crocidolite, 0.12 for amosite, and 0.03 for the Libby mixed amphiboles cohort. Significant heterogeneity was present for cohorts classed as chrysotile, with R_M values of 0.01 for chrysotile textiles cohorts and 0.0011 for other chrysotile-exposed cohorts. Average percentage excess lung cancer risks per unit cumulative exposure, R_L, were 4.3 for crocidolite and amosite combined, 0.82 for Libby. Very significant heterogeneity was present for chrysotile-exposed cohorts with R_L values spanning two orders of magnitude from 0.053 for the Balangero mine to 4.8 for the South Carolina textiles cohort. Best fitting models suggest a non-linear exposure-response in which the peritoneal mesothelioma risk is proportional to approximately the square of cumulative exposure. Pleural mesothelioma and lung cancer risk were proportion to powers of cumulative exposure slightly less than one and slightly higher than one respectively.

1. Introduction

In 2000, Hodgson and Darnton published a meta-analysis of asbestos-exposed worker cohorts for which quantitative data on asbestos exposure was available (Hodgson and Darnton, 2000). This included estimates of mesothelioma and lung cancer risk in relation to cumulative exposures to the three commercial asbestos types, crocidolite, amosite and chrysotile, and linear and non-linear exposure-response models that have been widely used for risk assessment.

There is a widely held scientific consensus that amphibole asbestos is much more potent in relation to mesothelioma than chrysotile. In 2000, Hodgson and Darnton found that much of the variation in the mesothelioma risk per unit exposure seen across studies could be explained by fibre mineral type alone with amosite and crocidolite fibres conferring a risk 100 and 500 times that of chrysotile respectively (for exposures typical of these historic settings).

In relation to lung cancer, the meta-analysis found that substantial variation in the risk per unit exposure remained after allowing for fibre type differences, and the extent to which amphibole confers a higher risk than chrysotile was more uncertain. The originally suggested summary risk estimates (Hodgson and Darnton, 2000) were between a factor of 10 and 50 times higher for amphibole compared with chrysotile.

After more than 20 years since the original publication, various updates to some of the included studies have been published, as well as additional studies that can now be incorporated into the meta-analysis. Here we report an update of the meta-analyses in the light of these latest

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available data, as well as a review of the evidence for a non-linear exposure-response. Resulting updated potency factors for mesothelioma and lung cancer are relevant to wider evidence about the mineralogical characteristics of elongate mineral particles and cancer risk.

2. Materials and methods

2.1. The H&D approach

The meta-analytical approach used by Hodgson and Darnton is described in detail in the original paper. Briefly, this was motivated by a concern to maximise the amount of evidence that could be incorporated whilst minimising the limitations arising from the underlying data. One approach is to derive cross-study summary estimates of exposure-response from any available within-study exposure-response relationships (Berman and Crump, 2008a). From a statistical point of view, study specific analyses have usually been more feasible for lung cancer than mesothelioma since the former is a relatively common malignancy. Given the sizes of some of the available studies, absolute numbers of mesothelioma were small in some cases even where asbestos exposures were high. Even for lung cancer, there are relatively few studies available with internal regression analyses based on quantitative assessments of cumulative exposure. Furthermore, such analyses can suffer from bias towards the null in situations where exposure is misclassified due to uncertainties associated with the underlying methods.

In view of these limitations, Hodgson and Darnton summarised measures of mesothelioma and excess lung cancer risk at the overall cohort-level in relation to average cumulative exposure, and then used these values to produce cross-study summary potency estimates (risk per unit of exposure) according to fibre type. In addition to these linear estimates of risk in terms of cumulative exposure, Hodgson and Darnton also derived non-linear exposure-response models derived from the cohort level data. These analyses were motivated *inter alia* by the observation that the proportion of pleural to peritoneal mesotheliomas was not constant over the range of exposures seen in the available studies, even though a linear relationship for the total mesothelioma risk in terms of cumulative exposure was consistent with the data. Non-linear relationships for excess lung cancer risk in terms of cumulative exposures were also explored. Here, we update both the linear potency estimates and re-fit the non-linear models for pleural and peritoneal mesothelioma as well as for lung cancer.

The method required identification of studies of asbestos-exposed cohorts which examined mesothelioma and lung cancer outcomes for which quantitative estimates of exposure were available as well as information about fibre type. Studies where the exposure was exclusively to one kind of fibre were particularly central to this approach with studies being classified as 'pure fibre' (crocidolite, amosite and chrysotile) or mixed fibre exposures.

2.1.1. Metrics

All of the cohort studies of asbestos-exposed workers included in the analysis reported Standardised Mortality Ratios (SMRs) for all causes of death and various specific disease subgroups including lung cancer. We define R_L as the excess of lung cancer deaths over expected per unit cumulative exposure, and R_M as the total (pleural and peritoneal) mesothelioma mortality as a proportion of expected mortality from all causes of death per unit cumulative exposure:

$$R_L = 100\ (O_L\text{-}E_L)/(E_L.X)$$

$$R_M = 100\ O_M/(E_{AC}.X)$$

where, O_L is the observed number of lung cancer deaths in a cohort, E_L the expected number of lung cancers, O_M is the observed number of mesothelioma deaths, E_{AC} the expected number of deaths from all causes (adjusted to allow for differences in the age at first exposure across cohorts), and X is the cumulative asbestos exposure measured via Phase Contrast Microscopy (PCM), or converted to PCM units (in f/cc. yrs).

If the effect of asbestos exposure is to multiply the underlying lung cancer risk – which is largely determined by cigarette smoking – then the relative risk of lung cancer in those exposed to asbestos vs those not exposed would be the same in smokers and non-smokers. In this situation, SMRs from different cohort studies with different smoking prevalences should reflect only the effect of asbestos providing the SMRs themselves are calculated using appropriate lung cancer reference rates – i.e. the reference population has the same smoking prevalence as the worker cohort.

Pleural mesothelioma in men has a particularly strong association with asbestos and mesothelioma overall is relatively rare in general populations even in countries that used asbestos extensively. This means the expected number of mesothelioma deaths in cohorts of the sizes being considered here would typically be very small. A lack of reliable reference mortality rates for the general population due to unavailability of cause of death codes prior to the use of the International Classification of Diseases volume 10 (ICD10) also means that calculation of expected numbers would have been problematic. Most investigators therefore just reported the number of observed deaths, usually based on a best evidence assessment of the cause, rather than a simple reliance on death certificate information.

The relationship between the incidence of mesothelioma and time since the start of asbestos exposure described by Peto implies that in the long-term mesothelioma rates increase rapidly and in proportion to the way deaths from all causes increase. Thus, provided there is sufficient follow-up since the start of exposure (at least 20 years, say), the observed number of mesothelioma deaths as a percentage of all-cause mortality will be a standardised measure of mortality across cohorts, provided that the workers started to be exposed at a similar age. This latter condition was not always the case in the available studies, but the expected mortality from all causes across the cohorts can be adjusted using lifetable methods to reflect that for a population followed up from a standard age. The most typical age at first exposure in the available studies was 30 and so this was used by Hodgson and Darnton.

Lung cancer deaths in cohort studies are typically identified based on the causes of death recorded on the death certificates (DC) rather than appealing to any other information such as autopsy reports. Where possible, Hodgson and Darnton excluded miscoded mesotheliomas from the observed lung cancer count and recalculated the SMRs prior to deriving R_L. Misclassification of pleural mesotheliomas as lung cancer is well-documented effect and counts of the number of mesotheliomas in cohorts were typically derived using the best evidence (BE) available. Leaving miscoded pleural mesotheliomas in the observed lung cancer count in a cohort with high asbestos exposure has the potential to substantial overestimate the SMR, whereas excluding them will tend to underestimate it. However, underestimation will only be to the extent that the effect of miscoding occurs on average in the population from which the reference rates were derived. This will be relatively minor given the much lower asbestos exposures on average across the population as a whole.

The coherence of the R_L and R_M measures when grouped according to the fibre type used in the studies was tested using Poisson regression methods. In the case of chrysotile exposure, we also examined subgroups based on the industrial process as a further potential explanatory variable. The extent of statistical heterogeneity within these groupings was tested by fitting a common value and testing the residual deviance between the observed and predicted number of mesothelioma or lung cancer deaths.

Separate Poisson regression models for pleural mesothelioma, peritoneal mesothelioma, and excess lung cancer in terms of cumulative asbestos exposure and fibre type were also refitted using the same formulation as in the original meta-analysis. Here the exposure-response relationships are allowed to be non-linear by fitting a parameter which is the power of cumulative exposure as follows:

Excess pleural mesothelioma, $P_r = A_i.X^r$

Excess peritoneal mesothelioma, $P_t = B_i.X^t$

Percentage excess lung cancer mortality, $P_l = C_i.X^l$

where, A_i, B_i and C_i are coefficients for fibre type and r, t and l are the 'slope' parameters and indicate a sub-linear slope where these are less than 1, and supra-linear if greater than 1.

3. Results

Hodgson and Darnton originally identified 17 published studies with sufficient data to be included in the meta-analysis. These studies collectively included 21 separate cohorts, of which, three were exposed predominantly to crocidolite fibre, two to amosite, six to chrysotile, and the remaining 10 to mixtures of more than one fibre type. The updated results for amphiboles now include an additional amosite-exposed cohort of manufacturing workers at Tyler, USA (Levin et al., 2016), a cohort of miners exposed to mixed amphiboles (tremolite, winchite, and richterite) in Montana, USA (Sullivan, 2007) (which will be will be referred to as 'Libby' in this paper), as well as updated results for the cohort of crocidolite miners at Wittenoom, Australia, based on further follow-up of that cohort (Musk et al., 2008). Three additional chrysotile exposed cohorts were available for inclusion in this analysis: a cohort of miners at Qinghai, China (Wang et al., 2013a), a cohort of textile manufacturing workers in North Carolina, USA (Loomis et al., 2009), and a cohort of factory workers at Chongqing, China (Wang et al., 2013b). Also, updated results based on further follow-up were available for the cohort of textile workers cohort in South Carolina, USA (Hein et al., 2007), the cohort of miners at Balangero, Italy (Pira et al., 2017), and the cohort manufacturing workers in Connecticut, USA (Finkelstein and Meisenkothen, 2010) (the latter update was for mesothelioma only).

Results for the cohorts classified as exclusively exposed to one of the three asbestos fibre types are shown in Table 1, for total (pleural and peritoneal) mesothelioma, and Table 2, for lung cancer. Results for cohorts exposed to mixtures of the fibre types have not been updated and are not shown. New or updated data since the original Hodgson and Darnton analysis have been shown in Tables 1 and 2 alongside the original data in brackets where applicable.

3.1. Total mesothelioma (pleural and peritoneal)

The original summary crocidolite R_M value was dominated by the Wittenoom cohort, which accounted for 72 out of the total of 97 for the three crocidolite cohorts in the analysis. The updated results include over four times as many mesotheliomas from Wittenoom than in the original review (316 vs 72) and no updates to the other two cohorts. The updated value of R_M is 0.51 (95% CI: 0.45–0.57), very close the original value of 0.52 (95% CI: 0.38–0.60), and the three crocidolite cohorts remain statistically homogeneous with an average of 0.52 (95% CI: 0.47–0.58), again close the original estimate of 0.51 (95% CI: 0.41–0.61).

For amosite, the inclusion of the Tyler cohort more than doubles the number of mesotheliomas on which the summary R_M estimate is based (44 vs 21 in the original review). The central estimate of R_M for the Tyler amosite cohort of 0.12 (95% CI: 0.073–0.17) is identical to that for the Paterson cohort, though exposure estimates for both cohorts are uncertain, with that for Paterson being derived from measurements made at Tyler. The updated estimated amosite summary value is very similar to the original: 0.11 (95% CI: 0.070–0.15) vs 0.10 (95% CI: 0.062–0.15) in the original analysis. The estimate of R_M for the Libby cohort of 0.030 (95% CI: 0.017–0.050) is about one quarter of the summary amosite value and not statistically consistent with it.

As a group, the seven amphibole cohorts are not statistically homogeneous, and separate groupings for crocidolite, amosite and Libby amphibole – with summary values of 0.52, 0.12 and 0.030 respectively – are more consistent with the underlying data.

Of the six chrysotile exposed cohorts in the original review, mesotheliomas were seen only at Quebec (33 deaths), the Balangero mine (2 deaths), and South Carolina (2 deaths). The original summary value of R_M for chrysotile (0.001, 95% CI: 0.0007–0.0014) was therefore heavily

Table 1
Exposure-specific total mesothelioma (pleural and peritoneal) mortality (RM) by cohort and fibre type group

Cohort	Label	Fibre	Process	Cumulative exposure (f/cc.yr)	Total mesothelioma deaths	Peritoneal mesothelioma deaths	R_M	(95% Confidence interval)
Wittenoom[6]	Wit(o)	o	M	23	(72) 316	(10) 48	(0.52) 0.51	(0.45 , 0.57)
SA mine[13]	SA(o)	o	M	16.4	20	2	0.59	(0.36 , 0.91)
Massachusetts[14]	Mas(o)	o	CF	120	5	3	0.68	(0.22 , 1.6)
Total crocidolite							**(0.51) 0.52**	**(0.47 , 0.58)**
SA mines[13]	SA(a)	a	M	23.6	4	1	0.060	(0.016 , 0.15)
Paterson[15]	Pat(a)	a	I	65	17	9	0.12	(0.068 , 0.19)
Tyler[4]	Tyl(a)	a	I	50	23	7	0.12	(0.073 , 0.17)
Total amosite							**(0.10) 0.11**	**(0.070 , 0.15)**
Libby[5]	Lib(la)	la	M	85	15	1	0.030	(0.017 , 0.050)
Quebec[16]	Que(y)	y	M	600	33	0	0.00090	(0.00064 , 0.0013)
Balangero[11]	Bal(y)	y	M	300	(2) 7	0	(0.0025) 0.0042	(0.0017 , 0.0088)
Qinghai[7]	Qin(y)	y	M	120	0	0	0	(0.000 , 0.0084)
New Orleans (plant 2)[17]	Nor(y)	y	C	22	0	0	0	(0.000 , 0.034)
S Carolina (women)[10]	SCf(y)	y	T	26	0	0	0	(0.000 , 0.021)
S Carolina (men)[10]	SCm(y)	y	T	28	(2) 3	(1) 1	(0.013) 0.016	(0.0032 , 0.045)
N Carolina[8]	NC(y)	y	T	68.3	8	0	0.010	(0.0042 , 0.019)
Chongqing[9]	Cho(y)	y	T	105.2	2	1	0.022	(0.000 , 0.081)
Connecticut[12]	Con(y)	y	F	46	(0) 2	0	(0) 0.0016	(0.00019 , 0.0057)
Total chrysotile							**(0.0010) 0.0014**	***P<0.001***
Excluding textiles							**(0.0010) 0.0011**	**(0.00079, 0.0014)**
Textiles only							**0.010**	**(0.0056 , 0.017)**

Table 2
Exposure-specific Lung cancer mortality (R_L) by cohort and fibre type group.

Cohort	Label	Fibre	Process	Cumul. Exp. (f/cc. yr)	Lung cancer deaths	Excess	R_L	95% Confidence interval	
Wittenoom (Musk et al., 2008)	Wit(o)	o	M	23	(87) 281	(38.3) 144.3	(3.4) 4.6	(3.6,	5.7)
SA mine (Sluis-Cremer et al., 1992)	SA(o)	o	M	16.4	19	8.8	5.2	(0.74,	12)
Massachusetts (Talcott et al., 1989)	Mas(o)	o	CF	120	8	7.4	10	(4.0,	21)
Total crocidolite							**(4.2) 4.8**	**(3.8,**	**5.9)**
SA mines (Sluis-Cremer et al., 1992)	SA(a)	a	M	23.6	21	6.5	1.9	(−0.44,	5.1)
Paterson (Seidman et al., 1986)	Pat(a)	a	I	65	98	77.5	5.8	(4.4,	7.4)
Tyler (Levin et al., 2016)	Tyl(a)	a	I	50	89	52.5	2.9	(1.9,	4.0)
Total amosite							**(5.2) 4.0**	**(3.3,**	**4.9)**
Crocidolite and amosite combined							**(4.8) 4.3**	**(3.7,**	**5.0)**
Libby (Sullivan, 2007)	Lib(la)	la	M	85	89	36.5	0.82	(0.43,	1.3)
Total amphibole (including Libby)							**2.7**	***P < 0.001***	
Quebec (Liddell et al., 1997)	Que(y)	y	M	600	587	155	0.060	(0.042,	0.079)
Balangero (Pira et al., 2017)	Bal(y)	y	M	300	(19) 53	(1.7) 7.3	(0.030) 0.053	(−0.044,	0.17)
Qinghai (Wang et al., 2013a)	Qin(y)	y	M	120	56	44.6	3.3	(2.3,	4.5)
New Orleans (plant 2) (Hughes et al., 1987)	Nor(y)	y	C	22	42	9.6	1.3	(−0.30,	3.4)
S Carolina (women) (Hein et al., 2007)	SCf(y)	y	T	26	(38) 61	(24.2) 33.5	(6.7) 4.7	(2.7,	7.1)
S Carolina (men) (Hein et al., 2007)	SCm (y)	y	T	28	(74) 116	(41.8) 66.4	(4.6) 4.8	(3.3,	6.4)
N Carolina (Loomis et al., 2009)	NC(y)	y	T	68.3	249	132.6	1.7	(1.3,	2.1)
Chongqing (Wang et al., 2013b)	Cho(y)	y	T	105.2	53	40	2.9	(2.0,	4.1)
Connecticut (Finkelstein and Meisenkothen, 2010)	Con(y)	y	F	46	49	13.2	0.80	(0.027,	1.8)
Total chrysotile							**(0.062) 0.078**	***P < 0.001***	
Quebec and Balangero only							**(0.060) 0.060**	**(0.043,**	**0.078)**
Mining cohorts							**0.064**	***P < 0.001***	
Textile cohorts							**2.2**	***P < 0.001***	
S Carolina and Chongqing only							**3.8**	**(3.0,**	**4.7)**

dominated by the Quebec cohort for which the cohort-specific R_M value (0.0009, 95% CI: 0.0006–0.0013) was less than one tenth of that for the South Carolina cohort (0.013, 95% CI: 0.0016–0.047, based on 2 deaths). The fact that the R_M values for the six cohorts were statistically homogeneous was largely due to the small numbers of deaths in cohorts other than Quebec. The updated analysis among the 9 chrysotile cohorts now includes up to 55 mesotheliomas (depending on how the results of the North Carolina textiles cohort are treated). The results for the Quebec miners are unchanged, but additional deaths – particularly those among the Balangero miners (7 deaths) and the inclusion of the North Carolina textiles cohort (8 deaths) – means that the Quebec results are now somewhat less dominant statistically, and the homogeneity across the group as a whole is lost. The overall average R_M value of 0.0014 is therefore not an adequate summary measure for chrysotile (P-value for homogeneity <0.001). Whether the North Carolina should be treated as entailing exposure only to pure chrysotile can reasonably be questioned due to some evidence of amphibole exposure at two of the four plants that contributed most of the person-years of observation (Garabrant, 2020). If this cohort is completely excluded, the remaining 7 cohorts still show very substantial heterogeneity (R_M = 0.0012, P-value for homogeneity <0.001).

With the exception of the South Carolina women, all four of the textile cohorts taken at face value have considerably higher R_M values, and are statistically homogeneous with a summary R_M value of 0.01 (95% CI: 0.0056–0.017). The remaining five cohorts are also statistically homogeneous with a summary R_M value around an order of magnitude lower than this of 0.0011 (95% CI: 0.00079–0.0014). Notably, no mesotheliomas were observed in the Qinghai miners' cohort which entailed substantial cumulative exposures to chrysotile with apparently low tremolite content and a very pronounced lung cancer excess.

3.2. Lung cancer

The updated results for lung cancer again show a similar picture to the original analysis. Overall, more variability is evident in the study specific R_L values than for R_M, and whereas there is reasonable consistency in the results for the amphibole cohorts, the chrysotile cohorts are much more variable with the study specific estimates spanning two orders of magnitude: the highest values being from the South Carolina cohorts and the lowest from the Quebec miners.

Additional follow-up on the Wittenoom miners led to an increased value for R_L and this increases the summary R_L estimate for crocidolite from 4.2 to 4.8 (95% CI: 3.8–5.9). In contrast, the inclusion of the Tyler cohort – which has a relatively low value of R_L – reduces the summary estimate for the three amosite cohorts to from 5.2 to 4.0 (95% CI: 3.3–4.9). The value for the Libby cohort is much lower at 0.82 (95% CI: 0.43–1.3). A combined summary estimate for crocidolite and amosite of 4.3 (95% CI: 3.7–5.0) is statistically homogeneous – and slightly lower than the equivalent estimate of 4.8 (95% CI: 3.9–5.8) from the original analysis – but the Libby value is not consistent with this.

Increased follow-up on the Balangero miners results in a higher R_L estimate (0.053, 95% CI: 0.044–0.17) than originally (0.030, 95% CI: −0.11–0.24) and this is now more in line with the value for the Quebec miners (0.06, 95% CI: 0.042–0.079). However, the R_L value for the Qinghai mines of 3.3 (95% CI: 2.3–4.5) is over 50 times the Quebec value and is more consistent with the R_L values from the South Carolina textiles cohorts (e.g. 4.8, 95% CI: 3.3–6.4, for S Carolina men) than the other two mines. The four textile cohorts now have R_L values of a similar order, though the somewhat lower value of 1.7 (95% CI: 1.3–2.1) means that they are not formally homogeneous as a group. In addition, the North Carolina cohort is marked by very substantial differences between the R_L value and a very much lower slope from internal exposure-

response regressions carried out for this cohort (Loomis et al., 2009). In summary, the average R_L values for the industrial groupings of mining cohorts (0.064) and textile manufacturing cohorts (2.2) are not supported by the updated data. Statistically consistent combined estimates for mines excluding Qinghai and for textiles excluding North Carolina of 0.060 (95% CI: 0.043–0.078) and 3.8 (95% CI: 3.0–4.7) respectively are shown in Table 2, though these rely on data-driven exclusions of each cohort from the industrial groupings.

3.3. Non-linear exposure-response analysis – mesothelioma

In the original meta-analysis, a model for pleural mesothelioma (expressed as a percentage of all-cause mortality) in terms of cumulative exposure and fibre type in which the exponent of cumulative exposure (i.e. the 'slope' parameter, r) was common for all three fibre types, but with separate coefficients for each, provided an adequate fit to the data for the pure fibre cohorts. The parameter estimates for this model were r = 0.75 for the slope, and 0.93, 0.13 and 0.0047 for the coefficients for crocidolite, amosite and chrysotile respectively (Fig. 1A, solid lines). These parameter values no longer provide an adequate fit to the updated dataset, excluding the Libby data point (Residual deviance, D = 35.8, degrees of freedom, df = 11, P < 0.001). Re-optimising this model – and including a separate coefficient for the Libby data point – gives a much lower value of r = 0.48 for the slope parameter, and 2.0, 0.45, 0.26, and 0.0028 for the coefficients for crocidolite, amosite, Libby and chrysotile respectively, but this model is still not an adequate fit (D = 29.2, df = 11, P = 0.002) (Fig. 1A, dashed lines). The more extreme non-linearity here is driven by the presence of the North Carolina cohort in the updated dataset which, with 8 observed mesotheliomas, carries enough weight statistically to conflict with the Quebec data point and push the value of the slope parameter substantially downwards.

If the model is fitted only to the amphibole cohorts, the best fitting model has a slope close to the original value (r = 0.77, D = 2.14, df = 3, P = 0.54) (Fig. 1B, solid lines), but constraining the model to a linear slope (r = 1) does not substantially degrade the fit (D = 2.66, df = 3, P = 0.45).

Excluding the Quebec data point from the full dataset improves the fit with a slope parameter close to linear (r = 0.94, D = 17.8, df = 10, P = 0.058) (Fig. 1B, hashed line); excluding the North Carolina data point instead of Quebec gives a similarly fitting model with parameters closer to their original values so that substantial non-linearity remains (r = 0.78, D = 18.1, df = 10, P = 0.053) (Fig. 1B, dashed/dotted line).

Another approach is to fit separate coefficients for two groupings of the cohorts categorised as chrysotile only: a 'high chrysotile' group (including the textiles cohorts) and a 'low chrysotile' group (including the mining and other factory cohorts). This again gives an adequate fit with a slope parameter close to the original value and with a coefficient for the high chrysotile group about 5 times that of the low chrysotile group (r = 0.82, D = 15.8, df = 10, P = 0.11) (Fig. 1C).

Overall, this analysis suggests that while the best fitting model for the amphibole data is non-linear, it is the data for cohorts categorised as chrysotile only – and in particular the inclusion of the Quebec data point that has a particularly strong influence on pushing the value of the slope parameter downwards, and this effect is strongest when both the North

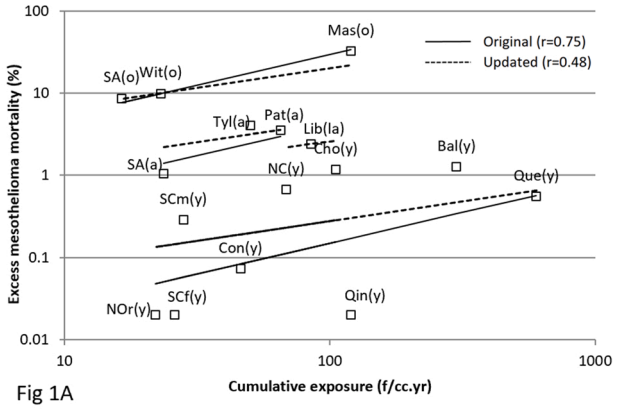

Fig 1A

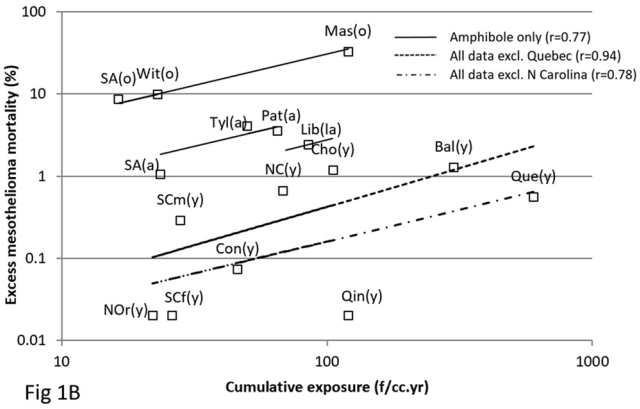

Fig 1B

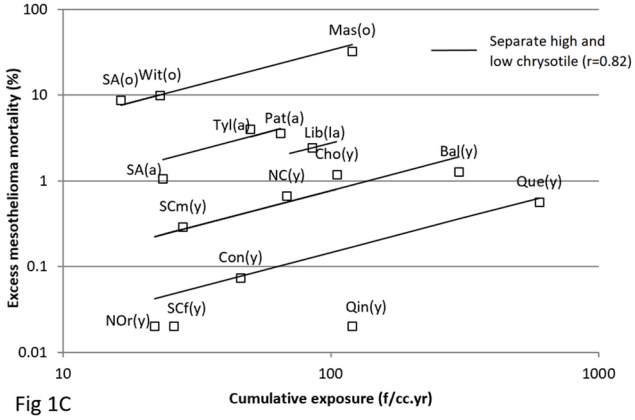

Fig 1C

Key to data points

Wit(o)	Wittenoom
SA(o)	SA crocidolite mines
Mas(o)	Massachusetts
SA(a)	SA amosite mines
Pat(a)	Paterson
Tyl(a)	Tyler
Lib(la)	Libby
Que(y)	Quebec
Bal(y)	Balangero
Qin(y)	Qinghai mine
Nor(y)	New Orleans (plant 2, y)
SCf(y)	S Carolina (women)
SCm(y)	S Carolina (men)
NC(y)	North Carolina
Cho(y)	Chongqing factory
Con(y)	Connecticut

Zero values are plotted at 0.02 on the vertical axis

Fig. 1. Excess pleural mesothelioma mortality. Note. Fibre-type coefficients for models are as follows: Fig. 1A: r = 0.75, 0.94 (o), 0.13 (a), 0.0047 (y); r = 0.48, 2.03 (o), 0.45 (a), 0.26 (la), 0.028 (y). Fig. 1B: r = 0.77, 0.89 (o), 0.16 (a), 0.079 (la); r = 0.94, 0.0056 (y, excl. Quebec); r = 0.78, 0.0044 (y, excl. N Carolina). Fig. 1C: r = 0.82, 0.77 (o), 0.13 (a), 0.064 (la), 0.018 (y, high), 0.0034 (y, low).

Carolina and Quebec cohorts are included in the dataset.

Only two peritoneal mesotheliomas were observed among cohorts categorised as chrysotile only in the updated dataset: one of the three mesotheliomas in men working in the South Carolina textiles factory and one of the two mesotheliomas among the Chongqing factory workers were reported as peritoneal cases. Modelling is therefore restricted to the amphibole cohorts (as in the original analysis). There were 71 deaths in total among the seven amphibole cohorts, 48 of which occurred among the Wittenoom crocidolite miners, with a further seven among the Tyler factor workers and one from the Libby cohort.

A model for peritoneal mesothelioma in terms of cumulative exposure with the same mathematical form as for pleural mesothelioma (a common slope parameter (t) for the different amphibole fibre types but separate coefficients for each) provided an adequate fit to the five amphibole cohorts in the original dataset, with parameter estimates of t = 2.1 for the slope, and 0.0022 and 0.0006 for the crocidolite and amosite coefficients respectively (D = 0.19, df = 2, P = 0.91). The model with these parameter values still provides a good fit to the updated data excluding the Libby cohort (D = 1.16, df = 3, P = 0.76). Re-optimising this model, and including a separate coefficient for the Libby data point, provides a good fit with the same value for the slope parameter of t = 2.1 and coefficients for crocidolite and amosite close to their original values (0.0026 and 0.00059 respectively) but a much lower coefficient for Libby (0.000017, i.e. a factor of 34 lower than amosite) (D = 0.41, df = 3, P = 0.94).

3.4. Non-linear exposure-response analysis – lung cancer

The observation of a non-linear exposure response relationships for pleural and peritoneal mesothelioma motivated the exploration of non-linear models for lung cancer in which the excess lung cancer was allowed to vary according to a power of cumulative exposure. Based on data for the five amphibole cohorts available at the time, a wide range of powers (from 1.1 to 2.1) were found to be statistically consistent with the data. For lung cancer, the amphibole data were considered together as a single group rather than fitting separate relationships for crocidolite and amosite (as for mesothelioma). Two of the five cohorts (Massachusetts and SA amosite) were particularly influential in pushing the slope parameter upwards to a more extreme value and, excluding these, the best fitting model had a slope of 1.4, with a value of 1 (i.e. linear) now within the range of possibilities. Given the uncertainties in the dataset, it was argued that the best choice of the value of the slope parameter could not solely be based on statistical grounds. At that time few studies had fitted relationships other than a linear exposure-response and concern about the plausibility of higher slope values led to the choice of a value of 1.3 as a best estimate, with a plausible range of from 1 to 1.6.

The updated amphibole data are shown in Fig. 2 and informally these suggest a model with common coefficient for crocidolite and amosite (as in the original analysis), but a separate coefficient for Libby, may be appropriate. Restricting the range of possible slopes to be between 1 and 1.6, as before, the best fitting model is for a slope of r = 1.2 with a coefficient of 2.1 for crocidolite/amosite and 0.34 for Libby, however, this is still not a particularly good fitting model (D = 16.9, df = 4, P = 0.002). Similarly fitting models can be found for slope parameter values between 1 and 1.5 by varying the other coefficients, but for slopes higher than 1.5 or less than 1, the fit is substantially worse. The fit is substantially improved if separate coefficients for crocidolite and amosite are allowed, but the best fitting version of this model has a much higher slope parameter value of r = 1.54 with coefficients for crocidolite, amosite and Libby of 0.84, 0.47, and 0.07 respectively (D = 7.75, df = 3, P = 0.051). Constraining the slope to lower values brings the coefficients for crocidolite and amosite closer together but degrades the fit: for example, with r = 1.3 (the preferred model in the original analysis) these have values of 1.8 and 1.2 respectively (D = 10.4, df = 3, P = 0.016) and with r = 1 (a linear model) the values are 4.8 and 4.0 respectively (D = 16.5, df = 4, P < 0.001).

In the original analysis, inconsistencies in the lung cancer data for cohorts categorised as chrysotile ruled out direct estimation of the exposure-response relationship. In fact, the much higher excess lung cancer risk per unit exposure for the South Carolina cohort than the Quebec cohort means that the best fitting model fitted to the data for the six pure-chrysotile cohorts in the original analysis had a negative slope value. In this context, the range of slopes from the amphibole data was assumed (r = 1 to r = 1.6). For the best fitting model in the original analysis (r = 1.3), the scaling coefficient was determined by setting the predicted risk at the median exposure (70 f/cc. yrs for chrysotile cohorts) to 0.1%, i.e. the best estimate of percentage excess risk per unit exposure from the original chrysotile cohorts considered. For the other models (r = 1 and r = 1.6), the corresponding scaling coefficients were set to give a predicted risk of 0.5% at 70 f/cc. yrs, i.e. the value regarded as the upper limit for the chrysotile risk in the original analysis.

Refitting the lung cancer model to the updated dataset again results in a negative slope if a single line is assumed, due to the influence of the Quebec cohort and the South and North Carolina cohorts. A model with two separate lines – a high coefficient for the group of textiles cohorts, but also including the Chongqing factor and the Qinghai mine, and a low coefficient for the remainder (including the Quebec and Balangero mine cohorts) – looks more plausible. Constraining the slope to r = 1 in such a model yields values of 2.4 and 0.06 for the high and low chrysotile

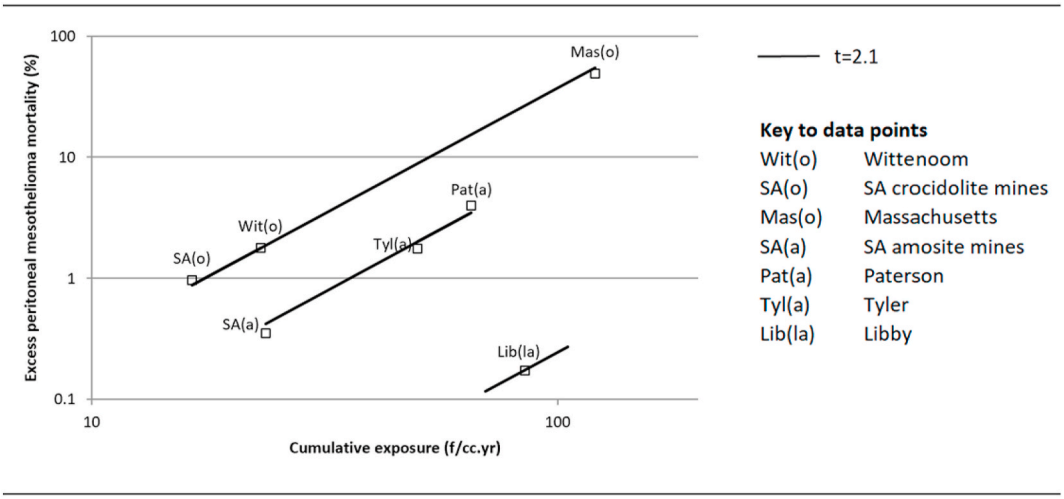

Fig. 2. Excess peritoneal mesothelioma mortality. Note. Fibre-type coefficients for model are as follows: r = 2.1, 0.0026 (o), 0.00059 (a), 0.000017 (la).

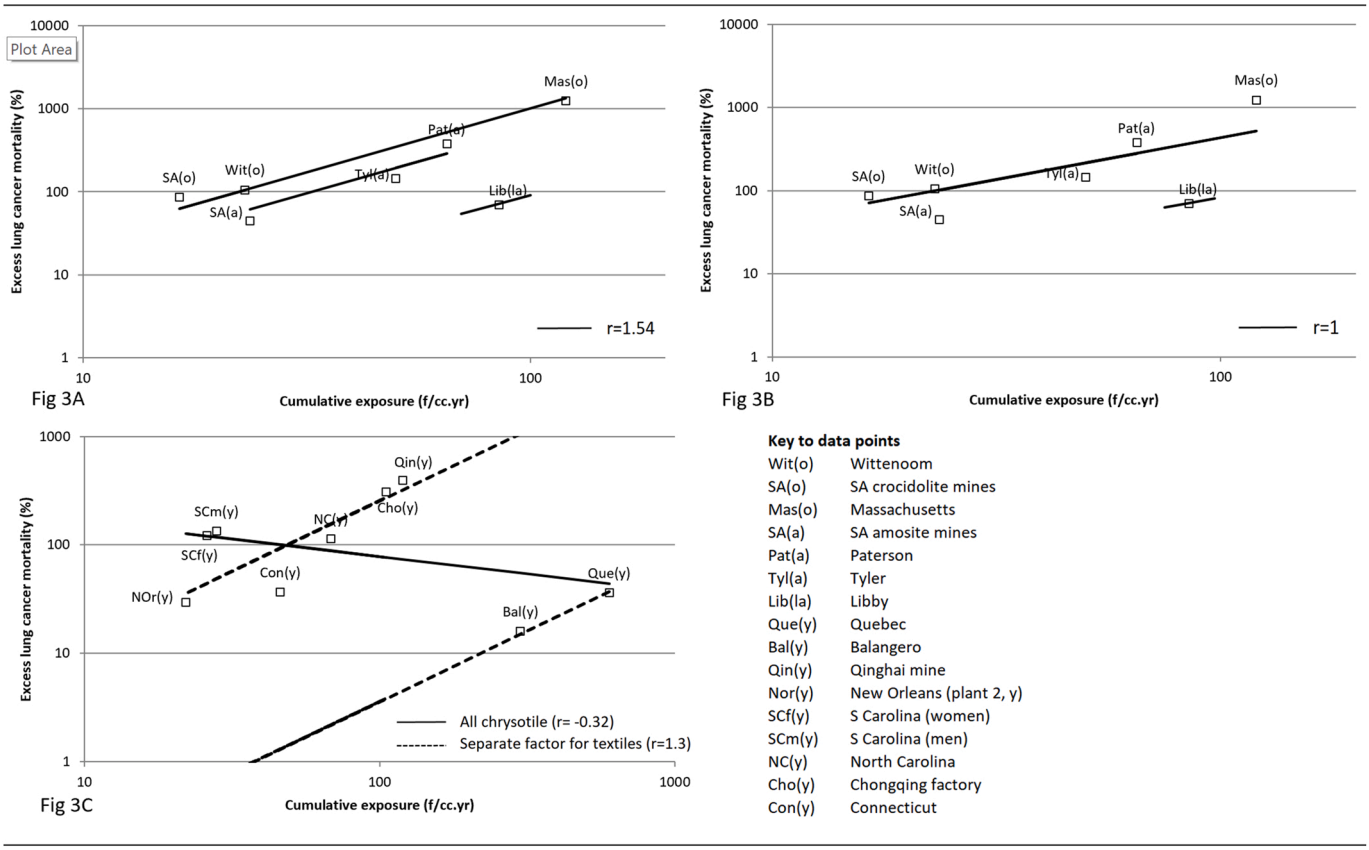

Fig. 3. Excess lung cancer mortality. Note. Fibre-type coefficients for models are as follows: Fig. 3A: r = 1.54, 0.84 (o), 0.47 (a), 0.07 (la). Fig. 3B: r = 1, 4.3 (o/a combined), 0.82 (la). Fig. 3C: r = −0.32, 341 (y, all); r = 1.3, 0.64 (y, textile), 0.009 (y, non-textile).

coefficients, and with r = 1.3 the values are 0.64 and 0.009 respectively, but the fit of both of these models is poor.

4. Discussion

The results of this analysis are broadly consistent with those of the original meta-analysis of Hodgson and Darnton published in 2000: fibre type largely explains differences in total (pleural plus peritoneal) mesothelioma risk per unit of cumulative exposure observed across cohort studies, whereas differences in excess lung cancer risk are less readily quantifiable appealing to fibre type alone – though fibre type nevertheless plays an important role. Non-linearity is still suggested in the exposure-response, particularly in the case of peritoneal mesothelioma.

Summary values for total excess mesothelioma (pleural and peritoneal combined) risk per unit cumulative exposure (R_M) for crocidolite and amosite asbestos of 0.52% and 0.11% were close to the values estimated in the original analysis. The value of R_M for the Libby cohort was around a quarter of the amosite value at 0.03%, and for cohorts categorised as chrysotile, estimates of 0.01% for textile manufacturing and 0.001% for other chrysotile exposure contexts were found. In broad terms, these results imply that a cumulative exposure of about 2 f/cc. yrs for crocidolite confers a lifetime mesothelioma risk of 1%, whereas exposures of 10 f/cc. yrs for amosite, 30 f/cc. yrs for the amphibole mixture at Libby, and between 100 and 1000 f/cc. yrs for chrysotile would be required to confer such a risk.

These very large differences in the mesothelioma risk per unit of exposure for amphibole – particularly crocidolite and amosite – compared with chrysotile imply there is potential for R_M estimates for cohorts classified as entailing exposure only to pure chrysotile to be biased upwards, perhaps considerably so, if small amounts of amphibole were also present. This could be due to the natural occurrence of amphiboles alongside chrysotile when mined, or the occasional specific use of amphiboles in processing and manufacturing contexts which may not be well known or documented. The inclusion of the North Carolina and the Chongqing cohorts increases the number of mesotheliomas arising from chrysotile textile contexts to 13 compared with two in the original analysis (both of which were from the South Carolina cohort). The increased weight from these additional cases means a single estimate of R_M combining all chrysotile cohorts is not now statistically homogeneous, and separate 'high' and 'low' chrysotile estimates of 0.01% (based on the textile cohorts) and 0.001% (based on other chrysotile cohorts) respectively are more appropriate simply taking the data at face value. Results are similar if the North Carolina cohort is treated as a mixed fibre type cohort and excluded from these analyses. Whether it is factors associated with the industrial context, or the potential for concomitant amphibole exposure in some or all of these cohorts, that produces an approximately 10-fold higher mesothelioma risk than the average seen in other contexts can reasonably be debated. Amphibole exposure may also have played a role in observed differences in risk between cohorts within the non-textile chrysotile group - for example, the presence of 'balangeroite' or commerical amphiboles could have influenced the mesothelioma risk among the Balangero miners though the extent of this in not clear.

The separate non-linear models for pleural and peritoneal mesothelioma imply that pleural cases form the majority of total cases except at very high cumulative amphibole exposures (and that no peritoneal cases are due to chrysotile). For example, at 100 f/cc. yrs exposure to amosite, using the models shown in Figs. 1C and 2, the pleural and peritoneal risk estimates are 5.7% and 8.5% of total mortality respectively, whereas at 10 f/cc. yrs the risk estimates are 0.9% and 0.07% respectively (i.e. 92%

of the total mesothelioma risk is due to pleural cases at 1 f/cc. yrs whereas only 40% is due to pleural cases at 100 f/cc. yrs). The total mesothelioma risk is effectively entirely determined by the pleural mesothelioma model at lower exposures (e.g. below about 1 f/cc. yrs) and this sub-linear model gives a progressively higher predicted risk than extrapolating the linear summary R_M values when progressing down the exposure scale. For example, for 1 f/cc. yrs exposure to amosite, the model shown in Fig. 1C gives a risk estimate of 0.13% which compares with an R_M value for amosite of 0.11%; at an order of magnitude below this, 0.1 f/cc. yrs, the equivalent estimates are 0.02% and 0.011% respectively – i.e. the non-linear model predicts a risk about twice that of the extrapolated R_M value.

The summary estimate of excess lung cancer risk per unit cumulative exposure (R_L) for crocidolite and amosite combined of 4.3% – slightly lower than the value of 4.8% estimated in the original analysis – implies that an exposure of around 25 f/cc. yrs of either crocidolite or amosite would double the baseline risk of lung cancer, whereas for the mixed amphiboles at Libby a much higher exposure of around 120 f/cc. yr would be required.

The very wide variation in the R_L values for cohorts categorised as chrysotile makes it more difficult to decide on an appropriate summary value for use in general risk estimation. In the original analysis we appealed to the evidence from mixed fibre cohorts (not presented here) to inform this choice. R_L values were generally substantially lower from mixed fibre settings – including textile production – than those for South Carolina, with a mean R_L of 0.32% and a median of 0.5% (excluding three cohorts thought likely to be atypical), about an order of magnitude lower than the amphibole cohorts. Taken together, these observations suggested that amphibole is making a much higher contribution than chrysotile to the observed lung cancer risk in the mixed fibre exposure settings, and so the average risk arising from chrysotile only exposure should be substantially lower than 0.32%. In this context we suggested that a value of 0.1% was a reasonable best estimate of R_L since this was consistent with the value from the Quebec mines of 0.06% (which has considerable weight statistically), though set slightly higher to allow for some additional risk arising from processed chrysotile fibre. A summary R_L five times higher than this of 0.5% was judged to be the highest arguable value.

This argument largely set aside the observations from South Carolina as being atypical and not generally appropriate for general risk estimation in settings involving pure chrysotile exposure, whilst also appealing to fibre type as the main explanation of the variability between other cohorts: if the South Carolina observations were discounted, there was a clearer association between excess lung cancer and fibre type. The inclusion in this analysis of three additional chrysotile exposed cohorts all with R_L values considerably higher than 0.5% means this is now less so, and the additional data may undermine the case for adopting a single chrysotile value much closer to that of the Quebec cohort for general risk estimation, unless the question of what factors other than fibre type play a significant role in lung cancer risk – and how commonly these need to be taken into account – is resolved. The role of fibre dimension has, in particular, been a major focus of inquiry by others (Loomis et al., 2010), and variation in typical fibre dimension characteristics of exposures within fibre type groupings potentially explains some of the remaining variability across cohorts after taking fibre type into account (Berman and Crump, 2008b). Whilst crudely grouping cohorts according to their industrial context might also potentially provide insight into the effect of other factors, in this analysis, such groupings still do not provide a consistent picture: for example, the Qinghai mining cohort has an R_L value much more similar to those of the South Carolina textiles cohorts than to the other mining cohorts, and the R_L value for the North Carolina textiles cohort is substantially lower than those for South Carolina (and the equivalent estimate from internal regression analyses of the North Carolina cohort lower still and in fact close to the value for the Quebec mines cohort).

While the original Hodgson and Darnton meta-analysis down-weighted the South Carolina observations, at least for the purposes of use in general risk estimation, others have adopted a meta-analytic approach which, through the application of study quality criteria, led to them playing a central role in summary risk estimates (Lenters et al., 2012). The study by Lenters et al. found that the average lung cancer risk was higher across studies satisfying each of five predefined study quality criteria than those that did not. Furthermore, this effect was found to be independent of fibre type, and when applying the quality criteria successively, a near monotonic relationship between the summary lung cancer risk values was found as studies were removed, and this was the case regardless of the order in which the criteria were applied. Their conclusion was that study quality is itself an important determinant of the observed lung cancer risk, that this should be taken into account by calculating the summary risk value over a much restricted pool of studies (only two studies, Libby and South Carolina meet all five criteria), and that their analyses cast doubt on the conclusion that the epidemiological evidence for lung cancer supports a difference in potency for the different fibre types.

This approach has appeal in apparently applying objective criteria which were determined in advance; however, it is not without important limitations as described, for example, by Hodgson in a detailed examination of the analysis (Hodgson et al., 2013). As well as highlighting some features of the analysis that tend to somewhat weaken the apparent effect between study quality and the size of the resulting summary risk value, he noted that while the association with study quality is not in doubt – and indeed would be expected on statistical grounds – at issue is how to take this effect into account appropriately. Subjective judgement again comes into this process, the approach of excluding a large proportion of the available studies being one option. This assumes that the true signal between exposure and effect is only quantifiable by restricting attention to the highest quality studies. In reality, however, all studies have limitations – some greater than others – and so the approach does not address the extent to which the restricted view of the effect based on higher quality studies could be uncertain or biased by not incorporating any signal – potentially still strong – from studies determined to be of lower quality. Hodgson also noted that when considered alongside the variables used to defined study quality, fibre type was still the one most strongly associated with the summary risk value; in simply excluding a large number of studies from its calculation, somewhat weaker associations for the quality-related variables are therefore effectively being prioritised over this important observation. These points also draw attention back to the large differences in the risk between the Quebec and South Carolina studies, where in fact the fibre type was the same but the processing was very different; while there is no doubt that the former study had more deficiencies that the latter, the question remains as to whether the South Carolina study produced a much higher risk estimate because of quality, or whether as a high quality study it simply provides a good picture of the effect in this very particular context.

The non-linear models for mesothelioma fitted here were motivated by the separate observations for pleural and peritoneal cases within studies, the latter only accounting for a small proportion of cases unless exposures were particularly high. This would be consistent with national-level data on mesothelioma incidence which show that most cases are pleural, reflecting the effect of a lower exposure averaged over the population as a whole. Given the uncertainties in the dataset used here, the non-linearity seen in the exposure-response relationship for pleural mesothelioma could reasonably be doubted, particularly in the light of other evidence for a linear relationship. For example, a linear relationship between amphibole lung burden and mesothelioma risk was found in a recent study in Britain (Gilham et al., 2016), and Korchevskiy et al. recently described how Peto's widely used model for mesothelioma incidence in terms of exposure intensity and a power of time (years) since the start of exposure (Peto et al., 1982) can be consistent with an apparently non-linear model for lifetime risk in terms of cumulative exposures if the variation in duration of exposures across

studies is not taken into account, as was the case in this meta-analysis (Korchevskiy and Korchevskiy, 2022). However, the models for peritoneal mesothelioma fitted here strongly suggest a non-linear relationship which is harder to dismiss, even in the context of the considerable uncertainties in the exposure and outcome data.

Other recent analyses have also suggested a non-linear exposure-response relationship between cumulative exposure and the relative risk of lung cancer. For example, a recent scientific report on asbestos by the European Chemicals Agency (ECHA) carried out meta-regression analyses using categorical data on cumulative exposure and lung cancer risk from pure-fibre and mixed-fibre asbestos exposed cohorts (European Chemicals Agency, 2021). The best fitting model was non-linear (sub--linear at lower exposures) but also included an intercept which could indicate the presence of confounding due to inappropriate reference rates in some studies or exposure misclassification leading to a flattening of the exposure-response slope. These models describe the average lung cancer risk across all fibre types and take into account variability across studies in a random effects framework that down-weights outlying observations. The evidence for a non-linear exposure-response relationship for lung cancer we report here is from the amphibole-exposed cohorts only, and while this also has a sub-linear shape, the risk increases more steeply with cumulative exposure than the models in the ECHA report.

5. Conclusions

These updated analyses continue to show substantial difference in the potency of crocidolite and amosite asbestos types (and the amphibole mixture at Libby) in relation to mesothelioma induction. Best fitting models suggest a non-linear exposure-response in which the long-term excess of mesothelioma depends on cumulative exposure to a power less than 1 for pleural mesothelioma and a power of around 2 for peritoneal mesothelioma. While other evidence may suggest that relationship for pleural mesothelioma may nevertheless be linear, the argument for a non-linear relationship for peritoneal mesothelioma is harder to dismiss. It was not possible to derive a single coefficient to describe the potency of chrysotile asbestos based on the available data for cohorts categorised as chrysotile only, but the range of potency values was substantially lower than for amphibole asbestos. For lung cancer, the very substantial variation in data for cohorts categorised as chrysotile did not provide a basis for deriving a single model for lung cancer in relation to chrysotile; updated data from crocidolite and amosite exposed cohorts continue to suggest a non-linear exposure-response and these models were consistent with there being a slight difference in potency between these fibre types.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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8. EA, 2021. Waste Classification. Guidance on the classification and assessment of waste (1st Edition v1.2GB)

Full Reference: EA, 2021. Waste Classification. Guidance on the classification and assessment of waste (1st Edition v1.2GB). Technical Guidance WM3, Environment Agency, Bristol, October 2021

[Extract: page 19]

Waste Classification

Guidance on the classification and assessment of waste (1st Edition v1.2.GB)

Technical Guidance WM3

SEPA
Scottish Environment Protection Agency

Environment Agency

Cyfoeth Naturiol Cymru
Natural

Further guidance on assessment

This chapter provides further guidance and examples to show how waste classification and assessment is applied to

1. Construction and demolition wastes containing asbestos
2. Waste containing coal tar
3. Waste soils
4. Waste oils and other wastes containing or contaminated with oil
   (a) Waste oils
   (b) and other wastes containing or contaminated with oil

1. Construction and demolition wastes containing asbestos

Asbestos is a naturally occurring silicate mineral and exists in a number of chemical types – for example **chrysotile** ('white'), **amosite** ('brown') and **crocidolite** ('blue') – either in a bonded or fibrous form. The fibres are very fine, less than 3 microns in diameter and respirable into the lung passageways where they can lodge indefinitely and penetrate tissue.

All forms of asbestos are classified the same way in the Mandatory Classification List (MCL) under the GB CLP Regulation:

- Carc. Cat 1A; H350, and
- STOT RE1; H372**

The assessment of asbestos containing waste considers both the presence of asbestos as

- fibres that are free and dispersed, and
- identifiable pieces of asbestos containing material

If the waste contains fibres that are free and dispersed then the waste will be hazardous if the waste as a whole contains 0.1% or more asbestos.

If the waste contains any identifiable pieces of suspected asbestos containing material they must be assessed as set out below. This would also apply to any dispersed fibres produced by deliberately breaking up such identifiable pieces.

Where the waste contains identifiable pieces of asbestos containing material (i.e. any particle of a size that can be identified as potentially being asbestos by a competent person if examined by the naked eye), then these pieces must be assessed separately. The waste is hazardous if the concentration of asbestos in the piece of asbestos containing material is 0.1% or more. The waste is regarded as a mixed waste and classified accordingly (see example 1 for advice on how to apply list of waste codes to mixed waste). The following codes should be assigned to the asbestos waste as appropriate:

- 17 06 05* Construction material containing asbestos MH
- 17 06 01* Insulation material containing asbestos MH

17 06 05* would normally be used in preference to 17 06 01* for the asbestos in asbestos contaminated soil and stones.

2. Waste containing coal tar

This example provides guidance on the classification of road asphalt waste containing coal tar (AWCCT) and other construction and demolition wastes containing coal tar and related materials.

This does not apply to wastes where coal tar is known not to be present.

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9. EA, 2013. Monitoring of particulate matter in ambient air around waste facilities. Technical Guidance Document (Monitoring) M17

Full Reference: EA, 2013. Monitoring of particulate matter in ambient air around waste facilities. Technical Guidance Document (Monitoring) M17. Environment Agency, Bristol, 2013.

[Extract: pages 35-38]

Technical Guidance Note (Monitoring) M17

Monitoring Particulate Matter in Ambient Air around Waste Facilities

Environment Agency

Version 2

July 2013

The first step to interpreting the results of monitoring is to compare upwind (background concentrations specific to that site) with downwind concentrations to determine the level of emission attributable to the composting facility (process contribution). The process contribution must not exceed the acceptable levels quoted above at the sensitive receptor. The operator would be expected to carry out up to four sampling campaigns a year to demonstrate bioaerosol levels are being maintained.

7.4 Fibres: Asbestos and Man-Made Mineral Fibres

7.4.1 The issue of fibres at waste facilities

At some waste facilities, e.g. landfill sites, particulate matter in the form of fibres may be encountered. This includes materials such as asbestos and man-made mineral fibres (MMMFs). Asbestos waste must be deposited in a landfill for hazardous waste, a site designed to accept asbestos only or in a separate cell in a landfill for non-hazardous waste, but only if the cell is sufficiently self-contained and the design provides a physical separation and isolates the asbestos so that it remains undisturbed[49]. To prevent the uncontrolled release of asbestos fibres there must be no drilling through asbestos cells.

There is also the legacy of asbestos/MMMF being released to air from contaminated land. We have conducted trials with the HSE to support development of guidance for assessing the risks from asbestos in contaminated soils but no published guidance is yet available; however, professional and industry bodies (CL:AIRE, the Environmental Industries Commission and the British Occupational Hygiene Society) are working together towards the development of practical and robust non-statutory industry guidance[50].

The epidemiological risk implications of fibres are due, in part, to their long, thin structure (aspect ratio) and, especially for asbestos fibres, their propensity to break down into ever finer, sharp fibres. The main health impacts from asbestos are from exposure that has occurred at work, rather than from non-occupational exposure. Workplace exposure to asbestos kills more people than any other single work-related illness. The diseases can take from 15-60 years to develop – so the person who has breathed in the fibres will not immediately aware of any change in their health. Asbestos can cause two main types of disease in humans: asbestosis (scarring of lung tissue) and cancer (particularly lung cancer and mesothelioma), as detailed in Box 7.1.

MMMFs can in some circumstances cause irritation of the skin and eyes and upper respiratory tract and such effects are discussed in further detail in HSE Guidance Note EH 46[51].

7.4.2 Summary of the measurement technique

There are no standard methods for monitoring fibres in ambient air around waste management facilities; therefore, procedures have been adopted based on modifications of published methods for occupational monitoring.

Manual sampling of fibres is undertaken in much the same way as for many other particulates, using air-sampling pumps and filters. A number of analytical end methods can then be used to identify and quantify the fibres that have been collected, as outlined in MDHS 87[52].

Box 7.1 Diseases from Asbestos Exposure

Asbestosis: A chronic lung ailment where the inhalation of fibres causes scarring and hardening of the lung tissue. Clinically similar to silicosis, the disease is progressive and rate of progression is related to exposure. There is a clear dose-response relationship and although incurable and irreversible, early diagnosis may halt the disease.

Lung Cancer: A malignant tumour of the lungs' air passages, and may spread to other parts of the body. It should be noted that there is a synergistic effect between smoking and asbestos – exposure of the two carcinogens together significantly increases the risk of developing lunch cancer. Similar to asbestosis, there appears to be a reasonable dose-response relationship.

Mesothelioma: This disease is still the dominant occupational cancer affecting cells that make up the lining around the outside of the lungs and inside the ribs (pleura) or around the abdominal organs (peritoneum). Although the risk appears to be increased with high and persistent exposure, there has been evidence that mesothelioma may be the result of relatively short exposures. The dose-response relationship is not clear and may possibly result from non-occupational exposure.

Asbestos

MDHS 87 outlines the two main methods of quantifying the asbestos that has been collected in air samples, optical microscopy and electron microscopy.

Optical microscopy is used as the routine approach for monitoring and the method given in HSE guidance HSG 248[#,53] is used basis for monitoring ambient air, although it should be noted that this method is designed for controlled conditions in premises and workplaces and dusty outdoor conditions cause problems. The procedure uses the membrane filter method, with low-flow sampling pumps and membrane filters (mixed esters of cellulose or cellulose nitrate with 0.8–1.2 μm pore size) held in electrically-conducting cylindrical cowled filter holders. Fibres collected on the cleared filter are then counted using phase contrast microscopy (PCM) to obtain the countable fibre number concentration in air. This method gives a lower detection limit of 0.01 fibres per millilitre of air (10,000 fibres per cubic metre) for a 25 mm diameter filter and a sampled air volume of 480 litres. However, this can be improved to some degree by increasing the sampled air volume, making it more suitable for measuring ambient environmental levels of asbestos.

PCM continues to be the analytical method of choice for occupational monitoring of asbestos, because of the following advantages over other methods:

- the technique is specific for countable fibres: non-fibrous particles are excluded from the count;
- the technique is relatively inexpensive;
- the analysis is quick and can be performed on-site for rapid determination of air concentrations of asbestos fibres; and
- the technique has continuity with historical epidemiological studies so that estimates of expected disease can be inferred from long-term determinations of asbestos exposures.

The main disadvantage of PCM is that it does not positively identify asbestos fibres. Other fibres that are not asbestos may be included in the count if deemed a countable fibre by HSG 248. A further disadvantage of PCM is that the smallest visible fibres are about 0.2 μm in diameter while the finest asbestos fibres may be as small as 0.02 μm in diameter. For

[#] HSG 248 consolidates and updates HSE technical guidance previously published as EH10, MDHS 39 and MDHS 77.

some exposures, substantially more fibres may be present than are actually counted. Other fibres can also interfere with counting, including fibreglass, anhydrite, plant fibres, perlite veins, gypsum, some synthetic fibres, membrane structures, sponge spicules, diatoms, micro-organisms and wollastonite. Positive identification of asbestos must be performed by dispersion staining or electron microscopy techniques. Fibre counting is not suited to very dusty atmospheres, and high levels of general environmental dust can render samples unreadable by PCM.

Electron microscopy is able to detect much smaller fibres than optical microscopy. Levels of electron microscope-visible fibres per cubic metre are reported to be in the range 40-100 fibres per m^3 ($0.04x10^{-3}$ to $0.1x10^{-3}$ fibres ml^{-1}) for remote areas and up to 2400 fibres per m^3 ($2.4x10^{-3}$ fibres ml^{-1}) in urban air. At these low levels, the scanning electron microscope (SEM) / transmission electron microscope (TEM) provides the best means of analysis. Quantification is by counting of fibres, but positive confirmation of fibres as asbestos on selected areas of the filter may be made by Selected Area Electron Diffraction (SAED) or Energy Dispersive X-ray Analysis (EDAX), which are facilities available on a TEM. This makes the electron microscope method preferable when there are significant levels of non-asbestos fibres in the air. British Standard BS ISO 10312 describes the standard method[54] for measuring asbestos fibres in ambient air using TEM. The SEM method[55] of measuring inorganic fibre particles is given by BS ISO 14966.

In summary, for monitoring around waste facilities the preferred method will usually be sampling onto membrane filters at about eight litres per minute for one hour, or two litres per minute over a four hour period to achieve a 480 litre sample volume, followed by fibre-counting by PCM in accordance with HSG 248. If difficulties with interferences are experienced with PCM, then TEM and/or EDAX should be used as the end method. One practical approach that can be taken is to divide the exposed filter paper into two halves and immediately analyse the first half by PCM; then, if necessary, the other half of the filter paper can later be analysed by scanning/transmission electron microscopy (SEM/TEM) to establish the PCM-equivalent asbestos fibres concentrations.

Several direct-reading instruments operating on the light scattering principle are used as portable fibre counters in occupational hygiene work, but their suitability for ambient applications is unproven. The instruments rely on being able to first align fibres before they pass into the optical sensor. However, they cannot match the performance of manual methods and are best used only for an indication of whether levels are increasing or decreasing[56].

Man-made mineral fibres

The UK occupational method MDHS 59[57] offers two approaches for monitoring man-made mineral fibre concentrations: sampling by cellulose ester filter followed by gravimetric determination; or sampling onto a filter followed by plasma ashing and fibre counting by polarised light microscopy. The gravimetric approach is not well suited to the ambient atmosphere because the method is non-specific and other atmospheric dusts would interfere significantly.

The fibre counting method is preferred for monitoring around waste facilities; it is similar in principle to that for asbestos and can be modified for ambient monitoring by increasing the sampled volume to provide an improved lower detection limit. As for asbestos, fibre counting is not suited to very dusty atmospheres and if difficulties with interferences are experienced with PCM, then TEM and/or EDAX should be used as the end method.

7.4.3 Guideline limits for fibres

Asbestos is a proven human carcinogen (IARC Group 1). No safe level can be proposed for asbestos because a threshold is not known to exist. Exposure should therefore be kept as low as possible[58] and asbestos should not be found above background levels at site boundaries. Further guidance will be available in the Technical Guidance Note for landfill sites, which should be available early in 2014.

Occupational exposure limits exist for MMMF (refer to the latest issue of Guidance Note EH 40[59] and to Operational Circular HSE OC 267/2[60]); but for ambient air, no EAL is currently listed in H1 - *Environmental Risk Assessment for Permits*. H1 recommends that in such cases, operators should discuss the requirement with the site inspector who, if necessary, can obtain appropriate advice.

8 Reporting Requirements

In order to allow proper checking and facilitate meaningful intercomparisons, monitoring reports providing data for site investigations should include the following information:

Front end/cover information

- The site address and name of the operator
- The type of development/process
- The planning consent reference
- Date of issue of the report
- Period covered
- Authors of the report
- Organisation submitting the report
- Evidence of quality check/authorised sign-off of the report

Introduction

- Scope and terms of reference of the monitoring and the report

Methodology

How was the measurement carried out?

- Statement on the standard published method, or in-house documented technical procedure, and the technique/principle used
- Summary of the technique and methodology used for both sampling and analysis
- Equipment type/make/models used
- Details of the monitoring locations on map that also shows the process/development

By whom?

For both the sampling and the analysis stages, needs to show:

- Who carried it out
- Belonging to which organisation (i.e. in-house or subcontracted outside)
- If the organisation carrying out the sampling and/or the analysis has UKAS or MCERTS accreditation, then this should be stated and the accreditation numbers given.

What quality accreditation is in place?

- The general QA system, if any, under which the organisation operates, e.g. ISO9001 quality management system

10. EA, 2009. Human health toxicological assessment of contaminants in soil

Full Reference: EA, 2009. Human health toxicological assessment of contaminants in soil, Science Report SC050021/SR2, Environment Agency, Bristol, January 2009

[Extract: pages 18, 35-36, 39, 58-59]

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Environment Agency

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Human health toxicological assessment of contaminants in soil

Science Report – Final SC050021/SR2

ea/br/e/sci/v1

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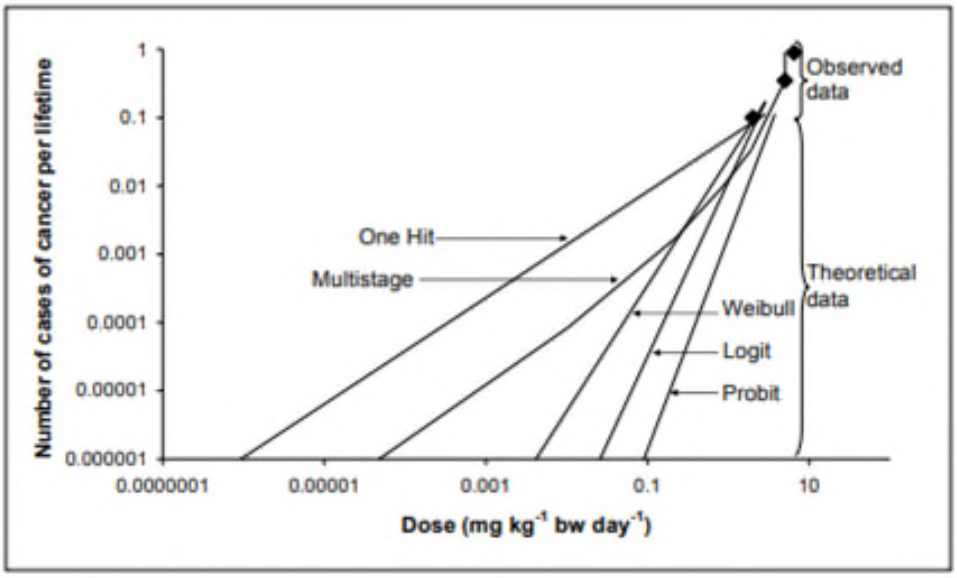

Figure 2.6 Example of variance of quantitative cancer risk models when modelling the same data set (modified from COC, 2004)

Non-quantitative extrapolation

The predominant alternative (non-quantitative) approach to setting HCVs for non-threshold carcinogens 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, and the use of expert judgement to derive a suitable margin (COC, 2004).

HCVs derived using this approach have previously been called **minimal risk levels**[15] by COC. COC (2004) defined 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 point of departure identified from the dose-response data. Where the assessment is based on animal data, it is usually not possible to identify a dose without discernible carcinogenic effect; effect level data are therefore used. Several indices of tumour production that may be used as the point of departure are commonly reported in the experimental carcinogenicity literature. The most common are the BMDL (as for threshold toxicity), the **TD_{50}**, and the **T25**. The TD_{50} is defined as the chronic dose rate that would induce tumours in a given target site(s) in 50% of the test animals at the end of a standard lifespan for the species, provided there were no tumours in control animals. However, since tumours unrelated to the test chemical often occur in control animals, the TD_{50} is better defined as the daily dose

[15] Note these are not the same as the minimal risk levels derived by the US ATSDR.

Table 3.4 Correction factors used to adjust adult MDI to younger age groups[25]

Age (years)	Typical bodyweight (kg) [a]	Correction factor for oral MDI	Typical inhalation rate (m^3 day^{-1}) [a]	Correction factor for inhalation MDI
0–1	6.3	0.53	8.7	0.51
1–2	10.2	0.66	13.4	0.80
2–3	13.0	0.65	13.0	0.77
3–4	15.5	0.65	12.5	0.74
4–5	17.3	0.74	12.5	0.74
5–6	19.7	0.74	12.5	0.74
6–7	22.5	0.74	12.9	0.76
7–8	25.4	0.80	12.9	0.76
8–9	27.8	0.80	12.9	0.76
9–10	32.3	0.80	12.9	0.76
10–11	35.7	0.80	12.9	0.76
11–12	40.8	0.81	14.4	0.85
12–13	45.5	0.81	14.4	0.85
13–14	50.5	0.81	14.4	0.85
14–15	57.8	0.81	14.4	0.85
15–16	60.1	0.88	14.4	0.85
16–59	76.6	1.00	17.1	1.00
60–70	76.8	1.00	14.2	1.00

[a] Default bodyweight and inhalation rate values (e.g. 70 kg and 20 m^3 day^{-1} for an adult) are usually used in converting toxicity data – which may come from another country or apply internationally – rather than using more specific values as presented here.

3.4.2 Non-threshold carcinogenicity

This section describes the methods used to derive an Index Dose (ID) – the term used herein to describe 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. The section also explains how IDs are used in the setting of SGVs.

Deriving an Index Dose

The extent and quality of data available determines which method should be followed in deriving an ID. Data from animal carcinogenicity bioassays may be used to derive an ID by BMD modelling (see Sections 2.2.1 and 2.2.5) of the tumour data and application

[25] Oral correction factors based on dietary surveys. Food consumption rates set by Byrom *et al.* (1995) have been widely used in regulatory risk assessments. However, these data apply only to a limited number of age classes. To correct for all age classes, the approach was applied to a wider analysis of the National Diet and Nutrition Survey (NDNS) data from 1995-2002 (Gregory *et al.*, 1995, 2000; Henderson *et al.*, 2002). NDNS data were corrected from consumer data to population data by multiplying by the fraction of consumers in each survey category. The Byrom *et al.* (1995) data for children less than one year old were used for age class one.

Inhalation correction factors are the ratio of the average male and female inhalation rates for each age class to the adult rate at age class 17 (age 16–59 years) and are based on the rates used by the CLEA model for residential land use to derive SGVs (Environment Agency, 2009; Lordo *et al.*, 2006).

of a large default uncertainty factor of 10,000 to the critical $BMDL_{10}$ (Defra, 2008b). Often more than one type of treatment-related tumour will have been produced in a carcinogenicity study, and sometimes more than one study will be available. Furthermore, BMD software packages contain several models that may each derive a statistically "acceptable" BMD_{10} and $BMDL_{10}$. Consequently, a number of $BMDL_{10}$ values may be produced for a contaminant, and expert judgement must be employed in selecting which should form the basis of the ID derivation. If it is not possible to derive a $BMDL_{10}$, the T25 (see Section 2.2.5) may be used instead, but a much larger uncertainty factor would be required.

This approach is less well developed for use with human data; therefore when sufficient human data are available, alternative approaches may be used, including quantitative dose-response modelling of suitable human cancer data (while acknowledging the imprecision of quantitative estimates of cancer risk; see Section 2.2.5). In such case, the ID should be based on estimates of the dose corresponding to an excess lifetime cancer risk of 1 in 100,000 (10^{-5}) (Defra, 2008b). Where human data are available, but have not undergone or 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 (see Section 2.2.5).

As for threshold contaminants, existing evaluations by authoritative groups will often provide a good foundation for a risk assessment of a non-threshold carcinogenic contaminant, and sometimes it may be appropriate to adopt HCVs already proposed by these bodies. Again, HCVs should not be adopted naively, and due consideration should be given to the suitability of the values (for example, use of an HCV based on quantitative cancer risk modelling of animal data would not normally be supported).[26]

Consideration of threshold effects

Whilst a serious adverse heath effect, cancer may not necessarily be the critical toxic effect of a non-threshold genotoxic carcinogen on which the HCV should be based. Such chemicals may also cause other, threshold, adverse effects; hence, for low potency carcinogens the threshold effects may drive the risk assessment.

It is essential, therefore, that risk assessments of genotoxic carcinogens, as for any other contaminant, methodically investigate all toxic endpoints before selecting the critical effect for deriving the HCV.

If a TDI, based on threshold critical toxicity, is proposed for a contaminant that also produces non-threshold effects (either for the same or for a different route of exposure), the ALARP principle (see Section 2.5) will nonetheless apply.

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[26] If a guideline for a non-threshold carcinogen has been produced under a different regulatory regime with UK jurisdiction that is less stringent than the derived ID, it may be considered disproportionate to enforce a stricter limit for contaminated land, and therefore inappropriate to set the SGV on the derived ID. In such instances, the ID and SGV may be set based on equivalence to the existing guideline. The guideline should be applicable to the UK population as a whole, and should relate to lifetime exposure. The UK Water Supply (Water Quality) Regulations 2000, for example, specify a limit of 10 $\mu g\ L^{-1}$ for arsenic in drinking-water (HMSO, 2000). In setting this standard, which is equivalent to an estimated excess lifetime cancer risk of about one in 1,000 (10^{-3}) derived from good epidemiology data, technical achievability and economic considerations (the ALARP principle) were taken into account in addition to health protection.

It would not, by contrast, be appropriate to set HCVs and SGVs based on equivalence to UK occupational standards for chemicals (Workplace Exposure Limits), since these relate only to persons of working age, and are based on working hours not continuous lifetime exposure.

This principle applies to all types of contaminants, but is expected to only have notable consequence for non-threshold carcinogens.

3.5.1 Risk characterisation of contaminants with a TDI

As discussed in Section 2.4.1, exposures equal to or less than the TDI are considered to be without appreciable health risk. Where an SGV is available for a threshold contaminant, soil contaminant concentrations equal to or less than the SGV would normally be considered similarly tolerable.[28]

Where a TDI is expected to be exceeded, this is undesirable but does not necessarily mean that adverse health effects will result. The likelihood and severity of health impacts from TDI exceedances need to be considered on a case-by-case basis and require expert judgement. Section 2.4.1 provides examples of some of the considerations that should form part of such an evaluation.

3.5.2 Risk characterisation of contaminants with an ID

IDs are derived for contaminants for which a threshold for adverse effects cannot be presumed. Exposure at the ID is therefore considered to carry some, albeit minimal and often unquantifiable, level of risk. Where exposures are predicted to be below the ID, the consequential risks are expected to be minimal, but the overriding risk management (see Section 2.5) requirement for exposures to be kept as low as reasonably practicable (ALARP) still applies.

Where an ID is exceeded, there will be an increased risk to health. The significance of this increased health risk requires expert judgement, but often will not be quantifiable.

3.5.3 Hazard and risk characterisation of mixtures of chemicals

Knowledge about the toxicology of a chemical comes mainly from studies in which relatively large doses of the substance are administered to experimental animals. In contrast, the human population is exposed to vast numbers of chemicals every day, including many priority soil contaminants. The possibility exists, therefore, that the cocktail of chemicals to which humans are exposed will have a greater cumulative effect on health than that predicted by risk assessments of individual chemicals.

The possible effects on toxicity resulting from the presence of other toxicants will not only depend on the number and identity of the chemicals, but also on their absolute concentrations and relative proportions. In addition, the toxicokinetics of different chemicals will determine whether non-concurrent external exposure will result in combined systemic exposure. Lasting effects of a chemical may also affect those of another even when the exposures do not overlap. With almost infinite permutations that could be envisaged, routine toxicity testing of chemical mixtures is clearly impossible. The pressures on the use of experimental animals – primarily ethical and financial – means that even rudimentary testing of a limited number of the chemicals in existence, to the extent that the findings would be sufficiently informative, is not viable.

Even epidemiology studies, while based on human subjects exposed to multiple chemicals, are of limited value in informing about chemical mixture toxicity, because the chemical exposures involved are usually ill-defined and there is often only sufficient

[28] It is possible for a high MDI to result in total exposure exceeding the TDI even when the SGV is not exceeded. However, in such an instance the exposure from the land would alone be considered tolerable (it would, in fact, be at most half the TDI, in theory) and the soil contamination would be contributing less than 50% to the total exposure (and therefore risk).

Health Criteria Value	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).
Hepatotoxicant	A substance capable of causing damage to the liver.
Independent action	See **simple dissimilar action**.
Index Dose	The term used in this report to refer to an estimate of the amount of a chemical soil contaminant (expressed as a daily intake **dose**) that can be experienced over a lifetime with minimal cancer risk.
Inhalable particle	A particle the size of which dictates that when inhaled it only reaches the upper respiratory tract. These particles generally have an **aerodynamic diameter** of 10–100 μm. Following deposition in the lung, they may be subject to **mucociliary clearance**, swallowing and oral absorption (cf. **respirable particle**).
Inhibition	An interaction in which one chemical acts to reduce the toxicity of another but is itself unaffected. The overall effect is therefore weaker than would be predicted from additivity.
Intake	The amount of a chemical entering the human body at the point of entry (that is, mouth, nose or skin) by ingestion, inhalation, or skin contact.
Interaction	The affecting of a chemical's behaviour by another chemical. Any effect of exposure to multiple chemicals that is not **simple similar action** (**dose addition**) or **simple dissimilar action** (**response addition**). The overall effect of an interaction may be one that is either stronger or weaker than would be predicted based on additivity, and the mechanism underlying the interaction may be at the chemical or biological level.
Interspecies variability	The variability (in chemical/toxic sensitivity) between members of different species, for example humans and other animals.
Intraspecies variability	The variability (in chemical/toxic sensitivity) among members of the same species. For example, variability within the human population (that results from factors such as genetic diversity, age, health status, personal habits, diet and smoking habits).
In vitro	Within an artificial environment such as a test tube (literally, "in glass").
In vivo	Within a living organism (literally, "in life").
Kinetics	See **toxicokinetics**.
Lowest-observed adverse effect level	The lowest concentration or amount of a substance, found by experiment or observation, which causes an **adverse effect** in the target organism distinguishable from normal (control) organisms of the same species and strain under the same defined conditions of exposure.
Local toxicity	Adverse effects of a chemical that are confined to the tissue(s) at the site of contact with the chemical. For example, lung cancer caused by inhaling asbestos fibres, or contact dermatitis caused by nickel.
Margin of Exposure	Ratio of the experimental point of departure (e.g. the critical NOAEL) to the theoretical, predicted, or estimated human exposure.

Masking	An **interaction** in which two or more chemicals produce functionally competing effects on the same organ system or the effects of one override the effects of another.
Mean daily intake	The average **intake** of a soil contaminant from other, non-soil, sources, expressed as an amount per day (e.g. µg day^{-1}). The mean daily intake is estimated for each **route of exposure** (oral and inhalation) and arises principally from exposure to the contaminant in food, water and air.
Mechanism of action	The detailed molecular and biochemical pathways and events initiated or altered by a chemical that give rise to its observed adverse effect(s) (cf. **mode of action**).
Mesothelioma	A malignant tumour (cancer) of the tissue membrane lining the chest cavity or abdominal cavity.
Minimal risk level	1. A level of exposure to a non-threshold carcinogen (expressed as a daily intake **dose**) that is considered to be associated with a negligible risk of cancer. Minimal risk levels are derived using non-quantitative risk assessment methods employing expert judgement. 2. An **ATSDR** estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), non-cancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic).
Mode of action	The collective key biochemical events initiated or altered by a chemical that result in the observed adverse effect (cf. **mechanism of action**).
Monotonic	Designating a sequence in which successive values either consistently increase or consistently decrease; they do not oscillate in relative value. Each member of a monotone increasing sequence is therefore equal to or greater than the preceding member; each member of a monotone decreasing sequence is equal to or less than the preceding member.
Mucociliary clearance	A process by which inhaled particles that have deposited on the mucous surface of the airways are removed with the mucous by the action of cilia (tiny hairs on the surface of the airways), often then becoming swallowed.
Mutagen	A chemical that can produce permanent heritable change in the amount or structure of the genetic material of cells or organisms (see **mutation**).
Mutation	A permanent change in the amount or structure of the genetic material of an organism, which may result in a heritable change in the characteristics of the organism. These alterations may involve individual genes, blocks of genes, or whole chromosomes. Mutations involving single genes may be a consequence of effects on single DNA bases (point mutations) or of larger changes, including deletions and rearrangements of DNA. Changes involving chromosomes as entities may be numerical or structural. A mutation in the **germ cells** of sexually reproducing organisms may be transmitted to the offspring, whereas a mutation that occurs in **somatic** cells may be transferred only to descendent daughter cells. Mutagenic chemicals may present a hazard to health since exposure to a mutagen carries the risk of inducing germ-line mutations, with the possibility of inherited disorders, and the risk of somatic mutations including those leading to cancer.
Negative synergy	See **antagonism**.

11. EU, 2023. Protection of workers from asbestos at work, EU Legislation in Progress Briefing, European Parliament

Full Reference: EU, 2023. Protection of workers from asbestos at work, EU Legislation in Progress Briefing, European Parliament. Access at https://www.europarl.europa.eu/RegData/etudes/BRIE/2023/749813/EPRS_BRI(2023)749813_EN.pdf

[Reference provided in full]

BRIEFING
EU Legislation in Progress

European Parliament

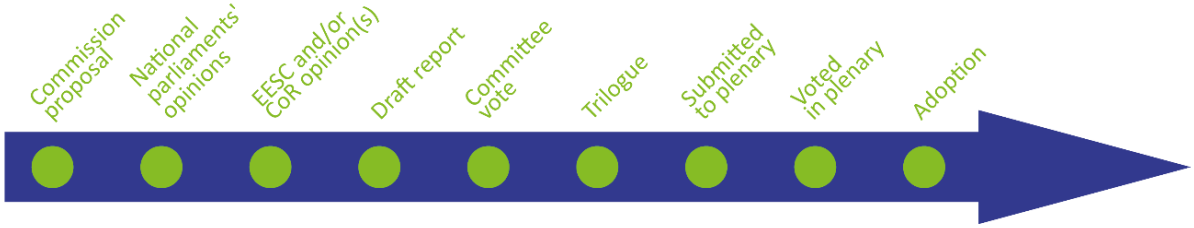
Protection of workers from asbestos at work

OVERVIEW

On 28 September 2022, the European Commission proposed a revision of the 2009 Directive on the protection of workers from the risk of exposure to asbestos. On 27 June 2023, after interinstitutional negotiations, the Council and the European Parliament reached a provisional agreement on the proposal. Parliament approved the agreed text on 3 October 2023; the Council followed on 23 October. The final act was signed on 22 November 2023. Member States have until 21 December 2025 to transpose the bulk of the provisions, with the remainder to be in national law by December 2029.

Despite the total ban on the use of asbestos in the EU, exposure to this carcinogenic fibre, which is still present in millions of buildings, kills more than 70 000 people a year in Europe. The new rules significantly lower the current asbestos limits and provide for more modern and accurate technologies to measure exposure levels to asbestos. They also provide for strengthened preventive and protective measures to improve worker protection, such as the adequate use of individual protective and respiratory equipment, the safe cleaning of clothing, a decontamination procedure, and high quality training requirements for workers.

Proposal for a directive of the European Parliament and of the Council amending Directive 2009/148/EC on the protection of workers from the risks related to exposure to asbestos at work		
Committee responsible:	Employment and Social Affairs (EMPL)	COM(2022) 489 28.9.2022
Rapporteur:	Véronique Trillet-Lenoir † (Renew, France)	2022/0298(COD)
Shadow rapporteurs:	Cindy Franssen (EPP, Belgium) Marianne Vind (S&D, Denmark) Sara Matthieu (Greens/EFA, Belgium) Joanna Kopcińska (ECR, Poland) Elena Lizzi (ID, Italy) Nikolaj Villumsen (The Left, Denmark)	Ordinary legislative procedure (COD) (Parliament and Council on equal footing – formerly 'co-decision')
Procedure completed.	Directive (EU) 2023/2668 OJ L, 30.11.2023.	



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PE 749.813 – December 2023

EN

Introduction

Asbestos is a highly dangerous, cancer-causing substance that is still present in many buildings in the European Union and is responsible for many avoidable deaths. When asbestos fibres are released and inhaled, for instance during renovations, it poses a threat for workers' health.

On 28 September 2022, the Commission presented a comprehensive approach to better protect people and the environment from asbestos and ensure an asbestos-free future. The package includes a communication on working towards an asbestos-free future, tackling asbestos in a comprehensive way, from improved diagnoses and treatment of diseases caused by asbestos, to identification and safe removal and waste treatment of asbestos; and a proposal to amend the Asbestos at Work Directive (2009/148/EC) to improve workers' protection by significantly lowering the occupational exposure limit (OEL) value for asbestos, given the latest scientific knowledge and technological developments.

Existing situation

The first EU action aimed at protecting workers from the specific risks of workplace exposure to asbestos dates back to 1983 and the adoption of Council Directive 83/477/EEC. The progressive ban on the use of asbestos in the EU began in 1988 with the prohibition of crocidolite (also called blue asbestos) This directive has been substantially amended several times to cover other asbestos-containing materials until its most recent codified version, Directive 2009/148/EC (the Asbestos at Work Directive). Since 2005, all forms of asbestos have been banned in the EU, for goods both produced in and imported into the EU.

The current OEL of 0.1 fibres/cm^3 in the 2009 directive was set in 2003, based on the scientific and technological knowledge available at that time. Some Member States have introduced stricter OELs in their national legislation (Germany, Denmark, France and the Netherlands, according to the Commission's proposal).

Parliament's starting position

On 20 October 2021, the European Parliament adopted a legislative-initiative resolution (INL) with recommendations to the Commission on protecting workers from asbestos. Stressing that the safe removal of asbestos is an urgent and difficult task, Parliament reiterated its call for a comprehensive European strategy for the removal of all asbestos in the EU, working across several policy areas and giving top priority to safe working conditions. The resolution was accompanied by recommendations for key elements that should be included in the strategy. On the update of Directive 2009/148/EC on the protection of workers from the risks related to exposure to asbestos at work, Parliament called on the Commission to update the exposure limits, which should be lowered to 0.001 fibres/cm^3 instead of the existing OEL for asbestos of 0.1 fibres/cm^3.

Preparation of the proposal

This initiative was included in the Commission's 2022 work programme (and 2023 as a priority pending proposal, and in the Joint Declaration on EU Legislative Priorities for 2023 and 2024). EU citizens also highlighted the importance of revising the Asbestos at Work Directive in the framework of the Conference on the Future of Europe.

The proposed directive is one of the flagship initiatives of the Europe's Beating Cancer plan. It is in line with the European Pillar of Social Rights (in particular its principle 10 on the right to a healthy, safe and well-adapted work environment) and also builds on the commitment the Commission made in the EU strategic framework on health and safety at work for 2021-2027 to further lower the OEL for asbestos in the revised Asbestos at Work Directive. It also takes into account the European Green Deal and the renovation wave strategy.

The proposal and the communication also address concerns expressed by the European Parliament in its legislative-initiative resolution of October 2021 (see above).

The proposal was accompanied by an impact assessment. EPRS published an initial appraisal of the impact assessment on the proposal in January 2023.

The changes the proposal would bring

The proposal includes a reduction in the exposure limit of asbestos at work to 10 times lower than the current value, based on the latest scientific and technological developments. The proposal aims to amend the Directive on Asbestos at Work (Directive 2009/148/EC) in particular as regards updating the limit value for asbestos, in order to protect workers against risks to their health and safety that can result from exposure to asbestos at work.

Under Directive 2009/148/EC, for all activities in which workers are or may be exposed to dust from asbestos or materials containing asbestos, exposure must be reduced to a minimum and in any case below the fixed binding OEL of 0.1 fibres/cm^3 as an 8-hour time-weighted average (TWA). Under the proposed directive, employers should ensure that no worker would be exposed to a higher airborne concentration of asbestos than 0.01 fibres/cm^3 as an 8-hour TWA. As it is possible to measure an OEL equal to 0.01 fibres/cm^3 with phase-contrast microscope (PCM), no transition period would be needed for the implementation of the revised OEL. Fibre counting would be carried out by PCM in accordance with the method recommended in 1997 by the World Health Organization (WHO)[1] or, wherever possible, any other method giving equivalent or better results, such as a method based on electron microscopy. The proposal also clarifies the obligation on employers to reduce the exposure of workers to dust from asbestos or materials containing asbestos at the place of work to a minimum, with the precision that in any case it must be as low a level as is technically possible, below the limit set by the proposal. Lastly, the obligation on employers to take all necessary steps to identify presumed asbestos containing materials before beginning demolition or maintenance work by obtaining information from the owners of the premises, would be extended to cover other relevant sources of information, such as relevant registers.

Advisory committees

In its opinion adopted on 15 December 2022 (rapporteur: Ellen Nygren, Workers – Group II, Sweden), the European Economic and Social Committee welcomes the Commission's ambition to reduce the incidence of cancer, and its specific efforts to minimise exposure to asbestos at work. It recommends that the technical limit value for asbestos exposure should ultimately be set at a lower level than the Commission is currently proposing. It proposes a broad public information campaign on asbestos and its risks. There is also a need for training for all workers at risk of exposure to asbestos, provided in the worker's native language or another language in which they are proficient. It calls on the EU and its Member States to actively promote an international ban on the use of asbestos and protection for workers dealing with existing asbestos during activities such as renovation, demolition and waste management.

The European Committee of the Regions (CoR) adopted its opinion on 16 March 2023 (rapporteur: Hanna Zdanowska, EPP, Poland). It welcomes the Commission's work on the revision of the Directive on the protection of workers from the risks related to exposure to asbestos at work. It joins the call for a European strategy for asbestos removal, which would ensure an integrated approach of different policies and implement systemic and effective solutions in all Member States on inventory, monitoring, safe disposal, storage and education (training) methods.

National parliaments

The deadline for the submission of reasoned opinions on the grounds of subsidiarity was 29 November 2022. No reasoned opinion on the grounds of subsidiarity was submitted within the given deadline.

Stakeholder views[2]

The deadline for feedback on the proposal following its adoption was 1 December 2022. In total, 10 contributions were received.

The European Federation of Building and Woodworkers (EFBWW) underlined that there is no safe exposure limit to protect workers completely from asbestos and that the Commission also needs to present a full approach to tackling the asbestos disaster in other policy areas (e.g. in the energy renovation of buildings). The European Trade Union Confederation (ETUC) regrets that the Commission has proposed a limit of only 0.01 fibres/cm^3, as called for by business associations. The European Federation of Public Service Unions (EPSU) said that EPSU would actively engage in this debate, together with the EFBWW, to lower the limit value alongside the effort of the ETUC. In a joint press release published on 26 April 2023, the EPSU, the EFBWW and the ETUC urged the Member States and the Commission to adapt the Commission's proposal in line with Parliament's more ambitious and holistic approach. On 27 June 2023, the EFBWW and ETUC welcomed the agreement reached in trilogue but expressed some disappointment at the length of the transition period chosen to achieve a more ambitious exposure value, the risks being implemented too late to protect those involved in building renovations part of the EU Green Deal. In a statement issued in April 2023, the European Construction Industry Federation (FIEC) expressed its concerns about the very significant financial impact of the revision on construction companies and their clients. On 5 June 2023, in a joint press release, FIEC and the European Builders Confederation recall that optimal protection conditions for the workforce will only be possible if the limit allows for realistic implementation, monitoring and evaluation by construction companies. The European Society for Medical Oncology welcomed the inclusion of the 0.001 fibres/cm^3 norm in the EMPL report as this legal limit is of crucial importance for preventing new cancer cases.

Legislative process

The European Commission adopted its proposal on 28 September 2022. On 8 December 2022, the Council adopted its position for negotiations with the European Parliament.

In Parliament, the Committee on Employment and Social Affairs (EMPL), responsible for the file, appointed Véronique Trillet-Lenoir (Renew, France) as rapporteur on 10 November 2022. EMPL adopted its report on 26 April 2023 with 40 votes in favour, none against and 7 abstentions. It calls on employers to reduce exposure to asbestos fibres to the lowest possible level and below an OEL of 0.001 fibres/cm^3 on an 8-hour average, while aiming to introduce the use of electron microscopy to efficiently detect thinner fibres, often the most carcinogenic. That revised OEL should apply after a transitional period. Until 4 years after the date of entry into force of the proposed directive, a transitional OEL, equal to 0.01 fibres/cm^3 as an 8-hour TWA, would apply, while EU countries would still be able to use the current PCM method. The report targets a harmonised EU approach in detection and prevention of asbestos, bringing the obligation for medical surveillance in line with scientific knowledge. It also sets out a list of means to avoid passive and secondary exposures to asbestos, such as the use of individual protective and respiratory equipment, the safe cleaning of clothing, and a mandatory decontamination procedure. Finally, the report includes a new annex on the minimum requirements for training for workers in specialised asbestos removal undertakings.

The mandate for negotiations was endorsed in plenary on 10 May. Parliament and Council representatives met in May to start trilogue discussions on the proposal. A second trilogue meeting took place on 15 June. On 27 June, the Council and Parliament reached a provisional agreement on the proposal. According to the agreed text, the OEL will go from 0.1 to 0.01 fibres of asbestos per cm^3 without a transition period. Following a maximum transition period of 6 years, Member States will have to use electron microscopy to detect fibres. They will have to decrease the level to 0.002 fibres of asbestos per cm^3 excluding thin fibres or to 0.01 fibres of asbestos per cm^3 including thin fibres. Under the new rules, undertakings that intend to carry out demolition or asbestos

removal work will be required to obtain permits from national authorities. Employers will also need to take steps to identify materials that could potentially contain asbestos before beginning demolition or maintenance work on premises built before the entry into force of the national asbestos ban. The new rules will also set out a list of ways to avoid exposure, such as the adequate use of individual protective and respiratory equipment, the safe cleaning of clothing, a decontamination procedure, and training requirements for workers. Member States will have to keep a register of all cases of medically diagnosed asbestos-related occupational diseases.

The rapporteur's untimely death on 9 August 2023 came before the text could be formally adopted. In the Council, the Permanent Representatives Committee approved the agreement resulting from interinstitutional negotiations on 19 July. In the European Parliament, EMPL approved the provisional agreement on 7 September 2023 (42 votes in favour, none against and 2 abstentions). On 3 October, Members adopted the agreement, with 614 votes in favour, 2 against and 4 abstentions. On 23 October, the Council gave its final green light to the text.

Directive (EU) 2023/2668 of the European Parliament and of the Council was signed on 22 November 2023, and published in the Official Journal of the EU on 30 November 2023. The Directive enters into force on 20 December 2023, and Member States have until 21 December 2025 to transpose it into national law (and until 21 December 2029 for certain provisions).

EUROPEAN PARLIAMENT SUPPORTING ANALYSIS

Amand-Eeckhout L., Protecting workers from asbestos, EPRS, European Parliament, October 2021.

Tuominen M., Protection of workers from the risks related to exposure to asbestos at work, EPRS, European Parliament, January 2023.

OTHER SOURCES

European Parliament, Protection of workers from asbestos, Legislative Observatory (OEIL).

European Parliament resolution of 20 October 2021 with recommendations to the Commission on protecting workers from asbestos (2019/2182(INL)).

ENDNOTES

[1] Determination of airborne fibre concentrations. A recommended method, by phase-contrast optical microscopy (membrane filter method), WHO, Geneva 1997 (ISBN 92 4 154496 1).

[2] This section aims to provide a flavour of the debate and is not intended to be an exhaustive account of all different views on the proposal. Additional information can be found in related publications listed under 'European Parliament supporting analysis'.

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Third edition. The 'EU Legislation in Progress' briefings are updated at key stages throughout the legislative procedure.

12. HMG, 2019. Analysis and Interpretation Methodology for the Soil Investigation at Grenfell Tower

Full Reference: HMG, 2019. Analysis and Interpretation Methodology for the Soil Investigation at Grenfell Tower, HM Government, London, October 2019. Access at https://www.gov.uk/guidance/grenfell-environmental-checks-stage-1-report#report-documents

[Reference provided in full]

HM Government

Analysis and Interpretation Methodology for the Soil Investigation at Grenfell Tower

Background

In October 2018, Government announced that additional environmental checks were to be carried out in and around the Grenfell Tower site. These proposals included a soil testing programme to check for contamination as a result of the fire.

A multiagency partnership (MAP) was formed to oversee an investigation of potential contamination of land and water as a result of the fire and to consider the potential risks to public health from the current use of the land surrounding the site (MHCLG 2018).

The investigation used the risk-based approach to the investigation of potential land contamination under *Part 2A of the Environmental Protection Act 1990* (*Part 2A*) and the *Statutory Guidance* (DEFRA 2012a), which informed the approach to the collection and interpretation of information and the conclusions drawn from the data.

AECOM, an environmental consultancy, was appointed in March 2019 (from a preselected panel of environment consultants on the ESPO Framework)[1] to carry out Stage 1 of the investigation, which consisted of the preliminary risk assessment, exploratory sampling and a Pilot Study at Waynflete Square. The objectives of the preliminary risk assessment were to:

- Summarise the environmental setting of Grenfell Tower and the surrounding area;
- Interpret existing factual information to identify potential *contaminant linkages*;[2]
- Undertake exploratory sampling to further characterise the presence of potential *contaminant linkages* within the geographical area of interest; and
- Prioritise the potential *contaminant linkages* that require further investigation and identify those where there is a reasonable possibility that a *significant contaminant linkage* may exist.

In addition, the aim of the Pilot Study at Waynflete Square was to better understand spatial variability in a small area close to Grenfell Tower and to demonstrate the subsequent process of site investigation and risk assessment as part of a detailed inspection of a particular area of land under *Part 2A*.

About this document

This document outlines the method used for the analysis and interpretation of the findings from Stage 1, in respect of human health, in accordance with *Part 2A*. However, it is important

[1] Eastern Shires Purchasing Organisation (ESPO) Framework 664 – Consultancy Services Lot 8b – Environmental & Sustainability.

[2] Under *Part 2A*, for a relevant risk to exist there needs to be one or more contaminant-pathway-receptor linkage(s) (a *contaminant linkage*) by which a relevant person might be affected. In other words, for a risk to health to exist there must be contaminants present in, on or under the land in a form and quantity that poses a hazard, and one or more pathways by which they might significantly harm people. See Appendix A1 for further information.

to note that the *Statutory Guidance* (DEFRA 2012a) is the definitive guide to investigation methods and the analysis and interpretation of data under *Part 2A*. References to specific paragraphs of the *Statutory Guidance* (DEFRA 2012a) are included in the text to help understanding (for example, SG 4.24, means paragraph 4.24). Although examples of the interpretation of specific *contaminant linkages* are provided throughout the text, the detailed information collected by this investigation is found in the comprehensive Technical Notes produced by AECOM for this project (see References for a list). Where legal and technical terms are not fully explained in the method statement, a definition has been included either in Appendix A or in separate Information Sheets.

Note about Quality Assurance and Quality Control

There are considerable inherent uncertainties in the collection, analysis, and interpretation of environmental data including soil contaminants. Strict quality assurance and quality control is essential to reduce uncertainty during investigation. AECOM carried out Stage 1 in accordance with good practice guidance including the relevant British Standards for site investigation (BS 10175:2011+A2:2017), soil sampling (BS ISO 18400-101:2017), and for the investigation of adverse impacts from fires (BS ISO 26367-1:2017 and BS ISO 26367-2:2017). Soil samples were collected, stored, and transported to the laboratory for chemical analysis in accordance with AECOM's Field Procedures and a project specific sampling protocol. Chemical analysis was conducted by an experienced laboratory using UKAS accredited methods traceable back to national/international standards where available. AECOM's work was overseen and all draft outputs were reviewed by MAP, the Science Advisory Group, and the National Quality Mark Scheme (NQMS) Suitably Qualified Person.

Method Statement for Stage 1

1. Scope set for the assessment
    a. *Part 2A* was the legal context (see Appendix A1)
    b. Investigation was carried out in accordance with good practice (see Information Sheet 1)
    c. Investigation in Stage 1 included a preliminary risk assessment and an illustrative generic quantitative risk assessment (the Pilot Study)[3]
    d. Investigation examined potential impact of fire emissions on land condition and potential risks to health.
2. A preliminary risk assessment was carried out (AECOM 2019k) that
    a. Identified Chemicals of Potential Concern (COPC) from fire emissions and their behaviour in the environment through a review of previously published studies (AECOM 2019b and e)
    b. Identified potential hazards to public health from human exposure to COPC in soil, the possible exposure pathways, and the most sensitive land-uses (AECOM 2019f)
    c. Identified potential routes for fire emissions to soil and likely patterns of deposition (AECOM 2019c, d, and i)

[3] This tiered approach to risk assessment was consistent with good practice (see Information Sheet 1) and formed part of the activities of *detailed inspection* under *Part 2A* (SG 2.9 – 2.15, SG 3.12 – 3.17).

d. Developed initial conceptual site model and identified potential *contaminant linkages* (SG 3.8 – 3.11). An example linkage is shown in Table 1.

e. Designed and executed exploratory soil sampling to refine the conceptual site model based on initial understanding of *contaminant linkages* (AECOM 2019a, 2019j)

f. Updated the initial conceptual site model for each potential *contaminant linkage*, identified major uncertainties, and assigned a qualitative ranking[4] of the likelihood that a reasonable possibility of significance exists for each *contaminant linkage* using the following factors:

    i. Chemical analysis results (percentage of non-detects, measured soil concentrations, and any spatial patterns consistent with fire emissions)

    ii. Generic screening criteria (GSC) including equivalent in-house values (see Information Sheet 2) for comparison with measured concentrations (AECOM 2019f) [5, 6]

    iii. *'Normal' levels* of COPC in soils (see Appendix A2) for comparison with measured concentrations (AECOM 2019g) [4, 5]

    iv. Additional lines of evidence for fire as the source of contamination (for example, the use of ratio plots for profiles of polycyclic aromatic hydrocarbons).

    v. Evidence gaps and other factors (qualitative degree of epistemic uncertainty)

Table 1: An example of a contaminant linkage

Linkage	Contaminant	Potential pathways	Receptor
1	Benzo[a]pyrene in ash and debris, deposited on surface soils by the fire	Ingestion of soil and soil derived indoor dust Inhalation of soil-derived dust (indoor and outdoor) Dermal contact with soil Dermal contact with soil-derived indoor dust Consumption of produce and attached soil Inhalation of vapours (indoor and outdoor)	Young female child (aged from birth – 6 years), potential resident and site user

g. Identified reasonably possible *significant contaminant linkages* for further investigation and identified any necessary work to address evidence gaps to resolve

[4] A qualitative ranking of the strength of evidence for each *contaminant linkage* for the reasonable *possibility of significant harm to human health* (SG 4.10 – 4.15). It was not a quantitative assessment of risk to public health, but a comparative ranking for prioritisation purposes only.

[5] Due to spatial variation and exploratory soil samples being taken over a wide area, it was not appropriate to draw any robust conclusions from a single sample at a single location. However, broader conclusions were drawn from the whole data set on the impact of fire emissions on soil contamination in the vicinity of the Grenfell tower.

[6] See Table 3 for examples for several contaminants.

major uncertainties. An illustrative example of a prioritisation matrix using the above criteria is shown in Table 2.

h. *Part 2A* requires the regulator to consider whether there are reasonable grounds for the existence of a *significant possibility of significant harm* before continuing with further inspection activities. Taking into account the uncertainties and gaps in the evidence, a regulator may rule out further inspection activities at this stage for contaminant linkages that it considers are unlikely to have a reasonable possibility of meeting the definition of a *significant contaminant linkage* (see Appendix A1).

Table 2: An illustrative example of a prioritisation matrix for potentially *significant contaminant linkages* at Grenfell **

Soil Data	Generic Screening Criteria (GSC)	Normal background levels	Linkage Ranking
Most if not all results at or less than suitable method detection limits (MDL) and/or sample depth and location inconsistent with potential exposure pathways	-	-	No further investigation required (evidence suggests that there is no reasonable possibility of a *significant contaminant linkage*)
Most results above MDL and sample depth and location consistent with potential exposure pathways, but no indication of spatial patterns or hot spot consistent with fire emissions	All results at or below a relevant GSC	All results considered to be within typical background levels	Low priority for further investigation (evidence suggests that there is unlikely to be a reasonable possibility of a *significant contaminant linkage*)
Most results above MDL and sample depth and location consistent with potential exposure pathways, but no indication of spatial patterns or hot spot consistent with fire emissions	Some results well-above a relevant GSC *	Some results above typical background levels	Medium priority for targeted further investigation (evidence suggests there could be a reasonable possibility of a *significant contaminant linkage*)
Results above MDL and sample depth and location consistent with potential exposure pathways. Results indicate a strong spatial pattern and/or hot spot(s) that are consistent with fire emissions	Majority of results above and many results well-above a relevant GSC *	Majority of results above typical background levels	High priority for further investigation (evidence suggests there could be a reasonable possibility of a *significant contaminant linkage*)
Results above MDL and sample depth and location consistent with potential exposure pathways. Results indicate a strong spatial pattern and/or hot spot(s) that are consistent with fire emissions	Majority of results well-above relevant GSC *	Majority of results well-above typical background levels	Highest priority for further investigation (evidence suggests there is a reasonable possibility of a *significant contaminant linkage*)

* SG 3.29 and foot notes conclude that the level of risk posed by land contamination depends on more than simply the contaminant concentration in soil. The question of how much above a GSC is no longer *Category 4* (there could still be a reasonable possibility of a *significant contaminant linkage*) depends on the specific GSC and the site circumstances built into it. The range given in the *Statutory Guidance* is from a few times higher to orders of magnitude higher.

** Not shown in the above matrix is the cross-cutting assessment of uncertainty and the identification of critical information gaps for each *contaminant linkage* by the assessor. If confidence in the assignment of a priority to a *contaminant linkage* is low, this may indicate the need for further investigation.

3. A generic quantitative risk assessment for the Pilot Study at Waynflete Square was carried out to illustrate the risk assessment process and investigation at Stage 2 (AECOM 2019l)

    a. Designed and executed soil sampling to inform a generic quantitative risk assessment for each potential *contaminant linkage* at the Pilot Study site (AECOM 2019a, 2019j)

    b. Reviewed laboratory results and divided into additional separate populations / data sets as appropriate (such as through taking into account differences in source zones and sample depths in accordance with good practice guidance)

    c. No clear spatial trends were observed in the data and it was possible to produce conventional statistics for several potential *contaminant linkages* in each data set including the range, the median, the mean, the 95th percentile upper confidence level of the mean (the UCL95). Results were also presented as simple box plots to aid presentation and understanding.

    d. Where conventional statistical methods are valid then the relevant summary statistics for each potential *contaminant linkage* can be compared with relevant information on (see examples in Table 3 and also Appendix A2 and Information Sheet 2):

        i. Generic screening criteria (GSC, AECOM 2019f)

        ii. *'Normal' levels* in soils (AECOM 2019g)

        iii. 'Estimated' levels of exposure from other sources, for example, ambient air and diet (for example, EFSA 2008, 2010 and 2018)

    The initial assessment compared the individual soil measurements for each location with the relevant information on a point-by-point basis. Subsequently, once analysis indicated that conventional statistics were valid, these were used as a secondary step to support interpretation of the findings.

    e. Where concentrations of soil contaminants measured were found at or below a GSC or within *normal levels* or where soil exposure was only a small proportion of total environmental exposure, the *contaminant linkage* was placed in *Category 4* (the *contaminant linkage* was considered not to pose a *significant possibility of significant harm* to health and was not a *significant contaminant linkage*).[7] Risks were also concluded to be absent if there was no longer considered to be a viable *contaminant linkage* (for example, no pathways for exposure to occur).

    f. Where the potential *contaminant linkage* could not immediately be placed in *Category 4*, it was not possible at this stage to assign it with any certainty to *Categories 1 – 4*.[8] If there was still a reasonable possibility that the *contaminant linkage* could be a *significant contaminant linkage* then further site investigation in the form of a detailed quantitative risk assessment will normally be undertaken to

[7] See Appendix A1 for a description of *Categories 1 – 4* in respect of the *significant possibility of significant harm* to human health from the *Statutory Guidance* (SG 4.17 – 4.29)

[8] SG 3.29 and foot notes conclude that the level of risk posed by land contamination will depend on more than simply the amount in soil. The question of how much above a GSC is no longer *Category 4* depends on the specific GSC and the site circumstances built into it. The range given in the *Statutory Guidance* is from a few times higher to orders of magnitude higher.

refine the outcome of the risk assessment (see Information Sheet 1). However, this additional investigation was beyond the scope of the Pilot Study.

g. In addition to any consideration of individual *contaminant linkages*, the possible combined effect of multiple *contaminant linkages* was also evaluated. In particular interactions and additive effects were considered from review of the toxicology (AECOM 2019f).[9] In some cases, the additivity of contaminants was considered within the derivation of the GSC (for example, benzo[a]pyrene and other carcinogenic polycyclic aromatic hydrocarbons or the combined effect of dioxins, furans and dioxin-like polychlorinated biphenyls).

h. Public health professionals were asked to assist with risk communication.

Table 3: Examples of relevant information used in the generic quantitative risk assessment for different contaminants

Contaminant	GSC (see Information Sheet 2)	Normal levels (see Appendix A2)	Estimated exposures from other sources (SG 4.21)
Benzo[a]pyrene	**C4SL** 5 mg/kg (Residential) 5.7 mg/kg (Allotments) 10 mg/kg (Open space) DEFRA (2014)	**NBC** 6.9 mg/kg Greater London soils (Vane et al. 2014)	**Dietary** 0.004 µg/kg bw/day (mean) 0.007 µg/kg bw/day (high) European mean and high level consumers (EFSA 2008)
Dioxins, furans and dioxin-like PCBs	**SGV** 0.008 mg/kg (Residential) 0.008 mg/kg (Allotments) Suitability will depend on congener profile Environment Agency (2009)	**Descriptors** 0.0002 – 0.011 mg/kg (Range) 0.001 mg/kg (Median) 0.0014 mg/kg (Mean) English urban soils (Environment Agency 2007a and b)	**Dietary** 0.6 – 1.5 pg TEQ/ kg bw/day (mean, median LB and UB) 1.9 – 3.5 pg TEQ/ kg bw/day (95^{th} P, median LB and UB) European mean and high level consumers (EFSA 2018)
Lead	**C4SL** 200 mg/kg (Residential) 80 mg/kg (Allotments) 630 mg/kg (Open space) DEFRA (2014)	**NBC** 820 mg/kg Urban domain (DEFRA 2012b)	**Dietary** 0.8 – 3.1 µg/kg bw/day (mean, LB and UB) 1.7 - 5.5 µg/kg bw/day (high) European mean and high level child (aged 1 – 7 years) consumers (EFSA 2010)

[9] The Committee on Toxicity of Chemicals in Food, Consumer Products, and the Environment (COT) has previously advised that interactions in mixtures at soil concentrations leading to exposures representing minimal or negligible risk (see Information Sheet 2) are unlikely (Environment Agency 2009a). However, exposures to mixtures at higher levels of exposure require consideration.

References

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AECOM, 2019b. Fire Chemistry Quick Scoping Evidence Review, Technical Note 4.

AECOM, 2019c. Fate of debris – deposition, spread, clean up, Technical Note 5.

AECOM, 2019d. Review of Met Office Air Dispersion Modelling, Technical Note 6.

AECOM, 2019e. Review of fire effluent environmental fate and transport, Technical Note 7.

AECOM, 2019f. COPC toxicity, Technical Note 8.

AECOM, 2019g. Published data on national and regional urban background concentrations, Technical Note 9.

AECOM, 2019h. Potential sources of urban soil pollution, Technical Note 13.

AECOM, 2019i. Collated community information, Technical Note 14.

AECOM, 2019j. Factual data from exploratory sampling and pilot study, Technical Note 15.

AECOM, 2019k. Preliminary risk assessment, Technical Note 16.

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Appendix A: Definitions

A1: Part 2A of the Environmental Protection Act 1990 and Statutory Guidance (Defra 2012a)

Part 2A of the Environmental Protection Act 1990 defines contaminated land in S. 78A as:

"Contaminated land" is any land which appears to the local authority in whose area it is situated to be in such a condition, by reason of substances in, on, or under the land, that –

a. *Significant harm is being caused or there is a significant possibility of such harm being caused; or*

b. *Significant pollution of controlled waters is being caused or there is a significant possibility of such pollution being caused.*

and, in determining whether any land appears to be such land, a local authority shall, subject to subsection (5) below, act in accordance with guidance issued by the Secretary of State in accordance with S. 78YA below with respect to the manner in which that determination is to be made.

Section 1 of the Statutory Guidance sets out the objectives of Part 2A:

1.1 This Guidance should be read and applied with Part 2A and the following points in mind.

1.2 England has a considerable legacy of historical land contamination involving a very wide range of substances. On all land there are background levels of substances, including substances that are naturally present as a result of our varied and complex geology and substances resulting from diffuse human pollution. On some land there are greater concentrations of contaminants, often associated with industrial use and waste disposal. In a minority of cases there may be sufficient risk to health or the environment for such land to be considered contaminated land.

1.3 Part 2A provides a means of dealing with unacceptable risks posed by land contamination to human health and the environment, and enforcing authorities should seek to find and deal with such land. Under Part 2A the starting point should be that land is not contaminated land unless there is reason to consider otherwise. Only land where unacceptable risks are clearly identified, after a risk assessment has been undertaken in accordance with this Guidance, should be considered as meeting the Part 2A definition of contaminated land.

1.4 The overarching objectives of the Government's policy on contaminated land and the Part 2A regime are:

(a) To identify and remove unacceptable risks to human health and the environment.

(b) To seek to ensure that contaminated land is made suitable for its current use.

(c) To ensure that the burdens faced by individuals, companies and society as a whole are proportionate, manageable and compatible with the principles of sustainable development.

1.5 Enforcing authorities should seek to use Part 2A only where no appropriate alternative solution exists. The Part 2A regime is one of several ways in which land contamination can be addressed. For example, land contamination can be addressed when land is developed (or redeveloped) under the planning system, during the building control process, or where action is taken independently by landowners. Other legislative regimes may also provide a means of dealing with land contamination issues, such as building regulations; the regimes for waste, water, and environmental permitting; and the Environmental Damage (Prevention and Remediation) Regulations 2009.

1.6 Under Part 2A, the enforcing authority may need to decide whether and how to act in situations where such decisions are not straightforward, and where there may be unavoidable uncertainty underlying some of the facts of each case. In so doing, the authority should use its judgement to strike a reasonable balance between: (a) dealing with risks raised by contaminants in land and the benefits of remediating land to remove or reduce those risks; and (b) the potential impacts of regulatory intervention including financial costs to whoever will pay for remediation (including the taxpayer where relevant), health and environmental impacts of taking action, property blight, and burdens on affected people. The authority should take a precautionary approach to the risks raised by contamination, whilst avoiding a disproportionate approach given the circumstances of each case. The aim should be to consider the various benefits and costs of taking action, with a view to ensuring that the regime produces net benefits, taking account of local circumstances.

Sections 3 and 4 of the Statutory Guidance set out some important definitions:

Current use

3.5 Under Part 2A, risks should be considered only in relation to the current use of the land. For the purposes of this Guidance, the "current use" means:

(a) The use which is being made of the land currently.

(b) Reasonable likely future uses of the land that would not require a new or amended grant of planning permission.

(c) Any temporary use to which the land is put, or is likely to be put, from time to time within the bounds of current planning permission.

(d) Likely informal use of the land, for example children playing on the land, whether authorised by the owners or occupiers, or not.

(e) In the case of agricultural land, the current agricultural use should not be taken to extend beyond the growing or rearing of the crops or animals which are habitually grown or reared on the land.

3.6 In assessing risks the local authority should disregard any receptors which are not likely to be present given the current use of the land or other land which might be affected. In considering the timescale over which a risk should be assessed the authority should take into account any evidence that the current use of the land will cease in the relevant foreseeable future (e.g. within the period of exposure assumed for relevant receptors in a contaminant linkage).

Contaminant linkages

3.8 Under Part 2A, for a relevant risk to exist there needs to be one or more contaminant-pathway-receptor linkages – "contaminant linkage" – by which a relevant receptor might be affected by the contaminants in question. In other words, for a risk to exist there must be contaminants present in, on or under the land in a form and quantity that poses a hazard, and one or more pathways by which they might significantly harm people, the environment, or property; or significantly pollute controlled waters. For the purposes of this Guidance:

> *(a) A "contaminant" is a substance which is in, on or under the land and which has the potential to cause significant harm to a relevant receptor, or to cause significant pollution of controlled waters.*
>
> *(b) A "receptor" is something that could be adversely affected by a contaminant, for example a person, an organism, an ecosystem, property, or controlled waters. The various types of receptors that are relevant under the Part 2A regime are explained in later sections.*
>
> *(c) A "pathway" is a route by which a receptor is or might be affected by a contaminant.*

3.9 The term "contaminant linkage" means the relationship between a contaminant, a pathway and a receptor. All three elements of a contaminant linkage must exist in relation to particular land before the land can be considered potentially to be contaminated land under Part2A, including evidence of the actual presence of contaminants. The term "significant contaminant linkage", as used in this Guidance, means a contaminant linkage which gives rise to a level of risk sufficient to justify a piece of land being determined as contaminated land. The term "significant contaminant" means the contaminant which forms part of a significant contaminant linkage.

3.10 In some cases the local authority may encounter land where risks are presented by groups of substances which are likely to behave in the same manner, or a substantially very similar manner, in relation to the risks they may present (e.g. as may be the case with organic substances found in oils). For the purposes of identifying and assessing contaminant linkages and taking regulatory decisions in relation to such linkages, the local authority may treat such groups of contaminants as being in effect a single contaminant and multiple contaminant linkages as being in effect a single contaminant linkage. The authority should only do this if there is a scientifically robust reason for doing so, and it should state clearly why this approach has been taken in relevant documentation (including the risk summary discussed later in this Section) if the land is later determined as contaminated land.

3.11 In considering contaminant linkages, the local authority should consider whether:

> *(a) The existence of several different potential pathways linking one or more potential contaminants to a particular receptor, or to a particular class of receptors, may result in a significant contaminant linkage.*
>
> *(b) There is more than one significant contaminant linkage on any land. If there are, the authority should consider whether or not each should be dealt with separately, since different people may be responsible for the remediation of individual contaminant linkages.*

Significant harm to human health

4.3 The paragraphs below set out categories of harm that should be considered to be significant harm to human health. In all cases the harm should be directly attributable to the effects of contaminants in, on or under the land on the body(ies) of the person(s) concerned.

4.4 Conditions for determining that land is contaminated land on the basis that significant harm is being caused would exist where: (a) the local authority has carried out an appropriate, scientific and technical assessment of all the relevant and available evidence; and (b) on the basis of that

assessment, the authority is satisfied on the balance of probabilities that significant harm is being caused (i.e. that it is more likely than not that such harm is being caused) by a significant contaminant(s).

4.5 The following health effects should always be considered to constitute significant harm to human health: 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.

4.6 Other health effects may be considered by the local authority to constitute significant harm. For example, a wide range of conditions may or may not constitute significant harm (alone or in combination) including: physical injury; gastrointestinal disturbances; respiratory tract effects; cardio-vascular effects; central nervous system effects; skin ailments; effects on organs such as the liver or kidneys; or a wide range of other health impacts. In deciding whether or not a particular form of harm is significant harm, the local authority should consider the seriousness of the harm in question: including the impact on the health, and quality of life, of any person suffering the harm; and the scale of the harm. The authority should only conclude that harm is significant if it considers that treating the land as contaminated land would be in accordance with the broad objectives of the regime as described in Section 1.

4.7 If the local authority decides that harm is occurring but it is not significant harm, it should consider whether such harm might be relevant to consideration of whether or not the land poses a significant possibility of significant harm (see sub-section 4.2 below). For example, this might be the case if there is evidence that the harm may be a precursor to, or indicative or symptomatic of, a more serious form of harm, or that repeated episodes of minor harm (e.g. repeated skin ailments) might lead to more serious harm in the longer term.

Possibility of significant harm to human health

4.10 In assessing the possibility of significant harm to human health from the land and associated issues, the local authority should act in accordance with the advice on risk assessment in Section 3 and the guidance in this section.

4.11 The term "possibility of significant harm" as it applies to human health, for the purposes of this guidance, means the risk posed by one or more relevant contaminant linkage(s) relating to the land. It comprises:

(a) The estimated likelihood that significant harm might occur to an identified receptor, taking account of the current use of the land in question.

(b) The estimated impact if the significant harm did occur i.e. the nature of the harm, the seriousness of the harm to any person who might suffer it, and (where relevant) the extent of the harm in terms of how many people might suffer it.

4.12 In estimating the likelihood that a specific form of significant harm might occur the local authority should, among other things, consider:

(a) The estimated probability that the significant harm might occur: (i) if the land continues to be used as it is currently being used; and (ii) where relevant, if the land were to be used in a different way (or ways) in the future having regard to the guidance on "current use" in Section 3.

(b) The strength of evidence underlying the risk estimate. It should also consider the key assumptions on which the estimate of likelihood is based, and the level of uncertainty underlying the estimate.

4.13 In some cases the local authority's assessment of possibility of significant harm may be based, solely or partially, on a possible risk that may exist if circumstances were to change in the future within the bounds of the current use of the land. For example, an assessment may be based on a

possible risk if a more sensitive receptor were to move onto the land at some point in the future. In such cases the authority should ensure that the possibility of the future circumstance occurring is taken into account in estimating the overall possibility of significant harm.

4.14 The local authority should estimate the timescale over which the significant harm might become manifest, to the extent that this is possible and practicable (and recognising that often it may only be possible and practicable to give a broad indication of the estimated timescale).

Categorisation of land for deciding whether a possibility of significant harm is significant (health)

4.17 In deciding whether or not land is contaminated land on grounds of significant possibility of significant harm to human health, the local authority should use the categorisations described in paragraphs 4.19 – 4.30 below. Categories 1 and 2 would encompass land which is capable of being determined as contaminated land on grounds of significant possibility of significant harm to human health. Categories 3 and 4 would encompass land which is not capable of being determined on such grounds.

4.18 In considering whether a significant possibility of significant harm exists, the local authority should consider the number of people who might be exposed to the risk in question and/or the number of people it estimates would be likely to suffer harm. In some cases, the authority may decide that this is not a particularly relevant consideration: it is quite possible that land could be determined as contaminated land on the basis of a significant possibility of significant harm to an individual or a small number of people. However in other cases the authority may consider that the number of people affected is an important consideration, for example if the number of people at risk substantially alters the authority's view of the likelihood of significant harm or the scale and seriousness of such harm if it did occur.

Category 1: Human Health

4.19 The local authority should assume that a significant possibility of significant harm exists in any case where it considers there is an unacceptably high probability, supported by robust science-based evidence that significant harm would occur if no action is taken to stop it. For the purposes of this Guidance, these are referred to as "Category 1: Human Health" cases. Land should be deemed to be a Category 1: Human Health case where:

> *(a) the authority is aware that similar land or situations are known, or are strongly suspected on the basis of robust evidence, to have caused such harm before in the United Kingdom or elsewhere; or*
>
> *(b) the authority is aware that similar degrees of exposure (via any medium) to the contaminant(s) in question are known, or strongly suspected on the basis of robust evidence, to have caused such harm before in the United Kingdom or elsewhere;*
>
> *(c) the authority considers that significant harm may already have been caused by contaminants in, on or under the land, and that there is an unacceptable risk that it might continue or occur again if no action is taken. Among other things, the authority may decide to determine the land on these grounds if it considers that it is likely that significant harm is being caused, but it considers either: (i) that there is insufficient evidence to be sure of meeting the "balance of probability" test for demonstrating that significant harm is being caused; or (ii) that the time needed to demonstrate such a level of probability would cause unreasonable delay, cost, or disruption and stress to affected people particularly in cases involving residential properties.*

Category 4: Human Health

4.20 The local authority should not assume that land poses a significant possibility of significant harm if it considers that there is no risk or that the level of risk posed is low. For the purposes of this Guidance, such land is referred to as a "Category 4: Human Health" case. The authority may decide that the land is a Category 4: Human Health case as soon as it considers it has evidence to this effect, and this may happen at any stage during risk assessment including the early stages.

4.21 The local authority should consider that the following types of land should be placed into Category 4: Human Health:

> *(a) Land where no relevant contaminant linkage has been established.*
>
> *(b) Land where there are only normal levels of contaminants in soil, as explained in Section 3 of this Guidance.*
>
> *(c) Land that has been excluded from the need for further inspection and assessment because contaminant levels do not exceed relevant generic assessment criteria in accordance with Section 3 of this Guidance, or relevant technical tools or advice that may be developed in accordance with paragraph 3.30 of this Guidance.*
>
> *(d) Land where estimated levels of exposure to contaminants in soil are likely to form only a small proportion of what a receptor might be exposed to anyway through other sources of environmental exposure (e.g. in relation to average estimated national levels of exposure to substances commonly found in the environment, to which receptors are likely to be exposed in the normal course of their lives).*

4.22 The local authority may consider that land other than the types described in paragraph 4.21 should be placed into Category 4: Human Health if following a detailed quantitative risk assessment it is satisfied that the level of risk posed is sufficiently low.

4.23 Local authorities may decide that particular land apparently matching the descriptions of paragraph 4.21 (b) or (d) immediately above poses sufficient risk to human health to fall into Categories other than Category 4. However, such cases are likely to be very unusual and the authority should take particular care to explain why the decision has been taken, and to ensure that it is supported by robust evidence.

Categories 2 and 3: Human Health

4.24 For land that cannot be placed into Categories 1 or 4, the local authority should decide whether the land should be placed into either: (a) Category 2: Human Health, in which case the land would be capable of being determined as contaminated land on grounds of significant possibility of significant harm to human health; or (b) Category 3: Human Health, in which case the land would not be capable of being determined on such grounds.

4.25 The local authority should consider this decision in the context of the broad objectives of the regime and of the Government's policy as set out in Section 1. It should also be mindful of the fact that the decision is a positive legal test, meaning that the starting assumption should be that land does not pose a significant possibility of significant harm unless there is reason to consider otherwise. The authority should then, in accordance with paragraphs 4.26 to 4.29 below, decide which of the following two categories the land falls into:

> *(a) Category 2: Human Health. Land should be placed into Category 2 if the authority concludes, on the basis that there is a strong case for considering that the risks from the land are of sufficient concern, that the land poses a significant possibility of significant harm, with all that this might involve and having regard to Section 1. Category 2 may include land where there is little or no direct evidence that similar land, situations or levels of exposure*

have caused harm before, but nonetheless the authority considers on the basis of the available evidence, including expert opinion, that there is a strong case for taking action under Part 2A on a precautionary basis.

(b) Category 3: Human Health. Land should be placed into Category 3 if the authority concludes that the strong case described in 4.25(a) does not exist, and therefore the legal test for significant possibility of significant harm is not met. Category 3 may include land where the risks are not low, but nonetheless the authority considers that regulatory intervention under Part 2A is not warranted. This recognises that placing land in Category 3 would not stop others, such as the owner or occupier of the land, from taking action to reduce risks outside of the Part 2A regime if they choose. The authority should consider making available the results of its inspection and risk assessment to the owners/occupiers of Category 3 land.

4.26 In making its decision on whether land falls into Category 2 or Category 3, the local authority should first consider its assessment of the possibility of significant harm to human health, including the estimated likelihood of such harm, the estimated impact if it did occur, the timescale over which it might occur, and the levels of certainty attached to these estimates. If the authority considers, on the basis of this consideration alone, that the strong case described in paragraph 4.25(a) does or does not exist, the authority should make its decision on whether the land falls into Category 2 or Category 3 on this basis regardless of the other factors discussed in paragraph 4.27.

4.27 If the authority considers that it cannot make a decision in line with paragraph 4.26, it should consider other factors which it considers are relevant to achieving the objectives set out in Section 1. This should include consideration of:

(a) The likely direct and indirect health benefits and impacts of regulatory intervention. This would include benefits of reducing or removing the risk posed by contamination. It would also include any risks from contaminants being mobilised during remediation (which would in any case have to be considered under other relevant legislation); and any indirect impacts such as stress-related health effects that may be experienced by affected people, particularly local residents. If it is not clear to the authority that the health benefits of remediation would outweigh the health impacts, the authority should presume the land falls into Category 3 unless there is strong reason to consider otherwise.

(b) The authority's initial estimate of what remediation would involve; how long it would take; what benefit it would be likely to bring; whether the benefits would outweigh the financial and economic costs; and any impacts on local society or the environment from taking action that the authority considers to be relevant.

4.28 In making its consideration in regard to paragraph 4.27(a) and (b), the local authority is not required to make a detailed assessment. For example, the consideration should not necessarily involve quantification of the impacts, particularly if the authority considers it is not possible or reasonable to do so, and the authority is not expected to produce a detailed cost-benefit or sustainability analysis. Rather it is expected to make a broad consideration of factors it considers relevant to achieving the aims of Section 1.

4.29 If, having taken the above factors into account, the local authority still cannot decide whether or not a significant possibility of significant harm exists, it should conclude that the legal test has not been met and the land should be placed in Category 3.

A2: 'Normal' levels of soil contamination

SG 3.21 to 3.26 (DEFRA 2012a) explain that *Part 2A* was "…*not intended to apply to land with levels of contaminants in soil that are commonplace and widespread throughout England or parts of it, and for which in the very large majority of cases there is no reason to consider that there is an unacceptable risk.*" Consequently, "*Normal levels of contaminants in soil should not be considered to cause land to qualify as contaminated land, unless there is a particular reason to consider otherwise.*"

"Normal" levels of contaminants in soil may result from:

> *(a) The natural presence of contaminants (e.g. caused by soil formation processes and underlying geology) at levels that might reasonably be considered typical in a given area and have not been shown to pose an unacceptable risk to health or the environment.*
>
> *(b) The presence of contaminants caused by low level diffuse pollution, and common human activity other than specific industrial processes. For example, this would include diffuse pollution caused by historic use of leaded petrol and the presence of benzo(a)pyrene from vehicle exhausts, and the spreading of domestic ash in gardens at levels that might reasonably be considered typical.*"

In order to assist local authorities to determine 'normal' concentrations for their local area, Defra published research on normal background concentrations (NBCs) for a range of trace elements and common diffuse urban pollutants including benzo[a]pyrene (DEFRA 2012c). For each contaminant, NBCs are "*attributed to different regions of the country for each contaminant based on factors that were observed to contribute to higher concentrations in some areas, referred to as domains.*" Data outside of the domains associated with higher concentrations is assigned to the principal domain. For each domain, the NBC is defined as the upper 95% confidence limit of the 95th percentile of the contaminant concentration for that domain. Additional data using the NBC method has also been published in the scientific literature and has been summarised for this investigation (AECOM 2019g).

Soil data for each chemical was compared with the NBC where available. Where soil concentrations were lower than the NBC, it was concluded that the levels are in the range seen in the respective domain (usually this was the urban domain, but others may be more representative of the Grenfell area). On this basis, and without further characterisation of the contamination, it was not possible to conclude with any certainty whether the contamination was the result of the fire or the result of natural or historic/diffuse anthropogenic pollution.

In many cases there was insufficient data to derive an NBC. Therefore AECOM made reasonable judgements on the basis of the available information (for example, a review of the available scientific literature and levels found elsewhere in the local area). This included:

- Comparing the range of soil concentrations found at the site with the range of values found in other similar locations (either locally or regionally – such as across London) using methods such as summary statistics and box-and-whisker plots.
- Comparing the average or median concentrations found at the site with representative values from other locations using methods such as summary statistics and box-and-whisker plots.

13. HSE, 2021. Asbestos: The Analysts' Guide, HSG248

Full Reference: HSE, 2021. Asbestos: The Analysts' Guide, HSG248, Second Edition, Health and Safety Executive, July 2021

[Provided in full as a Core Document]

14. HSL, 2006. Investigation of the chrysotile fibres in an asbestos cement sample

Full Reference: HSL, 2006. Investigation of the chrysotile fibres in an asbestos cement sample. HSL report 2007/11, Health and Safety Laboratory, 2006

[Extract: pages 24-28

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HEALTH & SAFETY LABORATORY

Investigation of the chrysotile fibres in an asbestos cement sample

HSL/2007/11

Project Leader: **G.Burdett**

Author(s): **G Burdett,**

Science Group: **Science Group 5**

6 RESULTS OF THE TEM ANALYSIS OF THE AIR SAMPLES

Due to the short sampling time (a few minutes) and the limited amount of material disturbed the air filter was relatively lightly loaded with particulate and fibre. Grid H4 was scanned for particles and fibres and examples of the types of fibres and particles seen are give below, along with examples of the EDXA spectra obtained.

Figure 7 - 11: Examples of particles, fibres and fibre bundles found on air sample grid H4 of Aims sample R28S2 V117s. All pictures are approximately the same magnification. (0_____5µm)

Figure 7: Spectrum from fibres along one edge of encapsulated chrysotile bundle.

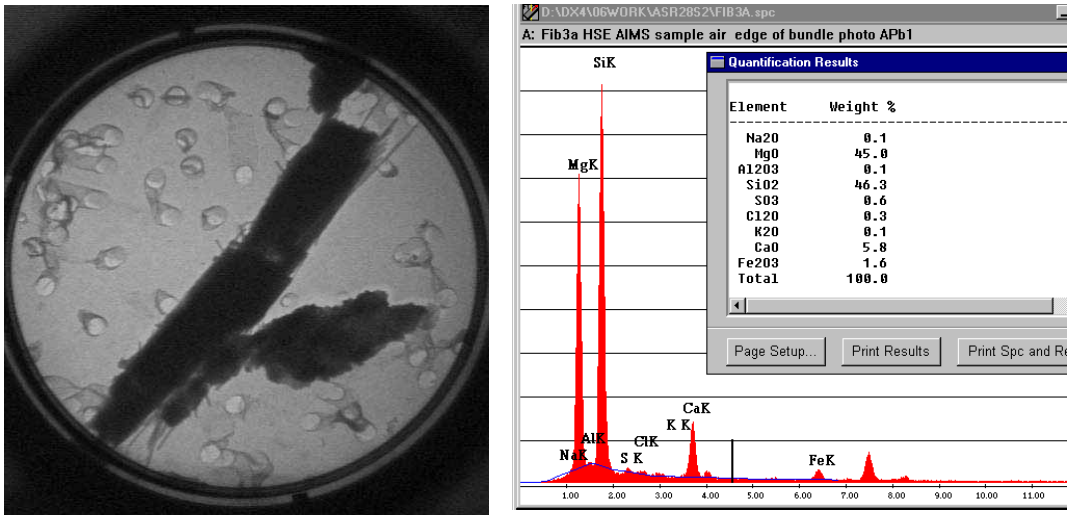


Figure 8: EDXA Spectrum of particle only

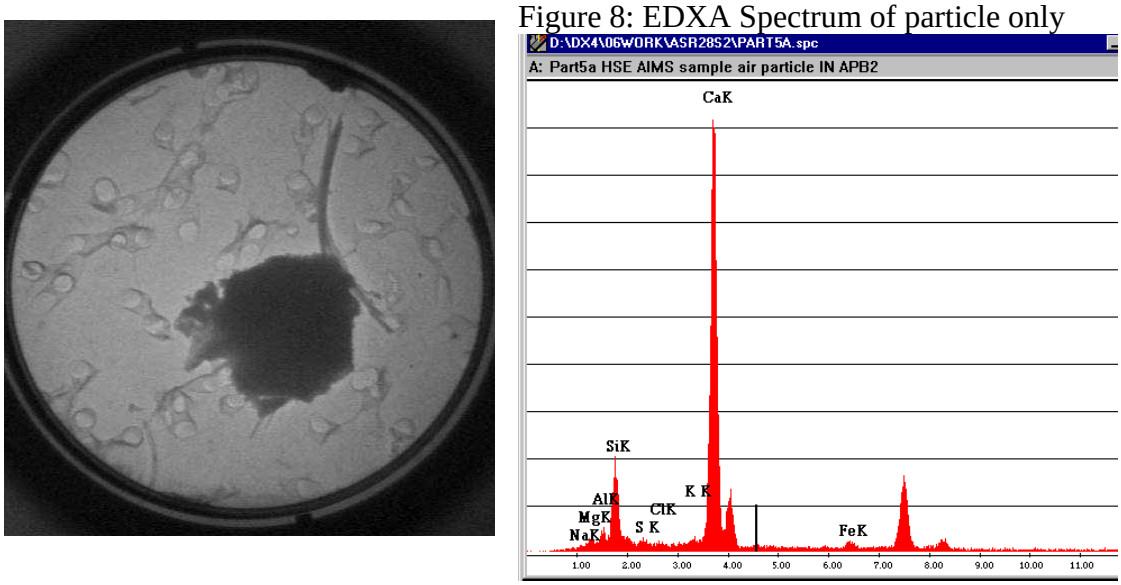
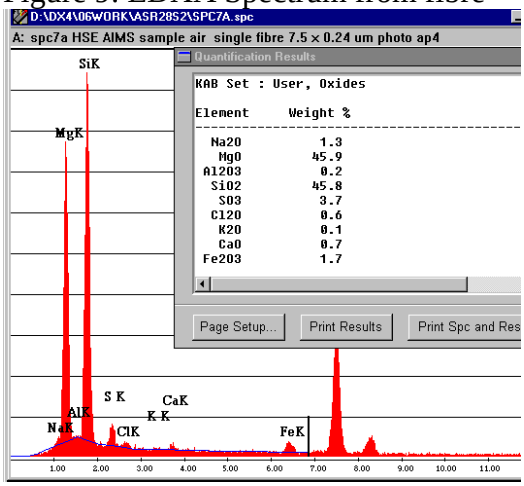


Figure 9: EDXA Spectrum from fibre



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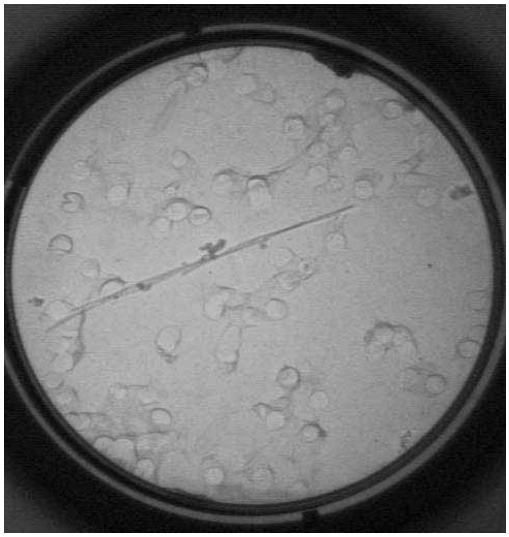
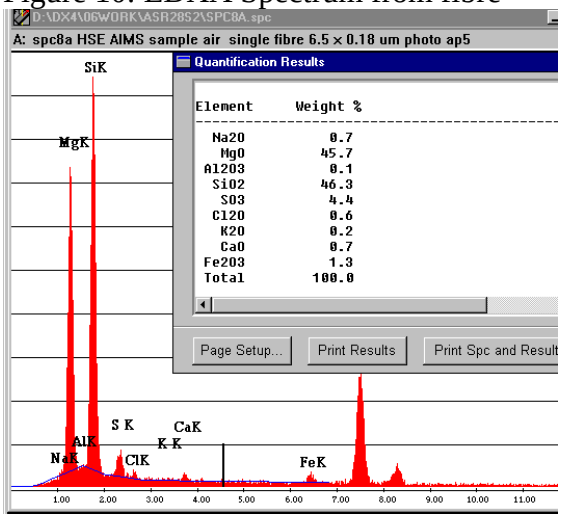


Figure 10: EDXA Spectrum from fibre



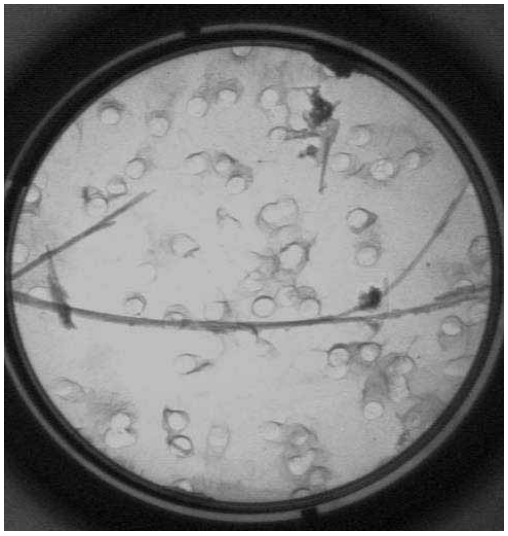
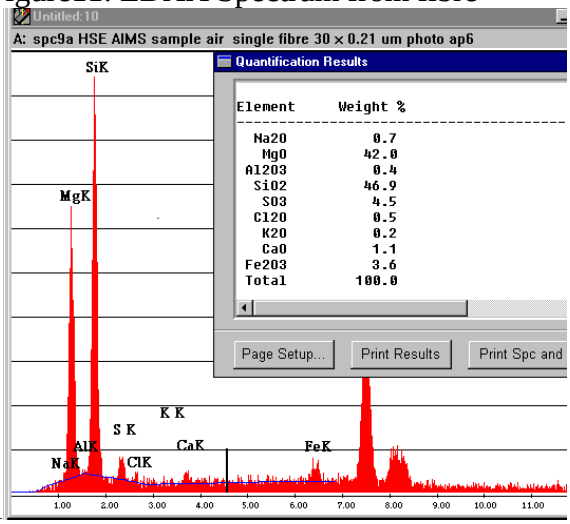


Figure11: EDXA Spectrum from fibre



1

It can be seen that the fine airborne fibres released usually had only a few small particles attached or overlying the fibres and the fibres found were essentially free of any coating (see figures 9 –11). Figure 7 showed a large fibre agglomerate, where most of the fibre is encapsulated in the cement matrix giving a higher Ca peak from the exposed end. Figure 8 gives the spectrum from the cement particle attached to the fibre. Examples of the energy dispersive x-ray spectra from the fibres (see figures 9 – 11) away from the vicinity of any attached particles gave chrysotile spectra similar to that from the standard and published information in tables 2 &3. The main change is a slight increase in the sulphur content.

The fibres in figures 7 – 11 were also analysed by electron diffraction and all gave the characteristic chrysotile SAED pattern.

7 RESULTS OF THE PLM ANALYSIS

Although PLM analysis was not carried out again on this sample of asbestos cement, the samples used are from the AIMS proficiency testing scheme and all 246 laboratories that analysed the samples reported the presence of chrysotile asbestos in the sample. It is therefore clear that the optical properties of the fibres in the sample were not significantly changed. If calcium had been adsorbed into the structure of the fibre the refractive index and hence dispersion staining colours used to measure the refractive index would also have been changed. That all 246 laboratories reported chrysotile asbestos, suggests that it was still present in this sample.

8 DISCUSSION

8.1 Evidence for changes to the chrysotile fibres

The fibres found in the bulk analysis and on the airborne filters were unambiguously chrysotile asbestos fibres, which showed no significant alteration. Often the dispersed fibres would have a few small particles attached or overlying the fibre but this was not extensive and cannot be viewed as anything other than attached particles of the cement matrix. The chemistry of the fibre was consistent with the chrysotile standard and published information from the main commercial chrysotile mines worldwide.

The tubular structure seen in many of the fibrils and the characteristic chrysotile asbestos diffraction patterns obtained are strong evidence against the adsorption of calcium into the chrysotile structure. If calcium were being adsorbed to form an altered mineral the d-spacings would also change. It is highly unlikely that the tubular structure would be able to accommodate the larger Ca atoms without some change in the structural and physical appearance of the fibres.

Chrysotile is well known to have poor resistance to acids and is capable of chemical change due to the loss of magnesium and hydroxide ions from the outer layers of the scroll structure. Acid attacked chrysotile shows clear damage to the fibril structure in the TEM and the EDX analysis shows a large reduction in the magnesium peak. This

was not observed for this sample but has been found in chrysotile fibres from water samples and from the weathered surface of AC sheets exposed to acid rain. When working with asbestos cement and breaking the material it was shown that unaltered chrysotile fibres were released.

Even when cement coated fibres occur it will be subject to weathering and will dissolve. This may also occur in the lung where fibres that reach the deep lung will be bathed in a constant flow of fluid and surfactants that coat the lung's surface. However, it was clear that the large majority of the chrysotile fibres in the asbestos cement sample analysed were unchanged and that unaltered chrysotile fibres were released to air.

This analysis and observation is consistent with other analyses of asbestos cement samples from other sources carried out by HSL.

8.2 Evidence for coating of fibres

The fibres inspected in the asbestos cement sample examined, appeared to be uncoated except that some cement particles from the matrix were present (see figure 2). These could not be described as a coating but were calcium-rich small particles and agglomerates , which were sometimes attached to the chrysotile fibres. The presence of the calcium-rich particles in the sample would also contribute to the presence of calcium in the EDX spectra.

Examples of coated fibres have been seen in other samples. Although some discreet particles were visible (see figure 3) it can also be seen that there is a more diffuse low electron density gel like coating on parts of the fibre, rather than discreet particles. In the particular fibre found, both calcium and aluminium were present in the spectrum. Pooley (2004) reported that the coated fibres had additional calcium and silicon but this could also be an artefact of the EDX analysis or due to magnesium depletion of the chrysotile fibre due to weathering. A calcium hydroxide gel is formed during the hydration process of Portland cement and could coat some of the individual fibres. Calcium hydroxide is the most water-soluble component of cement and is easily removed by normal weathering processes and presumably by immersion in water or lung fluids. If the coating on fibres is readily removed by water it is clear evidence that there is no overall change to the chemistry of the fibres.

In weathered samples of asbestos cement the chrysotile fibres are more resistant than the cement matrix so a surface layer of predominantly chrysotile fibres may form. PAH's have been reported to collect and concentrate on the exposed chrysotile fibres on the weathered surface of asbestos cement. Although the presence of moisture reduces this process it would also seem possible for exposed chrysotile fibres on the surface of weathered asbestos cement to be coated with PAH's.

Overall, it appears that the vast majority of fibres are uncoated and there is no evidence to support the claim that all the chrysotile has been chemically or structurally altered.

8.3 Evidence for airborne fibre release

The claim that respirable airborne chrysotile fibres are not able to be released from asbestos cement products was refuted by the individual airborne fibres sampled during the breaking of the test sample with a hammer. There are also a number of papers in the published peer reviewed scientific literature that have measured the airborne release of asbestos fibres from asbestos cement sheet as well as HSL's own database of measurements (see annex 1).

The measurements have been carried out by the regulatory method based on phase contrast microscopy (PCM) fibre counts (e.g. annex 1 to 83/477/EEC and HSG 248). This method counts all fibres of countable dimensions but does not identify individual fibres. The counting rules used for PCM analyses (prior to November 2006) do not allow the fibre to be counted if attached or overlain by a particle of >3 µm in width. Therefore the published data would not have counted any asbestos fibres attached to a cement particle >3 µm width, as it would be unlikely to reach the pulmonary region of the lung.

8.4 Evidence for loss of carcinogenic potency due to use of the asbestos in asbestos cement.

Although outside the scope of the current paper, it is worthwhile to note that there is substantial evidence from animal experiments that inhaled chrysotile fibres are carcinogenic. However, some authors have argued that the carcinogenic response from chrysotile is solely due to the "overload effect" on the lung and not the chrysotile fibre itself or is due solely (or in part) to the amphibole asbestos impurities in the chrysotile. Others accept that chrysotile is a cause of lung cancer but not mesothelioma, or that while chrysotile may be carcinogenic there is no evidence from animal experiments that the chrysotile fibres from asbestos cement are carcinogenic. It is not easy to unravel the claims and counter claims about the carcinogenic nature of chrysotile and it is noted that from the number of reviews on these issues over the last 2-3 years (e.g. Lemen, 2004; Le et al., 2004 and Yarborough, C. M. 2006) that the debate continues.

The claims that chrysotile in asbestos cement had reduced carcinogenic potency were particularly in vogue in the 1980's when prohibition on asbestos cement was being considered. These claims were specifically tested using sensitive interperitoneal injection into rats. No observed differences in the carcinogenic potencies were found between 20 year-old weathered chrysotile asbestos fibres from the surface of asbestos cement sheets and UICC chrysotile asbestos (Spurny, 1988). These results were supported by work carried out by Tiles and Beck (1990) who also reported that the carcinogenicity of the weathered asbestos-cement chrysotile fibres is comparable to that of standard chrysotile fibres following intraperitoneal (i.p.) application and even greater than the unaltered asbestos cement fibres taken from the core of the weathered sheets. The reason for these difference was attributed to the lower amount of chrysotile in the core sample (10%) as compared with the weathered surface sample (30%). However, the ability of the free chrysotile fibres on the weathered surface to

15. IAQM, 2016. IAQM guidance on the Assessment of Mineral Dust Impacts for Planning

Full Reference: IAQM, 2016. IAQM guidance on the Assessment of Mineral Dust Impacts for Planning, Version 1.1, Institute of Air Quality Management, London, September 2016

[Extract: pages 19-23]

IAQM Institute of Air Quality Management

Guidance on the Assessment of Mineral Dust Impacts for Planning

May 2016 (v1.1)

www.iaqm.co.uk

6. Factors influencing the risk of dust impacts

6.1 Categorising the Source, Pathway and Receptor Terms

The previous chapter described how the risk of dust impacts from any given minerals development proposal, and any resulting adverse effects, depended on: the level of dust emissions from the site (the Source); the effectiveness of transport through the air (the Pathway), and the sensitivity of surrounding land users (the Receptors) that could be exposed. The important factors that need to be considered in categorising the S, P and R terms are summarised below.

6.2 Factors Influencing the Residual Source Emissions

The scale and nature of the works will determine the level of residual (i.e. abated) dust emissions from fugitive sources, diffuse sources and, if applicable, point sources associated with the development. The judgement on the categorisation of the Source term will need to take into account the emission potential of each of the sources on the site (including source strength, frequency and duration) and how effectively they are likely to be controlled by designed–in measures proposed as part of the scheme. Specific factors include:

- the activities being undertaken (blasting, crushing, screening, methods of handling and storage, etc.);
- the types and properties of the materials involved;
- the size of the site and, specifically, the area of land being worked (and hence the quantities of materials involved and the number of vehicles and plant etc.);
- the durations and frequencies of the activities;
- the likely effectiveness of the dust control measures incorporated into the design of the submitted development scheme, including design features, management controls (ideally formalised within a Dust Management Plan) and, where appropriate, engineering controls;
- other mitigation measures applied to reduce or eliminate dust; and
- the meteorological conditions that can promote or inhibit the raising of dust at the source (high winds and rainfall, respectively).

Further information on these factors is given below.

6.2.1 Activities being undertaken

The following seven types of dust-generating activities on mineral extraction sites are likely to have the greatest potential for dust emissions:

a) Site preparation/restoration (including soil and overburden handling);

b) Mineral extraction (including blasting);

c) Materials handling (e.g. loading onto haul trucks or conveyors);

d) On-site transportation (haul roads);

e) Mineral processing (e.g. crushing and screening);

f) Stockpiling/exposed surfaces; and

g) Off-site transportation (e.g. leading to trackout onto external road network).

It is not usually possible to predict with any degree of certainty when particular work activities will take place and whether these will coincide with high-risk meteorological conditions (see further details below). It is usual therefore to make assumptions; a worst case would be to assume that for those periods when winds are blowing from the site to receptors, those specific site activities that generate dust will be occurring. In practice this is unlikely to always be the case.

6.2.2 Materials

The type of material being extracted and processed can have a significant influence on potential emissions. Sand and gravel deposits may possess an inherently high moisture content, which can cause particles to adhere and thereby affords a high degree of natural mitigation. However, this does not negate the potential for fugitive emissions from this material if it dries out, especially during high wind conditions. Conversely, the extraction and processing of hard rock such as granites and limestone can more readily generate dust, which requires appropriate mitigation.

Particle size distribution of the material is particularly important to dust emissions from vehicles passing over unpaved ground, as well as the speed and weight of the vehicle, the moisture content of the material, the distance covered and the frequency of vehicle movements.

High levels of PM_{10} may be associated with high levels of deposited dust. However, there is no direct correlation between

▲ **Image: © Hugh Datson, DustScan Ltd**

the two; indeed, as airborne particles fall out of the parcel of dust-laden air, the suspended PM concentration is reduced. The relative proportions of size fractions that deposit quickly compared to those that stay suspended for lengthy periods is determined by the materials and activities involved. Mineral type can dictate the potential influence on PM_{10}. Extraction of material with a high moisture content, such as sand and gravel, can potentially generate a smaller impact than the percussive processes associated with hard rock. The particle size and/or processes associated with specific minerals can also generate PM_{10}, for example, the inherently small particle size of clay.

6.2.3 Dust control measures incorporated into the design

Individual mineral site design and associated environmental management can significantly influence the fugitive emissions of dust generation. This can limit the capability of precise dust impact prediction as each site is distinct. Ideally, the various dust control measures and management controls should be described in a formal Dust Management Plan document (see **Appendix 6** for the IAQM's recommendation on what a DMP should contain).

6.2.4 Meteorological conditions

High wind speeds increase the likelihood of dust being raised and blown from the site. Dry materials are more easily raised into the air and so rainfall acts as a natural dust suppressant. High-risk meteorological conditions are, therefore, when the wind is coming from the direction of the dust source[28] at a sufficient strength, during periods of little or no rainfall (often taken as <0.2 mm per day) especially during periods when evaporation exceeds rainfall and drying conditions prevail. The threshold wind speeds for initiation of wind blow[29] can range from 2.4 m/s (Force 2, "light breeze") up to gale force, depending on the particle size and the condition of the surface[30] but moderate breeze, 5.5 m/s and above, is sometimes used as a general threshold. It is preferable to use a wind blow initiation wind speed specific to the mineral type.

Due to the variability of the weather, it is impossible to predict what the weather conditions will be when specific activities are being undertaken, so it is common practice to use either a worst-case approach (assuming the high-risk meteorological conditions exist for all working activities) or a probabilistic approach (assuming the high-risk meteorological conditions occur for a particular percentage of the time).

Impacts during the summer and winter months are generally different, and if it can be guaranteed that certain activities or those at a specific location will take place during a particular season (with this enforced through a planning condition, for

example), consideration could be given to using seasonal wind and rainfall data. However, this type of guarantee is not usual because the demand for minerals is not usually seasonal.

Large scale physical features such as rivers, valleys and hills can influence wind direction over a large area, as can be seen in the wind roses for certain Met Office meteorological stations, for example the influence of the Severn Estuary on wind at Bristol and Cardiff airports and the Pennines at Manchester Airport. Therefore the use of wind data from the nearest meteorological station to the site under consideration may be influenced by the terrain and not represent local conditions. Expert judgement is required to choose the most representative meteorological station, or whether there is a need for site specific data. This could be informed by looking for signs of the prevailing wind, such as the shape of trees, during the site visit.

6.3 Factors Influencing the Pathway

The primary factor influencing the Pathway is the distance between the sensitive receptor and the dust sources. However, other factors can cause a higher or a lower category to be assigned then would be the case based on distance alone. These factors include:

- orientation of receptors relative to the prevailing wind direction; and
- topography, terrain and physical features.

6.3.1 Distance between dust source and receptors

The dust that has become suspended in the air will dilute, disperse and deposit from the air (as deposited dust) with the resultant airborne PM concentration decreasing rapidly as a function of distance from its source (see **Appendix 2**). In general, smaller particles have the potential to be entrained within airflow for longer, thereby dispersing over a wider area.

6.3.2 Orientation of receptors relative to the prevailing wind direction

Dust impacts can occur in any direction from the site; they are, however, more likely to occur downwind of the prevailing wind direction and close to the boundary[31]. Although, overall, receptors in the prevailing downwind direction tend to be at higher risk of dust impact, this is a simplification: it should be noted the "prevailing" wind direction is usually the most frequent direction over a long period such as a year; whereas activity may only occur at a specific location over a period of weeks or months during which the most frequent wind direction might be quite different; furthermore, the most frequent wind direction may also not be the direction from which the wind speeds are highest[32]. The use of the prevailing wind direction in the assessment of risk is most useful, therefore, for activities of long duration such as processing carried out in dedicated areas, rather than activities such as extraction which may only occur at a specific location for a matter of weeks or months.

A more refined picture of this important factor in the effectiveness of the Pathway term can be obtained by considering the frequency that the receptor is downwind of the dust source. The percentage frequencies of winds blowing from the sources to the relevant receptors can be calculated from suitable meteorological data.

It should be noted that when strong winds occur from non-prevailing wind directions disamenity can occur if robust mitigation measures are not in place.

▲ Image: © Rachel McHale, SLR Consulting Limited

6.3.3 Terrain and physical features

The local terrain and natural and built features between the source and the receptor can variously act as barriers, reduce airborne concentrations due to impaction, lengthen pathways, affect air flow and increase or inhibit dispersion and dilution. Examples include trees and woodland, escarpments, hills and valleys, bunds, buildings/structures and trees.

6.4 Receptors

Boxes 3, 4 and **5** provide guidance on how to categorise the dust sensitivities of different receptors to disamenity, human health and ecological effects, respectively. These are the same categorisations used in other guidance (specifically the IAQM demolition and construction dust assessment guidance[8]), which is entirely appropriate as the dust sensitivity of a receptor is an inherent property and not one that is dependent on the type of development being assessed. As always, the specific circumstances should be taken into account and may mean that on some occasions particular receptors may not automatically fall into the example categorisations given in **Boxes 2**, **3** and **4**. Further discussion on this is given below.

A 'human receptor' refers to any location where a person may experience the disamenity effects of dust, or the health effects from exposure to PM_{10}. The latter should take account of the time period relevant to the air quality objectives, as defined in the Government's technical guidance for Local Air Quality Management[33].

In terms of disamenity effects, residential dwellings are considered highly sensitive. In some instances, industrial and commercial premises may be considered highly sensitive receptors if they are particularly vulnerable to soiling effects. The latter may include, for example, vehicle showrooms, food manufacturers and electronics manufacturers. The sensitivity will relate to the level of amenity that can be reasonably expected. For example, dwellings and schools are more sensitive than industrial units or farms. Care should be taken to ensure that the assessment takes into account whether exposure will arise in practice (e.g. computer chip manufacture is sensitive to dust and so premises are likely to have extensive dust filtering equipment, although the frequency of filter changes may need to be increased).

▲ Image: © Advance Environmental

An 'ecological receptor' refers to any sensitive habitat affected by dust deposition. This includes the direct impacts on vegetation or aquatic ecosystems, and the indirect impacts on fauna (e.g. on foraging habitats). For locations with a statutory designation, e.g. Special Areas of Conservation (SACs) and Sites of Special Scientific Interest (SSSIs), consideration should be given as to whether the particular site is sensitive to dust and this will depend on why it has been designated. Some non-statutory sites (i.e. local wildlife sites) and/or locations with very specific sensitivities may also be considered if appropriate. The level of dust deposition likely to lead to a change in vegetation is very high (over 1 $g/m^2/day$[34]) and the likelihood of a significant effect is therefore very low except on the sites with the highest dust release close to sensitive habitats. Notwithstanding this, the inclusion or exclusion of sites should be justified in the assessment.

[28] For receptors ≤30 m of the site, it has been assumed that they would be affected during any wind direction, which will be a conservative assumption.
[29] Wind blow is the suspension of dust by the wind from the exposed surfaces e.g. within the extraction area, and stockpiles.
[30] Arup Environmental, Ove Arup and Partner, 1995. The Environmental Effects of Dust from Surface Minerals Workings, HMSO, London (ISBN 11 75 3186 3).
[31] For receptors very close to sources the worst-case assumption, that they would be affected during any wind direction, could be made.
[32] High wind speeds, as well as raising dust (including that previously deposited), can better disperse and dilute the suspended dust.
[33] Department of the Environment, Food and Rural Affairs, 2016. Local Air Quality Management Technical Guidance LAQM.TG(16).
[34] Farmer, A M, 1993. The effects of dust on vegetation – a review. Environmental Pollution 79, 63-75.

Box 3. Sensitivities of People to Dust Soiling Effects

For the sensitivity of people and their property to soiling, the IAQM recommends that the air quality practitioner uses professional judgement to identify where on the spectrum between high and low sensitivity a receptor lies, taking into account the following general principles:

High sensitivity receptor

- users can reasonably expect[a] enjoyment of a high level of amenity; or
- the appearance, aesthetics or value of their property would be diminished by soiling; and the people or property would reasonably be expected[a] to be present continuously, or at least regularly for extended periods, as part of the normal pattern of use of the land.
- indicative examples include dwellings, medium and long term car parks[b] and car showrooms.

Medium sensitivity receptor

- users would expect [a] to enjoy a reasonable level of amenity, but would not reasonably expect to enjoy the same level of amenity as in their home; or
- the appearance, aesthetics or value of their property could be diminished by soiling; or
- the people or property wouldn't reasonably be expected [a] to be present here continuously or regularly for extended periods as part of the normal pattern of use of the land.
- Indicative examples include parks, and places of work.

Low sensitivity receptor

- the enjoyment of amenity would not reasonably be expected[a]; or
- there is property that would not reasonably be expecteda to be diminished in appearance, aesthetics or value by soiling; or
- there is transient exposure, where the people or property would reasonably be expected to be present only for limited periods of time as part of the normal pattern of use of the land.
- Indicative examples include playing fields, farmland (unless commercially-sensitive horticultural), footpaths, short term car parks[b] and roads.

[a.] People's expectations will vary depending on the existing dust deposition in the area.

[b.] Car parks can have a range of sensitivities depending on the duration and frequency that people would be expected to park their cars there, and the level of amenity they could reasonably expect whilst doing so. Car parks associated with work place or residential parking might have a high level of sensitivity compared to car parks used less frequency and for shorter durations, such as those associated with shopping or errands. Cases should be examined on their own merits.

16. IOM, 2005. An assessment of risks due to asbestos on farm tracks and rights of way in South Cambridgeshire

Full Reference: IOM, 2005. An assessment of risks due to asbestos on farm tracks and rights of way in South Cambridgeshire, Jones, AD, Cherrie, JW, Cowie, H, and Soutar, A. Research Report TM/05/07(rev), Institute of Occupational Medicine, Edinburgh, 2005.

[Extract: pages 9-11, 13-14, 27]

Research Report TM/05/07
December 2005

An assessment of risks due to asbestos on farm tracks and rights of way in South Cambridgeshire

AD Jones, JW Cherrie, H Cowie and A Soutar

Research Report

4 SUMMARY OF CONCENTRATION DATA

4.1 THE DATA AVAILABLE

The data on airborne concentrations comprise:

- measurements on six ROW, for weekly average concentrations, for the four weeks commencing 6th September 2004;
- weekly average concentrations from two ROW, from the pilot study in July 2004;
- concentrations measured by HSL on London Way in June 2001.

We used the first of these to develop a model of weather dependence. The latter two sets of data served to check the model. Then the model was used to predict annual average concentrations for each of the six ROW. The Pilot Study data also demonstrated that concentrations are low in wet weather. The HSL measurements provided information about the source of dust disturbance.

The samples in the 2004 sampling studies were all analysed by Scanning Electron Microscopy, which enabled asbestos fibres to be identified and distinguished from non-asbestos fibres.

In the sampling by HSL in 2001, a sample was analysed by Transmission Electron Microscopy (TEM) to estimate the relative proportions asbestos and non-asbestos fibres, and the other samples were counted by optical microscopy (which includes all fibres not just asbestos fibres).

4.2 DETECTION LIMITS AND AVERAGE CONCENTRATIONS

4.2.1 Detection limits and averages from combined samples

Where zero fibres are counted in examining a random selection of fields of view on the filter, then there is 95% confidence that the true mean count would be less than 3 fibres (from the Poisson statistics that describe the counts obtained on a subset of all possible fields of view). So where counts of asbestos fibres were 0, 1 or 2 fibres, they have been reported as being less than a detection limit corresponding to 3 fibres counted in the examined area of 1 mm^2 of the filter.

Where several samples were taken from a particular location, and it is reasonable to treat them as representing an average concentration for that place, then the data were combined as if they were for a single sample with the total volume of all the samples. This gives a lower detection limit. We have used this approach with the samples for each of the ROW. This calculation can lead to a measured concentration from the combined data even though the individual samples were all below the detection limit; this happens where the individual samples have 0, 1 or 2 fibres counted, but the total number of fibres for the set is 3 or more.

An average concentration calculated from the combined data for all the samples taken on a particular ROW is the best estimate of the average concentration that would be experienced if someone continuously walked up and down the ROW throughout the sampling period.

4.2.2 Average concentrations on the six ROW

In the September 2004 study, samples were collected with a total of 25 samplers located on the six ROW. There were 10 samplers on Shedbury Lane, 4 at Hill Top Farm, 4 at Newling Non-lets, 3 at London Way, 2 at Moor End lane, and 2 at Whaddon Estate Farm. As technical failures with some pumps caused a few samples to be lost from the collection programme, a total of 90 samples were evaluated.

When the data from all the individual samples on each ROW were combined, the average concentrations shown in Table 4.1 were obtained. The detection limits depend on the volume of air sampled in total and hence on the number of samples taken. As more samples were taken on some ROW than others, the detection limits were substantially different between ROW. Nine out of 24 average concentrations were below detection limits. Six of these were on two of the ROW (Hill Top Farm, and Whaddon Estate Farm). A measured average concentration (above detection limits) was obtained for all six ROW in Week 1. The number of average concentrations below detection limits increased progressively from week to week.

Table 4.1 Mean concentrations of airborne asbestos fibres from all the samples taken on the ROW during that week. Note that 9 out of 24 average concentrations were below detection limits.

Location	Mean Asbestos Fibre concentration for each week (fibres/ml)			
	Week 1	Week 2	Week 3	Week 4
Moor End Lane, Shepreth	0.00070	0.00013	<0.00002	<0.00002
Shedbury Lane	0.00022	0.00014	0.000033	0.000045
Newling Non-lets	0.00019	0.000021	0.000062	<0.000011
London Way	0.00017	0.000045	0.000045	0.000042
Hill Top Farm	0.000069	<0.000012	<0.00001	<0.00001
Whaddon Estate Farm	0.000064	<0.000019	<0.00002	<0.00004

In the Pilot study, each individual sample of airborne fibres indicated asbestos fibre concentrations below the detection limits. For those samples, the individual samples' detection limits ranged between 0.00003 and 0.0002 fibres/ml. When all data from the pilot trial were combined to give an overall estimate of fibre concentration, the value obtained (0.00001 fibres/ml) happened to be the same as that reported by the Health Effects Institute (HEI, 1991) as being the background rural level of airborne asbestos. The weather during the week of sampling in the Pilot trial was mostly damp with occasional light rain, and therefore may have been not conducive to release of airborne fibres from dust on ROW.

4.2.3 Asbestos type

Evidence on asbestos type was obtained partly by IOM's SEM measurements of the fibres sampled from the air, which distinguished chrysotile from amphibole. The type of amphibole was identified as amosite asbestos by polarising light microscope analysis of fibres in samples of loose dust from the track surface.

In the air samples:

- at Shedbury Lane, the asbestos fibres were mostly chrysotile;
- at Newling Non-Lets mostly amphibole,
- at Moor End Lane and London Way, about equally split between amphibole and chrysotile.
- At the other two ROW (Hill Top Farm and Whaddon Estate Farm), there were few asbestos fibres detected in the air samples – so there was limited data on the asbestos type - but chrysotile was more frequent than amphibole.

Our finding that the asbestos on London Way was about 50% chrysotile and about 50% amosite was supported by HSL's analysis of fibres on a sample they collected in 2001 (Chisholm, 2001). Their analysis by transmission electron microscopy (TEM), which was able to distinguish chrysotile, amosite, or crocidolite. showed that the fibres were mainly chrysotile or amosite.

The September 2004 sampling by IOM included not only air samples but also 48 samples of the dust from the track surface adjacent to air sampling positions on the ROW. These track surface dust samples were analysed by polarising light microscopy which distinguished the type of asbestos, chrysotile and type of amphibole. Chrysotile asbestos was detected in 46 samples. Amosite asbestos was detected in samples from Newling Non-lets and Moor End Lane. No crocidolite was detected in any of the surface dust samples. Therefore the amphibole asbestos detected by SEM on air samples was assumed to be amosite.

5 INFLUENCE OF WEATHER ON AIRBORNE FIBRE CONCENTRATION

5.1 INTRODUCTION

We used airborne fibre concentrations measured on six ROW in September 2004 and weather data from a local weather station (Iceni, at Royston) to estimate the dependence of airborne fibre concentration on weather conditions, and hence extrapolate to predict average fibre concentrations over the course of a year. The fibre concentration measurements comprised the average concentrations over the course of 7 day periods, for the weeks commencing September 6th, 13th, 20th, 27th in 2004.

These data from only four weeks are of course a limited amount of information about dependence on weather. Nevertheless, the four sampling weeks in 2004 provided some contrasting weather conditions. At the start of the month, there was dry sunny weather, and by the end of the month there were wet days.

We sought a model that would predict the variation in the relative level of airborne dust and fibres from day to day, due to weather factors. The predicted variation in daily average concentration would contribute to the predicted average over each week. The agreement between observed weekly averages and the relative levels predicted by the model would be the basis for confirming values assigned to parameters in the model.

The process of developing the model involved hypothesising a sensible dependence of relative level of airborne fibre concentration on each weather parameter, and then examining whether it produced predictions acceptably close to the observed data. Essentially, we set out assumptions about how we would expect weather to influence the relative airborne fibre concentrations. Then we expressed these in terms of a simple mathematical relationship which contained a few coefficients with values to be chosen (from a limited range of alternative possibilities). With limited data on relative dustiness of different weeks (i.e. fibre levels in Weeks 2, 3 and 4 compared to Week 1), the choice of values for these coefficients was based on empirical testing of trial models with the model outputs compared to this data.

Once the model had been selected, its validity was cross checked by comparing predictions with the data on fibre concentrations obtained separately in our pilot study in June 2004 and in June 2001 by HSL (Chisholm, 2001).

5.2 ASSUMPTIONS

The dust disturbed from the surface of the ROW could be dispersed by vehicular activity, pedestrians or by wind. In the measurements of concentrations of airborne dust and fibres on one of the ROW (London Way) conducted by HSL (Chisholm, 2001), vehicular movement was identified as the major cause of dust disturbance. Wind is a recognised mechanism of soil erosion, although there appears to be relatively little basis for predicting how much dispersion is produced. A recent paper (Lu, 2001) describes development towards a model of the wind erosion effect.

Our assumptions relating to the effect of weather are that:

- the source of dust, the track surface, would be affected by moisture content. A laboratory-based study on release of fibres from prepared and homogenised soil mixtures containing known amounts of asbestos showed that release of asbestos was greatly reduced by even a small percentage of moisture (Addison, 1988). Therefore:
    - days with rainfall less than 0.2 mm would be regarded as "dry days";
    - the fibre concentration on wet days would be small, probably negligible, compared to that on dry days.
    - a wet day is likely to affect the condition of the track surface on the following day. Therefore, a dry day preceded by a wet day would have lower concentration, on average, than a dry day preceded by a dry day.
- the hours of sunshine (in a day) would contribute to drying the track surface, and therefore release of airborne fibre would tend to be higher on days with a lot of sunshine.
- on most days, the wind will carry dust away from the track and therefore wind primarily dilutes the trackside concentrations. (There will be some days when wind dispersion contributes to trackside concentrations, for example if the wind is blowing along rather than at an angle to the track; however, we assume that those days will not have a large effect on the annual average trackside concentration when there are other greater and more regular sources of dust disturbance.)

The results obtained by HSL (Chisholm, 2001) on London Way indicated that the main source of disturbance of airborne dust was the passage of vehicles, during the period when they were sampling. We assume that this is generally the case throughout the year on this and other ROW where there is vehicular access (which includes the ROW in Table 4.1).

Weather is clearly the common factor that affected the change in relative level of airborne fibre concentration from week to week on the six ROW. There may have been other unrecorded factors (such as changes in traffic flow) which may have contributed, but they appear to have been negligible compared to the weather as the pattern of change was so broadly consistent among the ROW.

5.3 POSSIBLE WEATHER FACTORS

5.3.1 Weather parameters

The weather parameters that we assessed as potentially useful for describing the influence of weather on the day to day variation in average concentration during the measurement period included:

- mean wind speed for the day (in mph);
- hours of sunshine;
- rainfall (collected from 09:00 on that day to 09:00 on the following day; below 0.2 mm to define a dry day).

7 EXPOSURE SCENARIO

7.1 INTRODUCTION

The information needed to predict the risks to health from the asbestos exposure are the *duration of exposure*, the *annual cumulative exposure* (*E* in fibre.hours/ml) for each year of exposure, the *type of asbestos*, and the *age* of the person when first exposed. The *cumulative exposure E* is the airborne asbestos fibre concentration multiplied by the duration of the exposure. Where an individual has exposure at different concentrations over various periods, then a cumulative exposure is calculated for each period and then summation over such periods gives the cumulative exposure for the year.

Since *annual cumulative exposure* is the information needed, different patterns of exposure that amount to the same annual cumulative exposure are equivalent so far as predicted risk is concerned. For example, a cumulative exposure of 6 fibre.hours/ml could arise from exposure to 1 fibre/ml for 6 hours, or from exposure to 6 fibres/ml for 1 hour, or from 2 fibres/ml for 3 hours.

The information that we have about the concentrations on each of the six ROW comprises:

- average concentrations over night and day for 7-day periods, with data from four weeks in September 2004;
- a modelled dependence of airborne asbestos fibre concentration on weather;
- a predicted annual average concentration, also for the same sampling period (day and night).

To construct realistic estimates of the exposures, we also need to take account of:

- how concentration is likely to vary with activity and time of day and how that may be linked to the periods when people are on or near the ROW;
- the amount of time that people spend or are likely to spend on or near the ROW;
- how concentration varies with distance from the ROW.

The concentration measurements (of Table 4.1) are based on samples taken on filters mounted at about 1.5 m height above ground level and with the sampler placed as close to the trackside as safely possible, i.e. generally less than 1 m from the edge of the track. So they directly reflect the exposure of someone actually on or immediately beside the track. In theory, concentration diminishes approximately linearly with distance from a line source such as a long track. So concentrations more than 5 m from the track might well be less than a tenth of that measured within about 0.5 m of the track. Therefore, we would expect concentrations about 5 to 10 m away from the track to be (on average) less than a tenth of that at the trackside. Chisholm (2001) commented that a distance of 1 m from a source of dust release had given a tenfold difference in concentration.

We consider exposure scenario that include:

- residents regularly using the track, as their only access to and from their homes;

17. IOM, 1988. The release of dispersed asbestos fibres from soils

Full Reference: IOM, 1988. The release of dispersed asbestos fibres from soils, Addison J, Davies LST, Robertson A, Willey RJ, Historical Research Report TM/88/14, Institute of Occupational Medicine, Edinburgh, 1988

[Extract: pages 17-20]

WORKING FOR A HEALTHY FUTURE

HISTORICAL RESEARCH REPORT

Research Report TM/88/14
1988

The release of dispersed asbestos fibres from soils

Addison J, Davies LST, Robertson A, Willey RJ

World Health Organisation
Collaborating Centre
For Occupational Health

5. DISCUSSION

5.1 Airborne Fibre Concentrations from Dry Soils

Analysis of the data shows that the most important factor controlling airborne fibre concentrations in the experiments with dry loose aggregate mixtures was the bulk asbestos content. The results from both IOM and GCT studies show that, irrespective of fibre type or soil type, high airborne fibre concentrations (over 20 f ml^{-1}) can be generated from 1% asbestos in dry soil while restricting the respirable dust concentration to the nuisance dust occupational exposure limit (OEL) of 5 mg m^{-3} (HSE, 1987).

There was good agreement between the results from both sets of tests with dry soils. The similarity of the results for given dust concentrations and bulk asbestos contents suggests that the dust generation techniques were of secondary importance in establishing the relationship between dust and fibre concentrations.

The results for the chrysotile tests at GCT were somewhat lower than the equivalent IOM results and than the GCT results from the other fibre types. This may be the result of differing suppressant effects of particles on chrysotile as discussed later or to difficulties in dust generation.

The fibre concentrations measured in both studies are generally consistent with those reported Davis (1978) for 100% asbestos dust clouds produced by Timbrell Dust Generator where between 275 and 975 fibres ml^{-1} were found in a respirable mass concentration of 5 mg m^{-3} (i.e. 2.75 and 9.75 for 0.05 mg m^{-3}, i.e. 1% of 5 mg m^{-3}). There was a progressive reduction in airborne fibre concentrations at a given dust concentration with reducing amounts of asbestos in the mixtures but this reduction was not proportionate to the reduction in asbestos content below 0.1%. With 0.1%, and often 0.01%, of asbestos in soils the 0.5 f ml^{-1} Control Limit for chrysotile and the 0.2 f ml^{-1} Control Limit for crocidolite and amosite (HSE, 1987) could be exceeded while respirable dust concentrations were below 5 mg m^{-3}, the nuisance dust OEL. Similarly it is apparent that the clearance limit of 0.01 f ml^{-1} could be exceeded with any of the 0.01% and 0.001% asbestos mixtures if respirable dust concentrations approached the nuisance dust OEL.

There are problems of fibre counting with these low asbestos concentrations which make correct assessment of the potential hazards very difficult. For individual IOM tests, samples of between 8 and 24 litres of air were collected because of the presence of large amounts of other mineral dust. The fibre concentrations were calculated from cumulative counts over 6-8 samples collected sequentially during each test. The cumulative counts would then provide reasonable assessments of the fibre concentrations down to about 0.01 fibres per ml. This is borne out by the good agreement between the two sets of tests and the generally good repeatability of the experiments.

The airborne fibre concentrations associated with the blank soils were always lower than those associated with the test mixtures with the exception of the mixture of 0.001% amosite in clay. These background fibre concentrations while contributing substantially to some of the fibre concentrations from 0.001% asbestos mixtures measured by optical microscopy had only a limited effect on the overall results of the study.

The electron microscope examinations carried out on the IOM tests confirmed that asbestos fibres were present in the dust clouds produced from the low concentration mixtures and that there were few asbestiform minerals in the blank soils. The non-asbestos fibres which accounted for substantial proportions of the respirable fibres found by EM in samples collected from mixtures with low asbestos content consisted largely of elongated clay particles or chains of particles. It is likely that many of these would not have been counted as fibres by phase contrast optical microscopy; not because they were discriminated against during counting but rather because they would not have been perceived as fibres at all (Plate 1).

5.2 The Effects of Different Soil and Asbestos Types

The effects of fibre type and soil type on the airborne fibre concentrations are minor in comparison to the bulk asbestos content. However, the natures of the fibre and of the soil do have a real effect. For a given asbestos concentration in soil it is predicted from the model that the airborne fibre concentrations could differ by factors of around 5 according to the asbestos type/soil type combinations being tested (e.g. chrysotile in clay in comparison to crocidolite in sand).

In considering all asbestos fibres it is apparent that the increasing clay mineral content does have an effect on the normalised airborne fibre concentration. This could be due to various factors. It may be that the proportion of respirable dust in the clay mixture was higher and, therefore, to achieve a 5 mg m^{-3} respirable dust cloud would require smaller amounts of bulk mixture thus reducing the airborne fibre concentration with respect to respirable dust concentration. Alternatively there could be a dust suppressant effect from the clay particles binding on to the fibres. This could either prevent the fibres from being made airborne or otherwise reduce the sampling efficiency of particulate coated fibres.

Both these factors appear to be important. It seems as if there is a genuine suppressant effect with the clay minerals, both from the similarity of the ratios of respirable to total dust concentrations from the different soils and from the fact that the effect is most marked with chrysotile which is the fibre type most susceptable to entanglement (Plate 2). The effect is also quite marked for mixtures of chrysotile in intermediate soils which contain 25% clay. On the other hand, it is difficult to explain the variations in normalised fibre concentrations from the amosite mixtures in terms of binding effects as amosite is much less susceptible to entanglement of this type (Plate 3) because of its surface, shape and other physical properties (Hodgson, 1965).

There are consistent effects on the normalised airborne respirable fibre concentrations from different fibre types in a given soil. Crocidolite almost invariably produces higher normalised fibre concentrations than does chrysotile while the position of amosite varies with soil type. This may reflect an inherent ability for crocidolite to generate more airborne fibres per unit mass or it may be a difference in the suppressent effects of clay particles binding to the three different asbestos types as mentioned earlier. Given the established differences in the surface properties of chrysotile and the amphibole minerals in general (Hodgson 1965) and the tendency for chrysotile to produce more and finer fibres than amosite at least it is suggested that the differences in the normalised fibre concentrations for the three asbestos types are largely the result of the differing suppressant effects of the clay minerals.

5.3 Dust Generation

The choice of dust generation method could influence the results. No single generation method could be considered as representative of the wide range work practices which may produce dust on a contaminated site. The method of dust generation used at IOM (Modified Timbrell Dust Generator)(TDG) was recognised as moderately aggressive in comparison to other methods but was selected because of its lower tendency to blockage by grit particles and because of the necessity to generate dust at relatively constant concentrations over a four hour period. It operates by advancing a plug of the loosely packed material down a hollow tube into a small cylindrical chamber inside which a rotating vane scrapes dust from the front of the plug. A compressed air feed then lifts the dust to the input pipe of the chamber. This beating action of the vanes may release fibres from binding particles more effectively than other dust generation methods thus increasing airborne fibre concentrations.

The main difficulty arising from the use of the TDG appeared to occur when the face of the advancing soil plug collapsed (because of its lack of physical strength) leading to an increase in dust and fibre concentration within the chamber followed by a gradual reduction as shown by the SIMSLIN records. These variations could arise at any point in the dust generation and could not be avoided without artifically binding the soils. In spite of these difficulties, the final dust concentrations measured over the four hour periods were still close to the target concentration of 5 mg m^{-3} for respirable dust.

The fact that the dust concentrations could increase by a factor of 4 over a short period during the blank soil tests could account for most of the variability observed in the individual sample fibre concentrations. Very large differences between individual sample fibre concentrations were observed during runs with low asbestos content mixtures. These were primarily associated with the large statistical errors associated with counting low density samples (Crawford *et al*, 1984). The use of continuous sampling for gravimetric dust concentrations and sequential sampling for airborne fibre concentrations with the use of time weighted averages did much to reduce the effects of such variations, providing more reliable estimates of the normalised fibre concentrations. This is borne out by the repeat tests where the differences between test runs were generally small.

The transient dust cloud generation method used at GCT was simple in comparison to that used at IOM, and the sampling strategy, involving a 30 minute delay after dust generation to allow the dust to settle, was very different. The fact that the normalised fibre concentrations from the two tests are similar indicates that there may be a general relationship between respirable dust and airborne fibre concentrations. It is therefore possible that the normalised airborne fibre concentration is independent of the type of dust generation method adopted but further research would be required to confirm this.

5.4 Effects of the Addition of Water to the Asbestos/Soil Mixtures

The results from the GCT study show that the airborne fibre concentrations generated from contaminated soils are greatly reduced by the addition of water to the soil. The amount of water required to reduce levels to a given value depends primarily on the amount of contamination of the soil and to a lesser extent on the type of soil.

Starting with initial fibre concentrations of more than 5 f ml^{-1} from dry soil, the fibre concentrations can be reduced to less than the clearance indicator of 0.01 f ml^{-1} by the addition of between 20 and 50% water.

The introduction of the first 5 or 10% water had a greater effect than subsequent additions. The differences observed in the progressive additions would require further research to explain them. However, given the effectiveness of addition of large amounts of water to the mixtures it is unlikely that this factor would be of practical importance.

The work with the dry asbestos/soil mixtures has shown that significant airborne fibre concentrations can be generated from soil contaminated with very small traces of asbestos. In practice this would mean that virtually any work functions on a contaminated site which generate dust could liberate airborne asbestos concentrations greater than the normal clearance indicator or even the control limits for occupational exposure. Spraying the contaminated soil with sufficient water prior to the work can suppress the generation of respirable asbestos fibres. In most cases the level would be reduced well below the control limits of 0.5 f ml^{-1} and 0.2 f ml^{-1} and, by suitable and continued water treatment, the level would be reduced to that of the clearance indicator. Whilst not suggesting that this method should be used in place of respiratory protection and accepted asbestos work methods, it clearly can be used in conjunction with normal practices to reduce risk on asbestos working sites and surrounding areas.

The benefits of water addition however are emphasised by recent studies carried out in the USA which have demonstrated that the protection offered by high efficiency respirators is considerably less than previously believed (Myers and Peach, 1983).

18. ISO 14966:2019. Ambient air – Determination of numerical concentration of inorganic fibrous particles – Scanning electron microscopy method

Full Reference: ISO 14966:2019. Ambient air – Determination of numerical concentration of inorganic fibrous particles – Scanning electron microscopy method. Second edition. ISO, Geneva

[Reference not provided]

19. MECP, 2020. Ontario's Ambient Air Quality Criteria

Full Reference: MECP, 2020. Human Toxicology and Air Standards Section, Technical Assessment and Standards Development Branch, Ontario Ministry of the Environment, Conservation and Parks (MECP). 2020.. MECP, Toronto, ON, Canada.

https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria#section-4

[Extract: page 8]

HUMAN TOXICOLOGY and AIR STANDARDS SECTION

TECHNICAL ASSESSMENT and STANDARDS DEVELOPMENT BRANCH

ONTARIO MINISTRY of the ENVIRONMENT, CONSERVATION and PARKS

AMBIENT AIR QUALITY CRITERIA

May 1, 2020

No.	CASRN	Contaminant	AAQC ($\mu g/m^3$)	Averaging Time	Basis	Notes
24	7784-42-1	Arsine	10	½-Hour	Health	
25	1332-21-4	Asbestos (fibres > 5 µm in length)	0.04 fibres/cm^3	24-Hour	Health	
26	7440-39-3	Barium - total water soluble	10	24-Hour	Health	
27	71-43-2	Benzene	0.45	Annual	Health	
27	71-43-2	Benzene	2.3	24-Hour	Health	Converted from the annual AAQC to allow assessment of 24-hour air quality data
28	50-32-8	Benzo(a)pyrene [as a surrogate of total Polycyclic Aromatic Hydrocarbons (PAHs)]	0.00001	Annual	Health	B[a]P is used as a surrogate for the total carcinogenicity of PAHs This AAQC does not apply to naphthalene (CASRN 91-20-3) nor for any other PAH for which an AAQC may be derived separately
28	50-32-8	Benzo(a)pyrene [as a surrogate of total Polycyclic Aromatic Hydrocarbons (PAHs)]	0.00005	24-Hour	Health	Converted from the annual AAQC to allow assessment of 24-hour air quality data
29	65-85-0	Benzoic acid	700	24-Hour	Health	
30	95-16-9	Benzothiazole	70	24-Hour	Health	

20. NDEP, 2015. Technical Guidance for the Calculation of Asbestos Related Risk in Soils for the Basic Management Incorporated (BMI) Complex and Common Areas

Full Reference: NDEP, 2015. Technical Guidance for the Calculation of Asbestos Related Risk in Soils for the Basic Management Incorporated (BMI) Complex and Common Areas. P Black, M. S Balshi, R Perona, Nepture & Company Inc., for Nevada Division of Environmental Protection, February 2015.

[Extract; pages 8-11]

Technical Guidance for the Calculation of Asbestos Related Risk in Soils for the Basic Management Incorporated (BMI) Complex and Common Areas

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February, 2015

A quality assurance/quality control (QA/QC) program should be specified in the Quality Assurance Project Plan (QAPP) to provide an appropriate level of assurance that the data collected during sampling events are both reliable and usable for decision making purposes. Data validation should be conducted to determine compliance of QA/QC measures and achievement of the project data quality objectives (DQOs), and Data Usability should be completed prior to using the data in an ARR. Criteria that should be included in the subsequent Data Validation Summary Report (DVSR) are provided in Appendix A. The data should not be used for ARR assessment unless these criteria are satisfied.

Site-specific DQOs should be specified to provide the basis for sampling design and analysis as well as describing how the data will be used for evaluating ARR. The DQO process (USEPA, 2006) is an iterative tool that ensures the systematic application of the scientific method to environmental problems. It is a seven-step planning process for data collection in support of site-specific risk management decisions. This allows for proper planning of the project, including the identification of the types and quality of data required for decision-making purposes. Additionally, the DQO process is an effective means for determining the necessary amount and quality of data needed to support decision-making. This directly affects the outcome of the risk assessment.

For the BMI Complex and Common Areas, there are often few or no asbestos fibers found in a samples or collections of samples, especially post-remediation. However, even when the number of fibers observed is zero the reasonable maximum exposure (RME) concentration of fiber counts, which accounts for uncertainty, is nonzero and can result in calculation of an unacceptable ARR. As described in Section 5.0 of this guidance, implementation of the DQO process can help by ensuring that the number of samples is sufficient that the uncertainty in the outcome does not drive an unacceptable ARR. The DQO process steps should be documented in a detailed sampling and analysis plan (SAP), which should be prepared to guide data collection activities that meet the project-specific DQOs.

4.0 Risk Characterization

As noted above, the formulation for asbestos risk calculations is different than for chemical risks. The following subsections provide a brief overview of some methods for estimating ARR. Formulae used for characterizing risk for a variety of potential receptors are also provided.

4.1 Potentially Complete Exposure Pathways

The two exposure routes by which asbestos intake can occur are ingestion and inhalation. Dermal absorption of asbestos fibers does not occur, although dermal adherence of fibers may lead to secondary ingestion or inhalation (USDHHS, 2005). Asbestos ingestion has also raised concerns in the scientific community with respect to association with gastrointestinal cancer, laryngeal and pharyngeal cancer, and renal cancer. However,

many of these disease endpoints could not be directly linked to a cancer endpoint because of insufficient data (NAS, 2006). The USEPA publishes a maximum contaminant level (MCL) drinking water standard for asbestos fibers with length >10µm of 7 million fibers per liter (http://www.epa.gov/safewater/contaminants/index.html). This MCL is based upon increased risk of developing benign intestinal polyps. However, there are no drinking water sources at the BMI Complex that are contaminated with asbestos.

The exposure route that poses the greatest risk to human health at the BMI Complex is inhalation. Inhalation of asbestos fibers can lead to lung carcinoma and malignant mesothelioma (Bourdes et al., 2000; Pira et al., 2005). Specifically, the exposure pathway of asbestos inhalation following suspension of asbestos fibers from soil is the focus of this asbestos risk assessment guidance.

Receptor exposure scenarios that are considered in this guidance are construction worker, off-site resident, on-site resident, and commercial / industrial worker. The methods by which ARR is estimated for these scenarios are described below.

4.2 Sampling and Analysis Methods

The methods used for surface soil sampling for asbestos are outlined in the Standard Operating Procedures (SOP) 12 section of the December 2008 version of the *BRC Field Sampling and Standard Operating Procedures, BMI Common Areas, Clark County, Nevada* document. This document outlines the procedures for the collection of grab samples for determining moisture and silt content, composite sample collection, and quality control sampling. Taken from SOP-12, the collection procedures at the BMI Complex and Common Area consist of:

> *"Each selected sampling location is to serve as the center of a 50 feet by 50 feet sampling grid, which is to be further divided into four quadrant grid squares that are each 25 feet on a side. Grab samples for determination of moisture and silt content are to be collected from the center of the overall sampling grid. Samples to be collected for determination of asbestos content are to be composites constructed from four component samples with one component collected from a pre-selected, random location from within each of the four grid squares (quadrants) of the sampling grid."*

The modified elutriator method (Berman and Kolk, 2000) provides bulk measurements of asbestos structures that can be used for the prediction of airborne asbestos exposure. This method is a modified version of an earlier USEPA method (USEPA, 1997) that was developed to improve performance and reduce analysis costs. Soil samples are placed in a dust-generator to separate and concentrate the respirable fraction of the sample. The respirable fraction is deposited on a filter, which is then prepared for analysis by microscopy. This modified elutriator method is referenced for the acquisition of soil asbestos data to calculate ARR in Berman (2003a; 2003b; 2005).

Three main forms of microscopy have been used for measuring asbestos: ordinary light microscopy (OLM); phase contrast microscopy (PCM); and transmission electron microscopy (TEM). OLM is the most limited method as there can be no distinction made between mineralogies or morphologies. OLM is generally limited to detecting particles that are much larger than those detected using phase contrast and electron microscopy, which makes it the least useful of the readily available methods.

In the 1980s, the USEPA developed an approach for assessing ARR (Asbestos Health Effects Assessment Update, USEPA, 1986), which assumes no differences between the potencies of different asbestos types (amphibole and chrysotile). At the time, the most likely analytical method used for asbestos analysis was PCM. Unlike OLM, PCM is able to measure smaller asbestos structures and also determine their shape. However, PCM can only measure particles greater than 0.25 μm in diameter and 0.5 μm in length. This can result in underestimation of narrow asbestos particles, which may be important for accurately quantifying asbestos cancer risk (Berman and Crump 2003; Berman and Crump 2008a and 2008b). It has been shown in previous studies that PCM significantly underestimates asbestos fiber concentration in air when compared to TEM, primarily because of poor resolution (Perry, 2004). Other limitations of PCM include the inability to distinguish between particle mineralogy and in some instances the inability to distinguish between asbestiform and non-asbestiform particles. Depending on the sample matrix, this inability to clearly identify only asbestos fibers could potentially result in overestimation of the concentration of asbestos present on a filter. The possibility of either underestimation from poor resolution, or overestimation from misidentification of non-asbestiform particles, causes PCM to be an inaccurate method for estimation of asbestos concentrations.

Unlike other analytical techniques used for asbestos analysis, TEM is able to distinguish different fiber mineralogies and is able to reveal fibers that are less than 0.01 μm in diameter. As a consequence, different fiber size classes of both amphibole and chrysotile asbestos can be differentiated. Used in conjunction with the cancer potency factors described in Berman and Crump (2003), NDEP recommends the use of TEM for asbestos analysis.

NDEP notes that distinction between asbestos structures and fibers are not made in this guidance. NDEP recognizes that asbestos structures are measured using TEM (for example), and that structures can consist of several fibers. ARR is generally based on measurement of structures rather than fibers, but the terms are used interchangeably in this guidance.

4.3 Exposure Concentration Estimation

Asbestos soil measurements derived using the modified elutriator method can be combined with dust emission and dispersion models, which can then be used for predicting airborne exposures and associated risks. The details and protocols for this method are described in detail in Berman and Kolk (2000), and examples are provided in Berman (2003a; 2003b; 2005). The USEPA Particulate Emission Factor (PEF) model is used to estimate annual average concentrations of respirable particulates (approximately

10 μm and less) in ambient air (USEPA, 2002). The suitability of these generic particulate emission and dispersion models for predicting concentrations of asbestos fibers in air that are longer than 10 μm is defended in Berman and Kolk (2000) by reference to a study of dust emissions from two roads surfaced with asbestos-containing serpentine material.

The PEF model has two components. The first component is an atmospheric dispersion term (Q/C_a) that relates air concentrations to particulate emissions from soil. The second component is a particulate emission model related to some specific mechanism of soil disturbance. The PEF is calculated differently depending on the activities related to the exposure scenario.

The factor Q/C_a reflects the site location, local climate, surface area of the site that is under investigation, and the mechanism of dust dispersion (wind or construction). The dispersion factor is defined in USEPA (2002; Appendix D) as:

[Eq. 1]

$$\frac{Q}{C_a} = A * \exp[\frac{(\ln(A_{site}) - B)^2}{C}]$$

where A, B, and C are curve-fitting constants (unitless) tabulated in USEPA (2002) and A_{site} is the areal extent of the site or site contamination (acres). The dust emission and dispersion models needed for the construction worker, offsite resident, onsite resident, and commercial / industrial exposure scenarios are outlined in the following subsections.

4.3.1 Construction Worker PEF

The most significant pathway of asbestos exposure to construction workers is by inhalation of fugitive dust from traffic on unpaved roadways and wind erosion of surface soil (USEPA 2002). Construction workers are adults who are generally exposed over a shorter (sub-chronic; between 2 weeks and 7 years) exposure period than residents and commercial / industrial workers. Two PEFs are calculated for this scenario (one for overall construction activities and one for activity on unpaved roadways), which are then used to estimate the total outdoor ambient air dust concentration. The following subsections break the construction worker PEF calculations into three separate parts: 1) sub-chronic PEF for construction activities, 2) sub-chronic PEF for general vehicle traffic on unpaved roadways, and 3) total sub-chronic construction related PEF. As described in Section 5.3.2 of USEPA (2002), dust emissions from unpaved road traffic "typically contribute the majority of dust emissions during construction." The equations in *Part 1* are provided for use at the discretion of site managers should dust emissions from these activities be of particular concern at a site.

Part 1: Sub-chronic PEF for construction activities

The first part of the PEF for construction workers is the sub-chronic PEF for construction activities (PEF_{sc}). This is calculated according to Equation E-26 of USEPA (2002):

21. NICOLE, 2021. Asbestos in soil: A pan European perspective

Full Reference: NICOLE, 2021. Asbestos in soil: A pan European perspective. Network for Industrially Co-ordinated Sustainable Land Management in Europe, June 2021.

[Extract: pages 25-31, 35-52]

ASBESTOS IN SOIL

A pan european perspective

NICOLE

Network for Industrially Co-ordinated Sustainable Land Management in Europe

Hobmoor School – Birmingham, UK | Google Maps

Ramboll was commissioned by Balfour Beatty Construction Limited to develop and implement an asbestos remediation strategy to enable the construction of a new school.

Previously developed as industrial land, the historic review and site visit established significant volumes of demolition rubble from prefabricated buildings across the site. The proposed development included landscaping, sports areas and

Frequently occurring fragments of asbestos cement and AIB were discovered

Asbestos finds | Ramboll

earthworks reprofiling. This meant significant cut and fill works across the site with soil containing demolition rubble.

Asbestos Containing Material (ACM) was encountered during site clearance, so a specialist survey contractor was commissioned for soil sampling and perimeter air monitoring. The asbestos detected in this survey was asbestos cement (chrysotile), asbestos insulation board (amosite) and found in the topsoil till a depth of 1,00-1,50 meters. The pollutant linkages identified during construction and operation were potential exposure to free fibres from friable materials from the asbestos cement and insulation board.

The remedial options appraisal included:

- Dig contaminated soil and dump on site in vegetation strip; costs over £800 000,
- Hand pick asbestos material, capping with imported top soil (0,3 meters) and install a marker layer between clean top soil and contaminated soil underneath; costs approximately £500 000,
- Assess the risks of in situ reusing the top soil.

Pockets of asbestos covered much of the site at depths up to 5m.

Asbestos finds—hand picking | Ramboll

Processing plant | Ramboll

Based on the options appraisal a bespoke methodology was developed and a comprehensive worldwide review of asbestos legislation and guidelines was undertaken. The final remediation strategy designed comprised of:

1. Hand picking of asbestos cement and asbestos insulation board fragments,
2. Trommel sieving of soil on a 14 mm mesh,
3. Air monitoring for fibres across the perimeter of the site and in the "Control Zone",
4. Works carried out by a licensed contractor with a HSE approved asbestos methodology.

A dust and fibre release experiment was designed to estimate the potential fibre release during school operation, which could be released by soil derived indoor dust. This was done by simulating a realistic and real time situation. For this a 12 m^3 sealed enclosure was built into the school with an air lock entry. The soil in the sealed enclosure was vigorously disturbed to generate dust. The indoor air was monitored and sampled. The samples were tested with Phase Contrast Optical Microscopy (PCOM) analyses.

The remediation delivered a screened top soil which was suitable for re-use in the landscape area

Indoor air experiment | Ramboll

without requirement of a cover layer. The worst case activities were simulated and tested and concluded no residual fibres and low residual risks. All air monitoring results were below detection limit of the standard HSE method i.e. <0.01 f/ml during the earthworks. And the air testing experiment (samples repeatedly disturbed) did not generate airborne fibre concentrations above limit of detection of the standard HSE method (<0.01 f/ml).

The new school is in place and the landscaping offers a nice area around it.

Before and after construction | Ramboll

10 Risk-Based Soil Guidelines

There are few published guideline values for asbestos in soil in Europe. Those that are published are summarised below:

Country/ Region	Guideline Value	Additional Information
The Netherlands	Tier 1: 100mg/kg Tier 2: 1000mg/kg (non-friable) or 100mg/kg friable Tier 3: 10mg/kg respirable fibres	Soil Remediation Circular 2013 Annex 3. Concentrations defined as the sum of chrysotile + x10 amphibole and as the average dry weight concentration over a maximum spatial unit of 1000m2. Samples to be taken and analysed as per SIKB Protocol 2018 and NEN 5707.
Italy	1000mg/kg	D.Lgs 152/06. Analysis required to be either SEM for asbestos content <1% or DRX/FTIR for asbestos contents >1%.
Belgium/ Flanders	100mg/kg	Phase 1—minimum of two 10 litre sieved soil samples per 1000m2 of unpaved ground. If concentration < 100mg/kg or >70cm bgl, no action required. If >100mg/kg, further site-specific inspection (Phase 2) required. Concentrations defined as the sum of fixed fibres + x10 loose fibres.
Belgium/ Wallonia	100mg/kg	Concentrations defined as the sum of bonded fibres + x10 unbound fibres. If concentration is > 100mg/kg but <500mg/kg it is acceptable to use soil beneath 1m clean soil + geotextile.
Belgium/ Brussels	100mg/kg Intervention Value 80mg/kg Remediation Value	If the results obtained for a sample exceed the intervention standard for asbestos or if there is a question of pollution (in the sense of art. 3 25° of the Soil Ordinance), a detailed soil survey must be carried out.

Table 10.1 Published guidelines in Europe

11 Approaches to Risk Management

Risk perception and stakeholder acceptance of a risk-based approach to asbestos is potentially a far stronger driver of intervention than for many other soil contaminants. Zero tolerance or an abundance of caution towards asbestos can drive remediation towards "non-detect" solutions.

There are well established risk assessment decision frameworks available, for example the Australian, US EPA, Dutch, and Belgian approaches. What is not well understood is how often those frameworks are used past "Tier 1".

Is the challenge to prove the worth of the more detailed risk assessment Tiers? Is the scientific evidence sufficient to be able to persuade stakeholders that the risk is acceptable? Does the retention of asbestos-containing soils on-site leave constraints on land-use that is not cost-beneficial? Detailed risk assessment has its place and can be valuable in situations where it is not possible and not sustainable to remove the asbestos entirely. This is illustrated in the decision flowchart on the next page.

The difference in the prescriptive nature and detail of frameworks for individual countries and the sustainability of the output from those frameworks is worth further consideration.

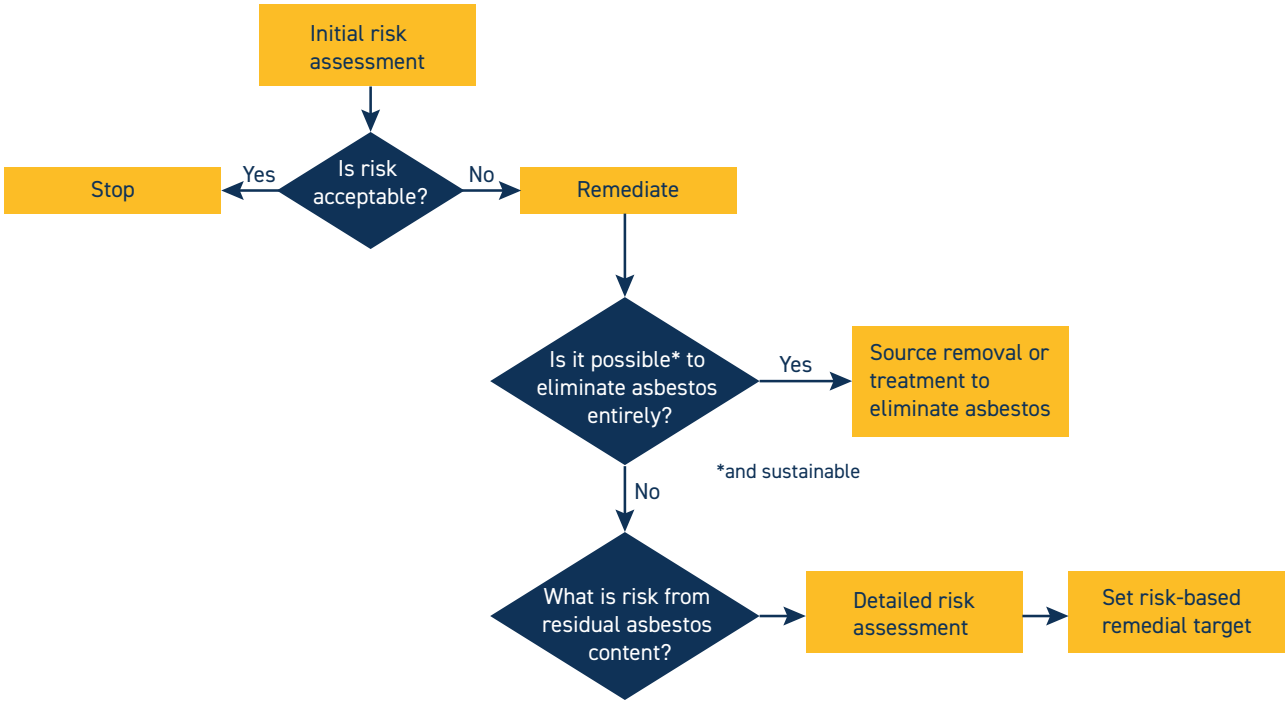


Figure 11.1 Approaches to Risk Management

13 Remediation Options

The most common remediation approach in many countries is still to “dig and dump” (i.e. excavate and dispose to an off-site landfill). A question is whether this is a sustainable approach? The risk is removed by removing the hazard (i.e. the source) but does the context of site use permit a lower impact solution?

The trigger for remediation is also different between countries. For example, mandatory testing for microscopic fibres in soil whenever a construction activity takes place versus action only if visible asbestos waste is encountered. In France, all road asphalt has to be tested for the presence of asbestos as part of any road improvement scheme.

From the questionnaire responses it is clear that there is substantial variation in remediation

Typical remediation earthworks activities in UK | AECOM

Damping down of stockpiled material with water spray | AECOM

triggers, in what restrictions and requirements the identified presence of asbestos introduces, and in the remediation standards enforced. Even if the value of the remediation standard appears at face value to be the same (for example for The Netherlands and Belgium), the detailed definition of that value is different.

What is generally recognised in the questionnaire responses is that the presence of asbestos in the ground can have a significant effect on land use and costs for remediation (either in the cost for remediating the asbestos itself as a risk and remediation driver, or in the additional cost for remediating a different risk driving contaminant because of the co-presence of asbestos).

There are a number of remediation options to consider, some more established than others. From a risk management perspective these options can be grouped as follows:

Monitor
- Risk assessment
- Monitoring strategy

Institutional Controls
- Land-use management
- Signs
- Fencing
- Permit control
- Land-use restrictions

Traditional Remediation Methods
- Excavation and disposal offsite
- In-situ containment (cover system)
- Hand-picking (ground or belt)
- Tilling
- Mechanical screening

Emerging/Innovative/ Alternative Methods
- Mechanical screening (advanced)
- Soil washing
- Vitrification
- ABCOV (acid destruction)
- Microwave destruction
- Modified low temperature thermal desorption
- Soil fungi
- Fine grinding
- Physical stabilisation
- Phytoremediation

The following scheme (next page) presents the risk management based considerations for the remedial options.

What is the context for the decision?	What is the risk characterisation?	Remediation options	Considerations for remediation options
Management of current situation (land condition and use)	Negligible risk and no regulatory driver for further action/intervention	Monitor	Monitoring locations and monitoring frequency Type of monitoring (realtime/continuous or spot monitoring, time duration, dust and/or fibres) Limit of detection and sensitivity of method (e.g. differentiation of fibre types and fibre sizes)
Regulatory intervention	Low risk - potential to manage risk without extensive remedial action	Institutional control	Is control of use/access of area practicable and achievable? Does it require reassurance boundary monitoring? Fencing, signage, specific PPE/RPE requirements
Preparation for site divestment/acquisition	Higher risk - requires more detailed consideration of remediation options	Remove	Can it be treated and re-used on-site? Can it be treated to reduce volume requiring disposal? Can it be treated to reduce handling/ transportation risk?
Preparation for site for new use		Cover	What level/degree of soil disturbance does this need to protect against? Durability. What ground access constraints are present which may restrict/constrain installation of cover (type, extent)?
Construction activity requiring asbestos containing soil to be excavated and/or constructed on		Ex-situ treatment	Treatment type - physical separation, chemical destruction, stabilisation. What is the required post-treatment specification for the material? What is the treatment capable of achieving?
		In-situ treatment	Treatment type - physical separation, chemical destruction, stabilisation. What is the required post-treatment specification for the material? What is the treatment capable of achieving?

Figure 14.2 Example of a Risk Management Decision Flowchart

John F Hunt demolished and remediated this former 44-acre foundry / iron works site in Ipswich. The mixed-use site also held two historic landfills containing inert and 'difficult' waste.

Part of the works involved the management of 35,000 m^3 of previously unidentified fibrous asbestos in soil. This unforeseen event had not been budgeted for and could have potentially rendered the project unviable. John F Hunt worked quickly and pragmatically with the client's consultants and regulators to agree a solution to enable the re-use of materials on site, making the necessary adjustments to the remedial design and Materials Management Plan.

An innovative process engineered approach of complex sorting and cement stabilisation of the

Futura Business Park – Ipswich, UK | John F Hunt

All forms of asbestos were discovered including crocidolite lagging.

Pockets of asbestos covered much of the site at depths up to 5m.

Asbestos finds | John F Hunt

soil was agreed with the regulators to derive site won engineered fill that was suitable for use.

Due to the nature of the asbestos, the remediation works were undertaken as Licensed Asbestos Works managed by John F Hunt.

Contaminated soil was fed into a three-way screener. The oversize material off the screener was proven to be suitable for re-use. The mid-size component was passed to an 'asbestos picking station' where six operatives hand removed visible asbestos products; in some instance the material was passed though the picking station twice to ensure the re-use criteria of <0.1% asbestos (w/w) was achieved. Fine material coming off the screener was passed to a mill unit where

2% cement was added. The stabilised fines were fed onto a stacking conveyor with misting sprays that deposited the material directly into the excavation.

Throughout the works the air was monitored by an independent Asbestos Analyst to demonstrate that the control measures were suitable.

The processed soil was tested to show compliance with the Remediation Strategy, following which it was placed and compacted to form a development platform 1.5m below the finished site level.

John F Hunt were able to successfully treat 65,000 tonnes of asbestos contaminated soil using innovative techniques that ultimately saved the client over £10,000,000 in disposal costs.

Processing plant | John F Hunt

A number of innovations in remediation have either been proposed and/or implemented by remediation specialists, as exemplified in some of the case studies included in this document and the listing of potential options on page 37. Innovation does not have to be a completely new technology, and can include the innovative use of an existing technology.

Examples of this include the use of:

- Cement impregnated geotextiles for cover systems (see photographs to the right)
- Low temperature driers or thermal desorption units to extract loose fibres by drying + extraction of airborne fibres
- Mechanical screening (dry and/or wet)

Installation of surface barrier geotextile | Curtis Barrier Intl

A comprehensive review of remediation technologies is provided in a report by Bureau KLB for the Dutch Ministry for Infrastructure and Water Management published in 2018. This was driven by the need to reduce the unsustainable volume of asbestos contaminated soils being disposed to landfill in the Netherlands.

Mechanical screening of excavated soil | AECOM

Remedial objectives can shape option choices. For example:

Remove ACM fragments and re-use remaining soil at depth on-site	Physical separation of ACMs using hand picking or mechanical screening?
Remove asbestos fibres and re-use remaining soil at surface on-site	Physical separation of fibres by soil washing or drying + vacuum extraction?
Treat soil + asbestos so that material is suitable for re-use	Stabilisation or fibre destruction technology?
Re-use on-site is not possible/ acceptable	Off-site disposal—can pre-treatment reduce cost by minimising hazardous waste volume?

Figure 13.1 Examples of choices for different Remedial objectives

Factors to consider in remedial selection can include:

- Types of asbestos present
- Levels of asbestos present
- Area / volume of impacted soil
- Timescales
- Client risk perception / avoid land blight
- Sustainability
- Presence of other contamination
- Current and/or proposed land-use
- Site location (and proximity to receptors)
- Occupational health constraints
- Remediation standard required
- Other requirements for soil (e.g. geotechnical)

Removing asbestos contaminated soil | NTP

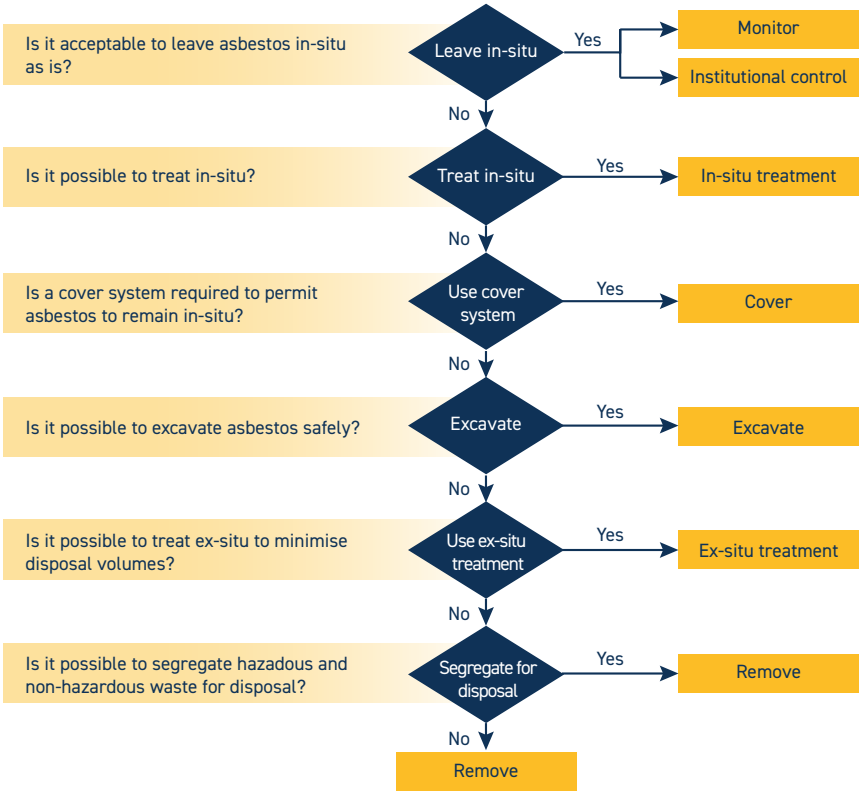


Figure 13.2 Example of a Remediation Decision Flowchart

14 Sustainable Remediation

Trommel screening of excavated soil | McAuliffe

Asbestos in soil remediation options should be considered in accordance with sustainable remediation frameworks (e.g. SuRF). Does the remediation approach represent the best solution when considering environmental, economic and social factors as agreed with stakeholders? How can successful remediation best be achieved with minimal environmental impact? What remedial solution delivers the greatest cost-benefit? Does the selected approach transfer impacts to future generations?

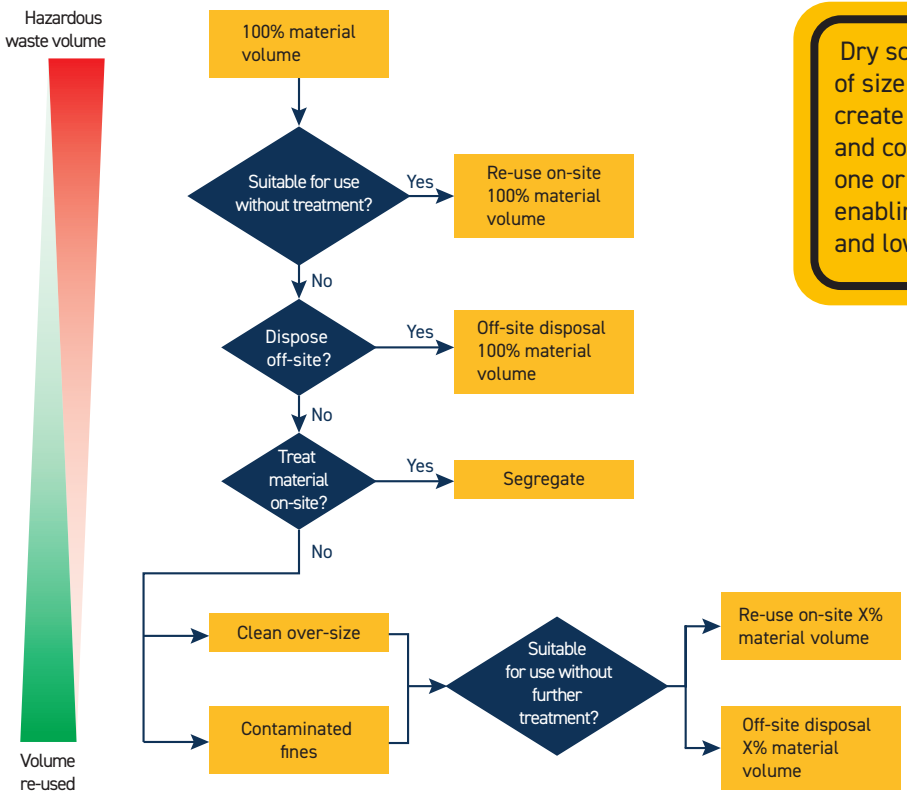
A simple example is the consideration of on-site physical separation to maximise the re-use of

Belt-picking station | McAuliffe


Hand picking of asbestos fragments on a belt | McAuliffe

material on-site and minimise off-site waste disposal. One way of viewing this is via a decision flowchart such as the examples on the following pages which illustrate the decision process and disposal volume reduction created by the adoption of mechanical separation treatment techniques. The use and sequencing of the material screening techniques will be influenced by a number of factors including:

- Cost of treatment versus cost of disposal
- Particle size distribution of material
- Remediation standard



Dry screening and separation of size fractions could create clean size-fractions and concentrate asbestos in one or more size fractions, enabling re-use of some material and lowering disposal volumes

Figure 14.1 An example of a treatment decision process for dry screening as a sustainable option

AECOM developed a remediation and excavated materials management strategy for the redevelopment of a former car part manufacturing facility located in the UK.

The presence of soil contaminants necessitated a remediation and earthworks strategy that had sustainability at its core: maximising reuse of site-won material, and minimising off-site disposal whist at the same time providing a safe development platform. The remediation strategy sought to first treat organic-based contamination through ex-situ bioremediation. Alongside the remediation works, an excavated materials management plan (MMP) was developed under the CL:AIRE Definition of Waste: Development Industry Code of Practice (Code of Practice) to support the earthworks design. Demolition of the former buildings and hard standing occurred alongside the soil remediation under separate contract by a third party. Four stockpiles of screened demolition materials (approx. 26,500 m^3) were prepared for re-use. However, these materials were subsequently found to contain a proportion of asbestos containing materials (ACM) which had in places also contaminated the ground as the stockpiles had been moved around by the contractor.

Asbestos finds | AECOM

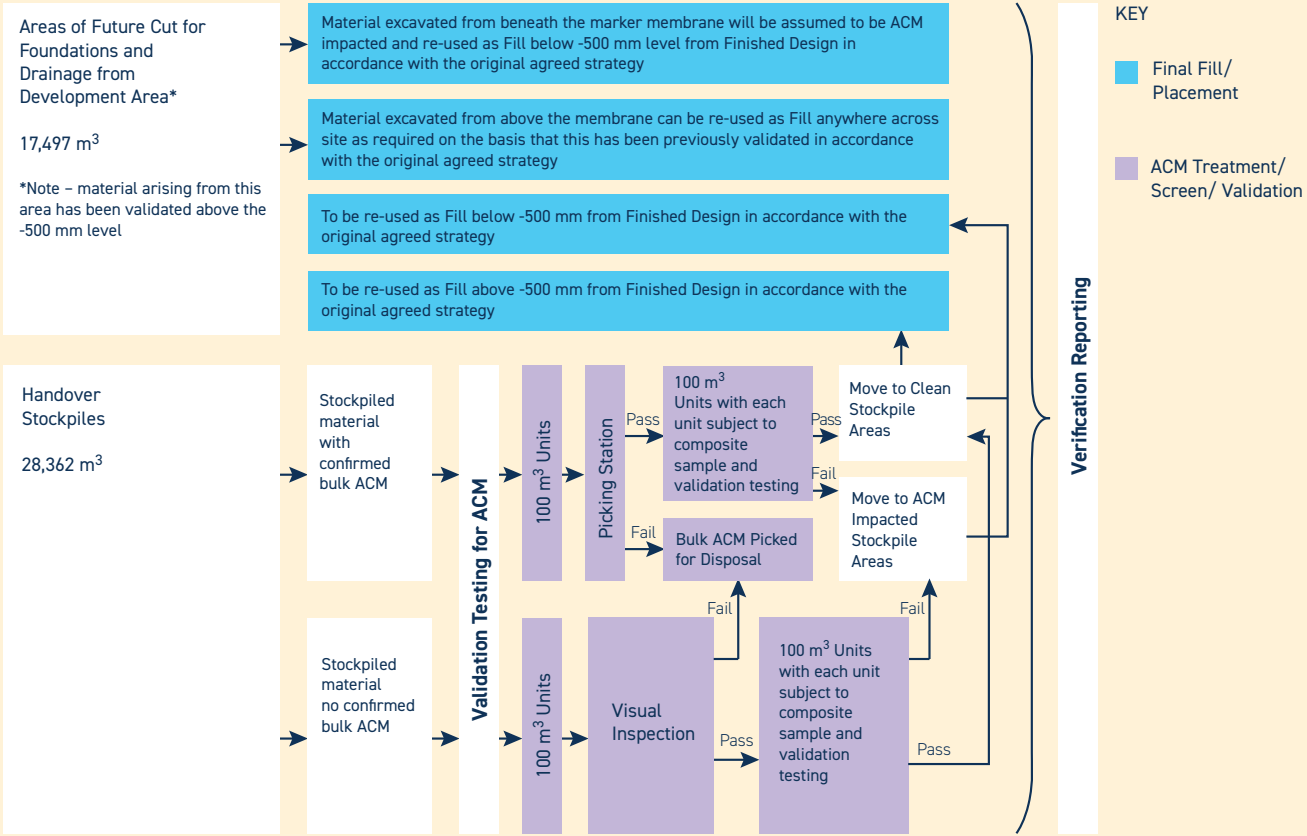


Figure C2.1 Material Management Flowchart

In order for the stockpiled materials to be re-used as part of the consented design a revised strategy was required to ensure the appropriate and safe re-use of these materials. AECOM prepared a detailed assessment on the levels of ACM and asbestos free fibres recorded in the materials and also quantified the level of risk posed by the materials. The soil re-use strategy was developed in accordance with the Control of Asbestos Regulations (2012) and the HSE Approved Code of Practice for managing and working with asbestos (ACoP L143) and gained regulatory agreement.

The strategy developed for the areas of impacted ground centred on a minimum of 500mm validated clean cover being placed below finished design level with the installation of a geotextile marker membrane at the interface of the clean cover and existing ground level. The strategy also made provision for selected 6F2 (UK highway's grade of aggregate) stockpiles impacted with asbestos to be

Installation of the cover system | AECOM

treated through mechanical screening, sorting and hand picking to generate screened material that met agreed validation criteria (<0.001% asbestos). The mechanical screening successfully separating the larger size fractions that were free of asbestos from the smaller size fractions where the asbestos tended to be. The treated larger size fractions could then be recrushed to produce graded material suitable for use in the development without restriction. Stockpiles that were not treated were tracked and used in dedicated areas of the development under 500mm of clean cover with geotextile marker membrane. In areas where soils containing ACM were placed beneath cover, the strategy set out the principles and expectations for a future site management strategy that would need to be adopted upon completion.

The approach taken at this site ensured that the excavated and site-won materials were managed sustainably on site, minimising potential off-site disposal and material import consistent with the original design aspirations and expectations attached to the planning consent.

22. RIVM, 2003. Assessment of the risks of soil contamination with asbestos

Full Reference: RIVM, 2003. Assessment of the risks of soil contamination with asbestos, F.A Swartjes, P.C Tromp, J.M Wezenbeek, RIVM report 711701034/2003

[Provided in full as a Core Document]

23. SCA, 2017 (now withdrawn). The Quantification of Asbestos in Soil and associated materials

Full Reference: SCA, 2017 (now withdrawn). The Quantification of Asbestos in Soil and associated materials (2017), Methods for the Examination of Waters and Associated Materials. Standing Committee of Analysts, April 2017.

[Extract: pages 16-18]

Standing Committee of Analysts

The Quantification of Asbestos in Soil (2017)

Methods for the Examination of Waters and Associated Materials

A1 The quantification of asbestos in soils and associated materials

A1.1 Scope

This method describes the quantification of the mass of asbestos in soil, construction materials/products, or associated materials. The method uses a gravimetric method for ACM and fibre bundles, plus dispersion and fibre counting for free fibres using Phase Contrast Microscopy, including calculations for the concentration of Total Fibres and Potentially Respirable Fibres, as appropriate.

Asbestos may be present in the form of different types of ACM, fibre bundles, or individual (free dispersed) fibres. This method seeks to address as wide a range of materials (matrices) as possible, initially by weighing the fragments of different ACMs and fibre bundles, and expressing their presumed asbestos content as a percentage by mass. The free fibres are dispersed and then filtered, prior to measurement and counting. These can also be expressed as a percentage by mass of the sample. The sum of the two results provides a quantitative measure of the total mass of asbestos in the sample expressed on a dry weight basis. The results may also be broken down and presented in a more detailed form, depending up the requirements of the client, to include type of asbestos fibre, type of matrix, respirable fibres, and percentage by mass of each type/fraction.

A1.2 Performance Characteristics of the Method

A1.2.1	Substances determined	Asbestos: chrysotile, crocidolite, amosite, and the asbestos forms of actinolite, anthophyllite and tremolite
A1.2.2	Type of sample	Ideally, approximately 1- 2 kg of soil or associated materials (1 litre tub)
A1.2.3	Basis of method	Visible fragments of ACM and fibre bundles are removed and determined gravimetrically, with free fibres dispersed, filtered, and measured and counted using PCM, if appropriate. The sum of the two results is calculated as % by weight of the original dried sample, plus the detailed composition may be reported, when required.
A1.2.4	Range of application	Gravimetric: 100 - 0.001 % Free fibres: 0.1- 0.001 %
A1.2.5	Calibration curve	Not applicable
A1.2.6	Standard deviation	Gravimetric: 15 % Free fibres: 30 %
A1.2.7	Limits of quantification	Gravimetric: 0.001 % Free fibres: 0.001 %

A1.3 Principle

A1.3.1 Stage 1 Identification of asbestos according to HSG248 (2016 draft)

The whole sample is examined visually inside a safety cabinet to identify if any visible suspect ACM are present, using the method stipulated in HSG248 (2016 draft). If no suspect asbestos containing materials are visible, a representative subsample (20 – 50 g) is collected in a petri dish for examination by stereo microscopy (x20 – x40). If no asbestos materials are found during this analysis, several small representative subsamples are examined on a microscope slide in a suitable RI medium and analysed at a higher magnification (x80 – x500) using PLM/PCM techniques, according to HSG248 (2016 draft). If no asbestos fibres are identified, the sample can be reported as No Asbestos Detected (NAD). If after careful searching, only 1 – 2 fibres/bundles are found, this can be reported as 'asbestos found at the limit of detection' (HSG248 (2016 draft) *, and it may not be necessary to continue with quantification. Asbestos fibres above this level should be identified before continuing with Stages 2 and 3 of the quantitative analysis. The oven dried weight of the sample is also determined, although the visual examination can be performed on the as-received or dried sample, depending upon suitability of the sample.

** This definition of trace is likely to change in final published version of HSG248*

A1.3.2 Stage 2 Gravimetric analysis

The whole as received sample is weighed and examined visually inside a safety cabinet. The oven dried weight of the sample is also determined – the visual examination can be performed on the as-received or dried sample.

Any items and/or fragments that may potentially contain asbestos fibres are identified, and a representative sample of each ACM type is removed from the sample for further identification, where suspect ACMs and fibres/fibre bundles are examined using stereomicroscopy and Polarised Light Microscopy to confirm the presence and type of asbestos as described in HSG248.

Materials confirmed as containing asbestos are removed, grouped according to material/asbestos type, weighed, and the mass percentage of ACM/fibre content of each material/asbestos type is calculated. For the purposes of grouping similar ACMs, analysts may assume asbestos fibre type based on the prior identification of representative similar materials in Stage 1. The overall asbestos content of the sample is based on the maximum asbestos content of the specific ACM types found as per HSG264 (See Appendix 4 in this document).

Once visible suspected ACM are removed, a representative sub-sample (20 – 50 g) of the remaining material should be selected by coning and quartering. This sub-sample is given a very detailed examination under stereomicroscopy (x40 - x 80) and any further smaller pieces of suspected ACM and or asbestos fibre bundles are removed for identification and weighing using a suitably sensitive balance. The mass percentage of each ACM/asbestos type in this fraction of the sample is calculated by expressing the weight of the recovered asbestos as a percentage of the dry weight of the sub-sample selected for detailed analysis. The overall asbestos content of the sample is then calculated.

If multiple (more than several) free dispersed fibres/bundles are identified as present,

then the Stage 3 dispersion/identification/counting method should be performed to ascertain the percentage of fibres, and/or the concentration of respirable fibres, present in the sample.

A1.3.3 **Stage 3 Free/dispersed fibre analysis**

Following the gravimetric method, a representative subsample (1 – 5 g) of the residue is weighed into a conical flask, and water added in the ratio of 1:200 solid to liquid (volume dependent on sample type). The suspension is mixed vigorously for a minimum of 30 seconds to ensure complete dispersion, allowed to settle for 10 seconds, and then a known quantity is filtered through a cellulose-ester filter (0.8 – 1.2 microns). The filter is then placed onto a microscope slide, allowed to dry, and then cleared and fixed using the acetone/triacetin method described in HSG248, (2016 draft). The slides are then evaluated using PCM/polariser/red tint plate to discriminate and quantify the asbestos fibres. From the number and size of the potential asbestos fibres observed on the slides the mass percentage of asbestos in the sample is estimated. The relative contribution to the overall mass percentage from presumed amphibole and serpentine asbestos may also be estimated.

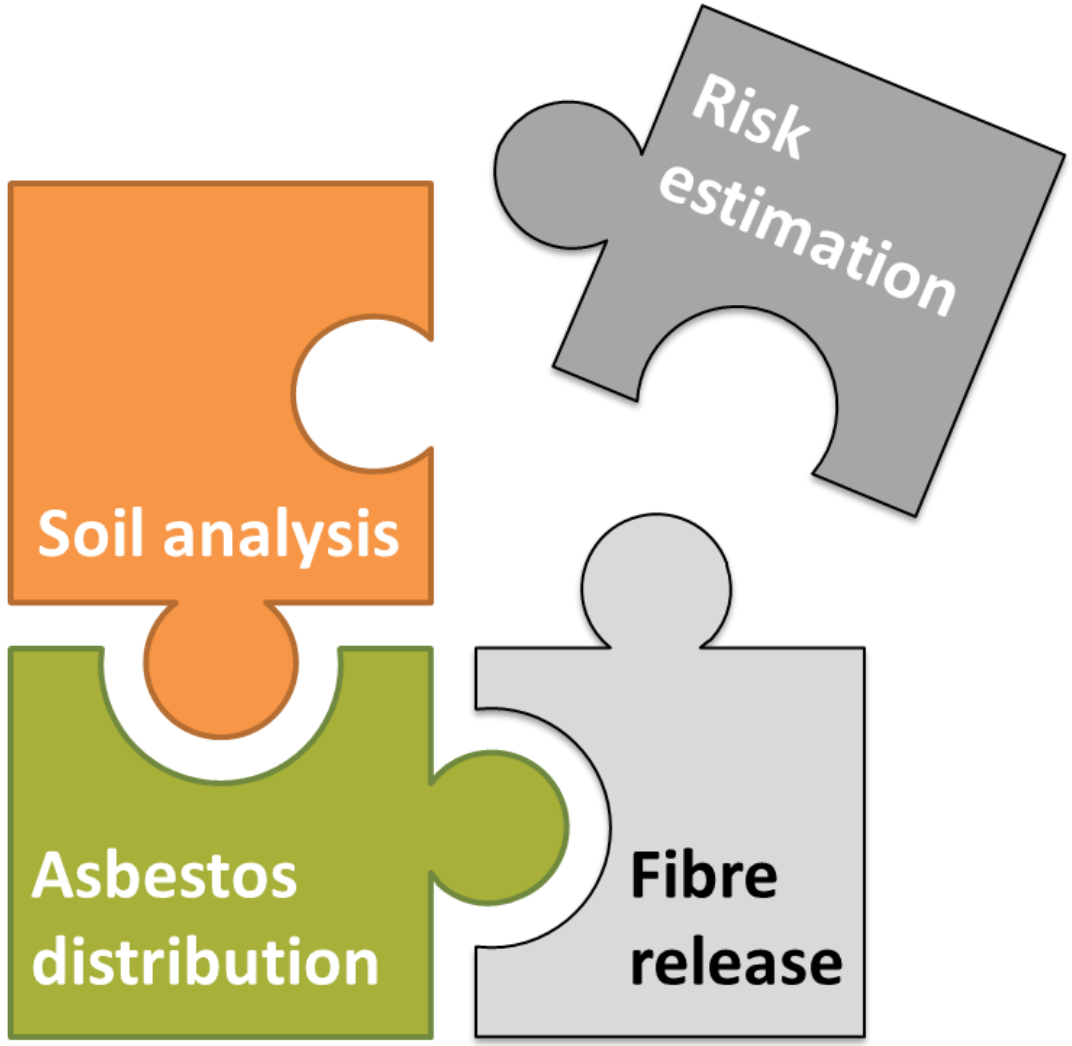
The sum of both ACM/visible fibres and free fibres should also be reported as the % asbestos content in the original sample on a dry weight basis.

24. SoBRA, 2021a. SoBRA Asbestos in Soil Human Health Risk Assessment (AiSHHRA) Toolbox

Full Reference: SoBRA, 2021a. SoBRA Asbestos in Soil Human Health Risk Assessment (AiSHHRA) Toolbox, SoBRA Asbestos Sub-Group, Society of Brownfield Risk Assessment, December 2021.

[Extract: slides unnumbered]

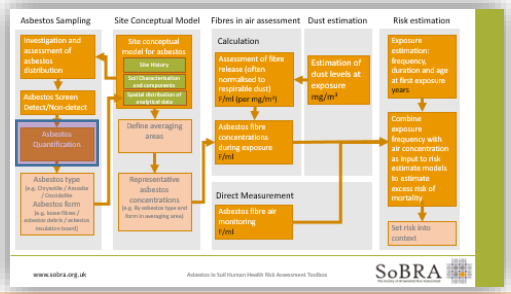
SoBRA Asbestos in Soil Human Health Risk Assessment (AiSHHRA) Toolbox

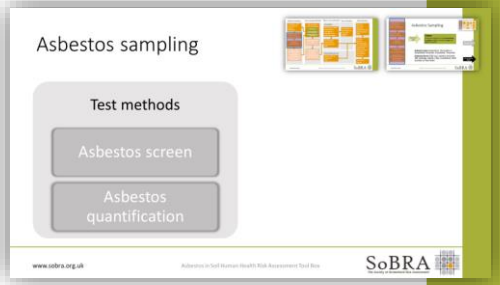


SoBRA asbestos sub-group

December 2021

Asbestos Quantification





Description: Stage 2 of the SCA BBM: The removal of asbestos containing material (ACM) and fibre bundles with identification and gravimetric analysis to determine percentage by weight.

Stage 3 of the SCA BBM: The dispersion and collection of free fibres followed by fibre identification, counting and measurement of fibres to determine percentage by weight (undertaken when the asbestos is not suitable for gravimetric quantification – if no ACM or fibre bundles are detected but free fibres are identified).

The sample size inspected at Stage 2 varies between laboratories ranging from the entire 1kg to a subsample of 100g or less; the majority of laboratories use the same sub-sample from Stage 2 for Stage 3.

Further information: Health and Safety Executive. (2021) Asbestos: The Analysts' Guide. HSG248 2nd edition May 2021. https://www.hse.gov.uk/pubns/books/hsg248.htm

SCA Blue Book (now withdrawn) https://www.claire.co.uk/home/news/59-sca-blue-book-method-the-determination-of-asbestos-in-soil-and-associated-materials-consultation-draft-now-available

SoBRA discussion paper on laboratory methods https://sobra.org.uk/?pmpro_getfile=1&file=2021/02/SoBRA-paper-on-laboratory-test-methods_Dec2020_final-for-publication&ext=pdf

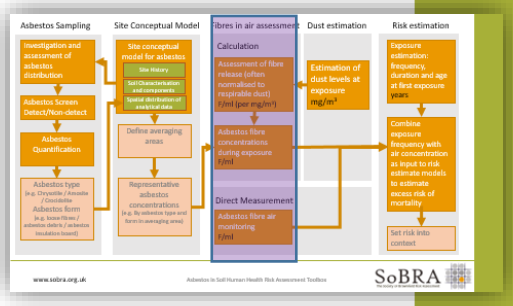
SoBRA comment: The current limits of quantification reported are typically 0.001 % w/w and the asbestos content is typically reported on a dry weight basis. It is rare that the quantified amounts are attributed to individual asbestos types or forms – a significant limitation in the reported results.

Be aware that where asbestos content is reported for an asbestos containing material, the "book value" for the percent of asbestos present in the material is often not consistent between laboratories. Also be aware that laboratories use different particle sizes for the Stage 3 analysis.

Recommendations for the reporting of Stage 2 and Stage 3 results are provided in the SoBRA discussion paper.

Asbestos in soil Output

Fibres in air assessment – Calculation



Calculation

Assessment of fibre release (often normalised to respirable dust)
F/ml (per mg/m³)

Asbestos fibre concentrations during exposure
F/ml

Input dust concentrations from Dust Estimation

Asbestos in Soil Dust Estimation from site data and CSM

Use to calculate cumulative exposures

Output

Fibre release x Dust concentration
OR air monitoring results

Weighted average asbestos concentration for each asbestos type and each zone (if defined)
f/ml

Assessment of fibre release

Asbestos air monitoring

Dust estimation

Risk estimation

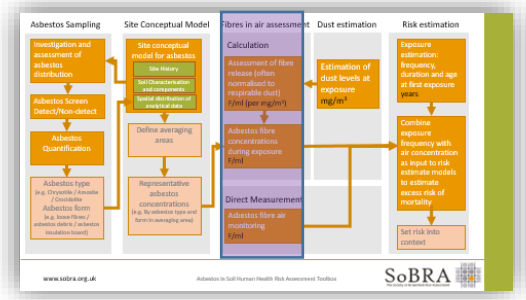
Direct Measurement

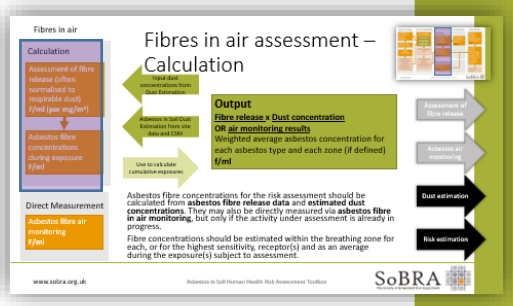
Asbestos fibre air monitoring
F/ml

Asbestos fibre concentrations for the risk assessment should be calculated from **asbestos fibre release data** and **estimated dust concentrations**. They may also be directly measured via **asbestos fibre in air monitoring**, but only if the activity under assessment is already in progress.

Fibre concentrations should be estimated within the breathing zone for each, or for the highest sensitivity, receptor(s) and as an average during the exposure(s) subject to assessment.

Assessment of fibre release





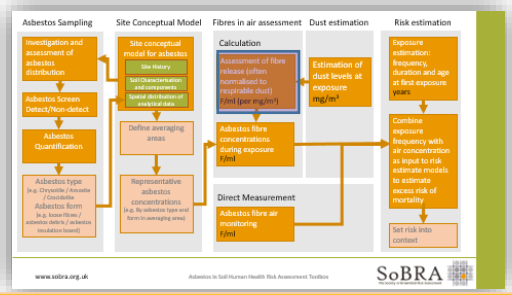
Test methods

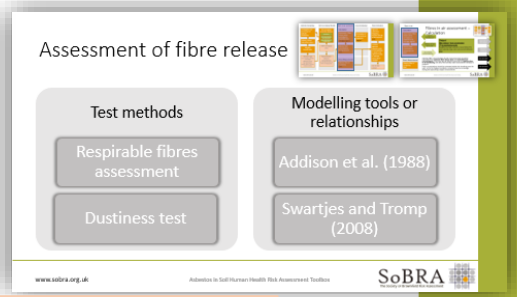
- Respirable fibres assessment
- Dustiness test

Modelling tools or relationships

- Addison et al. (1988)
- Swartjes and Tromp (2008)

Respirable fibres assessment





Description: Commonly used laboratory analytical methods (including the Blue Book) do not report respirable fibre concentrations as standard. Gravimetric analysis in particular does not distinguish between respirable and non-respirable fibres.

Respirable fibre assessment is a defined step in the Dutch (Ministry of Housing, Spatial Planning and the Environment (VROM)) guidance (Tier 3) and a small number of UK laboratories offer respirable fibre quantification as an optional additional step in asbestos quantification analysis. Typically this is reported as respirable fibres per gram of soil, and can be reported for a specific size fraction of soil if required.

Further information: Dutch Soil Remediation Circular 2013 Annex 3 [Note: this is now difficult to find online in English – this link provides information on the Circular but not access to the Circular itself] https://rwsenvironment.eu/subjects/soil/legislation-and/soil-remediation/

Commercial laboratory test method information

SoBRA comment: This can be an advantageous additional step if it is believed that a large percentage of the detected asbestos fibres are not respirable.

It can allow calculations to be done to estimate the potential respirable fibre concentrations in airborne dust if the test is done on the dust (e.g. PM_{10}) size fraction of the soil, thus either avoiding or providing an independent line of evidence to support the results of soil to air release estimates that are based on %wt/wt soil results.

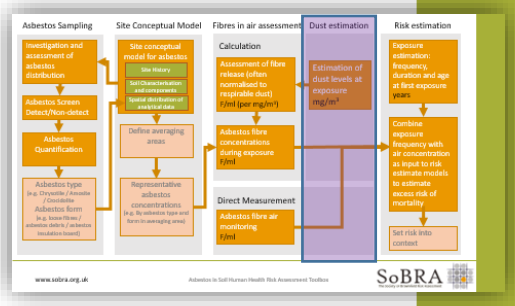
Like all risk assessment approaches based on soil sample data the assessment is only as good as the data on which it is based. This step is best done when robust data sets are available rather than at preliminary investigation stages.

The assessment requires a good understanding of whether the generation of additional respirable fibres is possible in the future as a result of future ground disturbance. Do the respirable fibre concentrations reported now represent what land users might be exposed to in the future. It is therefore of potentially greatest benefit when only loose free fibres have been detected.

Respirable fibres Output

Estimation of dust levels at exposure **mg/m³**

Dust estimation



Output dust levels for use Fibres in air assessment – calculations

Output

Average or worst-case dust concentration **mg/m³**

Respirable dust portion only

Highly dependent on moisture content and wind levels

Multiple values could be used in the assessment for a **sensitivity analysis**

Sense check calculated values or model results with published values

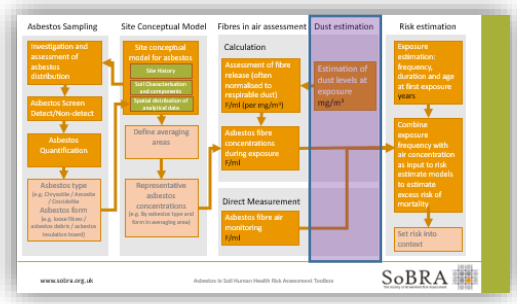
Dust concentrations should be estimated within the breathing zone for each receptor, or for the highest sensitivity receptor(s), and as an average during the exposure(s) subject to assessment.

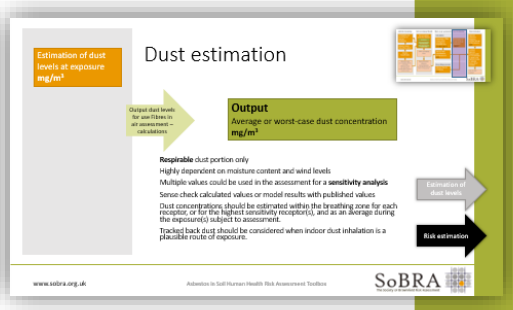
Tracked back dust should be considered when indoor dust inhalation is a plausible route of exposure.

Estimation of dust levels

Risk estimation

Estimation of dust levels





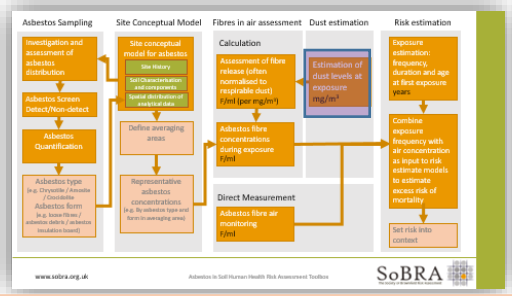
Modelling tools or relationships

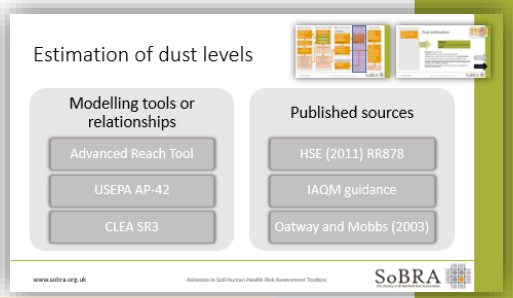
- Advanced Reach Tool
- USEPA AP-42
- CLEA SR3

Published sources

- HSE (2011) RR878
- IAQM guidance
- Oatway and Mobbs (2003)

USEPA AP-42





Description: AP-42: Compilation of Air Emissions Factors, is the primary source used by the US EPA for information concerning emissions factors, developed and compiled from test data and engineering estimates. Chapter 13.2 of AP-42 covers fugitive dust sources, estimating dust emissions from the disturbance of granular material, and consequently is the most relevant section of AP-42 for considering emissions from brownfield activities. The two main physical actions involved in this process are the pulverisation and abrasion of surface materials by the application of mechanical force, and the entrainment of dust particles by the action of turbulent air currents. The most relevant sub-sections area likely to be those on emissions from heavy construction operations, aggregate handling and storage piles, unpaved roads, and industrial wind erosion.

Further information:

AP42 https://www.epa.gov/air-emissions-factors-and-quantification/ap-42-compilation-air-emissions-factors

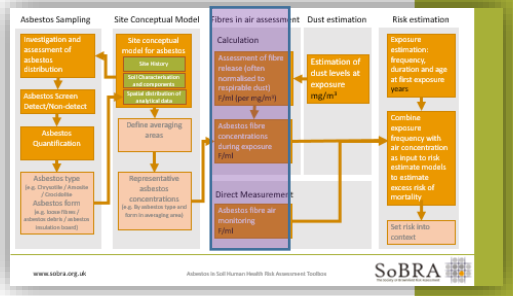
SoBRA comment: The AP-42 guidance is detailed, and is "tried and tested" having been originally published in the 1972 and last updated for Chapter 13: Miscellaneous Sources in 1995 and 2006. The algorithms for fugitive dust emissions allow for site-specific characteristics to be taken into account (such as material moisture content and particle size distribution). The guidance does not cover individual manual activities (rather focusing on larger site-wide mechanical activities), and care needs to be taken on parameter units (US imperial).

Dust Estimation Output

Direct Measurement

Asbestos fibre air monitoring
F/ml

Fibres in air assessment – Direct Measurement



Output

Air monitoring results

Weighted average asbestos concentration for each asbestos type and each zone (if defined)
f/ml

Assessment of fibre release

Asbestos air monitoring

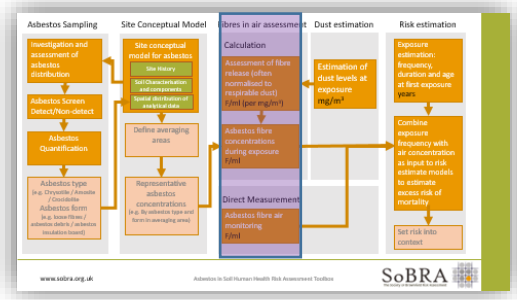
Dust estimation

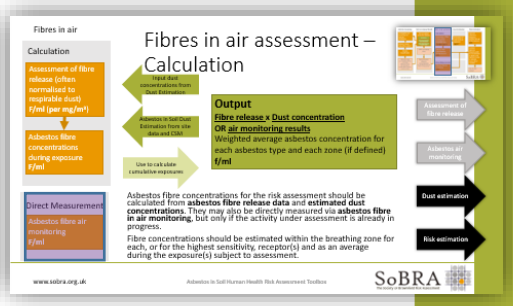
Risk estimation

Asbestos fibres may also be directly measured via **asbestos fibre in air monitoring**, but only if the activity under assessment is already in progress or by using activity based sampling methods.

Fibre concentrations should be estimated within the breathing zone for each receptor, or the highest sensitivity receptor(s), and as an average during the exposure(s) subject to assessment.

Direct measurement





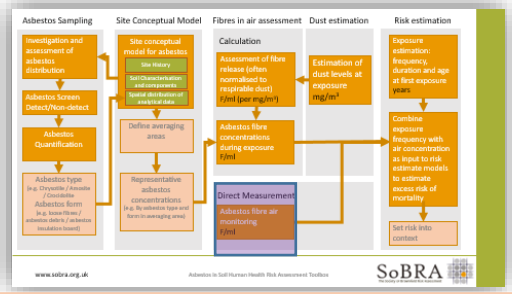
Air monitoring methods

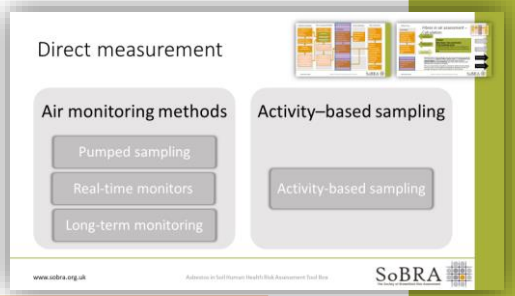
- Pumped sampling
- Real-time monitors
- Long-term monitoring

Activity–based sampling

- Activity-based sampling

Pumped Air Sampling





Description: Direct air measurement is achieved by collecting fibres on a filter by drawing a known volume of air through a pump. The filter is then examined by one of three different types of microscopy to count the airborne fibres over a specific area of the filter (graticules).

The principal guidance for the measurement of asbestos fibres in air is the HSE publication HSG248. This requires fibres to be counted if they are >5µm long and <3µm wide and length to width ratio of >3:1 using phase contrast microscopy (PCM). The limit of detection of the analysis can be reduced by sampling for a longer period of time, using duplicate or triplicate pumps, and/or by counting more graticule areas. Alternative scanning electron microscopy (SEM) or transmission electron microscopy (TEM) methods can also be used.

Further information: Health and Safety Executive, (2021). Asbestos: The Analysts' Guide. HSG248 2nd edition May 2021. https://www.hse.gov.uk/pubns/books/hsg248.htm

SoBRA (2015). Airborne Asbestos Fibre Monitoring Protocol for Earthwork Activities at Brownfield Sites. https://www.claire.co.uk/projects-and-initiatives/asbestos-in-soil?start=4

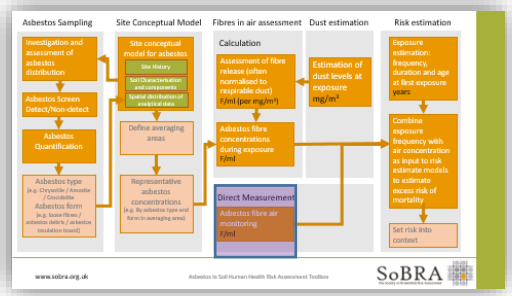
SoBRA comment: Measurement in accordance with HSG248 can result in fibres being counted but the concentration being reported as being below the method limit of quantification (typically 0.01f/ml – equivalent to 10,000 f/m^3). Below the limit of quantification does not in this case indicate an absence of asbestos fibres in air.

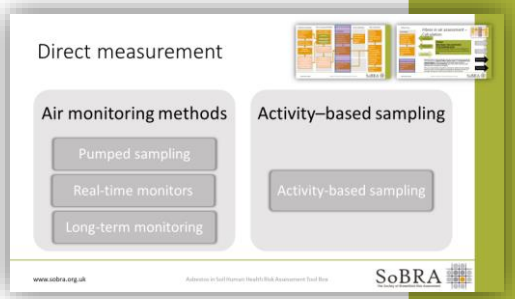
PCM alone cannot distinguish between asbestos and non-asbestos fibres (such as plant fibres). Electron microscopy analysis is required to positively confirm the presence of asbestos fibres following routine PCM evaluation if it is uncertain whether all fibres are likely to be asbestos. Duplicate sampling or splitting of sample filters is required for this. Note that because of the differing ability to identify asbestos fibres using the different microscopy techniques, measurements from each are not directly comparable.

Increasing sampling time can cause dust to obscure the filter thus preventing the counting of fibres present. The sampling approach therefore needs to be a careful balance of required limits of detection and predicted dust levels and the selection of the appropriate sampling equipment and analytical method. A protocol for air sampling is presented in the SoBRA (2015) Airborne Asbestos Fibre Monitoring Protocol for Earthwork Activities at Brownfield Sites.

Direct measurement Output

Long term air monitoring





Description: Long term low level asbestos in air monitoring may be required in certain circumstances, depending on the outcome of the asbestos in soils risk assessment, remedial works and any regulatory requirements. Such air quality monitoring is typically for reassurance purposes and potentially may be undertaken during one or more phases of the intrusive investigation, remediation or ongoing groundworks during development. It may be undertaken on-site or off-site.

Where monitoring is not for occupational exposure purposes and is to be used to support a risk assessment for future / current site use in relation to proposed or existing developments, it may be beneficial to report to 10 f/m^3 (0.00001 f/ml) using fibre-discriminatory SEM or TEM analysis. To achieve a lower detection limit, increased sampling flow rate and volume will be required.

Further information: SoBRA (2021). Discussion Paper on Guidelines for Airborne Concentrations of Asbestos Fibres in Ambient Air: Implications for Quantitative Risk Assessment. https://sobra.org.uk/resources/reports/

Nathanail, C P, Jones, A, Ogden, R, Robertson, A (2014). Asbestos in soil and made ground: a guide to understanding and managing risks (CIRIA C733).

SoBRA Airborne Asbestos Fibre Monitoring Protocol for Earthwork Activities at Brownfield Sites. (2015). https://www.claire.co.uk/projects-and-initiatives/asbestos-in-soil?start=4

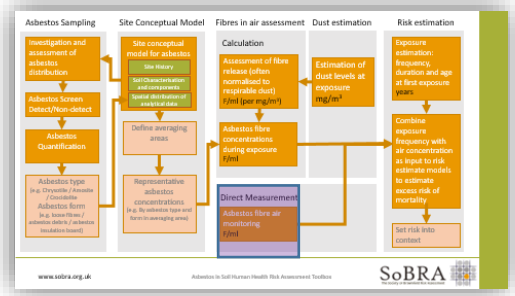
SoBRA comment: Prior to undertaking any long term air monitoring, the methodology of and rationale for the monitoring should be clearly documented, alongside the guidelines values to be used as trigger levels for any intervention. Background concentrations of asbestos in air may be required to benchmark the practicability of the proposed trigger levels for the monitoring.

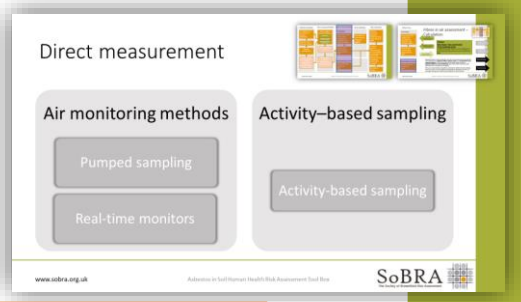
SEM and TEM analysis are more expensive than PCM (which gives a total fibre concentration rather than an asbestos fibre concentration) and currently only offered by specialist laboratories.

PCM, SEM and TEM results are not directly comparable and conversion factors are required. This is discussed in the SoBRA (2015) paper referenced above.

Direct measurement Output

Activity based sampling





Description: Activity based sampling (ABS) has the potential to be an important part of a staged approach to the assessment of health risk from the release of asbestos fibres resulting from the disturbance of ACM. It is capable of reducing the uncertainty in the estimation of fibre-release inherent in alternative theoretical approaches.

An option for ABS has been developed by SoBRA (2015). This procedure involves the raking of a minimum of 1m x 1m square of exposed bare soil using an ordinary garden rake, within a temporary enclosure secured to the ground to provide a reasonable seal. Static air samplers can be used to record airborne dust and fibre concentrations generated within the ABS enclosure whilst the raking activity is undertaken. Sampling and analysis of the soil layer being raked should be undertaken for asbestos, which may also include particle size distribution, FOC and soil moisture.

Further information:

SoBRA (2015). Design of an Activity-Based Sampling Protocol for the Testing of Asbestos Fibre Release Potential from Residential Garden Soil. https://sobra.org.uk/resources/reports/

US EPA (2007). Standard Operating Procedure 2084:2007. Activity based air sampling for asbestos. https://semspub.epa.gov/work/HQ/174392.pdf

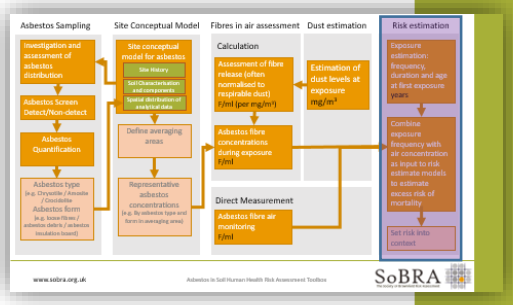
SoBRA comment: The primary objective of the ABS protocol is to provide a reasonable worst-case estimate of current and future fibre-release and subsequent localised airborne fibre concentrations that might be possible as a result of soil disturbance. If ABS is proposed, the practical and regulatory constrains of the Control of Asbestos Regulations 2012 and any other Health and Safety requirements must be considered in advance.

The location of any ABS is important – need to be confident that soil being tested is representative of exposure model. ABS is limited to the environmental conditions encountered at the time of sampling. This can mean that reasonable worst-case conditions can be missed leading to increased uncertainty in how to interpret the results and use as a line of evidence.

A distinct advantage of ABS is the reassurance it can give to stakeholders as a line of evidence that is based on actual site conditions.

Direct measurement Output

Estimation of risk



Exposure estimation: frequency, duration and age at first exposure **years**

Calculation of cumulative exposure **F/ml.years**

Fibre concentrations from Fibres in air assessment

Estimation of risk (Excess Mortality Risk)

Estimate exposure to calculate cumulative exposure from fibre concentrations during exposures

Then

Use the Hodson and Darnton model – to get excess mortality risk

Output

Excess Mortality Risk

Needs to be put in context

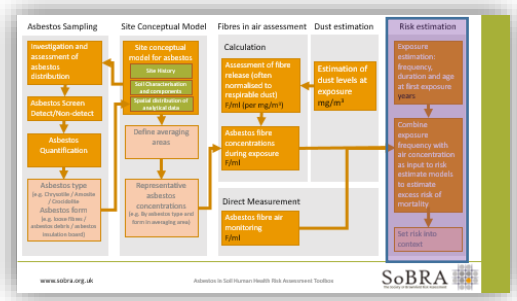
Exposure estimation

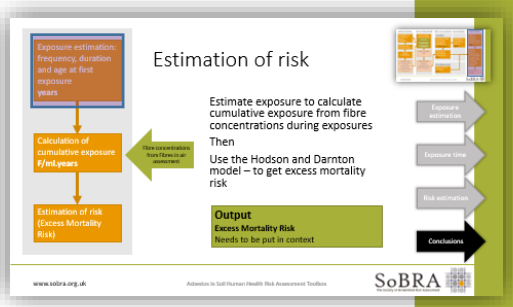
Exposure time

Risk estimation

Conclusions

Exposure estimation





Generally not measured, more likely to be modelled or assumed, though site survey may be used to determine land use scenario.

Consider number of dry dusty days from local weather data for outdoor exposure.

For indoor exposure the amount of tracked back soil should be assessed.

Define exposure scenario using either generic or site specific exposure parameters

UK Contaminated Land Exposure Assessment (CLEA) model[1] (or UK C4SL Main Report[2] or USEPA Exposure Factors Handbook (EFH)[3]) exposure duration assumptions for site scenarios

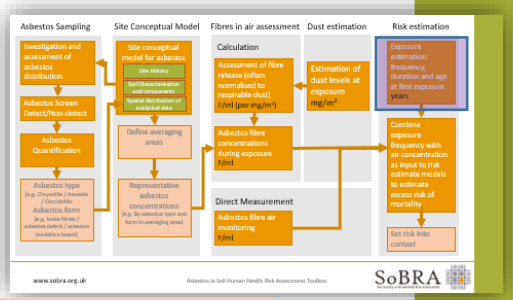
Determine likely age at first exposure

exposure frequency x exposure duration = total duration of exposure (years)
Determine for all (or highest risk) receptors and **age** at first exposure

Risk estimation

1. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/455747/LIT_10167.pdf and https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/291014/scho0508bnqw-e-e.pdf
2. https://www.claire.co.uk/projects-and-initiatives/category-4-screening-levels
3. https://www.epa.gov/expobox/about-exposure-factors-handbook

Estimation of exposure time



Description: The exposure time should consider both exposure to asbestos from soil disturbance outdoors and soil dust tracked back into buildings.

The exposure should consider the age at first exposure, and the exposure frequency for each five year tranche should be based on appropriate estimate for the use being assessed.

For generic UK land uses such as residential, public open space or commercial end uses refer the C4SL Main Report or CLEA model guidance.

Further information:

Environment Agency (2009). Updated technical background to the CLEA model. Science Report: SC050021/SR3
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/291014/scho0508bnqw-e-e.pdf

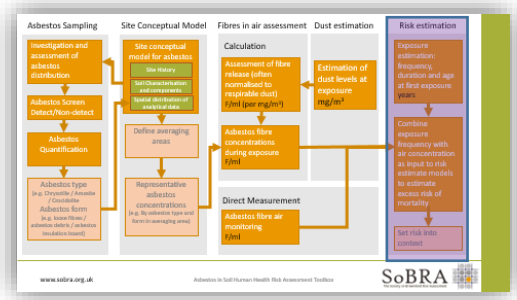
Environment Agency (2009). CLEA Software Handbook. Science Report: SC050021/SR4.
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/455747/LIT_10167.pdf

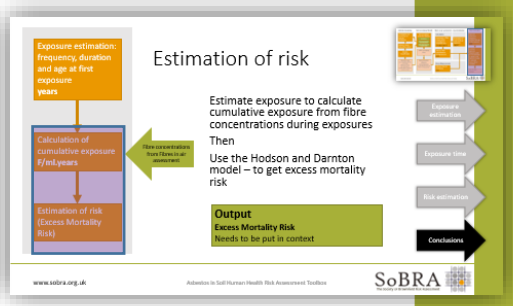
CL:AIRE (2014). SP1010 Development of Category 4 Screening Levels Main Report https://www.claire.co.uk/projects-and-initiatives/category-4-screening-levels

Exposure Output

SoBRA comment: For outdoor exposure consideration may be required of wet days when no or little dust is generated. Care should be taken to ensure that if wet days with no dust are to be considered that this is accounted for in the asbestos in air concentration used (i.e. consider time-weighted average concentration).

Risk estimation





Use exposure over time, asbestos type and age at first exposure to determine the risk of mortality.

Use Hodgson and Darnton (2000)[1] meta-analysis of multiple epidemiological studies

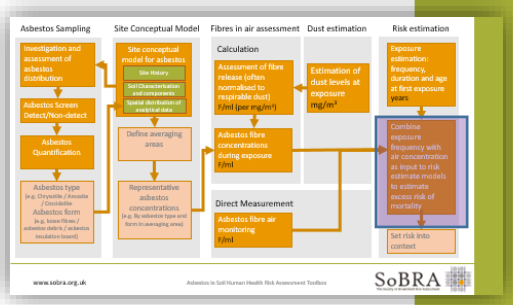
SoBRA Excel based spreadsheet tool implementing the Hodgson & Darnton algorithms is available on the SoBRA website https://sobra.org.uk/resources/reports/

Input parameters:
Cumulative exposure, **asbestos type**, **age** at first exposure
Output:
Mortality rate (e.g. 1 in a million)

Conclusions

1. Hodgson & Darnton (2000) Hodgson, J.T. and Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Annals of Occ. Hyg., Volume 44, No 8, pages 565-601.

Risk estimation from asbestos in air



Description: There are a number of models that can be used to estimate the risk to people from asbestos in air. These models consider the risks from mesothelioma and from lung cancer, and are derived based on empirical fits to epidemiological data. Differences in output often relate to how individual studies are interpreted.

These models broadly have similar inputs including:

1. The form of asbestos
2. The age of first exposure
3. The concentrations of asbestos and the exposure time and
4. The expected lifetime of the exposed individuals (to allow latency factors to be considered).

The asbestos concentrations and exposure time is generally combined to produce an cumulative exposure, however the way these are treated in each model may differ, for instance the Hodson and Darnton model applies an age adjustment factor to the cumulative exposure in each 5 year tranche.

The output is a risk. This is typically a risks of mortality rather than the risk of getting mesothelioma or lung cancer. The mortality takes into account the expected lifetime of the individual. For the lung cancer the exposed population may be important as for instance smokers have a higher risk than non-smokers.

Risk estimation Output

Further information: SoBRA (2021). Discussion Paper on Guidelines for Airborne Concentrations of Asbestos Fibres in Ambient Air: Implications for Quantitative Risk Assessment

SoBRA (2021). Excel calculation Sheet for risk from cumulative exposure to asbestos in air. https://sobra.org.uk/resources/reports/

SoBRA comment: The SoBRA discussion paper has adopted the linear Hodgson and Darnton model for mesothelioma and non-linear model for lung cancer. The Hodgson and Darnton model was chosen as it is the model used by the UK Health and Safety Executive (HSE).

An Excel tool has been produced by SoBRA that uses the Hodgson and Darnton model to estimate risk of mortality for each asbestos type from the data on the exposure to asbestos in air. The aim of the model is to provide a readily-available tool to users which improves consistency in calculations and avoids having to interpret and re-create the algorithms presented in the original paper.

25. SoBRA, 2021b. Discussion Paper on Guidelines for Airborne Concentrations of Asbestos Fibres in Ambient Air

Full Reference: SoBRA, 2021b. Discussion Paper on Guidelines for Airborne Concentrations of Asbestos Fibres in Ambient Air: Implications for Quantitative Risk Assessment. The Society of Brownfield Risk Assessment, January 2021.

[Extract: pages 1-15, Annex 1, Annex 4, Annex 5]

Discussion Paper on Guidelines for Airborne Concentrations of Asbestos Fibres in Ambient Air: Implications for Quantitative Risk Assessment

Authors: Simon Cole[1], Simon Hay[2], Barry Mitcheson[3]

Introduction

This paper is an update to that first published in 2017[4]. This updated version includes risk estimates calculated using SoBRA's workbook for the calculation of risk estimates using the linear as well as the non-linear version of the Hodgson & Darnton model as well as updates on the age adjustment calculations.

- Previous published use of the models published by H&D (2000), such as that in CIRIA C733 (2014) have adopted the non-linear version of the H&D model for pleural mesothelioma. Based on the outcomes of a SoBRA asbestos sub-group workshop in April 2019 it was agreed that the linear version of the pleural mesothelioma model is likely to be the more appropriate version for use in estimating risk from low environmental exposures.[5]
- Age adjustment is an important consideration for the use of the H&D models. In this update it is made clearer that the age adjustment factors should be applied to the summed exposure and risk for each 5yr tranche, and the adjusted risk summed for all the relevant 5yr tranches (refer to Annex 2 for details).

This paper has been prepared by a sub-set of the SoBRA asbestos risk assessment working group, to document the results of research and evaluation undertaken over the past year on air quality thresholds for asbestos in ambient air. The scope of this paper includes:

- Presentation of a selection of internationally published air quality guidelines together with the data upon which these are based
- Using the data that underpins some of those air quality guidelines, modelling has been undertaken with two different approaches to demonstrate how the modelling approach adopted alters the air quality guideline. Similarly, the sensitivity to the land use adopted upon the air quality guideline has also been evaluated.
- Information on existing background concentrations to set the calculated air quality guidelines into context
- Implications for current risk assessment approaches adopted in the UK
- Recommendations on next steps for consideration by the asbestos risk assessment working group members.

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[1] AECOM
[2] Arcadis
[3] Wood
[4] Baker, K., Cole, S., Hay, S., Mitcheson, B., Thomas, L., Discussion Paper on Guidelines for Airborne Concentrations of Asbestos Fibres in Ambient Air: Implications for Quantitative Risk Assessment, Society of Brownfield Risk Assessment, December 2017
[5] Reference to the use of the linear model for pleural mesothelioma has been abbreviated in the remainder of this paper as "H&D linear model". Note that this abbreviation encompasses the continued use of the non-linear model variants for peritoneal mesothelioma and lung cancer.

This paper forms one part of SoBRA's efforts to support the wider objectives of the Joint Industry Working Group[6] and support industry in the risk assessment and risk management of asbestos in soil. SoBRA identified in 2013[7] that establishing appropriate air quality values is a key component of the risk-based management process.

Background

Asbestos poses a risk to people when it is airborne, and the fibres inhaled can result in diseases including mesothelioma and lung cancer. In the UK, while there are workplace exposure levels for asbestos fibres in air supporting the assessment and removal of asbestos containing materials in buildings, there is no current consensus on which air quality guidelines should be used to assess potential risks from exposure to asbestos in soils by the general population. This in turn means that there is no UK regulatory or industry-agreed good practice for the assessment of risks from asbestos in soils, which are being - or could be - released to air and subsequently inhaled. The White Paper has been prepared as an evidence base, with the aim of supporting a cross-industry working group in developing good practice for assessment of potential risks from asbestos at sites affected by land contamination.

There are a range of existing air quality guidelines for asbestos fibres, provided both by international bodies (e.g. the World Health Organisation) and national bodies (e.g. Health Council of the Netherlands). There are also different approaches in literature for calculating air quality guidelines for asbestos fibres, dependent on the exposure scenario under consideration. This paper summarises a selection of internationally published air quality guidelines, together with the data upon which these are based, as well as calculating air quality guidelines using two different modelling approaches from literature. The existing air quality guidelines and calculated guidelines have been compared alongside published ambient background airborne concentrations to understand variability in thresholds for asbestos in air and the potential practicalities of those guidelines.

For the purposes of this White Paper, the authors adopted risk of death from cancer[8] of 1 in 100,000 as a risk level to allow true comparison of the different air quality guidelines and calculation methodologies. Appreciating that the UK approach to assessing carcinogens is based on minimal risk rather than a defined risk level, adoption of a 1 in 100,000 risk level was felt to be a reasonable starting point for comparison purposes. While there is ongoing debate regarding non-cancer effects from exposure to asbestos fibres, the authors agreed that the focus of the White Paper should be on cancer effects (mesothelioma and lung cancer) given the weight of evidence from epidemiological studies.

The authors note that for the risk assessment community to be able to draw conclusions as to risks from asbestos in soils, it is also important to reduce the uncertainty and lack of science relating to the relationship between asbestos in soil and asbestos fibres in air. However, this is subject to

[6] Joint Industry Working Group on Asbestos in Soil and Construction & Demolition Materials (www.claire.co.uk/asbestos)
[7] Requirements for further research in to the release of asbestos from soil, SoBRA, October 2013
[8] As noted in Environment Agency (2009) Human Health toxicology assessment of contaminants in soil Science report SC050021/SR2- "…where human data is available, it may be possible to model both risk of cancer (e.g. excess lifetime risk of cancer) and risk of death from cancer. These are sometimes used as though they are synonymous which they are not; their interrelation depends on the survival/fatality rate for malignancy. For example, fatality rates for non-melanoma skin cancer are quite low in western countries (a few percent) while for lung cancer they are high…" The use of risk of death from cancer assesses the whole risk and enables the effect on mortality from the delayed development of cancer later in life to be considered.

further research and will form a separate White Paper. This paper does however provide a preliminary consequence evaluation for one commonly adopted approach for soil risk.

Reporting conventions for asbestos fibres in air do vary and are not necessarily consistent with the definitions used for asbestos content in asbestos in bulk materials or soils[9]. The units used in this paper to compare and contrast guidelines are fibres per metres cubed (f/m^3) and we have chosen where possible to quote values as f/m^3 measured by Transmission Electron Microscopy (TEM). Where source literature quotes concentrations as measured by Phase Contrast Microscopy (PCM) the data have been converted using the adopted convention that TEM = 2 x PCM based on the approaches taken by WHO[10] and RIVM[11]. It is accepted that the conversion between PCM and TEM or SEM is complex and that there is no universally accepted conversion factor. Published values have varied from 1.7 – 4, and up to 30 in one study[12], and ATSDR[13] published a range of 19-76 for "all fibres" and a more restricted range of 1.4-3.2 for respirable fibres. However, an assumption on conversion is necessary to be able to compare and contrast guidelines quoted in the two different methods and is also required when converting PCM-based epidemiological data into TEM-defined guidelines (such as those adopted in the US and The Netherlands). RIVM for example adopts a x2 conversion from PCM to SEM, and WHO also adopts a x2 conversion for the purposes of defining air quality guidelines. It is noted however that ATSDR adopted a 1984 NRC recommendation to use a conversion factor of TEM = 60 x PCM for use in the conversion of historic ambient air measurements. Given this variability in conversion factors we have not converted reported background air concentrations where quoted in this paper. Where source literature quotes values in fibres per millilitre (f/ml) this is stated and the data has been converted using the conversion f/m^3 = f/ml x 1,000,000.

Existing Guidelines

Air quality guidelines protective at population level have been proposed by various organisations[14]; a number of these are summarised in Figure 1 with the raw data provided in Table 1. The organisations were selected based on their influence at an international level (e.g. WHO and USEPA) and the authors' knowledge of research in this field. The list is therefore not exhaustive. Further detail is provided in Annex 1.

[9] In terms of airborne fibres, the critical distinction from a risk perspective is between respirable fibres (those most likely to remain in the lung) and non-respirable fibres (those that are more likely to be expelled from the lung). The current convention in the UK is to define a countable asbestos fibre as one which is longer than 5µm, with an average width less than 3µm and having an aspect ratio greater than 3:1. RIVM (2003) defines respirable fibres as having a diameter smaller than 3µm and a length less than 200µm. The US EPA (2008) recommend a variation to ISO10312:1995 such that fibres are counted based on a length greater than 0.5µm and an aspect ratio of 3:1 or greater using TEM or based on a length greater than 5µm and an aspect ratio of 3:1 or greater for PCM. WHO (1986) recommends a width range of between 0.25µm and 3µm.

[10] WHO (2000) Air quality guidelines for Europe, second edition, WHO Regional Publications, European Series, No. 91, World Health Organization Regional Office for Europe, Copenhagen

[11] RIVM (2003) Table 2.1 in Assessment of the risks of soil contamination with asbestos, RIVM report 711701034/2003

[12] Boulanger et al (2014) Quantification of short and long asbestos fibers to assess asbestos exposure: a review of fiber size toxicity, Environmental Health, 13:59

[13] ATSDR (2001) Toxicological Profile for Asbestos, Agency for Toxic Substances and Disease Registry, September 2001

[14] World Health Organisation (WHO), US Environmental Protection Agency (USEPA), Health Effects Institute (HEI), Dutch National Institute for Public Health and the Environment (RIVM), Health Council of the Netherlands (HCN)

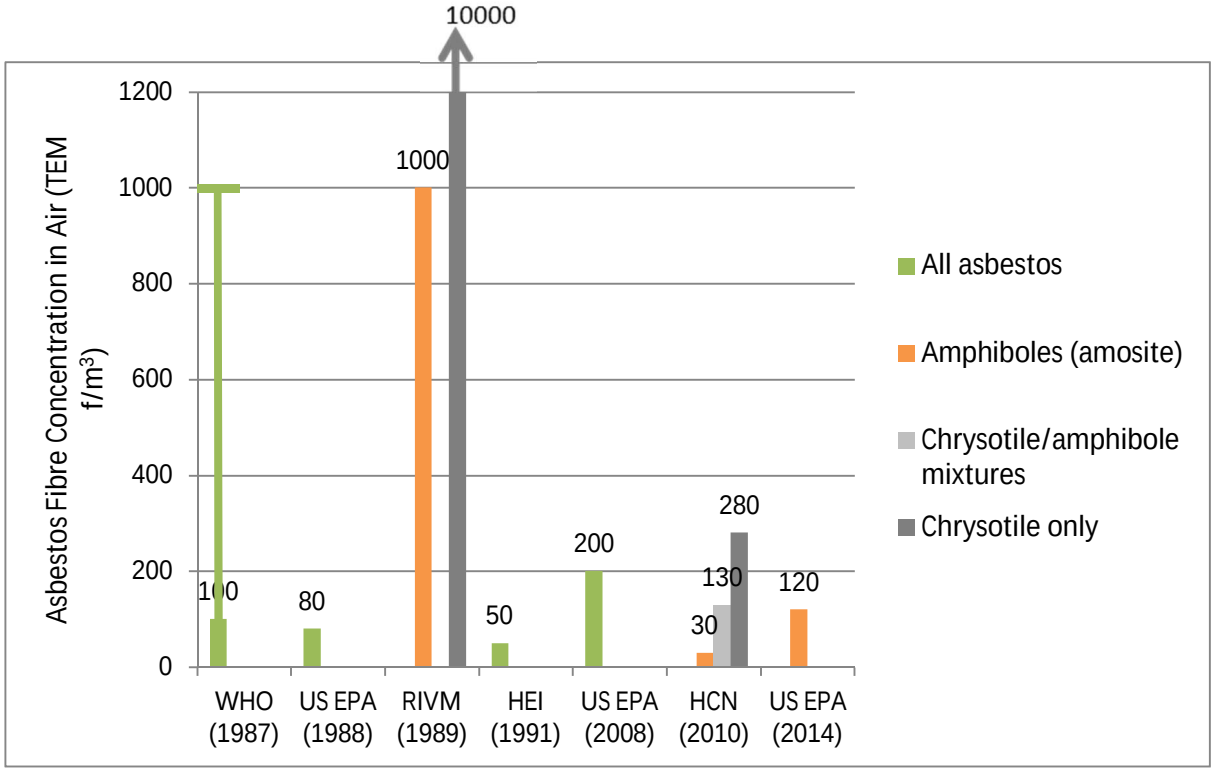

Figure 1 Comparison of Asbestos in Air Quality Guidelines (Selected International and National Organisations)[a]

Table 1 Summary of Asbestos Air Quality Guidelines (Selected International and National Organisations)

Organisation	Air Quality Guideline (f/m³)[a]			
	All asbestos types/not specified	Amphiboles[15] (amosite)	Chrysotile / amphibole mixtures	Chrysotile only
WHO (1987)	100-1000	-	-	-
US EPA (1988)	80	-	-	-
US EPA (2008)	200	-	-	-
US EPA (2014)	-	120	-	-
HEI (1991)	50		-	-
RIVM (1987)	-	1000	-	10000
HCN (2010)	-	30	130	280

Notes

[a] All published guidelines adjusted by the authors to reflect a 1 in 100,000 risk of death from cancer from developing mesothelioma and lung cancer and quoted in f/m³ TEM

[15] The use of "amphiboles" is the convention used by RIVM, although the assumption in its use is that the asbestos is amosite

Figure 1 and Table 1 illustrate that RIVM (1989) and the upper guideline from WHO (1987) are considerably higher than the remaining guidelines, which range from 30 to 280 f/m^3 with the lowest value applicable for amphiboles and the highest value applicable for chrysotile.

USEPA Asbestos Working Group

Black (pers comm, 2016) highlighted to the authors that the USEPA criteria in Table 1 are undergoing review within the USEPA Asbestos Working Group[16]. The expectation is that the air quality guidelines for asbestos will be reduced, with the potential for additional non-asbestos fibre types to be included for consideration when evaluating risks from dust and fibres. This information, combined with review of Table 1, indicates a general trend towards lower air quality guidelines for asbestos from those developed in the 1980s.

Modelling Approaches

Two alternative modelling options have been identified as being used by UK practitioners for calculating air quality guidelines for the protection at general population level, using the available epidemiological data and modified according to the exposure scenario under consideration. These modelled approaches are:

- Hodgson and Darnton (2000) algorithms for mesothelioma and lung cancer estimation adopted by the UK HSE (method adopted in CIRIA C733).
- Algorithms for mesothelioma and lung cancer commonly adopted by the US EPA, Berman and Crump, HEI and HCN.

Examples of how these two approaches can be used to calculate air concentrations that represent a given risk to land users in a UK context are provided in Annexes 2 and 3. The modelling outputs are summarised in Table 2. The conceptual exposure models adopted were based on the exposure parameters in the C4SL project (Defra SP1010, 2014) in relation to receptor ages and land-use characterisation. Further details on the exposure assumptions are provided in the model annexes.

[16] The understanding of the authors is that this review is still to be finalised/published.

Modelling Results

The calculations have been completed for residential, commercial, and public open space land uses, with the results summarised in Table 2 below. The risk of death from cancer for this exercise was set at 1 in 100,000. Other risk levels could be adopted, and the air concentrations re-calculated in accordance with the approach outlined in this paper. B&C and HEI calculations remain unchanged from the original version of this paper. The H&D calculations have all be re-done using the new SoBRA calculation spreadsheet.

Table 2 Summary of Calculated Exposure (Air) Concentrations for Residential, Commercial and Public Open Space Scenarios Indicative of risk of death from cancer of 10^{-5}

	Calculated Air Concentration (f/m³ TEM)														
Land-use	Residential					Commercial					Public Open Space				
Modelling Approach	B&C[a]	B&C[b]	HCN	H&D Non-linear[c]	H&D Linear[c]	B&C[a]	B&C[b]	HCN	H&D Non-linear[c]	H&D Linear[c]	B&C[a]	B&C[b]	HCN	H&D Non-linear[c]	H&D Linear[c]
Chrysotile	2600	-	400	400	3600	24000	-	3640	6400	34000	280000^	-	48000	36000	420000^
Mixed fibres	-	-	80	-	-	-	-	960	-	-	-	-	12800	-	-
Amphiboles (amosite)	14	40	12	5*	48*	200	400	200	80*	560*	1100	2600	1040	420*	4600*

Notes

[a] using Table 7-17 PCME coefficients (Berman & Crump 2003)

[b] using Seidman coefficients in Berman & Crump 2003)

[c] using best estimates from the non-linear and linear models (Hodgson & Darnton 2000). Calculations performed in PCM units (refer to Annex 2) and converted here to TEM using the adopted conversion factor of x2

*Based on H&D estimates for amosite

^ Exceeds the Control Limit of 0.1f/ml but is based on a 2hr exposure duration

The differences in values between chrysotile and amphibole values suggest a potency difference of between approximately 18x and 255x. Further discussion on asbestos fibre potency is provided in Annex 4

Comparison of Model Calculations with Published Air Quality Guidelines

Based on the calculation examples above for residential land-use, Figure 2 compares the existing air quality guidelines (from Table 1) and calculated residential air concentrations in Table 2. This indicates the potential range in values that the UK could adopt, based on an ECLR of 1 in 100,000 for residential land use. The comparison indicates that there is relatively low variation for chrysotile if the value for chrysotile using the Berman and Crump coefficients is excluded. It also indicates that there is relatively low variation for amphiboles, but that the air quality values for amphiboles are typically lower by an order of magnitude or more than for chrysotile. It also shows that UK-based assumptions on early life exposure adjustment and mortality rates using a linear model for pleural risk can result in higher values than those published by the original authors (for example the SoBRA amphibole value of 48f/m^3 by H&D method compared to 12 f/m^3 by HCN method and the HCN published value of 30f/m.

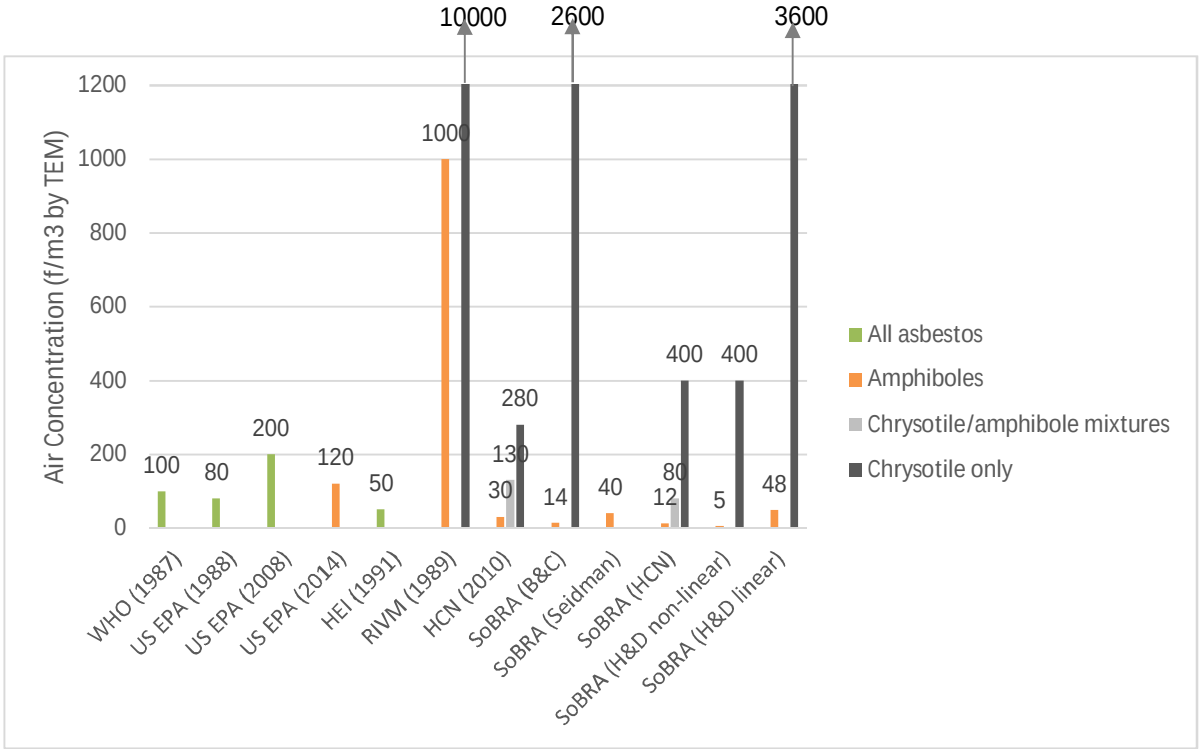

Figure 2 Comparison of Asbestos in Air Quality Guidelines (Selected International and National Organisations)

Model Sensitivity to Air Concentration and Exposure Frequency/Duration

The H&D and HEI models present different exposure/risk relationships and therefore exhibit different sensitivities to changes in air concentration and or frequency and duration of exposure. The H&D relationship includes a power relationship based on cumulative exposure (concentration x duration) although following discussion with the author a linear model for the more dominant pleural mesothelioma risk has been adopted as the preferred approach, whereas the HEI model is a combination of a linear relationship for air concentration and a power relationship for time.

Figures 3 and 4 below provide examples of model sensitivity to changes in air concentration and exposure time based on a single scenario of an adult exposed to amosite.

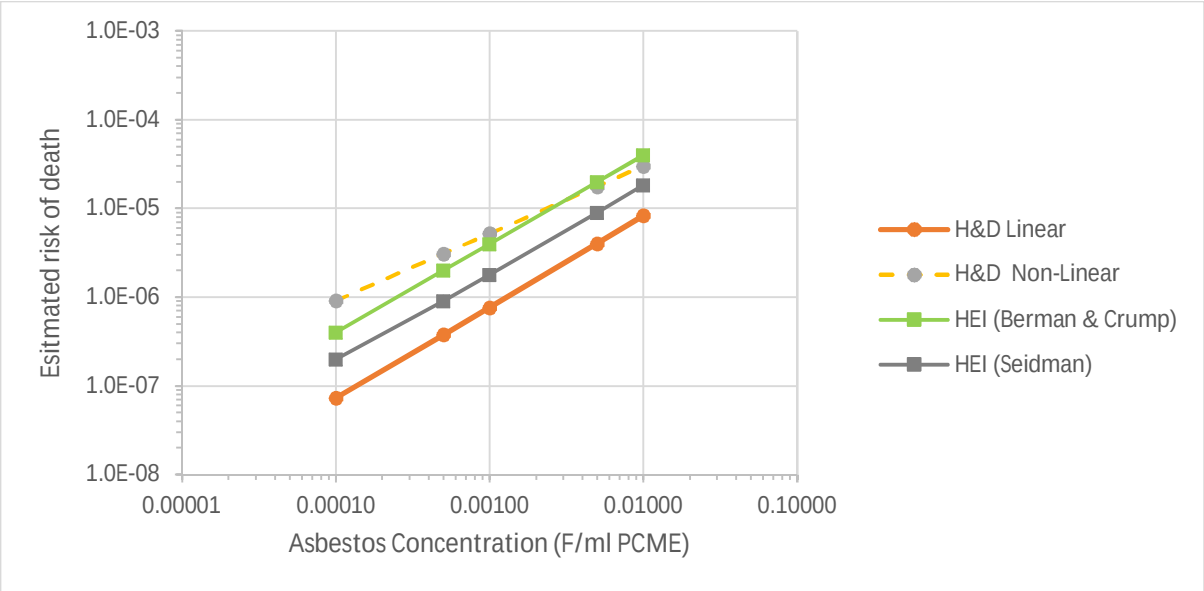

Figure 3 Sensitivity of Risk Models to Changes in Air Concentration

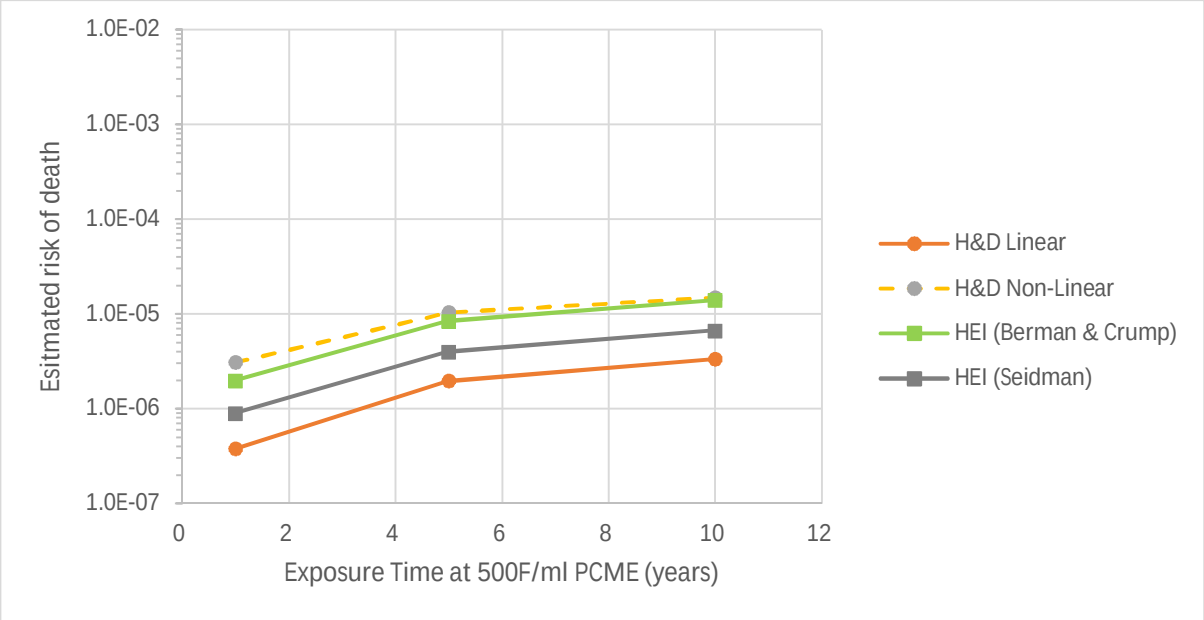

Figure 4 Sensitivity of Risk Models to Changes in Exposure Duration

Principal model assumptions[17] for Figures 3 and 4 above are an adult aged 30 at start of exposure with risk persisting for 80 years.

Figures 3 and 4 show that it is possible to estimate the significance of individual changes to default exposure assumptions; for example, increasing or decreasing the time weighted average exposure

[17] The H&D linear model refers to the use of the linear model for the more dominant pleural mesothelioma risk but the peritoneal and lung cancer risk models remain non-linear in these calculations, The H&D non-linear model uses the non-linear model for pleural, peritoneal and lung cancer risks

concentration or increasing or decreasing the duration of exposure, without needing to undertake detailed modelling. Detailed modelling is likely to be required for changes in multiple parameters.

Background Concentrations

The authors found that research into background concentrations of airborne asbestos fibres in ambient air (indoors and/or outdoors) is limited and is largely restricted to studies undertaken before prohibitions on the importation and use of asbestos containing products came into force. Of the most recent reviews, CIRIA (2014) published a summary of identified studies (Table 6.1 of C733), The Health and Safety Executive (HSE) Working Group on Action to Control Chemicals (WATCH) reviewed background concentrations in 2010, and the HCN also reviewed at background concentrations in 2010.

The conclusion of HCN (based on unpublished reference measurements in uncontaminated urban and non-urban areas by TNO, taken as part of investigations into workplace exposure) was that current background levels in the outdoor atmosphere in the Netherlands are likely to be 10-20 f/m^3 (as measured by TEM). This expected range is narrower although still potentially consistent with the assumption in CIRIA (2014) that outdoor concentrations in rural and urban areas in the UK are likely to be below 100f/m^3 (as measured by PCM).

The Institute for Environment and Health published a study of background air concentrations in the UK in 1997. This concluded that outdoor ambient concentrations were generally in the range 1-100 f/m^3, and indoor concentrations were mostly below 200 f/m^3, rising to approximately 500 f/m^3 for buildings containing asbestos in good condition (all values measured by PCM).

WATCH summarised available published information from the UK, Europe and US; the values quoted reasonably consistent with the ranges noted above. Critically the studies did not typically distinguish between asbestos type so detailed comparison with the air guideline values is not possible, however, taking a likely range between 1 and 100 f/m^3, it is evident from Figure 2 that the guidelines for amphiboles (primarily amosite) and the guidelines for mixtures of asbestos types are likely to be within the range of anticipated background concentrations (noting these concentrations are for all asbestos fibres and not specifically for amphiboles). It is likely on the other hand that ambient background concentrations are likely to be lower than the guideline values for chrysotile.

Further information is on published background concentrations are provided in Annex 5.

Implications for QRA

This White Paper highlights that:

- There appears to be good consensus in literature that air quality guidelines for amphibole should be lower than chrysotile, typically by up to two orders of magnitude;
- There is variation but not significant differences (i.e. an order of magnitude of more) in air quality guidelines for general population exposure (assuming sensitive receptor exposure, such as residential land use) when existing air quality guidelines are compared to the guidelines calculated by the authors using different methods[18];
- The existing and calculated air quality guidelines for chrysotile are consistent, or higher, than literature-reported background concentrations of asbestos fibres in air;

[18] With the exception of the calculation undertaken using Berman and Crump co-efficient for chrysotile

- The existing and calculated air quality guidelines for amphiboles are typically lower than literature-reported background concentrations of asbestos fibres in air, however little is known as to the extent to which amphiboles contribute to the reported background values;
- There appears to be a downward trend in air quality guidelines when comparing those derived in the 1980s to those derived in the 2000s;
- The use of the Hodgson & Darnton non-linear model for pleural mesothelioma results in lower air guideline values compared to the use of alternative models (including the H&D linear pleural mesothelioma model);
- The adoption of more recently published air quality guidelines for amphiboles could lead to more stringent assessment and clean-up goals for asbestos at land contamination sites (compared to use of WHO (1987) for example);
- Conversely the adoption of more recently published air quality guidelines for chrysotile could lead to less stringent assessment and clean-up goals for asbestos at land contamination sites
- The adoption of older air quality guidelines should therefore be carefully considered before use in risk-based decision making;
- The adoption of more recently published air quality values for amphiboles likely presents a challenge for existing air sampling and analysis methods and puts into question the practicability of these values – can they be reliably determined?;
- Occupational monitoring/sampling techniques are likely in most cases to be inappropriate for use as a line of evidence in assessments of potential exposure against air quality values such as those referenced in this paper; and
- The use of the HSE clearance indicator level of 0.01 f/ml PCMe (20,000 f/m^3 by TEM) is outside the range of air values for chrysotile quoted in Figure 2 (240-10,000 f/m^3) and significantly outside the range for amphiboles (3-1000 f/m^3).

To illustrate what (if any) implication there may be to the use of one of the most widely adopted guidelines in Europe (Annex 3 of the Dutch Soil Remediation Circular), the graph on which the Dutch guidelines are based is reproduced below with the calculated air values in this paper added.

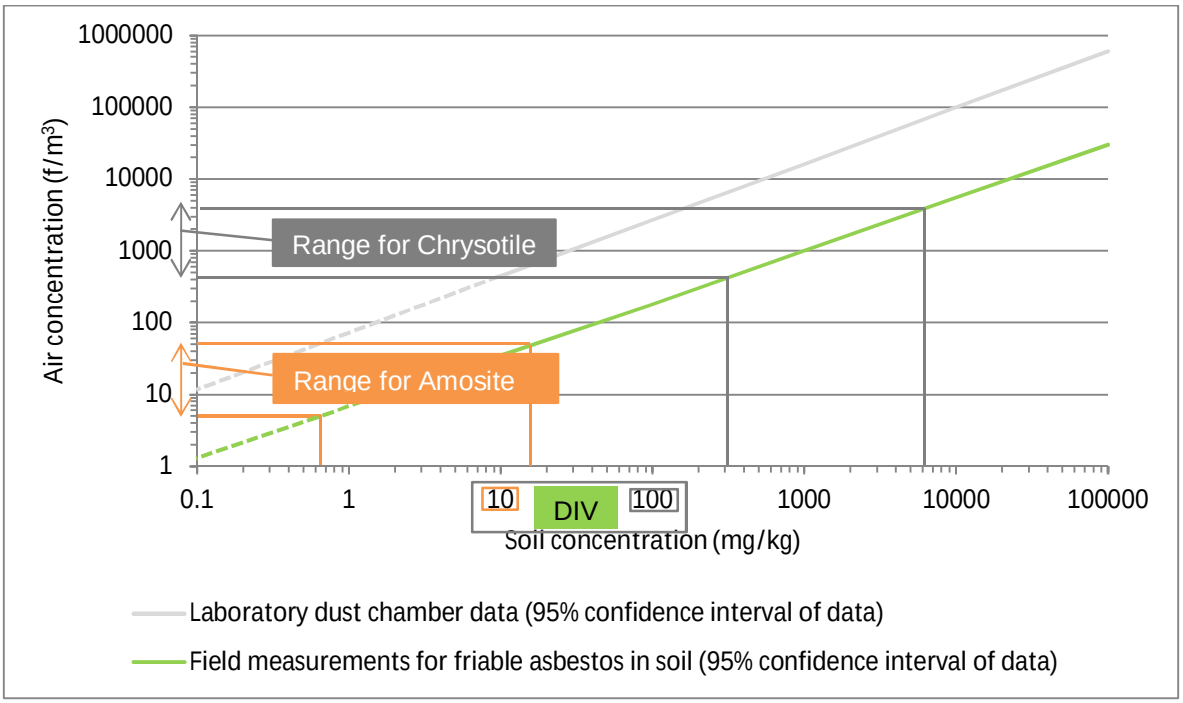

Figure 5 Implication of calculated air values on use of Dutch Guidelines

Figure 5 indicates that the Dutch Intervention Value (DIV) for chrysotile in soil of 100mg/kg (0.01%wt/wt) is likely to remain precautionary (i.e. based on the graphical correlation air concentrations should remain below all calculated air guidelines) unless conditions similar to laboratory conditions prevail. The DIV for amosite of 10mg/kg (0.001%wt/wt) however is unlikely to be similarly precautionary.

In relation to the quantitative risk assessment approach advocated in CIRIA C733 it is evident from the calculations presented in this paper that the Hodgson & Darnton non-linear algorithms for all forms of asbestos-related cancer is likely to produce higher risk estimates than those calculated using alternative risk algorithms such as those advocated by HCN and Berman & Crump. The use of the linear version of the H&D model for pleural mesothelioma in conjunction with the non-linear variants for peritoneal mesothelioma and lung cancer however produces similar to lower risk estimates compared to HCN and B&C.

Recommendations

The authors believe that the information in this White Paper should be used as supporting evidence for the Joint Industry Working Group to formulate a position regarding air quality guidelines for asbestos in the UK. There remain a number of issues that need to be resolved before an air guideline value can be proposed:

1. Should the UK adopt existing air quality guidelines to air assessment of chronic risks from asbestos at land contamination sites? If yes:
    a. Which guidelines could be adopted?
    b. Should a range in guidelines be adopted?
    c. Should the analysis comprise PCM or TEM?
    d. What risk level should be adopted?
    e. Should provision be made for modification to air quality guidelines based on non-residential exposure scenarios?
2. Should the UK adopt a framework for calculating air quality guidelines? If yes:
    a. Which approach should be adopted?
    b. Should the analysis comprise PCM or TEM?
    c. What level of risk should be adopted?
3. Should background concentrations be incorporated into the assessment process in the UK? If yes:
    a. Is more data needed before an approach can be formulated?
    b. Should published background concentrations be used to bench-mark the air quality guidelines?
4. Can we make a clear distinction between the relative risks from chrysotile and amphiboles given the relative abundance of the former compared to the latter, and thereby lead to different risk management approaches?

It is recommended that the linear version of the H&D model for pleural mesothelioma is used to estimate risk and calculate air guideline values in conjunction with the non-linear variants for peritoneal mesothelioma and lung cancer. SoBRA has developed an excel-based tool to implement both the non-linear and linear versions of the H&D model. This model is provided free to use via the SoBRA website.

It is evident from the assessment presented in this paper that there is a clear requirement for further research into background air concentrations in the UK. This is needed to be able to benchmark the practicability of proposed air guidelines. It is also evident that a step change in air monitoring practice is required; with a move away from the use of occupational monitoring techniques that typically report to 10000f/m^3 (0.01f/ml) and use non-fibre-discriminatory PCM analysis to methods capable of measuring down to at least 10f/m^3 using fibre-discriminatory SEM or TEM analysis (as advocated by the authors of CIRIA C733).

<u>Limitations</u>

This white paper has been developed by members of the SoBRA Asbestos-in-soil sub-group acting in a voluntary capacity, and details the views of the individual authors, not those of their employers. It is provided freely on the SoBRA website to help promote discussion on what should constitute good practice in assessing the health risk from asbestos-contaminated soil in the UK. Users of this paper must satisfy themselves that the content is appropriate for the intended use and no guarantee of accuracy or suitability is made.

Feedback

Feedback on this paper is welcomed and should be submitted to SoBRA at info@sobra.org.uk.

January 2021

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Annex 1 – Published Air Quality Guidelines

Organisation/Reference	Air Quality Guideline (f/m^3)	Comments
WHO (2000)	100-1000	Based on toxicological review published in 1987. The review noted that amphiboles were more potent than chrysotile but did not differentiate between asbestos type in the calculations as a precaution. Calculations based on Peto (1984) model for mesothelioma and US EPA (1985) model for lung cancer. Final recommendation that $500f/m^3$ (PCM) equated to an approximate risk of mesothelioma of 10^{-5}-10^{-} and 10^{-6}-10^{-5} for lung cancer. A "best" estimate for lifetime exposure to $100f/m^3$ (PCM) may be $2x10^{-5}$; suggesting an AQG set at 10^{-5} could be 50 f/m^3 (PCM).
US EPA (1988)	80	Inhalation unit risk of 0.23 per f/ml (PCM) based on a 1988 toxicological review available on IRIS. This equates to an air concentration of 40f/ml (PCM) for cancer risk of 10^{-5}. No asbestos type, with calculations based on assumption that airborne fibres are amosite or mixed amosite.
US EPA (2008)	200	This framework document sets out an approach to setting land-use specific air quality guidelines. Based on the IRIS (1988) IUR adjusted for less than lifetime exposure. Value quoted to the left is for baseline residential exposure (30 years exposure from birth) adopting an ELCR of 10^{-5}.
US EPA (2014)	120 RfC 180	Inhalation unit risk of 0.17 per f/ml (PCM) based on the Libby mine cohort only (exposure primarily to tremolite). A reference concentration for non-carcinogenic health effects of $90f/m^3$ (PCM) also established based on the risk of pleural thickening.
HEI (1991)	50	The authors concluded that 100f/ml (PCM) equated to a risk of death of $4x10^{-5}$ for mesothelioma and lung cancer from mixed fibre exposures.
RIVM (1989)	1000 amphiboles 10,000 chrysotile	Based on WHO (1987) toxicological evaluation. Assessment distinguished between amphibole and chrysotile fibres, using an assumption that lung cancer was attributable to chrysotile exposure and mesothelioma only attributable to amphibole exposure. Maximum permissible risk levels were stated for a cancer risk of 10^{-4} and negligible risk levels stated for a cancer risk of 10^{-6} based on the upper values in the fibre concentration ranges for those risk levels. Values quoted to left are the mid-point values for a cancer risk of 10^{-5} to maintain consistency with other values quoted.

Organisation/Reference	Air Quality Guideline (f/m^3)	Comments
HCN (2010)	30 amphiboles 130 mixed fibres 280 chrysotile	Proposed new MPR and NR levels based on a new meta-analysis of the epidemiological data. Values quoted to left are for a mid-point cancer risk of 10^{-5} based on the combination of lung cancer and mesothelioma risk. Risk of mesothelioma differentiated across three categories; no differentiation made for lung cancer risk (i.e. risk the same irrespective of asbestos type)

All AQG are guidelines and are non-statutory. All AQG values quoted as measured by TEM unless stated otherwise. Conversion between PCM and TEM where required based on commonly adopted approximation of TEM = 2x PCM

Annex 4 - Fibre potency

One of the reasons for the differences in risk estimates from different epidemiological evaluations is the difference in approach taken to accounting for fibre potency. This typically takes two forms; the asbestos fibre type, and the fibre size (often described in terms of length and width).

Hodgson & Darnton (2000) suggested a potency difference based on asbestos type of 1:100:500 for mesothelioma for chrysotile:amosite:crocidolite. This however is only relevant to the range of occupational exposures measured in the occupational cohorts. The ratio is more like 1:10:100 at lower environmental exposure levels.

RIVM (2003) evaluated potency based on fibre asbestos type and fibre dimension as follows:

Chrysotile	Fibre length >5um	1
Amphiboles	Fibre length >5um	10
Chrysotile	Fibre length <5um	0.1
Amphiboles	Fibre length <5um	1

The Health Council of the Netherlands in 2010 re-evaluated the epidemiological data and provided a revised analysis of potency differences. It concluded on a potency ratio of 1:2:10 for chrysotile:mixed fibres:amphiboles.

ATSDR (2001) concluded that mineral type and fibre size were of prime importance to health risk, and that long fibres were more carcinogenic than short ones.

US EPA (1986) concluded that crocidolite was 2-4 times more potent than chrysotile for mesothelioma but that the difference may be overstated by differences in fibre size distribution in the exposures received by the occupational cohorts.

The World Health Organisation (1987) and US EPA (1986) chose not to distinguish between fibre potency when developing guideline values and unit risks for air concentrations and as a result these values can be taken to be associated with amphibole exposure.

Berman & Crump (2008) looked more closely at accounting for fibre mixtures and fibre size distributions in the historic occupational cohort data. In doing so they produce very different exposure-risk coefficients to those based solely on the reported air concentration. They suggest a potency ratio of at least 1:200 for chrysotile:amphibole mesothelioma risk.

Although focus can sometimes be on mesothelioma, lung cancer risk can be an important factor at low concentrations. CIRIA (2014) provides a summary of the different potencies for mesothelioma and lung cancer based on the HEI and H&D models:

Mesothelioma	HEI	1:3.2
	H&D	1:100*
Lung cancer	HEI	1:4
	H&D	1:10-50

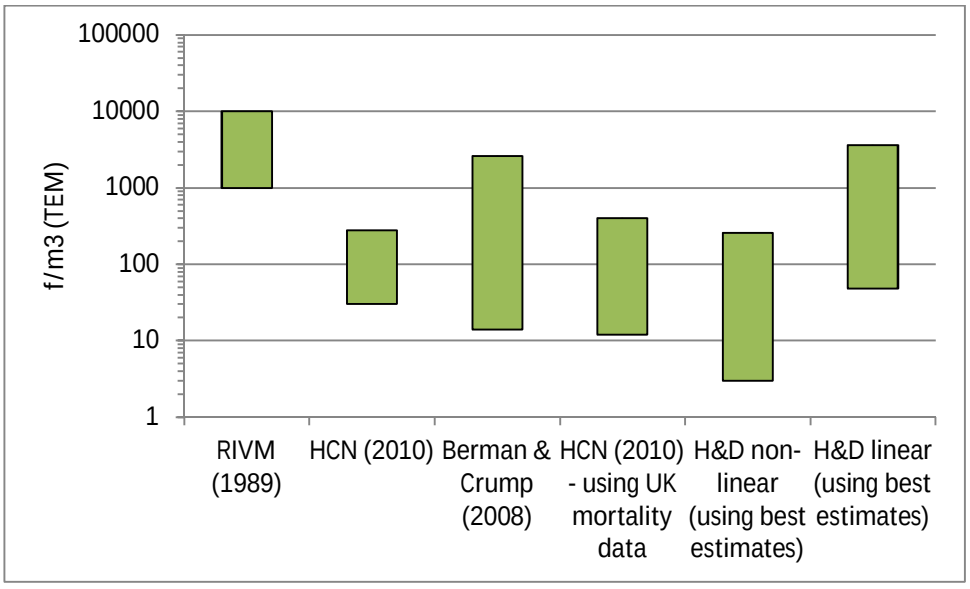
Ratios expressed as chrysotile:amphiboles

The results of SoBRA's modelling using the HEI and H&D models, accounting for the summation of mesothelioma and lung cancer risk suggests the following:

Berman & Crump (2008) analysis taking into account fibre size distribution and fibre mixture of original occupational exposure (adjusted coefficients)	1:185
HCN (use of unadjusted cohort coefficients and UK mortality data)	1:35
Hodgson&Darnton (non-linear) (best estimates)	1:80 (residential scenario)
Hodgson&Darnton (linear) (best estimates)	1:75 (residential scenario)

Potency ratio expressed as chrysotile:amphibole

These different potency ratios have a strong influence on the model outputs, but don't in themselves dictate the values since the ratios are relative. The graph below illustrates the range in ambient air concentrations associated with a risk of 10-5 (the upper value in the range being for chrysotile and the lower value in the range being for amphiboles).



Annex 5 – Background Air Concentrations

Background air concentrations from the UK HSE's Working Group on Action to Control Chemicals (WATCH), outdoor and indoor concentrations reported in CIRIA C733 and information from the Health Council of the Netherlands has been reviewed and is presented in this annex. It should be noted that the authors have not undertaken an exhaustive literature review; there may be additional published data that is not included here.

UK HSE WATCH

The UK HSE's WATCH looked at background air concentrations during its consideration of asbestos between 2007 and 2011. The summary of the information is reproduced below:

WATCH 2010-02-Annex 3 Table 1: Update of publications giving quantitative airborne asbestos fibre measurements in buildings during normal occupation since the HEI-AR review on asbestos in the non-occupational environment (units are in PCM equivalent fibres/m^3).

Study	No of buildings or people and (air samples)	Types of buildings / or person sampled	Arithmetic average indoor in asbestos containing buildings (F/m^3)	Arithmetic average outdoors (F/m^3)	Special observations
HEI – Review (1992) Non litigation data	198 (1377)	All buildings (occupied). Including:	270	~10 rural ~100 urban	Includes some maintenance and custodial work and cable pulling gave highest value. Excluding highest value (sample) average becomes:
		Schools and colleges	510	-	0.00038 (mechanical room)
		Residences	190	-	-
		Public and commercial	200	-	0.00008 (during cable pulling)
HEI Review – Litigation data	171	Schools and colleges	110	50	-
	10	Residences	BLD	-	-

Study	No of buildings or people and (air samples)	Types of buildings / or person sampled	Arithmetic average indoor in asbestos containing buildings (F/m^3)	Arithmetic average outdoors (F/m^3)	Special observations
	50	Public and commercial	60	-	-
Review by MRC Institute for Environmental Health (1997)		Buildings containing asbestos material	~500	0 – ~200	Review and tabulation of previous individual studies carried out no detailed calculation of averages.
Schneider et al. (1996)	5 (40)	School children	44	-	Personal sampling, sample changed once in 24 hours for day and night
	5 (40)	Retired persons	66	-	-
	5 (40)	Office workers	10	-	-
	5(40)	Taxi drivers	105	-	-
Italian Schools Campopiano et al. (2004)	59 (132)	Schools during normal occupation	83% <400 Max 2200 Average ~250	-	Building containing ACMs (vinyl tile and Asbestos cement) Chrysotile only found although AC had amosite
US schools and public buildings with asbestos containing materials. Lee and Van Orden, (2008). 752	752 (3978)	All indoor	120	-	Max for building = 0.004 90% of buildings had no PCME asbestos fibres detected
	752 (1678)	All Outdoor	-	20	-
	371	Schools during normal occupation	100	-	-
	752	All buildings	80	-	-

Study	No of buildings or people and (air samples)	Types of buildings / or person sampled	Arithmetic average indoor in asbestos containing buildings (F/m^3)	Arithmetic average outdoors (F/m^3)	Special observations
Polish city urban air asbestos measurements outside asbestos cement buildings. Krakowiak et al. (2009)	27 (41)	Close to degraded AC buildings	-	1800	SEM study debris on ground limited analytical sensitivity no averages given.
	24 (42)	100-500 m from buildings	-	<1000	-
	11 (17)	Close to buildings with no ACMs		<1000	
UK Schools with CLASP construction. Burdett et al. (2009)	7 (28)	Schools during normal occupation after remediation / sealing gaps	Average <50	Not done	Mainly asbestos insulating boards in columns with metal cladding around it
	1 (8)	Office in normal use sampled during day time for 4 weeks	Average <30	Not done	Mainly asbestos insulating boards in columns

CIRIA C733 Table 6.1 Background asbestos concentrations reported in indoor and outdoor air

Outdoor air [1,2]	
Rural areas (remote from asbestos emission sources)	Below 100 f/m^3 (0.0001 f/ml)
Urban areas	General levels may vary from below 100 to 1000 f/m^3
Near various emission sources the following figures have been measured as yearly averages	• downwind from an asbestos-cement plant 300m: 2200 f/m^3, at 700 m: 800 f/m^3, at 1000 m: 600 f/m^3 • at a street crossing with heavy traffic 900 f/m^3 • on an express-way, up to 3300 f/m^3
Indoor air [1,2]	
In buildings without specific asbestos sources	Concentrations are generally below 1000 f/m^3
In buildings with friable asbestos	Concentrations vary irregularly, usually less than 1000 F/m^3 are found but in some cases exposure reaches 10000 F/m^3 (values measured by PCM)

Notes:

[1] All reported concentrations are based on measurements by electron microscopy methods, except where stated otherwise.

[2] The data represents a range of different sampling and analytical techniques and was collected for a variety of purposes. A direct comparison between different values is not appropriate.

[3] Much of this data relates to measurements collected in the 1980s. The more stringent restrictions and controls implemented in many countries since then mean that current background concentrations would be expected to be lower than those cited.

The information summarised in Table 6.1 is attributed in CIRIA C733 to WHO (2000) Air quality guidelines for Europe. Second edition, European series, No. 91, Regional Office for Europe, World Health Organization, Copenhagen, Denmark (ISBN: 9-28901-358-3), however, the authors of this paper note that this information is not contained in this WHO publication, rather in WHO (1986) International Programme on Chemical Safety, Environmental Health Criteria 53, Asbestos and other natural mineral fibres.

Health Council of the Netherlands

Health Council of the Netherlands (2010) quotes RIVM (1987) Basisdocument Asbest, Rapport nr. 758473006, Sloof, W. and P.J Blokzijl (eds.). RIVM (1987) data summary reproduced in RIVM 758473013 (1989) (English translation). Measurements by Den Boeft and Lanting made between 1978 and 1980:

Summary of Health Council of the Netherlands (2010) based on measurements in 1978 and 1980

Outdoor	
Rural	100-1000 f/m^3
Towns	1000-10,000 f/m^3
Near sources	10,000-100,000 f/m^3
Indoor	
Living area	100-1000 f/m^3
Factories with sprayed asbestos	<1000 – 600,000 f/m^3

Boeft, J. Den and R.W Lanting. Asbest en andere minerale vezels in de buitenlucht Orienterende metingen van concentratieniveaus in Nederland, IMG-TNO rapport G 856

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Full Reference: Swartjes & Tromp, 2008. A Tiered Approach for the Assessment of the Human Health Risks of Asbestos in Soils, Frank A. Swartjes & Peter C. Tromp, Soil and Sediment Contamination: An International Journal, 17:2, 137-149

[Reference provided in full]

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A Tiered Approach for the Assessment of the Human Health Risks of Asbestos in Soils

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A tiered approach for the assessment of human health risks of soil contamination with asbestos has been developed. When in a specific tier the human health risk can not be rejected, the assessment in the following tier has to be performed. Because the risks of asbestos are caused by inhalation of asbestos fibers, the emission of fibers from soil to air is the determining factor. In Tier 0 of the tiered approach a generic soil quality standard is used. This Intervention Value is 100 mg/kg$_{soil,dw}$ asbestos equivalents (0.01% by weight), i.e. the sum of the concentration of chrysotile asbestos and ten times the concentration of amphibole asbestos, for bound (non friable) as well as for friable asbestos. Tiers 1 to 3 are site-specific. Tier 1 concerns a simple qualitative testing procedure, in which the potential or probability for emission of asbestos fibers from soil to air is assessed. In Tier 2 the respirable fraction in the soil and house dust, which relates to the potential *site-specific exposure to humans, is determined and tested. Finally, when the risk can not be excluded, the concentration of asbestos fibers in outdoor and/or indoor air has to be measured and tested according to a standardized procedure, in Tier 3.*

Keywords Asbestos, soil contamination, Intervention Value, risk assessment, chrysotile, amphibole

1. Introduction

Asbestos is often found in the soil or on the soil surface. Therefore, it is essential to have a testing framework that permits assessment of risks related to the presence of asbestos in or on the soil. According to the Dutch Soil Protection Act, risks of soil contamination must be assessed on the basis of a generic soil quality standard (Intervention Value) and, if these values are exceeded, on the basis of a site-specific risk assessment (Swartjes, 1999). The risks of asbestos to the ecosystem are negligible. Risks of dispersal only occur through wind blow, not via leaching into the groundwater. The main concern of asbestos in soil is the risk to human health.

2. Procedure

To be able to assess the human health risks of asbestos in soil in a scientifically based and efficient way a tiered approach has been developed. This tiered approach is described in

The authors thank the Dutch Ministry of Housing, Spatial Planning and the Environment (VROM) of the Netherlands, Directorate General for the Environment (DGM), Directorate of Soil, Water and Rural Areas (BWL), on which account this investigation has been performed.

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this paper. It contains the scientific foundation for the *Intervention Value*, used in Tier 0, and a procedure for *site-specific assessment* of soils contaminated with asbestos, as Tiers 1 to 3 of the tiered approach. Successively, in each tier the degree of conservatism decreases, while site-specificism increases. As a consequence, complexity and hence effort needed also increase in each tier. When in a specific tier the human health risks can not be rejected, the assessment in the following tier has to be performed. The underlying principle is: "simple when possible and complex when necessary."

3. Human Health Risks

3.1. Human Health Criteria for Asbestos in Air

The risks after oral intake, i.e. cancer to the gastrointestinal tract, are assumed negligible. The major effects on human health after inhalation of asbestos fibers are (ATSDR, 1995):

- mesothelioma (cancer of the pulmonary membrane and peritoneum);
- asbestosis (brown lung disease);
- increased risks for bronchial carcinoma (lung cancer).

The latency period between first exposure to asbestos and the manifestation of a disease can be substantial, i.e. up to several decades. Acute (short-term) exposure to asbestos is considered irrelevant. The effects on human health depend on the type of asbestos (chrysotile or amphibole asbestos), dimensions (diameter and length) of the asbestos fibers, period of exposure, the durability and fissility of the asbestos fibers, the concentrations to which humans are exposed and individual human characteristics.

The Dutch Health Council considered the carcinogenic potency of fibers with a length smaller than 5 μm 10 times less then the carcinogenic potency of fibers with a length larger than 5 μm (Dutch Health Council, 1988). Besides, the carcinogenic potency of amphibole fibers were assumed 10 times higher then the carcinogenic potency of chrysotile fibers. This resulted in the following differentiated asbestos equivalent factors (related to a chrysotile fiber with a length >5 μm):

- 1 chrysotile fiber, length >5 μm: equivalence factor 1;
- 1 chrysotile fiber, length <5 μm: equivalence factor 0.1;
- 1 amphibole fiber, length >5 μm: equivalence factor 10;
- 1 amphibole fiber, length <5 μm: equivalence factor 1.

Besides, the Dutch Ministry of Housing, Spatial Planning and the Environment formulated human health quality objectives (Ministry of VROM, 1991). On the basis of the above-mentioned equivalence factors the following human health quality criteria in air were defined as yearly average values:

- Negligible Risk level: 1,000 fiber equivalents/m^3_{air};
- Maximum Permissible Risk level: 100,000 fiber equivalents/m^3_{air}.

3.2. Human Exposure to Asbestos in Soil

3.2.1. Emission. Because the risks of asbestos are caused by inhalation of asbestos fibers, the emission of fibers from soil to air is the determining factor. The emission is strongly influenced by the characteristics of the asbestos-containing materials, like (the degree of) friability, the type of asbestos (chrysotile or amphibole) and the amount of respirable

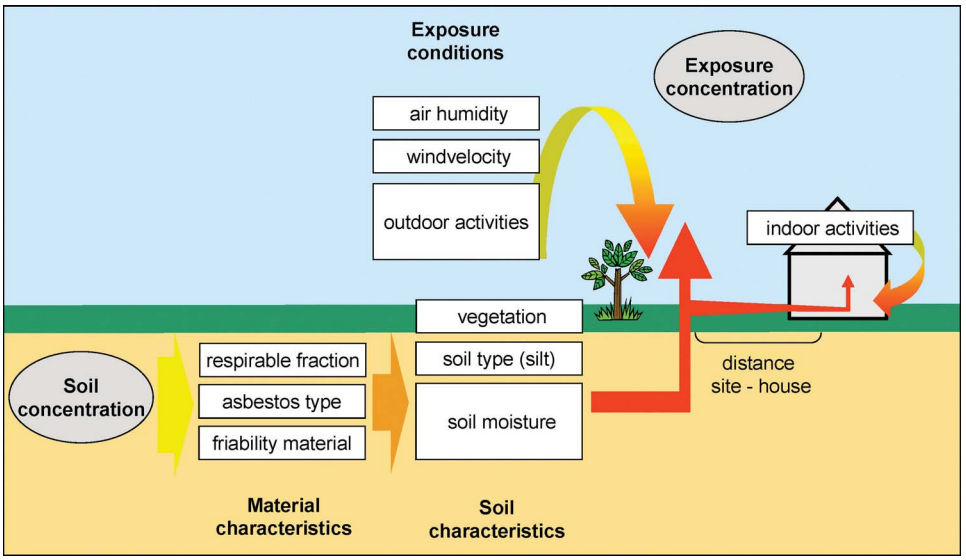

Figure 1. Factors that play a role in the emission of asbestos fibers.

fibers (fibers smaller than 200 μm) (Lee, 1985) in soil. In addition, emission of asbestos fibers depends on a large number of site-specific factors, which can be subdivided in soil characteristics, weather influences, indoor and outdoor activities on the site, and position and extent of the contamination (see Figure 1). The major soil characteristic is the humidity of the soil (Tromp, 2002; Addison *et al.*, 1988).

Two types of exposure are relevant (see also Figure 1), i.e. inhalation of asbestos fibers in outdoor air (*direct exposure*) and inhalation of asbestos fibers in indoor air, after "tracking in" of asbestos fibers, possibly attached to soil particles (*indirect exposure*). The intensity of inhalation depends on residence time, human activity (breathing rate) and the height above soil level where inhalation takes place.

3.2.2. The CSOIL Exposure Model. With the CSOIL model (Van den Berg, 1995; Otte *et al.*, 2001) the exposure of soil-born contaminants to humans that live, work, or recreate on a contaminated site can be calculated. The model is used for the derivation of the human health part of the remediation standards in The Netherlands, i.e. the Intervention Values and, in combination with measurements in the contact media, for the calculation of the site-specific exposure as the basis for the determination of the remediation urgency (Swartjes, 1999). However, there are at least two problems with the calculation of the human exposure to asbestos in soils using CSOIL:

- In CSOIL the calculation of the distribution of contaminants over the soil compartments is based on the fugacity theory (Mackay *et al.*, 1985). Subsequently, transport of volatile contaminants from the pore water phase and the soil gas phase into (indoor and outdoor) air is calculated from convection-diffusion equations. Both elements, the fugacity theory and convective-diffusive transport, are not applicable to asbestos fibers.
- To assess the exposure to asbestos, the influence of the activity on the site and the humidity of the soil on the emission of asbestos into the air should be included at a minimum. However, these factors are not incorporated in the CSOIL exposure model.

Moreover, no reliable quantitative relationships are known between both factors and the respirable fiber concentration in the air.

4. Experimental Data

An inventory has been made of the measured asbestos concentrations in air from own experiments (Tromp, 2002) and data from the literature. The result of this inventory was a database of more than 1000 measured data. These data resulted from *worst case* simulation experiments (simulated activities using a wind blower with dry soil and loose asbestos fibers) and field experiments from daily practice activities (driving on contaminated roads and digging, dumping and sifting of humid soil with a mixture of friable and bound asbestos). During these activities air sampling was performed using both personal air samples (in the breathing zone of the workers) and stationary air samples near the activities. Most of the analyses were performed with scanning electron microscopy in combination with energy dispersive X-ray analysis (SEM/EDX) in conformity with ISO 14966 (ISO, 2002). The *worst case* simulation experiments were performed with known amounts of asbestos in soil. For most of the field experiments the average concentration of asbestos on the site was determined with a visual inspection and a soil sampling program according to the Dutch standard protocol NEN 5707 (NEN, 2003). The soil samples were dried and subsequently divided into sieve fractions. The separate sieve fractions were weighed and spread out into thin layers and inspected by stereomicroscopy for the presence of suspected asbestos containing particles and fiber structures. Using polarized light microscopy, these selected particles were analyzed for asbestos. On the basis of the weight of the sieve fractions and the asbestos containing materials and the estimated asbestos percentage in these materials, the total concentration of asbestos was calculated in $mg/kg_{soil,dw}$.

The results are presented in Figure 2, which shows the airborne asbestos concentration during the simulation experiments and field measurements with friable and bound asbestos (chrysotile and amphibole asbestos), as a function of asbestos concentration in soil. The fiber concentrations in this graph concern average values of several comparable measurements. Also the 95% confidence intervals of the average airborne asbestos concentrations of the comparable measurements are given.

Figure 2 shows that the average asbestos concentration increases with asbestos concentration in soil. However, the 95% confidence intervals are rather large. In the field experiments with friable asbestos, for example, the highest measured airborne concentration at a soil concentration of 100 $mg/kg_{soil,dw}$ is higher than the lowest measured airborne concentration at a soil concentration of 10,000 $mg/kg_{soil,dw}$. In general, the *worse case* simulation experiments give higher airborne asbestos concentrations, while the field measurements with bound asbestos give, according to expectation, relatively low airborne asbestos concentrations. Besides, the following conclusions can be drawn from Figure 2:

- Fiber concentrations in the air exceeding the Maximum Permissible Risk level (100,000 fiber equivalents/m^3_{air}) are only found for highly contaminated soils, i.e. at soil concentration in excess of 10,000 $mg/kg_{soil,dw}$[1] (1%), and materials with

[1]Actually, 100,000 fiber/m^3_{air} is not exceeded at soil concentrations in excess of 100,000 $mg/kg_{soil,dw}$ of asbestos fibers. However, a concentration of 100,000 fiber *equivalents*/m^3_{air} (the Maximum Permissible Risk level) could theoretically be exceeded at a soil concentration of 10,000 $mg/kg_{soil,dw}$ of asbestos, when all asbestos fibers concern amphibole fibers with a length >5 μm).

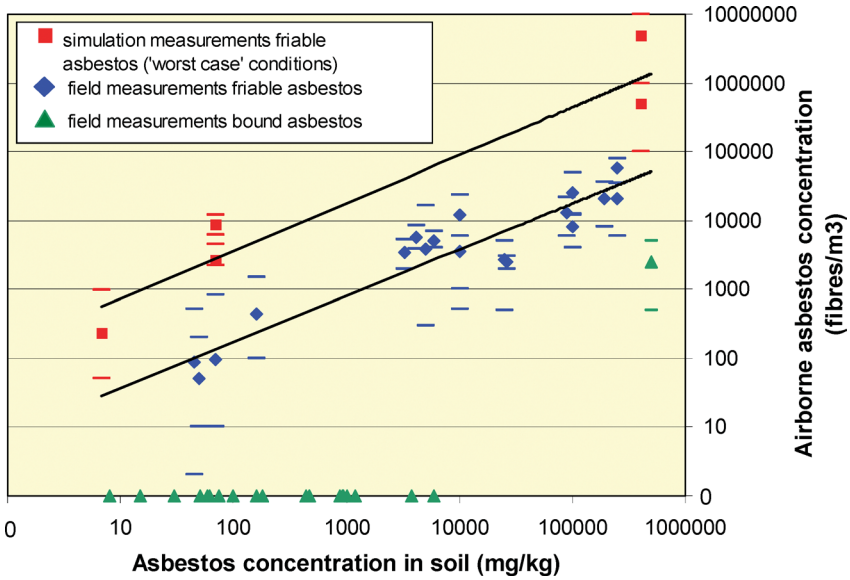

Figure 2. Average airborne asbestos concentrations from several comparable measurements (*symbols*) in fibers/m^3_{air} and 95% confidence intervals (*hyphens*), from *worst case* simulation experiments (*squares*) and from field measurements with friable (*diamonds*) and bound (*triangles*) asbestos, as a function of asbestos concentration in soil. Straight lines represents the 95% confidence intervals of all data.

friable asbestos. In such situations, even the slightest activity in combination with dry air (no *worst case* conditions) is sufficient to exceed the Negligible Risk level in the air (1000 fibre equivalents/m^3_{air}).

- For less contaminated soils with bound asbestos (less than 10,000 mg/$kg_{soil,dw}$ (1%)) no airborne asbestos fibers were found. For less contaminated soils with friable asbestos materials (less than 100 mg/$kg_{soil,dw}$ (0.01%)) the MPR risk level in the air is never exceeded and the NR level in the air is hardly exceeded. The same conclusion holds in case of activities such as digging, dumping and sifting.

In Figure 3 the average airborne asbestos fiber equivalent concentration with increasing distance to the emission source is given, calculated with the emission model PLUIM-PLUS (TNO, 1989), for an asbestos concentration in soil of 100,000 mg/$kg_{soil,dw}$ and a corresponding airborne concentration near the source of approximately 10,000–100,000 fibers equivalents/m^3_{air} (see Figure 2). The horizontal line in Figure 3 at an average airborne fiber concentration of 1000 equivalents/m^3_{air} represents the Negligible Risk level. Although the emission model was validated for gasses, aerosols and particles smaller than 10 μm (PM_{10}), it is assumed to be suited for the calculation of airborne asbestos fibers, because analyses showed that the aerodynamic diameter of airborne fibers is always smaller than 10 μm. This assumption was validated using measurements in air in the vicinity of asbestos industrial activities in the Netherlands (Tempelman *et al.*, 1981). Nevertheless, the PLUIM-PLUS emission model is only used to have an indication of the influence of the distance to the source on the airborne asbestos concentration and not for the derivation of soil quality criteria.

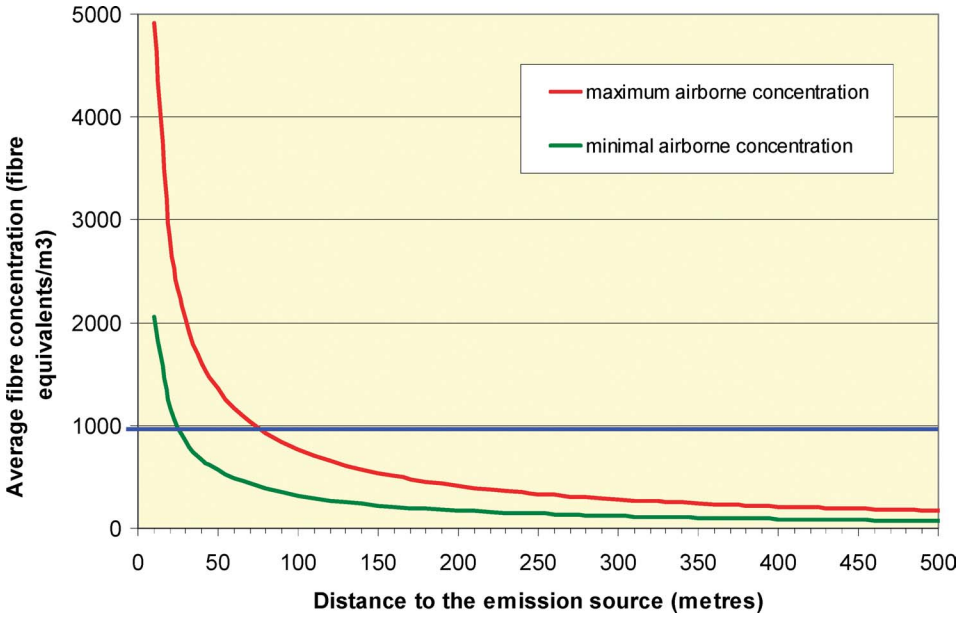

Figure 3. Airborne asbestos fiber equivalent concentration, calculated with the emission model PLUIM-PLUS, with increasing distance to the emission source (asbestos contaminated soil with an asbestos concentration of 100,000 mg asbestos/$kg_{soil,dw}$). The bold horizontal line represents the Negligible Risk level.

From Figure 3 the following conclusions can be drawn:

- The airborne fiber concentration decreases sharply with distance and is generally lower than the Negligible Risk level at a distance of more than circa 100 meters from the source.
- Exceeding of the Maximum Permissible Risk level in air can virtually only be measured close to the asbestos source and with intensive activity, such as digging, dumping and traffic at the site.

Furthermore, experimental data on the relation between soil humidity and the relative asbestos emissions have been evaluated (Tromp, 2002; Addison *et al*., 1988; see Figure 4). Data of three experiments have been included in this graph, i.e. dust cloud simulation experiments with a sandy and an average soil (25% clay and 75% sand) and a laboratory experiment with a sandy soil.

From Figure 4 it can be concluded that the humidity of the soil and the type of soil have a substantial influence on the emission of asbestos fibers. In an outdoor situation in the Netherlands, a typical level of humidity of the soil is approximately 10%. Compared to dry soil, the airborne fiber concentration for a soil with humidity of 10% is more than 1 order of magnitude lower.

5. Generic Soil Standard (Tier 0)

According to the Dutch Soil Protection Act an Intervention Value, being a generic soil quality standard independent of land-use and human behavior on the site, is used to trigger

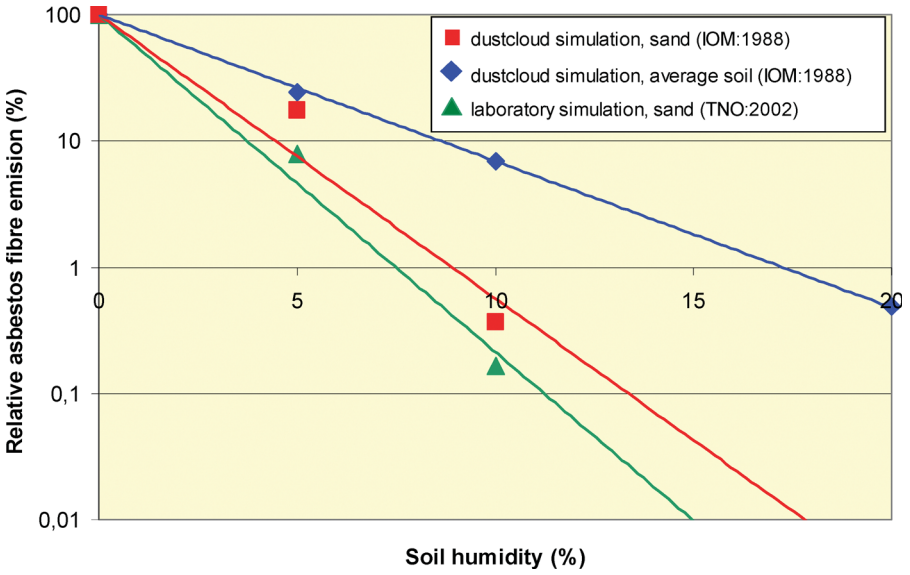

Figure 4. Relative airborne asbestos fiber emission during several simulation experiments with soil with a different level of humidity (data from Tromp, 2002; Addison *et al*., 1988).

possible remediation (Swartjes, 1999). If the resulting average concentration exceeds the Intervention Value the site is labeled as "seriously contaminated." This implies that the site in principle has to be remediated and the urgency of remediation has to be determined on the basis of a site-specific risk assessment.

5.1. Determination of Asbestos in Soil

When the presence of asbestos is suspected, a site survey has to be performed in which the asbestos concentration will be determined. The determination of asbestos in soil has to be performed in conformity with the Dutch standard protocol NEN 5707 (Tromp and Tempelman, 1994; NEN, 2003). All aspects of the soil survey have been included in these Dutch standards, i.e. investigation strategy, visual inspection, soil sampling and laboratory analyses. The survey includes the three following stages:

1. *A preliminary survey*. The objective of the survey is the formulation of a hypothesis about the nature and the spatial distribution of the contamination of asbestos in soil on the basis of historical information.
2. *An exploratory survey*. The objective of this survey is the verification of the hypothesis from the preliminary survey, with little effort of investigation. The survey consists of a visual inspection of the soil surface and random sampling combined with a visual inspection of the sampled soil.
3. *An in-depth survey*. The objective of this survey is the determination of the average concentration of asbestos in soil per spatial unit of 1000 m^2. The extent of the asbestos contamination is determined by means of an extended visual inspection of the soil surface and systematic sampling of the deeper soil layers, followed by laboratory analysis of the sampled soil.

5.2. History

In 1993, a so-called *ad hoc Intervention Value*[2] for asbestos of 100 mg/$kg_{soil,dw}$ up to 2000 mg/$kg_{soil,dw}$[3] (0.01% – 0.2%) was derived in the Netherlands, depending on the type of asbestos. The shape and length of the asbestos fibers were not taken into account. It was concluded that the calculation with the CSOIL exposure model, which was the basis of this ad hoc Intervention Value, must be considered of limited significance. This ad hoc Intervention Value was never formalized. Alternatively, the zero-level was generally accepted in practice. In the Occupational Safety and Health Decree of 1999 (letter to the Dutch Parliament), the residual concentration standard for bound asbestos was increased from 0 to 10 mg/$kg_{soil,dw}$. For friable asbestos the zero-level was maintained. This decision was based on the data available from the results of available experiments at that time, including a safety factor. Subsequently, the residual concentration standard was also declared applicable to the utilization and re-use of soil materials in de *Ministerial Circular on Target and Intervention Values Soil Remediation* (Ministry of VROM, 2000).

5.3. Intervention Value

In 2003 and 2004, an extended analysis of the experimental asbestos data, as described in chapter 4, was performed. From these data it can be concluded that with respect to the "standard" Dutch situation a suitable Intervention Value for friable asbestos is 100 mg/$kg_{soil,dw}$ for the sum of the concentration of chrysotile asbestos (also serpentine asbestos or white asbestos) and 10 times the concentration of amphibole asbestos (other asbestos types). At this concentration, it is unlikely that the Negligible Risk level in air is exceeded, even under worst case conditions. The value of 100 mg/$kg_{soil,dw}$ (asbestos equivalents) was incorporated in the interim policy on asbestos in soils, sediments, dredge materials and demolition waste (granules) (Ministry of VROM, 2002). A "standard" situation implies circumstances under which there are no systematic activities, like digging, dumping or sifting of soil material, and the (top) soil (layer) is relatively wet for most of the year.

In the case of bound asbestos, the concentration in the air will hardly ever exceed the Negligible Risk level. However, because it is difficult to determine when bound asbestos turns into friable asbestos due to aging (weathering and erosion), it was proposed to include this nuance in the *site-specific* risk assessment only. As a consequence, the above-mentioned value of 100 mg/$kg_{soil,dw}$ (asbestos equivalents) is valid for bound as well as for friable asbestos. The value also applies to the residual concentration for the recycling of soil material, dredging and demolition waste (granules) and as criterion for remediation of roads and private property. The Intervention Value of 100 mg/$kg_{soil,dw}$ (asbestos equivalents) was incorporated in the new Dutch soil policy on soil contamination (Ministry of VROM, 2006).

6. Site-Specific Risk Assessment (Tiers 1 to 3)

In this chapter a protocol is described for the assessment of site-specific human health risks in case of soil contamination with asbestos, with the purpose to determine the urgency of

[2]An ad hoc Intervention Value is derived by the National Institute of Public Health and the Environment (RIVM) for a specific case for a contaminant for which no Intervention Value has been incorporated in the Ministerial Circular on Target Values and Intervention Values for soil remediation (Ministry of VROM, 2000). Such an ad hoc Intervention Value is only valid for this specific case and, hence, has a lower status; requirements to the scientific foundation and review procedure for *ad-hoc* Intervention Values are less strict.

[3]In analogy with concentrations of other contaminants, the concentration of asbestos in soil is expressed as weight of asbestos per kilogram dry soil: mg/$kg_{soil,dw}$.

remediation. Analogous to the Remediation Urgency Methodology for other contaminants, an unacceptable site-specific human health risk is assumed, unless there is evidence to the contrary ("risk, unless....") (Swartjes, 1999). The protocol comprises three tiers (Ministry of VROM, 2004; Swartjes *et al*., 2003):

- Tier 1: simple qualitative testing: assessing the potential or probability of human exposure to asbestos;
- Tier 2: determinating and testing the respirable asbestos fraction in soil;
- Tier 3: measuring the concentration of asbestos fibers in outdoor and/or indoor air.

When in a specific tier the human health risks can not be rejected, the assessment in the following tier has to be performed. In case it can not be refuted in Tier 3, there is an unacceptable site-specific human health risk. The underlying principle of the tiered approach is: "simple when possible and complex when necessary."

Since reliable qualitative relations between important site-specific parameters like wind velocity (distribution with depth) and activity on the site with soil to air fiber migration are lacking, no calculation procedures (like human exposure models) have been used in any of the three tiers.

The tiered approach has been incorporated in the new Dutch soil policy on soil contamination with the purpose to determine the urgency of remediation, on the basis of site-specific human health risks (Ministry of VROM, 2006).

6.1. Simple Qualitative Testing (Tier 1)

For simple qualitative testing in Tier 1 it is examined if exposure to asbestos fibers is possible or likely for the specific site. No additional experimental research has to be performed. The Tier 1 assessment can be performed on the basis of the measured asbestos concentration in soils, the condition of asbestos (bound or friable) and the layout of the location (type of soil surface, presence of buildings or vegetation, intended land development). When one or more of the following criteria are met, exposure to asbestos is impossible or unlikely and human health risks can be excluded:

- Asbestos is only present under buildings, paved areas or a water body (in sediment), on condition that no excavation or dredging activities are expected.
- Asbestos is present at a soil depth of more than 0.5 meter, on condition that no excavation activities are expected.
- The site is permanently, year round, completely covered with vegetation.
- For *bound (non friable) asbestos* only: the average soil concentration does not exceed 1,000 mg/$kg_{soil,dw}$ (asbestos equivalents), on condition that the bound materials are not seriously weathered or eroded. The decision on the degree of erosion is subjective. However, in this simple testing procedure in Tier 1 a material is considered non-friable if it cannot be broken manually. For friable asbestos no additional simple testing is performed, because the same soil concentration as the Intervention Value (100 mg/$kg_{soil,dw}$ asbestos equivalents) is used as criterion.

6.2. Determination of the Respirable Fraction in Soil (Tier 2)

Because the inhalation of asbestos fibers is the only relevant exposure route concerning human health, the potential of asbestos fibres in soil for inhalation is investigated in Tier 2, regardless whether or not the fibers actually are emitted into the air. To this purpose, a

distinction is made in *respirable* fibers and all other remaining forms of asbestos in soil. The upper dimension for respirability of fibers corresponds with a fiber diameter of 3 μm and a fiber length of 200 μm (ATSDR, 1995). Fibers smaller than these dimension are contributed to the respirable fraction. Since emission in needed for exposure trough inhalation, the respirable fraction relates to the *potential* site-specific exposure to humans, independent of actual site use or site-specific factors.

The determination of the respirable concentration of asbestos fibers in the upper soil layer takes place in conformity with the Dutch standard protocol NEN 5707 (Tromp and Tempelman, 1994). This standard describes a sedimentation procedure in water, in which the respirable fraction is separated from the coarse particles. Subsequently, the separated fraction is filtrated through a gold coated Nuclepore filter. This filter is analyzed with scanning electron microscopy in combination with energy dispersive X-ray analysis (SEM/EDX) in conformity with ISO 14966 (ISO, 2002).

When "tracking in" of asbestos fibers (attached to soil particles) to the indoor environment cannot be excluded, the amount of asbestos in house dust must additionally be determined. Since it is assumed that bound asbestos degrades by indoor human activity, both asbestos fibers and asbestos conglomerates are measured. The sampling of house dust takes place with adhesive tape on horizontal surfaces with visible dust. The analysis of deposited asbestos fibers in house dust takes place with SEM/EDX in conformity with the Dutch standard protocol NEN 2991 (NEN, 2005). For testing the amount of respirable fibers in soil or dust, a limit value of $4.3 * 10^{10}$ fiber equivalents in soil is used. This value corresponds to a limit value of respirable fibers of 10 $mg/kg_{soil,dw}$ (asbestos equivalents) (Swartjes *et al*., 2003). For testing the amount of asbestos fibers and asbestos conglomerates in indoor dust, a limit value of 100 fibers/cm^2 is used (NEN, 2005).

6.3. Measurement of the Concentration of Asbestos Fibers (Tier 3)

Tier 3 focuses on the actual presence of asbestos fibers in air. To this purpose the asbestos concentration in air has to be measured additionally.

6.3.1. Outdoor Air. For measuring the asbestos fiber concentration in outdoor air, two options are given:

- measurement of the concentration of asbestos fibers in the outdoor air at the site, under "standardized *realistic worst case* circumstances" (Tier $3_{outdoors}$a);
- measurement of the concentration of asbestos fibers in a laboratory simulation, under *worst case* circumstances (availability test; Tier $3_{outdoors}$b).

Measurements in the outdoor air are performed at a height of 1.5 meters above the soil surface, during simulation of "daily practice activities" in the soil (digging, dumping and sifting). A *worst case* location at the site is selected according to the highest asbestos concentration in soil. During measurements the weather has to be dry, just like the three previous days, with a moderate wind speed as a minimum (wind velocity maximal 4 Beaufort).

Measurements in a laboratory simulation are performed in a 1–2 m^3 test chamber. First, a *worst case* sample of the most contaminated location is taken to the laboratory. After drying the sample, the soil is spread out in the test chamber. During measurement, activity is simulated with a fan in such a way that the airspeed on the soil surface amounts to 3–5 meters/second.

Like in Tier 2, analyses of the asbestos fiber concentration are performed with SEM/EDX in conformity with ISO 14966 (ISO, 2002). For testing, a limit value of 1,000 fiber equivalents/m^3_{air} is used, i.e. at the Negligible Risk level.

To facilitate the choice between these options, the advantages and disadvantages of both methods are listed in Swartjes *et al*. (2003). The major advantage of measurements on location is that this represents real life conditions. The biggest disadvantage is that the possibility for performance depends on weather conditions. Another disadvantage is the lack of a standardized protocol for the simulation of "daily practice activities." For measurements during *worst case* laboratory simulations, it is the question if the results are representative for outdoor conditions. On the other hand, the testing procedure is simple, standardized (test results are comparable) and the performance is not hampered by weather conditions.

6.3.2. Indoor Air. The site-specific measurement of the concentration of asbestos fibers in indoor air (Tier $3_{indoors}$) should be performed in houses or other buildings adjacent (no more then 100 meters) to the contaminated site, only when the contamination includes friable asbestos. In that case, the concentration of asbestos fibers in indoor air should be measured under "standardized conditions," in conformity with the Dutch standard protocol NEN 2991 (NEN, 2005) and ISO 14966 (ISO, 2002). Air samplers are located in room units in which an asbestos contamination of house dust is determined. Furthermore, preference is given to room units with high exposure potential (due to the location, accessibility, user activities, etc.). The duration of measurements is 6–8 hours and during measurements simulation of "daily practice activities" are performed. Again, for testing a limit value of 1,000 fiber equivalents/m^3_{air} is used (Negligible Risk level).

7. Conclusions and Recommendations

7.1. Conclusions

A tiered approach for the assessment of human health risks of soil contamination with asbestos has been developed. In Tier 0 a generic soil quality standard (Intervention Value) is derived from measured data. This value is 100 $mg/kg_{soil,dw}$ asbestos equivalents (0.01% by weight), i.e. the sum of the concentration of chrysotile asbestos and ten times the concentration of amphibole asbestos, for bound (non friable) as well as for friable asbestos. Tiers 1 to 3 are site-specific. Tier 1 concerns a simple qualitative testing procedure, in which the potential or probability for emission of asbestos fibers from soil to air is assessed. In Tier 2 the respirable fraction in the soil and house dust, which relates to the *potential* site-specific exposure to humans, is determined and tested. Finally, when the risk can not be excluded, the concentration of asbestos fibers in outdoor and/or indoor air has to be measured and tested according to a standardized procedure, in Tier 3.

7.2. Recommendations

The following recommendations for further research in the future are given:

- An extended (international) database should be created containing soil and air concentrations, information on the type of asbestos and on the measurement conditions. To improve assessment of the emission of asbestos fibers, specific

supplemental measurements should be performed, particularly in the range of 100 en 10,000 mg/$kg_{soil,dw}$(0.01%–1%).

- In order to incorporate the influence of site-specific factors on human-health risks, further research or the relationship between soil and weather characteristics, activity on the site and the emission of asbestos fibers to the air should be investigated. Also the impact of changes in land-use on these factors, and hence on the emission of asbestos fibers to the air, must be investigated. The ultimate goal should be a qualitative description of the influence of these factors. Since it is doubtful whether the derivation of a quantitative description is possible, a feasibility study for the derivation of qualitative and quantitative relationships could be done first.

Furthermore, research should focus on the following topics:

- The influence of "tracking in" of asbestos fibers from soil to the indoor environment (attached to shoes and, to a lesser extent, to clothing) on indoor exposure and the influence of the material and soil characteristics such as soil humidity on this process.
- The transition of bound asbestos to friable asbestos (relevant processes/activities, time span).
- Evaluation of the quality and practical implementation of the measurement procedures for assessment of outdoor concentrations of asbestos fibers (at the site and through laboratory simulation) and of indoor concentrations of asbestos fibers, which are part of Tier 3 of the assessment of site-specific human health risks.

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[Extract: pages 64-65]

OLEM Directive No. 9200.0-90

FRAMEWORK FOR INVESTIGATING ASBESTOS-CONTAMINATED COMPREHENSIVE ENVIRONMENTAL RESPONSE, COMPENSATION AND LIABILITY ACT SITES

PREPARED BY THE
ASBESTOS COMMITTEE OF THE
TECHNICAL REVIEW WORKGROUP
OF THE OFFICE OF LAND AND EMERGENCY MANAGEMENT
U. S. ENVIRONMENTAL PROTECTION AGENCY

2021

7.4 Risk Characterization

The purpose of risk characterization is to summarize and combine outputs of the exposure and toxicity assessments performed within the HHRA to provide a quantitative assessment of site-related risks. The risk characterization step also identifies contamination with concentrations which exceed acceptable levels, defined by the NCP as an excess lifetime cancer risk greater than $1 \times 10^{-6} - 1 \times 10^{-4}$ or an HI greater than 1. Risks that exceed these benchmarks must be highlighted in the HHRA for risk management consideration. The TRW Asbestos Committee website (https://www.epa.gov/superfund/asbestos-superfund-sites-cleanup-examples) has a variety of examples in risk assessments that include these and other exposure scenarios. The table below provides a summary of the risks and hazards identified for the example scenarios illustrated above:

Scenario	Fiber Type	Risk	Hazard
Recreational Exposure - Adult	General Asbestos	5×10^{-5}	N/A
Recreational Exposure - Child	General Asbestos	7×10^{-5}	N/A
Combined Residential Ambient Air and Gardening Exposure - Adult	General Asbestos	1×10^{-4}	N/A
Recreational Exposure - Adult	LAA	4×10^{-5}	N/A
Recreational Exposure - Child	LAA	2×10^{-5}	N/A
Combined Residential Ambient Air and Gardening Exposure - Adult	LAA	1×10^{-4}	N/A
Continuous Exposure (lifetime)	LAA	N/A	9
Continuous Exposure (residential)	LAA	N/A	3
Simultaneous Exposure	LAA	N/A	4
Sequential Exposure	LAA	N/A	5

7.5 Identifying the Air Action Level

OSWER Directive 9345.4-05 (U.S. EPA, 2004) recommends the development of risk-based, site-specific air action levels (*i.e.*, LOCs) to determine if response actions for asbestos in soil/debris should be undertaken. Because inhalation is the exposure pathway of concern for asbestos, an action (or screening) level for asbestos in air is an appropriate metric for site managers in making the determination of whether a response action, no action, or further, more detailed investigation at a given site is warranted.

It is recommended that the action level for asbestos in air be carefully considered to ensure that it is appropriate for the site. As discussed in Section 3.1.2 and 4.3.5, the air action level, or LOC, may be useful in guiding the data collection effort for site investigations as they can support the identification of appropriate detection levels for establishing DQOs. Technical and statistical issues should be carefully considered in determining whether the average air concentration from ABS can be compared to these risk-based action levels for asbestos in air (*e.g.*, it would not be

appropriate to compare air concentrations generated by a short-term ABS scenario, such as raking or lawn mowing, with an air action level which assumes a continuous residential exposure scenario). The following subsections provide a range of air action values that may be useful for different site-specific circumstances, given the toxicity and exposure parameters for the various fiber types previously described.

7.5.1 General Asbestos

A risk-based action level for general asbestos (*e.g.*, chrysotile) in air may be calculated by rearranging the standard risk equation to compute the concentration of asbestos in air that corresponds to a specified risk level for a specified exposure scenario of concern as follows:

$$\text{LOC (s/cc)} = \frac{\text{Target Risk}}{[\text{IUR} \bullet \text{TWF}]} \qquad \text{(Eq. 35)}$$

Example Calculation:
The following site-specific LOC can be calculated using a hypothetical scenario including exposure for 1-hour/day, 156 days/year for 24 years beginning at age 20:

$$\text{TWF} = \text{ET}/24\ \text{hours} \bullet \text{EF}/365\ \text{days}$$
$$= 1\ \text{hour}/24\ \text{hours} \cdot 156\ \text{days}/365\ \text{days} = 0.018$$

$$\text{IUR} = 0.068\ (\text{f/cc})^{-1}\ \text{(from Table H-4)}$$

Assuming a target risk of $1x10^{-6}$:

$$\text{LOC (s/cc)} = 1x10^{-6} / [0.068\ (\text{f/cc})^{-1} \bullet 0.018] = 0.0008\ \text{s/cc}$$

7.5.2 Libby Amphibole Asbestos

For sites where the mineral fibers are determined to be LAA, the LOC can be determined by both cancer risk and non-cancer hazard. The carcinogenic LOC is determined by rearranging the risk equation in the same way shown above for general asbestos. For LAA, however, there is no adjustment for time from first exposure due to the derivation of the Libby IUR.

Example Calculation:
A hypothetical site-specific LOC using the same exposure parameters in the previous example (i.e., exposure for 1-hour/day, 156 days/year for 24 years beginning at age 20) would be calculated as:

$$\text{TWF} = \text{ET}/24\ \text{hours} \bullet \text{EF}/365\ \text{days} \bullet \text{ED}/70\ \text{years}$$
$$= 1\ \text{hour}/24\ \text{hours} \cdot 156\ \text{days}/365\ \text{days} \cdot 24\ \text{years}/70\ \text{years} = 0.0061$$

$$\text{IUR} = 0.17\ (\text{f/cc})^{-1}$$

28. US EPA, 2006. AP-42 Compilation of Air Pollutant Emission Factors

Full Reference: US EPA, 2006. AP-42 Compilation of Air Pollutant Emission Factors, Fifth Edition, Volume 1 Chapter 13: Miscellaneous Sources, US Environmental Protection Agency, 2006

[Extract: pages 13.2-1 – 13.2-2, 13.2.1-1 – 13.2.1-2, 13.2.1-4 – 13.2.1-6, 13.2.1-10, 13.2.2-1 – 13.2.2-5, 13.2.3-1 – 13.2.3-7, 13.2.4-1 – 13.2.4-6, 13.2.5-1 – 13.2.5-15]

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COMPILATION OF AIR POLLUTANT EMISSION FACTORS

VOLUME I: STATIONARY POINT AND AREA SOURCES

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13.2 Fugitive Dust Sources

Significant atmospheric dust arises from the mechanical disturbance of granular material exposed to the air. Dust generated from these open sources is termed "fugitive" because it is not discharged to the atmosphere in a confined flow stream. Common sources of fugitive dust include unpaved roads, agricultural tilling operations, aggregate storage piles, and heavy construction operations.

For the above sources of fugitive dust, the dust-generation process is caused by 2 basic physical phenomena:

1. Pulverization and abrasion of surface materials by application of mechanical force through implements (wheels, blades, etc.).

2. Entrainment of dust particles by the action of turbulent air currents, such as wind erosion of an exposed surface by wind speeds over 19 kilometers per hour (km/hr) (12 miles per hour [mph]).

In this section of AP-42, the principal pollutant of interest is PM-10 — particulate matter (PM) no greater than 10 micrometers in aerodynamic diameter (μmA). Because PM-10 is the size basis for the current primary National Ambient Air Quality Standards (NAAQS) for particulate matter, it represents the particle size range of the greatest regulatory interest. Because formal establishment of PM-10 as the primary standard basis occurred in 1987, many earlier emission tests have been referenced to other particle size ranges, such as:

TSP Total Suspended Particulate, as measured by the standard high-volume ("hi-vol") air sampler, has a relatively coarse size range. TSP was the basis for the previous primary NAAQS for PM and is still the basis of the secondary standard. Wind tunnel studies show that the particle mass capture efficiency curve for the high-volume sampler is very broad, extending from 100 percent capture of particles smaller than 10 μm to a few percent capture of particles as large as 100 μm. Also, the capture efficiency curve varies with wind speed and wind direction, relative to roof ridge orientation. Thus, high-volume samplers do not provide definitive particle size information for emission factors. However, an effective cut point of 30 μm aerodynamic diameter is frequently assigned to the standard high volume sampler.

SP Suspended Particulate, which is often used as a surrogate for TSP, is defined as PM with an aerodynamic diameter no greater than 30 μm. SP may also be denoted as PM-30.

IP Inhalable Particulate is defined as PM with an aerodynamic diameter no greater than 15 μm IP also may be denoted as PM-15.

FP Fine Particulate is defined as PM with an aerodynamic diameter no greater than 2.5 μm. FP may also be denoted as PM-2.5.

The impact of a fugitive dust source on air pollution depends on the quantity and drift potential of the dust particles injected into the atmosphere. In addition to large dust particles that

settle out near the source (often creating a local nuisance problem), considerable amounts of fine particles also are emitted and dispersed over much greater distances from the source. PM-10 represents a relatively fine particle size range and, as such, is not overly susceptible to gravitational settling.

The potential drift distance of particles is governed by the initial injection height of the particle, the terminal settling velocity of the particle, and the degree of atmospheric turbulence. Theoretical drift distance, as a function of particle diameter and mean wind speed, has been computed for fugitive dust emissions. Results indicate that, for a typical mean wind speed of 16 km/hr (10 mph), particles larger than about 100 μm are likely to settle out within 6 to 9 meters (20 to 30 feet [ft]) from the edge of the road or other point of emission. Particles that are 30 to 100 μm in diameter are likely to undergo impeded settling. These particles, depending upon the extent of atmospheric turbulence, are likely to settle within a few hundred feet from the road. Smaller particles, particularly IP, PM-10, and FP, have much slower gravitational settling velocities and are much more likely to have their settling rate retarded by atmospheric turbulence.

Control techniques for fugitive dust sources generally involve watering, chemical stabilization, or reduction of surface wind speed with windbreaks or source enclosures. Watering, the most common and, generally, least expensive method, provides only temporary dust control. The use of chemicals to treat exposed surfaces provides longer dust suppression, but may be costly, have adverse effects on plant and animal life, or contaminate the treated material. Windbreaks and source enclosures are often impractical because of the size of fugitive dust sources.

The reduction of source extent and the incorporation of process modifications or adjusted work practices, both of which reduce the amount of dust generation, are preventive techniques for the control of fugitive dust emissions. These techniques could include, for example, the elimination of mud/dirt carryout on paved roads at construction sites. On the other hand, mitigative measures entail the periodic removal of dust-producing material. Examples of mitigative control measures include clean-up of spillage on paved or unpaved travel surfaces and clean-up of material spillage at conveyor transfer points.

13.2.1 Paved Roads

13.2.1.1 General

Particulate emissions occur whenever vehicles travel over a paved surface such as a road or parking lot. Particulate emissions from paved roads are due to direct emissions from vehicles in the form of exhaust, brake wear and tire wear emissions and resuspension of loose material on the road surface. In general terms, resuspended particulate emissions from paved roads originate from, and result in the depletion of, the loose material present on the surface (i.e., the surface loading). In turn, that surface loading is continuously replenished by other sources. At industrial sites, surface loading is replenished by spillage of material and trackout from unpaved roads and staging areas. Figure 13.2.1-1 illustrates several transfer processes occurring on public streets.

Various field studies have found that public streets and highways, as well as roadways at industrial facilities, can be major sources of the atmospheric particulate matter within an area.[1-9] Of particular interest in many parts of the United States are the increased levels of emissions from public paved roads when the equilibrium between deposition and removal processes is upset. This situation can occur for various reasons, including application of granular materials for snow and ice control, mud/dirt carryout from construction activities in the area, and deposition from wind and/or water erosion of surrounding unstabilized areas. In the absence of continuous addition of fresh material (through localized track out or application of antiskid material), paved road surface loading should reach an equilibrium value in which the amount of material resuspended matches the amount replenished. The equilibrium surface loading value depends upon numerous factors. It is believed that the most important factors are: mean speed of vehicles traveling the road; the average daily traffic (ADT); the number of lanes and ADT per lane; the fraction of heavy vehicles (buses and trucks); and the presence/absence of curbs, storm sewers and parking lanes.[10]

The particulate emission factors presented in a previous version of this section of AP-42, dated October 2002, implicitly included the emissions from vehicles in the form of exhaust, brake wear, and tire wear as well as resuspended road surface material. EPA included these sources in the emission factor equation for paved roads since the field testing data used to develop the equation included both the direct emissions from vehicles and emissions from resuspension of road dust.

This version of the paved road emission factor equation only estimates particulate emissions from resuspended road surface material[28]. The particulate emissions from vehicle exhaust, brake wear, and tire wear are now estimated separately using EPA's MOVES[29] model. This approach eliminates the possibility of double counting emissions. Double counting results when employing the previous version of the emission factor equation in this section and MOVES to estimate particulate emissions from vehicle traffic on paved roads. It also incorporates the decrease in exhaust emissions that has occurred since the paved road emission factor equation was developed. Earlier versions of the paved road emission factor equation includes estimates of emissions from exhaust, brake wear, and tire wear based on emission rates for vehicles in the 1980 calendar year fleet. The amount of PM released from vehicle exhaust has decreased since 1980 due to lower new vehicle emission standards and changes in fuel characteristics.

13.2.1.2 Emissions And Correction Parameters

Dust emissions from paved roads have been found to vary with what is termed the "silt loading" present on the road surface. In addition, the average weight and speed of vehicles traveling the road influence road dust emissions. The term silt loading (sL) refers to the mass of silt-size material (equal to or less than 75 micrometers [µm] in physical diameter) per unit area of the travel surface. The total road surface dust loading consists of loose material that can be collected by broom sweeping and vacuuming of the traveled portion of the paved road. The silt fraction is determined by measuring the proportion of the loose dry surface dust that passes through a 200-mesh screen, using the ASTM-C-136 method. Silt loading is the product of the silt fraction and the total loading, and is abbreviated "sL". Additional details on the sampling and analysis of such material are provided in AP-42 Appendices C.1 and C.2.

The surface sL provides a reasonable means of characterizing seasonal variability in a paved road emission inventory. In many areas of the country, road surface loadings [11-21] are heaviest during the late winter and early spring months when the residual loading from snow/ice controls is greatest. As noted earlier, once replenishment of fresh material is eliminated, the road surface loading can be expected to reach an equilibrium value, which is substantially lower than the late winter/early spring values.

13.2.1.3 Predictive Emission Factor Equations[10,29]

The quantity of particulate emissions from resuspension of loose material on the road surface due to vehicle travel on a dry paved road may be estimated using the following empirical expression:

$$E = k\,(sL)^{0.91} \times (W)^{1.02} \qquad (1)$$

where: E = particulate emission factor (having units matching the units of k),
k = particle size multiplier for particle size range and units of interest (see below),
sL = road surface silt loading (grams per square meter) (g/m^2), and
W = average weight (tons) of the vehicles traveling the road.

It is important to note that Equation 1 calls for the average weight of all vehicles traveling the road. For example, if 99 percent of traffic on the road are 2 ton cars/trucks while the remaining 1 percent consists of 20 ton trucks, then the mean weight "W" is 2.2 tons. More specifically, Equation 1 is *not* intended to be used to calculate a separate emission factor for each vehicle weight class. Instead, only one emission factor should be calculated to represent the "fleet" average weight of all vehicles traveling the road.

The particle size multiplier (k) above varies with aerodynamic size range as shown in Table 13.2.1-1. To determine particulate emissions for a specific particle size range, use the appropriate value of k shown in Table 13.2.1-1.

To obtain the total emissions factor, the emissions factors for the exhaust, brake wear and tire wear obtained from either EPA's MOBILE6.2 [27] or most recent MOVES [29] software model should be added to the emissions factor calculated from the empirical equation.

Table 13.2.1-1. PARTICLE SIZE MULTIPLIERS FOR PAVED ROAD EQUATION

Size range[a]	Particle Size Multiplier k[b]		
	g/VKT	g/VMT	lb/VMT
PM-2.5[c]	0.15	0.25	0.00054
PM-10	0.62	1.00	0.0022
PM-15	0.77	1.23	0.0027
PM-30[d]	3.23	5.24	0.011

[a] Refers to airborne particulate matter (PM-x) with an aerodynamic diameter equal to or less than x micrometers.

[b] Units shown are grams per vehicle kilometer traveled (g/VKT), grams per vehicle mile traveled (g/VMT), and pounds per vehicle mile traveled (lb/VMT). The multiplier k includes unit conversions to produce emission factors in the units shown for the indicated size range from the mixed units required in Equation 1.

[c] The k-factors for $PM_{2.5}$ were based on the average $PM_{2.5}$:PM_{10} ratio of test runs in Reference 30.

[d] PM-30 is sometimes termed "suspendable particulate" (SP) and is often used as a surrogate for TSP.

Equation 1 is based on a regression analysis of 83 tests for PM-10.[3, 5-6, 8, 27-29, 31-36] Sources tested include public paved roads, as well as controlled and uncontrolled industrial paved roads. The majority of tests involved freely flowing vehicles traveling at constant speed on relatively level roads. However, 22 tests of slow moving or "stop-and-go" traffic or vehicles under load were available for inclusion in the data base.[32-36] Engine exhaust, tire wear and break wear were subtracted from the emissions measured in the test programs prior to stepwise regression to determine Equation 1.[37, 39] The equations retain the quality rating of A (D for PM-2.5), if applied within the range of source conditions that were tested in developing the equation as follows:

Silt loading:	0.03 - 400 g/m^2 0.04 - 570 grains/square foot (ft^2)
Mean vehicle weight:	1.8 - 38 megagrams (Mg) 2.0 - 42 tons
Mean vehicle speed:	1 - 88 kilometers per hour (kph) 1 - 55 miles per hour (mph)

The upper and lower 95% confidence levels of equation 1 for PM_{10} is best described with equations using an exponents of 1.14 and 0.677 for silt loading and an exponents of 1.19 and 0.85 for weight. Users are cautioned that application of equation 1 outside of the range of variables and operating conditions specified above, e.g., application to roadways or road networks with speeds above 55 mph and average vehicle weights of 42 tons, will result in emission estimates with a higher level of uncertainty. In these situations, users are encouraged to consider an assessment of the impacts of the influence of extrapolation to the overall emissions and alternative methods that are equally or more plausible in light of local emissions data and/or ambient concentration or compositional data.

To retain the quality rating for the emission factor equation when it is applied to a specific paved road, it is necessary that reliable correction parameter values for the specific road in question be determined. With the exception of limited access roadways, which are difficult to sample, the collection and use of site-specific silt loading (sL) data for public paved road emission inventories are strongly recommended. The field and laboratory procedures for determining surface material silt content and surface dust loading are summarized in Appendices C.1 and C.2. In the event that site-specific values cannot be obtained, an appropriate value for a paved public road may be selected from the values in Table 13.2.1-2, but the quality rating of the equation should be reduced by 2 levels.

Equation 1 may be extrapolated to average uncontrolled conditions (but including natural mitigation) under the simplifying assumption that annual (or other long-term) average emissions are inversely proportional to the frequency of measurable (> 0.254 mm [0.01 inch]) precipitation by application of a precipitation correction term. The precipitation correction term can be applied on a daily or an hourly basis [26, 38].

For the daily basis, Equation 1 becomes:

$$E_{ext} = [\, k\,(sL)^{0.91} \times (W)^{1.02} \,]\,(1 - P/4N) \qquad (2)$$

where k, sL, W, and S are as defined in Equation 1 and

E_{ext} = annual or other long-term average emission factor in the same units as k,

P = number of "wet" days with at least 0.254 mm (0.01 in) of precipitation during the averaging period, and

N = number of days in the averaging period (e.g., 365 for annual, 91 for seasonal, 30 for monthly).

Note that the assumption leading to Equation 2 is based on analogy with the approach used to develop long-term average unpaved road emission factors in Section 13.2.2. However, Equation 2 above incorporates an additional factor of "4" in the denominator to account for the fact that paved roads dry more quickly than unpaved roads and that the precipitation may not occur over the complete 24-hour day.

For the hourly basis, equation 1 becomes:

$$E_{ext} = [\, k\,(sL)^{0.91} \times (W)^{1.02} \,]\,(1 - 1.2P/N) \qquad (3)$$

where k, sL, W, and S are as defined in Equation 1 and

E_{ext} = annual or other long-term average emission factor in the same units as k,

P = number of hours with at least 0.254 mm (0.01 in) of precipitation during the averaging period, and

N = number of hours in the averaging period (e.g., 8760 for annual, 2124 for season 720 for monthly)

Note: In the hourly moisture correction term (1-1.2P/N) for equation 3, the 1.2 multiplier is applied to account for the residual mitigative effect of moisture. For most applications, this equation will produce satisfactory results. Users should select a time interval to include sufficient "dry" hours such that a reasonable emissions averaging period is evaluated. For the special case where this equation is used to calculate emissions on an hour by hour basis, such as would be done in some emissions modeling situations, the moisture correction term should be modified so that the moisture correction "credit" is applied to the first hours following cessation of precipitation. In this special case, it is suggested that this 20% "credit" be applied on a basis of one hour credit for each hour of precipitation up to a maximum of 12 hours.

Note that the assumption leading to Equation 3 is based on analogy with the approach used to develop long-term average unpaved road emission factors in Section 13.2.2.

Figure 13.2.1-2 presents the geographical distribution of "wet" days on an annual basis for the United States. Maps showing this information on a monthly basis are available in the *Climatic Atlas of the United States*[23]. Alternative sources include other Department of Commerce publications (such as local climatological data summaries). The National Climatic Data Center (NCDC) offers several products that provide hourly precipitation data. In particular, NCDC offers *Solar and Meteorological Surface Observation Network 1961-1990* (SAMSON) CD-ROM, which contains 30 years worth of hourly meteorological data for first-order National Weather Service locations. Whatever meteorological data are used, the source of that data and the averaging period should be clearly specified.

It is emphasized that the simple assumption underlying Equations 2 and 3 has not been verified in any rigorous manner. For that reason, the quality ratings for Equations 2 and 3 should be downgraded one letter from the rating that would be applied to Equation 1.

Table 13.2.1-3 (Metric And English Units). TYPICAL SILT CONTENT AND LOADING VALUES FOR PAVED ROADS AT INDUSTRIAL FACILITIES [a]

Industry	No. of Sites	No. Of Samples	Silt Content (%)		No. of Travel Lanes	Total Loading x 10^{-3}			Silt Loading (g/m^2)	
			Range	Mean		Range	Mean	Units[b]	Range	Mean
Copper smelting	1	3	15.4-21.7	19.0	2	12.9 - 19.5 45.8 - 69.2	15.9 55.4	kg/km lb/mi	188-400	292
Iron and steel production	9	48	1.1-35.7	12.5	2	0.006 - 4.77 0.020 -16.9	0.495 1.75	kg/km lb/mi	0.09-79	9.7
Asphalt batching	1	3	2.6 - 4.6	3.3	1	12.1 - 18.0 43.0 - 64.0	14.9 52.8	kg/km lb/mi	76-193	120
Concrete batching	1	3	5.2 - 6.0	5.5	2	1.4 - 1.8 5.0 - 6.4	1.7 5.9	kg/km lb/mi	11-12	12
Sand and gravel processing	1	3	6.4 - 7.9	7.1	1	2.8 - 5.5 9.9 - 19.4	3.8 13.3	kg/km lb/mi	53-95	70
Municipal solid waste landfill	2	7		-	2	-			1.1-32.0	7.4
Quarry	1	6		-	2	-			2.4-14	8.2
Corn wet mills	3	15		-	2	-			0.05 – 2.9	1.1

[a] References 1-2,5-6,11-13. Values represent samples collected from *industrial* roads. Public road silt loading values are presented in Table-13.2.1-2. Dashes indicate information not available. [b] Multiply entries by 1000 to obtain stated units; kilograms per kilometer (kg/km) and pounds per mile (lb/mi).

13.2.2 Unpaved Roads

13.2.2.1 General

When a vehicle travels an unpaved road, the force of the wheels on the road surface causes pulverization of surface material. Particles are lifted and dropped from the rolling wheels, and the road surface is exposed to strong air currents in turbulent shear with the surface. The turbulent wake behind the vehicle continues to act on the road surface after the vehicle has passed.

The particulate emission factors presented in the previous draft version of this section of AP-42, dated October 2001, implicitly included the emissions from vehicles in the form of exhaust, brake wear, and tire wear as well as resuspended road surface material[25]. EPA included these sources in the emission factor equation for unpaved public roads (equation 1b in this section) since the field testing data used to develop the equation included both the direct emissions from vehicles and emissions from resuspension of road dust.

This version of the unpaved public road emission factor equation only estimates particulate emissions from resuspended road surface material [23, 26]. The particulate emissions from vehicle exhaust, brake wear, and tire wear are now estimated separately using EPA's MOBILE6.2 [24]. This approach eliminates the possibility of double counting emissions. Double counting results when employing the previous version of the emission factor equation in this section and MOBILE6.2 to estimate particulate emissions from vehicle traffic on unpaved public roads. It also incorporates the decrease in exhaust emissions that has occurred since the unpaved public road emission factor equation was developed. The previous version of the unpaved public road emission factor equation includes estimates of emissions from exhaust, brake wear, and tire wear based on emission rates for vehicles in the 1980 calendar year fleet. The amount of PM released from vehicle exhaust has decreased since 1980 due to lower new vehicle emission standards and changes in fuel characteristics.

13.2.2.2 Emissions Calculation And Correction Parameters[1-6]

The quantity of dust emissions from a given segment of unpaved road varies linearly with the volume of traffic. Field investigations also have shown that emissions depend on source parameters that characterize the condition of a particular road and the associated vehicle traffic. Characterization of these source parameters allow for "correction" of emission estimates to specific road and traffic conditions present on public and industrial roadways.

Dust emissions from unpaved roads have been found to vary directly with the fraction of silt (particles smaller than 75 micrometers [μm] in diameter) in the road surface materials.[1] The silt fraction is determined by measuring the proportion of loose dry surface dust that passes a 200-mesh screen, using the ASTM-C-136 method. A summary of this method is contained in Appendix C of AP-42. Table 13.2.2-1 summarizes measured silt values for industrial unpaved roads. Table 13.2.2-2 summarizes measured silt values for public unpaved roads. It should be noted that the ranges of silt content vary over two orders of magnitude. Therefore, the use of data from this table can potentially introduce considerable error. Use of this data is strongly discouraged when it is feasible to obtain locally gathered data.

Since the silt content of a rural dirt road will vary with geographic location, it should be measured for use in projecting emissions. As a conservative approximation, the silt content of the parent soil in the area can be used. Tests, however, show that road silt content is normally lower than in the surrounding parent soil, because the fines are continually removed by the vehicle traffic, leaving a higher percentage of coarse particles.

Other variables are important in addition to the silt content of the road surface material. For example, at industrial sites, where haul trucks and other heavy equipment are common, emissions are highly correlated with vehicle weight. On the other hand, there is far less variability in the weights of cars and pickup trucks that commonly travel publicly accessible unpaved roads throughout the United States. For those roads, the moisture content of the road surface material may be more dominant in determining differences in emission levels between, for example a hot, desert environment and a cool, moist location.

The PM-10 and TSP emission factors presented below are the outcomes from stepwise linear regressions of field emission test results of vehicles traveling over unpaved surfaces. Due to a limited amount of information available for PM-2.5, the expression for that particle size range has been scaled against the result for PM-10. Consequently, the quality rating for the PM-2.5 factor is lower than that for the PM-10 expression.

Table 13.2.2-1. TYPICAL SILT CONTENT VALUES OF SURFACE MATERIAL ON INDUSTRIAL UNPAVED ROADS[a]

Industry	Road Use Or Surface Material	Plant Sites	No. Of Samples	Silt Content (%)	
				Range	Mean
Copper smelting	Plant road	1	3	16 - 19	17
Iron and steel production	Plant road	19	135	0.2 - 19	6.0
Sand and gravel processing	Plant road	1	3	4.1 - 6.0	4.8
	Material storage area	1	1	-	7.1
Stone quarrying and processing	Plant road	2	10	2.4 - 16	10
	Haul road to/from pit	4	20	5.0-15	8.3
Taconite mining and processing	Service road	1	8	2.4 - 7.1	4.3
	Haul road to/from pit	1	12	3.9 - 9.7	5.8
Western surface coal mining	Haul road to/from pit	3	21	2.8 - 18	8.4
	Plant road	2	2	4.9 - 5.3	5.1
	Scraper route	3	10	7.2 - 25	17
	Haul road (freshly graded)	2	5	18 - 29	24
Construction sites	Scraper routes	7	20	0.56-23	8.5
Lumber sawmills	Log yards	2	2	4.8-12	8.4
Municipal solid waste landfills	Disposal routes	4	20	2.2 - 21	6.4

[a]References 1,5-15.

The following empirical expressions may be used to estimate the quantity in pounds (lb) of size-specific particulate emissions from an unpaved road, per vehicle mile traveled (VMT):

For vehicles traveling on unpaved surfaces at industrial sites, emissions are estimated from the following equation:

$$E = k\,(s/12)^a (W/3)^b \quad (1a)$$

and, for vehicles traveling on publicly accessible roads, dominated by light duty vehicles, emissions may be estimated from the following:

$$E = \frac{k\,(s/12)^a (S/30)^d}{(M/0.5)^c} - C \quad (1b)$$

where k, a, b, c and d are empirical constants (Reference 6) given below and

E = size-specific emission factor (lb/VMT)
s = surface material silt content (%)
W = mean vehicle weight (tons)
M = surface material moisture content (%)
S = mean vehicle speed (mph)
C = emission factor for 1980's vehicle fleet exhaust, brake wear and tire wear.

The source characteristics s, W and M are referred to as correction parameters for adjusting the emission estimates to local conditions. The metric conversion from lb/VMT to grams (g) per vehicle kilometer traveled (VKT) is as follows:

1 lb/VMT = 281.9 g/VKT

The constants for Equations 1a and 1b based on the stated aerodynamic particle sizes are shown in Tables 13.2.2-2 and 13.2.2-4. The PM-2.5 particle size multipliers (k-factors) are taken from Reference 27.

Table 13.2.2-2. CONSTANTS FOR EQUATIONS 1a AND 1b

Constant	Industrial Roads (Equation 1a)			Public Roads (Equation 1b)		
	PM-2.5	PM-10	PM-30*	PM-2.5	PM-10	PM-30*
k (lb/VMT)	0.15	1.5	4.9	0.18	1.8	6.0
a	0.9	0.9	0.7	1	1	1
b	0.45	0.45	0.45	-	-	-
c	-	-	-	0.2	0.2	0.3
d	-	-	-	0.5	0.5	0.3
Quality Rating	B	B	B	B	B	B

*Assumed equivalent to total suspended particulate matter (TSP)
"-" = not used in the emission factor equation

Table 13.2.2-2 also contains the quality ratings for the various size-specific versions of Equation 1a and 1b. The equation retains the assigned quality rating, if applied within the ranges of source conditions, shown in Table 13.2.2-3, that were tested in developing the equation:

Table 13.2.2-3. RANGE OF SOURCE CONDITIONS USED IN DEVELOPING EQUATION 1a AND 1b

Emission Factor	Surface Silt Content, %	Mean Vehicle Weight		Mean Vehicle Speed		Mean No. of Wheels	Surface Moisture Content, %
		Mg	ton	km/hr	mph		
Industrial Roads (Equation 1a)	1.8-25.2	1.8-260	2-290	8-69	5-43	4-17[a]	0.03-13
Public Roads (Equation 1b)	1.8-35	1.4-2.7	1.5-3	16-88	10-55	4-4.8	0.03-13

[a] See discussion in text.

As noted earlier, the models presented as Equations 1a and 1b were developed from tests of traffic on unpaved surfaces. Unpaved roads have a hard, generally nonporous surface that usually dries quickly after a rainfall or watering, because of traffic-enhanced natural evaporation. (Factors influencing how fast a road dries are discussed in Section 13.2.2.3, below.) The quality ratings given above pertain to the mid-range of the measured source conditions for the equation. A higher mean vehicle weight and a higher than normal traffic rate may be justified when performing a worst-case analysis of emissions from unpaved roads.

The emission factors for the exhaust, brake wear and tire wear of a 1980's vehicle fleet (*C*) was obtained from EPA's MOBILE6.2 model [23]. The emission factor also varies with aerodynamic size range

13.2.3 Heavy Construction Operations

13.2.3.1 General

Heavy construction is a source of dust emissions that may have substantial temporary impact on local air quality. Building and road construction are 2 examples of construction activities with high emissions potential. Emissions during the construction of a building or road can be associated with land clearing, drilling and blasting, ground excavation, cut and fill operations (i.e., earth moving), and construction of a particular facility itself. Dust emissions often vary substantially from day to day, depending on the level of activity, the specific operations, and the prevailing meteorological conditions. A large portion of the emissions results from equipment traffic over temporary roads at the construction site.

The temporary nature of construction differentiates it from other fugitive dust sources as to estimation and control of emissions. Construction consists of a series of different operations, each with its own duration and potential for dust generation. In other words, emissions from any single construction site can be expected (1) to have a definable beginning and an end and (2) to vary substantially over different phases of the construction process. This is in contrast to most other fugitive dust sources, where emissions are either relatively steady or follow a discernable annual cycle. Furthermore, there is often a need to estimate areawide construction emissions, without regard to the actual plans of any individual construction project. For these reasons, following are methods by which either areawide or site-specific emissions may be estimated.

13.2.3.2 Emissions And Correction Parameters

The quantity of dust emissions from construction operations is proportional to the area of land being worked and to the level of construction activity. By analogy to the parameter dependence observed for other similar fugitive dust sources,[1] one can expect emissions from heavy construction operations to be positively correlated with the silt content of the soil (that is, particles smaller than 75 micrometers [µm] in diameter), as well as with the speed and weight of the average vehicle, and to be negatively correlated with the soil moisture content.

13.2.3.3 Emission Factors

Only 1 set of field studies has been performed that attempts to relate the emissions from construction directly to an emission factor.[1-2] Based on field measurements of total suspended particulate (TSP) concentrations surrounding apartment and shopping center construction projects, the approximate emission factors for construction activity operations are:

E = 2.69 megagrams (Mg)/hectare/month of activity
E = 1.2 tons/acre/month of activity

These values are most useful for developing estimates of overall emissions from construction scattered throughout a geographical area. The value is most applicable to construction operations with: (1) medium activity level, (2) moderate silt contents, and (3) semiarid climate. Test data were not sufficient to derive the specific dependence of dust emissions on correction parameters. Because the above emission factor is referenced to TSP, use of this factor to estimate particulate matter (PM) no greater than 10 µm in aerodynamic diameter (PM-10) emissions will result in conservatively high

estimates. Also, because derivation of the factor assumes that construction activity occurs 30 days per month, the above estimate is somewhat conservatively high for TSP as well.

Although the equation above represents a relatively straightforward means of preparing an areawide emission inventory, at least 2 features limit its usefulness for specific construction sites. First, the conservative nature of the emission factor may result in too high an estimate for PM-10 to be of much use for a specific site under consideration. Second, the equation provides neither information about which particular construction activities have the greatest emission potential nor guidance for developing an effective dust control plan.

For these reasons, it is strongly recommended that when emissions are to be estimated for a particular construction site, the construction process be broken down into component operations. (Note that many general contractors typically employ planning and scheduling tools, such as critical path method [CPM], that make use of different sequential operations to allocate resources.) This approach to emission estimation uses a unit or phase method to consider the more basic dust sources of vehicle travel and material handling. That is to say, the construction project is viewed as consisting of several operations, each involving traffic and material movements, and emission factors from other AP-42 sections are used to generate estimates. Table 13.2.3-1 displays the dust sources involved with construction, along with the recommended emission factors.[3]

In addition to the on-site activities shown in Table 13.2.3-1, substantial emissions are possible because of material tracked out from the site and deposited on adjacent paved streets. Because all traffic passing the site (i. e., not just that associated with the construction) can resuspend the deposited material, this "secondary" source of emissions may be far more important than all the dust sources actually within the construction site. Furthermore, this secondary source will be present during all construction operations. Persons developing construction site emission estimates must consider the potential for increased adjacent emissions from off-site paved roadways (see Section 13.2.1, "Paved Roads"). High wind events also can lead to emissions from cleared land and material stockpiles. Section 13.2.5, "Industrial Wind Erosion", presents an estimation methodology that can be used for such sources at construction sites.

13.2.3.4 Control Measures[4]

Because of the relatively short-term nature of construction activities, some control measures are more cost effective than others. Wet suppression and wind speed reduction are 2 common methods used to control open dust sources at construction sites, because a source of water and material for wind barriers tend to be readily available on a construction site. However, several other forms of dust control are available.

Table 13.2.3-2 displays each of the preferred control measures, by dust source.[3-4] Because most of the controls listed in the table modify independent variables in the emission factor models, the effectiveness can be calculated by comparing controlled and uncontrolled emission estimates from Table 13.2.3-1. Additional guidance on controls is provided in the AP-42 sections from which the recommended emission factors were taken, as well as in other documents, such as Reference 4.

Table 13.2.3-1. RECOMMENDED EMISSION FACTORS FOR CONSTRUCTION OPERATIONS[a]

Construction Phase	Dust-generating Activities	Recommended Emission Factor	Comments	Rating Adjustment[b]
I. Demolition and debris removal	1. Demolition of buildings or other (natural) obstacles such as trees, boulders, etc.			
	a. Mechanical dismemberment ("headache ball") of existing structures	NA		—
	b. Implosion of existing structures	NA		—
	c. Drilling and blasting of soil	Drilling factor in Table 11.9-4		-1
		Blasting factor NA	Blasting factor in Tables 11.9-1 and 11.9-2 not considered appropriate for general construction activities	NA
	d. General land clearing	Dozer equation (overburden) in Tables 11.9-1 and 11.9-2		-1/-2[c]
	2. Loading of debris into trucks	Material handling emission factor equation in Section 13.2.4		-0/-1[c]
	3. Truck transport of debris	Unpaved road emission factor in Section 13.2.2, or paved road emission factor in Section 13.2.1		-0/-1[c]
	4. Truck unloading of debris	Material handling emission factor equation in Section 13.2.4	May occur offsite	-0/-1[c]

Table 13.2.3-1 (cont.).

Construction Phase	Dust-generating Activities	Recommended Emission Factor	Comments	Rating Adjustment[b]
II. Site Preparation (earth moving)	1. Bulldozing	Dozer equation (overburden) in Tables 11.9-1 and 11.9-2		-1/-2[c]
	2. Scrapers unloading topsoil	Scraper unloading factor in Table 11.9-4		-1
	3. Scrapers in travel	Scraper (travel mode) expression in Tables 11.9-1 and 11.9-2		-0/-1[c]
	4. Scrapers removing topsoil	5.7 kg/vehicle kilometer traveled (VKT) (20.2 lb/vehicle mile traveled [VMT])		E[d]
	5. Loading of excavated material into trucks	Material handling emission factor equation in Section 13.2.4		-0/-1[c]
	6. Truck dumping of fill material, road base, or other materials	Material handling emission factor equation in Section 13.2.4	May occur offsite	-0/-1[c]
	7. Compacting	Dozer equation in Tables 11.9-1 and 11.9-2	Emission factor downgraded because of differences in operating equipment	-1/-2[c]
	8. Motor grading	Grading equation in Tables 11.9-1 and 11.9-2		-1/-2[c]

Table 13.2.3-1 (cont.).

Construction Phase	Dust-generating Activities	Recommended Emission Factor	Comments	Rating Adjustment[b]
III. General Construction	1. Vehicular traffic	Unpaved road emission factor in Section 13.2.2, or paved road emission factor in Section 13.2.1		-0/-1[c] -0/-1[c]
	2. Portable plants			
	a. Crushing	Factors for similar material/operations in Section 11.19.2		-1/-2[c]
	b. Screening	Factors for similar material/operations in Section 11.19.2		-1/-2[c]
	c. Material transfers	Material handling emission factor equation in Section 13.2.4		-0/-1[c]
	3. Other operations	Factors for similar material/operations in the Mineral Products Industry, Chapter 11 of this document		—

[a] NA = not applicable.
[b] Refers to how many additional letters the emission factor should be downrated (beyond the guidance given in the other sections of AP-42) for application to construction activities. For example, "-2" means that an A-rated factor should be considered of C quality in estimating construction emissions. All emission factors assumed to have site-specific input values; otherwise, additional downgrading of one letter should be employed. Note that no rating can be lower than E.
[c] First value for cases with independent variables within range given in AP-42 section; second value for cases with at least 1 variable outside the range.
[d] Rating for emission factor given. Reference 5.
[e] In the event that individual operations cannot be identified, one may very conservatively overestimate PM-10 emissions by using Equation 1.

Table 13.2.3-2. CONTROL OPTIONS FOR GENERAL CONSTRUCTION OPEN SOURCES OF PM-10

Emission Source	Recommended Control Method(s)
Debris handling	Wind speed reduction Wet suppression[a]
Truck transport[b]	Wet suppression Paving Chemical stabilization[c]
Bulldozers	Wet suppression[d]
Pan scrapers	Wet suppression of travel routes
Cut/fill material handling	Wind speed reduction Wet suppression
Cut/fill haulage	Wet suppression Paving Chemical stabilization
General construction	Wind speed reduction Wet suppression Early paving of permanent roads

[a] Dust control plans should contain precautions against watering programs that confound trackout problems.

[b] Loads could be covered to avoid loss of material in transport, especially if material is transported offsite.

[c] Chemical stabilization usually cost-effective for relatively long-term or semipermanent unpaved roads.

[d] Excavated materials may already be moist and not require additional wetting. Furthermore, most soils are associated with an "optimum moisture" for compaction.

References For Section 13.2.3

1. C. Cowherd, Jr., *et al., Development Of Emissions Factors For Fugitive Dust Sources*, EPA-450/3-74-03, U. S. Environmental Protection Agency, Research Triangle Park, NC, June 1974.

2. G. A. Jutze, *et al., Investigation Of Fugitive Dust Sources Emissions And Control*, EPA-450/3-74-036a, U. S. Environmental Protection Agency, Research Triangle Park, NC, June 1974.

3. *Background Documentation For AP-42 Section 11.2.4, Heavy Construction Operations*, EPA Contract No. 69-D0-0123, Midwest Research Institute, Kansas City, MO, April 1993.

4. C. Cowherd, *et al., Control Of Open Fugitive Dust Sources*, EPA-450/3-88-008, U. S. Environmental Protection Agency, Research Triangle Park, NC, September 1988.

5. M. A. Grelinger, *et al., Gap Filling PM-10 Emission Factors For Open Area Fugitive Dust Sources*, EPA-450/4-88-003, U. S. Environmental Protection Agency, Research Triangle Park, NC, March 1988.

13.2.4 Aggregate Handling And Storage Piles

13.2.4.1 General

Inherent in operations that use minerals in aggregate form is the maintenance of outdoor storage piles. Storage piles are usually left uncovered, partially because of the need for frequent material transfer into or out of storage.

Dust emissions occur at several points in the storage cycle, such as material loading onto the pile, disturbances by strong wind currents, and loadout from the pile. The movement of trucks and loading equipment in the storage pile area is also a substantial source of dust.

13.2.4.2 Emissions And Correction Parameters

The quantity of dust emissions from aggregate storage operations varies with the volume of aggregate passing through the storage cycle. Emissions also depend on 3 parameters of the condition of a particular storage pile: age of the pile, moisture content, and proportion of aggregate fines.

When freshly processed aggregate is loaded onto a storage pile, the potential for dust emissions is at a maximum. Fines are easily disaggregated and released to the atmosphere upon exposure to air currents, either from aggregate transfer itself or from high winds. As the aggregate pile weathers, however, potential for dust emissions is greatly reduced. Moisture causes aggregation and cementation of fines to the surfaces of larger particles. Any significant rainfall soaks the interior of the pile, and then the drying process is very slow.

Silt (particles equal to or less than 75 micrometers [μm] in diameter) content is determined by measuring the portion of dry aggregate material that passes through a 200-mesh screen, using ASTM-C-136 method.[1] Table 13.2.4-1 summarizes measured silt and moisture values for industrial aggregate materials.

Table 13.2.4-1. TYPICAL SILT AND MOISTURE CONTENTS OF MATERIALS AT VARIOUS INDUSTRIES[a]

Industry	No. Of Facilities	Material	Silt Content (%)			Moisture Content (%)		
			No. Of Samples	Range	Mean	No. Of Samples	Range	Mean
Iron and steel production	9	Pellet ore	13	1.3 - 13	4.3	11	0.64 - 4.0	2.2
		Lump ore	9	2.8 - 19	9.5	6	1.6 - 8.0	5.4
		Coal	12	2.0 - 7.7	4.6	11	2.8 - 11	4.8
		Slag	3	3.0 - 7.3	5.3	3	0.25 - 2.0	0.92
		Flue dust	3	2.7 - 23	13	1	—	7
		Coke breeze	2	4.4 - 5.4	4.9	2	6.4 - 9.2	7.8
		Blended ore	1	—	15	1	—	6.6
		Sinter	1	—	0.7	0	—	—
		Limestone	3	0.4 - 2.3	1.0	2	ND	0.2
Stone quarrying and processing	2	Crushed limestone	2	1.3 - 1.9	1.6	2	0.3 - 1.1	0.7
		Various limestone products	8	0.8 - 14	3.9	8	0.46 - 5.0	2.1
Taconite mining and processing	1	Pellets	9	2.2 - 5.4	3.4	7	0.05 - 2.0	0.9
		Tailings	2	ND	11	1	—	0.4
Western surface coal mining	4	Coal	15	3.4 - 16	6.2	7	2.8 - 20	6.9
		Overburden	15	3.8 - 15	7.5	0	—	—
		Exposed ground	3	5.1 - 21	15	3	0.8 - 6.4	3.4
Coal-fired power plant	1	Coal (as received)	60	0.6 - 4.8	2.2	59	2.7 - 7.4	4.5
Municipal solid waste landfills	4	Sand	1	—	2.6	1	—	7.4
		Slag	2	3.0 - 4.7	3.8	2	2.3 - 4.9	3.6
		Cover	5	5.0 - 16	9.0	5	8.9 - 16	12
		Clay/dirt mix	1	—	9.2	1	—	14
		Clay	2	4.5 - 7.4	6.0	2	8.9 - 11	10
		Fly ash	4	78 - 81	80	4	26 - 29	27
		Misc. fill materials	1	—	12	1	—	11

[a] References 1-10. ND = no data.

13.2.4.3 Predictive Emission Factor Equations

Total dust emissions from aggregate storage piles result from several distinct source activities within the storage cycle:

1. Loading of aggregate onto storage piles (batch or continuous drop operations).
2. Equipment traffic in storage area.
3. Wind erosion of pile surfaces and ground areas around piles.
4. Loadout of aggregate for shipment or for return to the process stream (batch or continuous drop operations).

Either adding aggregate material to a storage pile or removing it usually involves dropping the material onto a receiving surface. Truck dumping on the pile or loading out from the pile to a truck with a front-end loader are examples of batch drop operations. Adding material to the pile by a conveyor stacker is an example of a continuous drop operation.

The quantity of particulate emissions generated by either type of drop operation, per kilogram (kg) (ton) of material transferred, may be estimated, with a rating of A, using the following empirical expression:[11]

$$E = k(0.0016) \frac{\left(\frac{U}{2.2}\right)^{1.3}}{\left(\frac{M}{2}\right)^{1.4}} \text{ (kg/megagram [Mg])} \quad (1)$$

$$E = k(0.0032) \frac{\left(\frac{U}{5}\right)^{1.3}}{\left(\frac{M}{2}\right)^{1.4}} \text{ (pound [lb]/ton)}$$

where:

E = emission factor
k = particle size multiplier (dimensionless)
U = mean wind speed, meters per second (m/s) (miles per hour [mph])
M = material moisture content (%)

The particle size multiplier in the equation, k, varies with aerodynamic particle size range, as follows:

Aerodynamic Particle Size Multiplier (k) For Equation 1				
< 30 μm	< 15 μm	< 10 μm	< 5 μm	< 2.5 μm
0.74	0.48	0.35	0.20	0.053[a]

[a] Multiplier for < 2.5 μm taken from Reference 14.

The equation retains the assigned quality rating if applied within the ranges of source conditions that were tested in developing the equation, as follows. Note that silt content is included, even though silt content does not appear as a correction parameter in the equation. While it is reasonable to expect that silt content and emission factors are interrelated, no significant correlation between the 2 was found during the derivation of the equation, probably because most tests with high silt contents were conducted under lower winds, and vice versa. It is recommended that estimates from the equation be reduced 1 quality rating level if the silt content used in a particular application falls outside the range given:

Ranges Of Source Conditions For Equation 1			
Silt Content (%)	Moisture Content (%)	Wind Speed	
		m/s	mph
0.44 - 19	0.25 - 4.8	0.6 - 6.7	1.3 - 15

To retain the quality rating of the equation when it is applied to a specific facility, reliable correction parameters must be determined for specific sources of interest. The field and laboratory procedures for aggregate sampling are given in Reference 3. In the event that site-specific values for

correction parameters cannot be obtained, the appropriate mean from Table 13.2.4-1 may be used, but the quality rating of the equation is reduced by 1 letter.

For emissions from equipment traffic (trucks, front-end loaders, dozers, etc.) traveling between or on piles, it is recommended that the equations for vehicle traffic on unpaved surfaces be used (see Section 13.2.2). For vehicle travel between storage piles, the silt value(s) for the areas among the piles (which may differ from the silt values for the stored materials) should be used.

Worst-case emissions from storage pile areas occur under dry, windy conditions. Worst-case emissions from materials-handling operations may be calculated by substituting into the equation appropriate values for aggregate material moisture content and for anticipated wind speeds during the worst case averaging period, usually 24 hours. The treatment of dry conditions for Section 13.2.2, vehicle traffic, "Unpaved Roads", follows the methodology described in that section centering on parameter p. A separate set of nonclimatic correction parameters and source extent values corresponding to higher than normal storage pile activity also may be justified for the worst-case averaging period.

13.2.4.4 Controls[12-13]

Watering and the use of chemical wetting agents are the principal means for control of aggregate storage pile emissions. Enclosure or covering of inactive piles to reduce wind erosion can also reduce emissions. Watering is useful mainly to reduce emissions from vehicle traffic in the storage pile area. Watering of the storage piles themselves typically has only a very temporary slight effect on total emissions. A much more effective technique is to apply chemical agents (such as surfactants) that permit more extensive wetting. Continuous chemical treating of material loaded onto piles, coupled with watering or treatment of roadways, can reduce total particulate emissions from aggregate storage operations by up to 90 percent.[12]

References For Section 13.2.4

1. C. Cowherd, Jr., *et al., Development Of Emission Factors For Fugitive Dust Sources*, EPA-450/3-74-037, U. S. Environmental Protection Agency, Research Triangle Park, NC, June 1974.

2. R. Bohn, *et al., Fugitive Emissions From Integrated Iron And Steel Plants*, EPA-600/2-78-050, U. S. Environmental Protection Agency, Cincinnati, OH, March 1978.

3. C. Cowherd, Jr., *et al., Iron And Steel Plant Open Dust Source Fugitive Emission Evaluation*, EPA-600/2-79-103, U. S. Environmental Protection Agency, Cincinnati, OH, May 1979.

4. *Evaluation Of Open Dust Sources In The Vicinity Of Buffalo, New York*, EPA Contract No. 68-02-2545, Midwest Research Institute, Kansas City, MO, March 1979.

5. C. Cowherd, Jr., and T. Cuscino, Jr., *Fugitive Emissions Evaluation*, MRI-4343-L, Midwest Research Institute, Kansas City, MO, February 1977.

6. T. Cuscino, Jr., *et al., Taconite Mining Fugitive Emissions Study*, Minnesota Pollution Control Agency, Roseville, MN, June 1979.

7. *Improved Emission Factors For Fugitive Dust From Western Surface Coal Mining Sources*, 2 Volumes, EPA Contract No. 68-03-2924, PEDCo Environmental, Kansas City, MO, and Midwest Research Institute, Kansas City, MO, July 1981.

8. *Determination Of Fugitive Coal Dust Emissions From Rotary Railcar Dumping*, TRC, Hartford, CT, May 1984.

9. *PM-10 Emission Inventory Of Landfills In the Lake Calumet Area*, EPA Contract No. 68-02-3891, Midwest Research Institute, Kansas City, MO, September 1987.

10. *Chicago Area Particulate Matter Emission Inventory — Sampling And Analysis*, EPA Contract No. 68-02-4395, Midwest Research Institute, Kansas City, MO, May 1988.

11. *Update Of Fugitive Dust Emission Factors In AP-42 Section 11.2*, EPA Contract No. 68-02-3891, Midwest Research Institute, Kansas City, MO, July 1987.

12. G. A. Jutze, *et al., Investigation Of Fugitive Dust Sources Emissions And Control*, EPA-450/3-74-036a, U. S. Environmental Protection Agency, Research Triangle Park, NC, June 1974.

13. C. Cowherd, Jr., *et al., Control Of Open Fugitive Dust Sources*, EPA-450/3-88-008, U. S. Environmental Protection Agency, Research Triangle Park, NC, September 1988.

14. C. Cowherd, *Background Document for Revisions to Fine Fraction Ratios &sed for AP-42 Fugitive Dust Emission Factors*. Prepared by Midwest Research Institute for Western Governors Association, Western Regional Air Partnership, Denver, CO, February 1, 2006.

13.2.5 Industrial Wind Erosion

13.2.5.1 General[1-3]

Dust emissions may be generated by wind erosion of open aggregate storage piles and exposed areas within an industrial facility. These sources typically are characterized by nonhomogeneous surfaces impregnated with nonerodible elements (particles larger than approximately 1 centimeter [cm] in diameter). Field testing of coal piles and other exposed materials using a portable wind tunnel has shown that (a) threshold wind speeds exceed 5 meters per second (m/s) (11 miles per hour [mph]) at 15 cm above the surface or 10 m/s (22 mph) at 7 m above the surface, and (b) particulate emission rates tend to decay rapidly (half-life of a few minutes) during an erosion event. In other words, these aggregate material surfaces are characterized by finite availability of erodible material (mass/area) referred to as the erosion potential. Any natural crusting of the surface binds the erodible material, thereby reducing the erosion potential.

13.2.5.2 Emissions And Correction Parameters

If typical values for threshold wind speed at 15 cm are corrected to typical wind sensor height (7 - 10 m), the resulting values exceed the upper extremes of hourly mean wind speeds observed in most areas of the country. In other words, mean atmospheric wind speeds are not sufficient to sustain wind erosion from flat surfaces of the type tested. However, wind gusts may quickly deplete a substantial portion of the erosion potential. Because erosion potential has been found to increase rapidly with increasing wind speed, estimated emissions should be related to the gusts of highest magnitude.

The routinely measured meteorological variable that best reflects the magnitude of wind gusts is the fastest mile. This quantity represents the wind speed corresponding to the whole mile of wind movement that has passed by the 1 mile contact anemometer in the least amount of time. Daily measurements of the fastest mile are presented in the monthly Local Climatological Data (LCD) summaries. The duration of the fastest mile, typically about 2 minutes (for a fastest mile of 30 mph), matches well with the half-life of the erosion process, which ranges between 1 and 4 minutes. It should be noted, however, that peak winds can significantly exceed the daily fastest mile.

The wind speed profile in the surface boundary layer is found to follow a logarithmic distribution:

$$u(z) = \frac{u^*}{0.4} \ln\frac{z}{z_o} \qquad (z > z_o) \qquad (1)$$

where:

u = wind speed, cm/s
u^* = friction velocity, cm/s
z = height above test surface, cm
z_o = roughness height, cm
0.4 = von Karman's constant, dimensionless

The friction velocity (u^*) is a measure of wind shear stress on the erodible surface, as determined from the slope of the logarithmic velocity profile. The roughness height (z_o) is a measure of the roughness of the exposed surface as determined from the y intercept of the velocity profile, i. e., the height at which the wind speed is zero. These parameters are illustrated in Figure 13.2.5-1 for a roughness height of 0.1 cm.

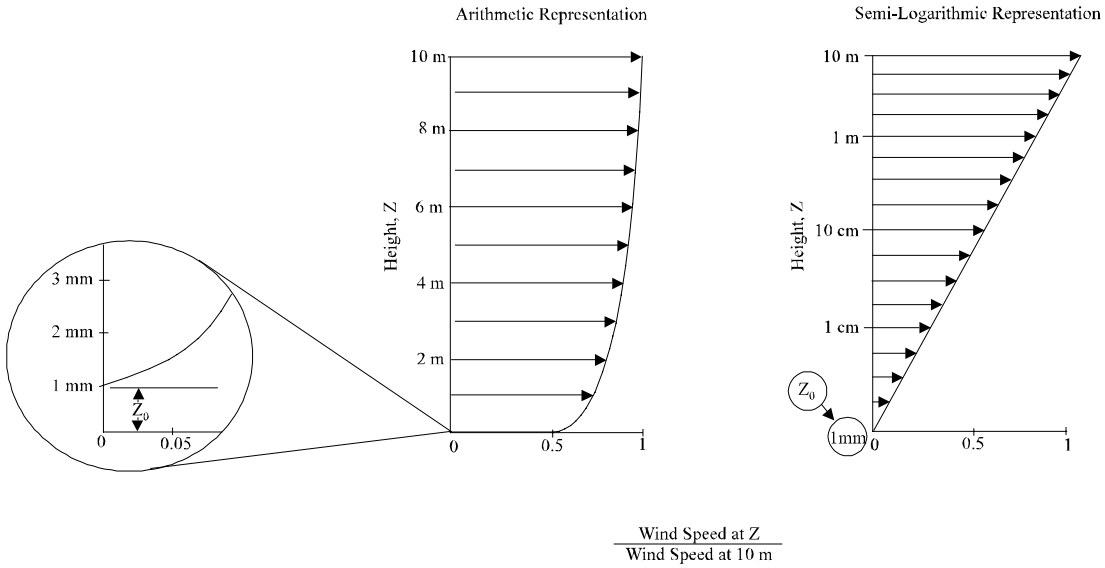


Figure 13.2.5-1. Illustration of logarithmic velocity profile.

Emissions generated by wind erosion are also dependent on the frequency of disturbance of the erodible surface because each time that a surface is disturbed, its erosion potential is restored. A disturbance is defined as an action that results in the exposure of fresh surface material. On a storage pile, this would occur whenever aggregate material is either added to or removed from the old surface. A disturbance of an exposed area may also result from the turning of surface material to a depth exceeding the size of the largest pieces of material present.

13.2.5.3 Predictive Emission Factor Equation[4]

The emission factor for wind-generated particulate emissions from mixtures of erodible and nonerodible surface material subject to disturbance may be expressed in units of grams per square meter (g/m^2) per year as follows:

$$\text{Emission factor} = k \sum_{i=1}^{N} P_i \qquad (2)$$

where:

k = particle size multiplier
N = number of disturbances per year
P_i = erosion potential corresponding to the observed (or probable) fastest mile of wind for the ith period between disturbances, g/m^2

The particle size multiplier (k) for Equation 2 varies with aerodynamic particle size, as follows:

Aerodynamic Particle Size Multipliers For Equation 2			
30 µm	<15 µm	<10 µm	<2.5 µm
1.0	0.6	0.5	0.075[a]

[a] Multiplier for < 2.5 um taken from Reference 11.

This distribution of particle size within the under 30 micrometer (µm) fraction is comparable to the distributions reported for other fugitive dust sources where wind speed is a factor. This is illustrated, for example, in the distributions for batch and continuous drop operations encompassing a number of test aggregate materials (see Section 13.2.4).

In calculating emission factors, each area of an erodible surface that is subject to a different frequency of disturbance should be treated separately. For a surface disturbed daily, N = 365 per year, and for a surface disturbance once every 6 months, N = 2 per year.

The erosion potential function for a dry, exposed surface is:

$$P = 58\,(u^* - u_t^*)^2 + 25\,(u^* - u_t^*) \qquad (3)$$

$$P = 0 \text{ for } u^* \leq u_t^*$$

where:

u^* = friction velocity (m/s)
u_t = threshold friction velocity (m/s)

Because of the nonlinear form of the erosion potential function, each erosion event must be treated separately.

Equations 2 and 3 apply only to dry, exposed materials with limited erosion potential. The resulting calculation is valid only for a time period as long or longer than the period between disturbances. Calculated emissions represent intermittent events and should not be input directly into dispersion models that assume steady-state emission rates.

For uncrusted surfaces, the threshold friction velocity is best estimated from the dry aggregate structure of the soil. A simple hand sieving test of surface soil can be used to determine the mode of the surface aggregate size distribution by inspection of relative sieve catch amounts, following the procedure described below.

FIELD PROCEDURE FOR DETERMINATION OF THRESHOLD FRICTION VELOCITY
(from a 1952 laboratory procedure published by W. S. Chepil):

1. Prepare a nest of sieves with the following openings: 4 mm, 2 mm, 1 mm, 0.5 mm, and 0.25 mm. Place a collector pan below the bottom (0.25 mm) sieve.

2. Collect a sample representing the surface layer of loose particles (approximately 1 cm in depth, for an encrusted surface), removing any rocks larger than about 1 cm in average physical diameter. The area to be sampled should be not less than 30 cm by 30 cm.

3. Pour the sample into the top sieve (4-mm opening), and place a lid on the top.

4. Move the covered sieve/pan unit by hand, using a broad circular arm motion in the horizontal plane. Complete 20 circular movements at a speed just necessary to achieve some relative horizontal motion between the sieve and the particles.

5. Inspect the relative quantities of catch within each sieve, and determine where the mode in the aggregate size distribution lies, i. e., between the opening size of the sieve with the largest catch and the opening size of the next largest sieve.

6. Determine the threshold friction velocity from Table 13.2.5-1.

The results of the sieving can be interpreted using Table 13.2.5-1. Alternatively, the threshold friction velocity for erosion can be determined from the mode of the aggregate size distribution using the graphical relationship described by Gillette.[5-6] If the surface material contains nonerodible elements that are too large to include in the sieving (i. e., greater than about 1 cm in diameter), the effect of the elements must be taken into account by increasing the threshold friction velocity.[10]

Table 13.2.5-1 (Metric Units). FIELD PROCEDURE FOR DETERMINATION OF THRESHOLD FRICTION VELOCITY

Tyler Sieve No.	Opening (mm)	Midpoint (mm)	u_t^* (cm/s)
5	4		
9	2	3	100
16	1	1.5	76
32	0.5	0.75	58
60	0.25	0.375	43

Threshold friction velocities for several surface types have been determined by field measurements with a portable wind tunnel. These values are presented in Table 13.2.5-2.

Table 13.2.5-2 (Metric Units). THRESHOLD FRICTION VELOCITIES

Material	Threshold Friction Velocity (m/s)	Roughness Height (cm)	Threshold Wind Velocity At 10 m (m/s)	
			z_o = Act	z_o = 0.5 cm
Overburden[a]	1.02	0.3	21	19
Scoria (roadbed material)[a]	1.33	0.3	27	25
Ground coal (surrounding coal pile)[a]	0.55	0.01	16	10
Uncrusted coal pile[a]	1.12	0.3	23	21
Scraper tracks on coal pile[a,b]	0.62	0.06	15	12
Fine coal dust on concrete pad[c]	0.54	0.2	11	10

[a] Western surface coal mine. Reference 2.
[b] Lightly crusted.
[c] Eastern power plant. Reference 3.

The fastest mile of wind for the periods between disturbances may be obtained from the monthly LCD summaries for the nearest reporting weather station that is representative of the site in question.[7] These summaries report actual fastest mile values for each day of a given month. Because the erosion potential is a highly nonlinear function of the fastest mile, mean values of the fastest mile are inappropriate. The anemometer heights of reporting weather stations are found in Reference 8, and should be corrected to a 10-m reference height using Equation 1.

To convert the fastest mile of wind (u^+) from a reference anemometer height of 10 m to the equivalent friction velocity (u^*), the logarithmic wind speed profile may be used to yield the following equation:

$$u^* = 0.053\ u_{10}^+ \quad (4)$$

where:

u^* = friction velocity (m/s)

u_{10}^+ = fastest mile of reference anemometer for period between disturbances (m/s)

This assumes a typical roughness height of 0.5 cm for open terrain. Equation 4 is restricted to large relatively flat piles or exposed areas with little penetration into the surface wind layer.

If the pile significantly penetrates the surface wind layer (i. e., with a height-to-base ratio exceeding 0.2), it is necessary to divide the pile area into subareas representing different degrees of exposure to wind. The results of physical modeling show that the frontal face of an elevated pile is exposed to wind speeds of the same order as the approach wind speed at the top of the pile.

For 2 representative pile shapes (conical and oval with flattop, 37-degree side slope), the ratios of surface wind speed (u_s) to approach wind speed (u_r) have been derived from wind tunnel studies.[9] The results are shown in Figure 13.2.5-2 corresponding to an actual pile height of 11 m, a reference (upwind) anemometer height of 10 m, and a pile surface roughness height (z_o) of 0.5 cm. The measured surface winds correspond to a height of 25 cm above the surface. The area fraction within each contour pair is specified in Table 13.2.5-3.

Table 13.2.5-3. SUBAREA DISTRIBUTION FOR REGIMES OF u_s/u_r[a]

Pile Subarea	Percent Of Pile Surface Area			
	Pile A	Pile B1	Pile B2	Pile B3
0.2a	5	5	3	3
0.2b	35	2	28	25
0.2c	NA	29	NA	NA
0.6a	48	26	29	28
0.6b	NA	24	22	26
0.9	12	14	15	14
1.1	NA	NA	3	4

[a] NA = not applicable.

The profiles of u_s/u_r in Figure 13.2.5-2 can be used to estimate the surface friction velocity distribution around similarly shaped piles, using the following procedure:

1. Correct the fastest mile value (u^+) for the period of interest from the anemometer height (z) to a reference height of 10 m u_{10}^+ using a variation of Equation 1:

$$u_{10}^+ = u^+ \frac{\ln (10/0.005)}{\ln (z/0.005)} \quad (5)$$

   where a typical roughness height of 0.5 cm (0.005 m) has been assumed. If a site-specific roughness height is available, it should be used.

2. Use the appropriate part of Figure 13.2.5-2 based on the pile shape and orientation to the fastest mile of wind, to obtain the corresponding surface wind speed distribution (u_s^+)

$$u_s^+ = \frac{(u_s)}{u_r} \quad u_{10}^+ \quad (6)$$

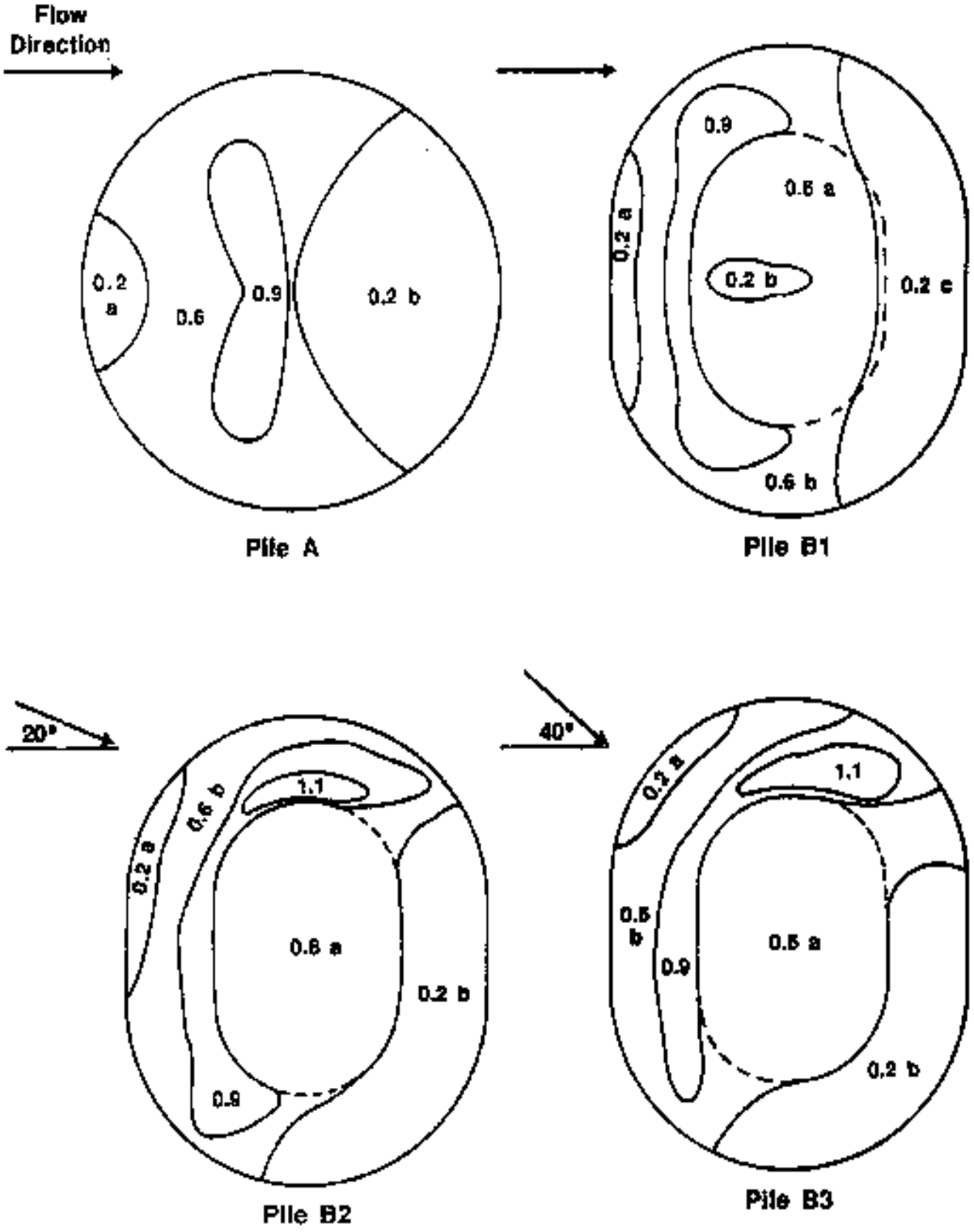


Figure 13.2.5-2. Contours of normalized surface windspeeds, u_s/u_r.

3. For any subarea of the pile surface having a narrow range of surface wind speed, use a variation of Equation 1 to calculate the equivalent friction velocity (u^*):

$$u^* = \frac{0.4 u_s^+}{\frac{25}{\ln 0.5}} = 0.10 u_s^+ \quad (7)$$

From this point on, the procedure is identical to that used for a flat pile, as described above.

Implementation of the above procedure is carried out in the following steps:

1. Determine threshold friction velocity for erodible material of interest (see Table 13.2.5-2 or determine from mode of aggregate size distribution).

2. Divide the exposed surface area into subareas of constant frequency of disturbance (N).

3. Tabulate fastest mile values (u^+) for each frequency of disturbance and correct them to 10 m (u^+_{10}) using Equation 5.5

4. Convert fastest mile values (u_{10}) to equivalent friction velocities (u^*), taking into account (a) the uniform wind exposure of nonelevated surfaces, using Equation 4, or (b) the nonuniform wind exposure of elevated surfaces (piles), using Equations 6 and 7.

5. For elevated surfaces (piles), subdivide areas of constant N into subareas of constant u^* (i. e., within the isopleth values of u_s/u_r in Figure 13.2.5-2 and Table 13.2.5-3) and determine the size of each subarea.

6. Treating each subarea (of constant N and u^*) as a separate source, calculate the erosion potential (P_i) for each period between disturbances using Equation 3 and the emission factor using Equation 2.

7. Multiply the resulting emission factor for each subarea by the size of the subarea, and add the emission contributions of all subareas. Note that the highest 24-hour (hr) emissions would be expected to occur on the windiest day of the year. Maximum emissions are calculated assuming a single event with the highest fastest mile value for the annual period.

The recommended emission factor equation presented above assumes that all of the erosion potential corresponding to the fastest mile of wind is lost during the period between disturbances. Because the fastest mile event typically lasts only about 2 minutes, which corresponds roughly to the half-life for the decay of actual erosion potential, it could be argued that the emission factor overestimates particulate emissions. However, there are other aspects of the wind erosion process that offset this apparent conservatism:

1. The fastest mile event contains peak winds that substantially exceed the mean value for the event.

2. Whenever the fastest mile event occurs, there are usually a number of periods of

slightly lower mean wind speed that contain peak gusts of the same order as the fastest mile wind speed.

Of greater concern is the likelihood of overprediction of wind erosion emissions in the case of surfaces disturbed infrequently in comparison to the rate of crust formation.

13.2.5.4 Example 1: Calculation for wind erosion emissions from conically shaped coal pile

A coal burning facility maintains a conically shaped surge pile 11 m in height and 29.2 m in base diameter, containing about 2000 megagrams (Mg) of coal, with a bulk density of 800 kilograms per cubic meter (kg/m$^{3)}$ (50 pounds per cubic feet [lb/ft^3]). The total exposed surface area of the pile is calculated as follows:

Coal is added to the pile by means of a fixed stacker and reclaimed by front-end loaders operating

$$S = \pi r \sqrt{r^2 + h^2}$$
$$= 3.14(14.6)\sqrt{(14.6)^2 + (11.0)^2}$$
$$= 838\ m^2$$

at the base of the pile on the downwind side. In addition, every 3 days 250 Mg (12.5 percent of the stored capacity of coal) is added back to the pile by a topping off operation, thereby restoring the full capacity of the pile. It is assumed that (a) the reclaiming operation disturbs only a limited portion of the surface area where the daily activity is occurring, such that the remainder of the pile surface remains intact, and (b) the topping off operation creates a fresh surface on the entire pile while restoring its original shape in the area depleted by daily reclaiming activity.

Because of the high frequency of disturbance of the pile, a large number of calculations must be made to determine each contribution to the total annual wind erosion emissions. This illustration will use a single month as an example.

Step 1: In the absence of field data for estimating the threshold friction velocity, a value of 1.12 m/s is obtained from Table 13.2.5-2.

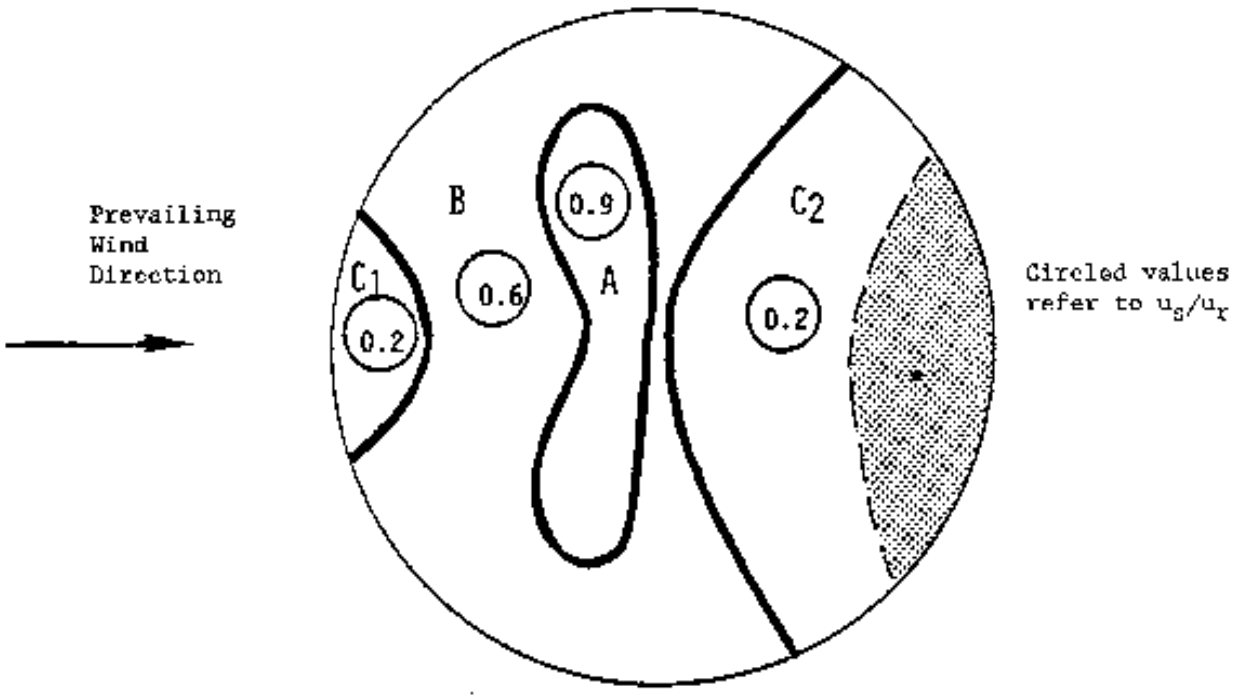
Step 2: Except for a small area near the base of the pile (see Figure 13.2.5-3), the entire pile surface is disturbed every 3 days, corresponding to a value of N = 120 per year. It will be shown that the contribution of the area where daily activity occurs is negligible so that it does not need to be treated separately in the calculations.

Step 3: The calculation procedure involves determination of the fastest mile for each period of disturbance. Figure 13.2.5-4 shows a representative set of values (for a 1-month period) that are assumed to be applicable to the geographic area of the pile location. The values have been separated into 3-day periods, and the highest value in each period is indicated. In this example, the anemometer height is 7 m, so that a height correction to 10 m is needed for the fastest mile values. From Equation 5,

$$u_{10}^{+} = u_{7}^{+} \left(\frac{\ln (10/0.005)}{\ln (7/0.005)} \right)$$

$$u_{10}^{+} = 1.05\ u_{7}^{+}$$

Step 4: The next step is to convert the fastest mile value for each 3-day period into



* A portion of C_2 is disturbed daily by reclaiming activities.

Area ID	$\frac{u_s}{u_r}$	Pile Surface %	Pile Surface Area (m^2)
A	0.9	12	101
B	0.6	48	402
$C_1 + C_2$	0.2	40	335
		Total	838

Figure 13.2.5-3. Example 1: Pile surface areas within each wind speed regime.

Local Climatological Data

Monthly Summary

Wind						
			Fastest Mile			
Resultant Dir.	Resultant Speed M.P.H.	Average Speed M.P.H.	Speed M.P.H.	Direction		Date
13	14	15	16	17		22
30	5.3	6.9	9	36		1
01	10.5	10.6	(14)	01		2
10	2.4	6.0	10	02		3
13	11.0	11.4	16	13		4
12	11.3	11.9	15	11		5
20	11.1	19.0	(29)	30		6
29	19.6	19.8	(30)	30		7
29	10.9	11.2	17	30		8
22	3.0	8.1	15	13		9
14	14.6	15.1	23	12		10
29	22.3	23.3	(31)	29		11
17	7.9	13.5	23	17		12
21	7.7	15.5	18	18		13
10	4.5	9.6	(22)	13		14
10	6.7	8.8	13	11		15
01	13.7	13.8	(21)	36		16
33	11.2	11.5	15	34		17
27	4.3	5.8	12	31		18
32	9.3	10.2	14	35		19
24	7.5	7.8	(16)	24		20
22	10.3	10.6	16	20		21
32	17.1	17.3	(25)	32		22
29	2.4	8.5	14	13		23
07	5.9	8.8	15	02		24
34	11.3	11.7	(17)	32		25
31	12.1	12.2	16	32		26
30	8.3	8.5	16	26		27
30	8.2	8.3	(13)	32		28
33	5.0	6.6	10	32		29
34	3.1	5.2	9	31		30
29	4.9	5.5	8	25		31
For the Month:						
30	3.3	11.1	31	29		
———	———	———	Date: 11			

Figure 13.2.5-4. Example daily fastest miles wind for periods of interest.

equivalent friction velocities for each surface wind regime (i. e., u_s/u_r ratio) of the pile, using Equations 6 and 7. Figure 13.2.5-3 shows the surface wind speed pattern (expressed as a fraction of the approach wind speed at a height of 10 m). The surface areas lying within each wind speed regime are tabulated below the figure.

The calculated friction velocities are presented in Table 13.2.5-4. As indicated, only 3 of the periods contain a friction velocity which exceeds the threshold value of 1.12 m/s for an uncrusted coal pile. These 3 values all occur within the $u_s/u_r = 0.9$ regime of the pile surface.

Table 13.2.5-4 (Metric And English Units). EXAMPLE 1:
CALCULATION OF FRICTION VELOCITIES

	u_7^+		u_{10}^+		$u^* = 0.1u^+$ (m/s) s		
3-Day Period	mph	m/s	mph	m/s	u_s/u_r: 0.2	u_s/u_r: 0.6	u_s/u_r: 0.9
1	14	6.3	15	6.6	0.13	0.40	0.59
2	29	13.0	31	13.7	0.27	0.82	1.23
3	30	13.4	32	14.1	0.28	0.84	1.27
4	31	13.9	33	14.6	0.29	0.88	1.31
5	22	9.8	23	10.3	0.21	0.62	0.93
6	21	9.4	22	9.9	0.20	0.59	0.89
7	16	7.2	17	7.6	0.15	0.46	0.68
8	25	11.2	26	11.8	0.24	0.71	1.06
9	17	7.6	18	8.0	0.16	0.48	0.72
10	13	5.8	14	6.1	0.12	0.37	0.55

Step 5: This step is not necessary because there is only 1 frequency of disturbance used in the calculations. It is clear that the small area of daily disturbance (which lies entirely within the $u_s/u_r = 0.2$ regime) is never subject to wind speeds exceeding the threshold value.

Steps 6 and 7: The final set of calculations (shown in Table 13.2.5-5) involves the tabulation and summation of emissions for each disturbance period and for the affected subarea. The erosion potential (P) is calculated from Equation 3.

For example, the calculation for the second 3-day period is:

$$P = 58(u^* - u_t^*)^2 + 25(u^* - u_t^*)$$

$$P_2 = 58(1.23 - 1.12)^2 + 25(1.23 - 1.12)$$

$$= 0.70 + 2.75 = 3.45 \text{ g/m}^2$$

Table 13.2.5-5 (Metric Units). EXAMPLE 1: CALCULATION OF PM-10 EMISSIONS[a]

3-Day Period	u^* (m/s)	$u^* - u_t^*$ (m/s)	P (g/m^2)	ID	Pile Surface Area (m^2)	kPA (g)
2	1.23	0.11	3.45	A	101	170
3	1.27	0.15	5.06	A	101	260
4	1.31	0.19	6.84	A	101	350
TOTAL						780

[a] Where $u_t^* = 1.12$ m/s for uncrusted coal and k = 0.5 for PM-10.

The emissions of particulate matter greater than 10 µm (PM-10) generated by each event are found as the product of the PM-10 multiplier (k = 0.5), the erosion potential (P), and the affected area of the pile (A).

As shown in Table 13.2.5-5, the results of these calculations indicate a monthly PM-10 emission total of 780 g.

13.2.5.5 Example 2: Calculation for wind erosion from flat area covered with coal dust

A flat circular area 29.2 m in diameter is covered with coal dust left over from the total reclaiming of a conical coal pile described in the example above. The total exposed surface area is calculated as follows:

$$s = \frac{\pi}{4} d^2 = 0.785 (29.2)^2 = 670 \text{ m}^2$$

This area will remain exposed for a period of 1 month when a new pile will be formed.

Step 1: In the absence of field data for estimating the threshold friction velocity, a value of 0.54 m/s is obtained from Table 13.2.5-2.

Step 2: The entire surface area is exposed for a period of 1 month after removal of a pile and N = 1/yr.

Step 3: From Figure 13.2.5-4, the highest value of fastest mile for the 30-day period (31 mph) occurs on the 11th day of the period. In this example, the reference anemometer height is 7 m, so that a height correction is needed for the fastest mile value. From Step 3 of the previous example, $u_{10}^+ = 1.05\ u_7^+$, so that $u_{10}^+ = 33$ mph.

Step 4: Equation 4 is used to convert the fastest mile value of 14.6 m/s (33 mph) to an equivalent friction velocity of 0.77 m/s. This value exceeds the threshold friction velocity from Step 1 so that erosion does occur.

Step 5: This step is not necessary, because there is only 1 frequency of disturbance for the entire source area.

Steps 6 and 7: The PM-10 emissions generated by the erosion event are calculated as the product of the PM-10 multiplier (k = 0.5), the erosion potential (P) and the source area (A). The erosion potential is calculated from Equation 3 as follows:

$$P = 58(u^* - u_t^*)^2 + 25(u^* - u_t^*)$$

$$P = 58(0.77 - 0.54)^2 + 25(0.77 - 0.54)$$

$$= 3.07 + 5.75$$

$$= 8.82 \text{ g/m}^2$$

Thus the PM-10 emissions for the 1-month period are found to be:

$$E = (0.5)(8.82 \text{ g/m}^2)(670 \text{ m}^2)$$

$$= 3.0 \text{ kg}$$

References For Section 13.2.5

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29. US EPA, 1988. Asbestos; CASRN 1332-21-4, Integrated Risk Information System (IRIS) Chemical Assessment Summary

Full Reference: US EPA, 1988. Asbestos; CASRN 1332-21-4, Integrated Risk Information System (IRIS) Chemical Assessment Summary, US Environmental Protection Agency National Center for Environmental Assessment, 1988

[Reference provided in full]

Asbestos; CASRN 1332-21-4

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Asbestos

File First On-Line 09/26/1988

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	not evaluated	
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	yes	09/26/1988

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Asbestos
CASRN — 1332-21-4

Not available at this time.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Asbestos
CASRN — 1332-21-4

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Asbestos
CASRN — 1332-21-4
Last Revised — 09/26/1988

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

NOTE: The carcinogen assessment summary for asbestos may change in the near future pending the outcome of a further review now being conducted by the CRAVE Work Group.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — A; human carcinogen

Basis — Observation of increased mortality and incidence of lung cancer, mesotheliomas and gastrointestinal cancer in occupationally exposed workers are consistent across investigators and study populations. Animal studies by inhalation in two strains of rats showed similar findings for lung cancer and mesotheliomas. Animal evidence for carcinogenicity via ingestion is limited (male rats fed intermediate-range chrysotile fibers; i.e., >10 um length, developed benign polyps), and epidemiologic data in this regard are inadequate.

II.A.2. Human Carcinogenicity Data

Sufficient. Numerous epidemiologic studies have reported an increased incidence of deaths due to cancer, primarily lung cancer and mesotheliomas associated with exposure to inhaled asbestos. Among 170 asbestos insulation workers in North Ireland followed for up to 26 years, an increased incidence of death was seen due to all cancers (SMR=390), cancers of the lower respiratory tract and pleura (SMR=1760) (Elmes and Simpson, 1971) and mesothelioma (7 cases). Exposure was not quantified.

Selikoff (1976) reported 59 cases of lung cancer and 31 cases of mesothelioma among 1249 asbestos insulation workers followed prospectively for 11 years. Exposure was not quantified. A retrospective cohort mortality study (Selikoff et al., 1979) of 17,800 U.S. and Canadian asbestos insulation workers for a 10-year period using best available information (autopsy, surgical, clinical) reported an increased incidence of cancer at all sites (319.7 expected vs. 995 observed, SMR=311) and cancer of the lung (105.6 expected vs. 486 observed, SMR=460). A modest increase in deaths from gastrointestinal cancer was reported along with 175 deaths from mesothelioma (none expected). Years of exposure ranged from less than 10 to greater than or equal to 45. Levels of exposure were not quantified. In other epidemiologic studies, the increase for lung and pleural cancers has ranged from a low of 1.9 times the expected rate, in asbestos factory workers in England (Peto et al., 1977), to a high of 28 times the expected rate, in female asbestos textile workers in England (Newhouse et al., 1972). Other occupational studies have demonstrated asbestos exposure-related increases in lung cancer and mesothelioma in several industries including textile manufacturing, friction products manufacture, asbestos cement products, and in the mining and milling of asbestos. The studies used for the inhalation quantitative estimate of risk are listed in the table in Section II.C.2.

A case-control study (Newhouse and Thompson, 1965) of 83 patients with mesothelioma reported 52.6% had occupational exposure to asbestos or lived with asbestos workers compared with 11.8% of the controls. Of the remaining subjects, 30.6% of the mesothelioma cases lived within one-half mile of an asbestos factory compared with 7.6% of the controls.

The occurrence of pleural mesothelioma has been associated with the presence of asbestos fibers in water, fields and streets in a region of Turkey with very high environmental levels of naturally-occurring asbestos (Baris et al., 1979).

Kanarek et al. (1980) conducted an ecologic study of cancer deaths in 722 census tracts in the San Francisco Bay area, using cancer incidence data from the period of 1969-1971. Chrysotile asbestos concentrations in drinking water ranged from nondetectable to 3.6E+7 fibers/L. Statistically significant dose-related trends were reported for lung and peritoneal cancer in white males and for gall bladder, pancreatic and peritoneal cancer in white females. Weaker correlations were reported between asbestos levels and female esophageal, pleural and kidney cancer, and stomach cancer in both sexes. In an extension of this study, Conforti et al. (1981) included cancer incidence data from the period of 1969-1974. Statistically significant positive associations were found between asbestos concentration and cancer of the digestive organs in white females, cancers of the digestive tract in white males and esophageal, pancreatic and stomach cancer in both sexes. These associations appeared to be independent of socioeconomic status and occupational exposure to asbestos.

Marsh (1983) reviewed eight independent ecologic studies of asbestos in drinking water carried out in five geographic areas. It was concluded that even though one or more studies found an association between asbestos in water and cancer mortality (or incidence) due to neoplasms of various organs, no individual study or aggregation of studies exists that would establish risk levels from ingested asbestos. Factors confounding the results of these studies include the possible underestimates of occupational exposure to asbestos and the possible misclassification of peritioneal mesothelioma as GI cancer.

Polissar et al. (1984) carried out a case-control study which included better control for confounding variables at the individual level. The authors concluded that there was no convincing evidence for increased cancer risk from asbestos ingestion. At the present time, an important limitation of both the case-control and the ecologic studies is the short follow-up time relative to the long latent period for the appearance of tumors from asbestos exposure.

II.A.3. Animal Carcinogenicity Data

Sufficient. There have been about 20 animal bioassays of asbestos. Gross et al. (1967) exposed 61 white male rats (strain not reported) to 86 mg chrysotile asbestos dust/cu.m for 30 hours/week for 16 months. Of the 41 animals that survived the exposure period, 10 had lung cancer. No lung cancer was observed in 25 controls.

Reeves (1976) exposed 60-77 rats/group for 4 hours/day, 4 days/week for 2 years to doses of 48.7-50.2 mg/cu.m crocidolite, 48.2-48.6 mg/cu.m amosite and 47.4-47.9 mg/cu.m chrysotile. A

5-14% incidence of lung cancer was observed among concentration groups and was concentration-dependent.

Wagner et al. (1974) exposed CD Wistar rats (19-52/group) to 9.7-14.7 mg/cu.m of several types of asbestos for 1 day to 24 months for 7 hours/day, 5 days/week. A duration-dependent increased incidence of lung carcinomas and mesotheliomas was seen for all types of asbestos after 3 months of exposure compared with controls.

F344 rats (88-250/group) were exposed to intermediate range chrysotile asbestos (1291E+8 f/g) in drinking water by gavage to dams during lactation and then in diet throughout their lifetime (NTP, 1985). A statistically significant increase in incidence of benign epithelial neoplasms (adenomatous polyps in the large intestine) was observed in male rats compared with pooled controls of all NTP oral lifetime studies (3/524). In the same study, rats exposed to short range chrysotile asbestos (6081E+9 f/g) showed no significant increase in tumor incidence.

Ward et al. (1980) administered 10 mg UICC amosite asbestos 3 times/week for 10 weeks by gavage to 50 male F344 rats. The animals were observed for an additional 78-79 weeks post-treatment. A total of 17 colon carcinomas were observed. This result was statistically significant compared with historical controls; no concurrent controls were maintained.

Syrian golden hamsters (126-253/group) were exposed to short and intermediate range chrysotile asbestos at a concentration of 1% in the diet for the lifetime of the animals (NTP, 1983). An increased incidence of neoplasia of the adrenal cortex was observed in both males and females exposed to intermediate range fibers and in males exposed to short range fibers. This increase was statistically significant by comparison to pooled controls but not by comparison to concurrent controls. NTP suggested that the biologic importance of adrenal tumors in the absence of target organ (GI tract) neoplasia was questionable.

II.A.4. Supporting Data for Carcinogenicity

Sincock (1977) reported an increased number of chromosomes and chromosome breaks after passive inclusion of asbestos with CHO-K1 cells. Chamberlain and Tarmy (1977) reported asbestos not to be mutagenic for E. coli or S. typhimurium. A positive response was unlikely, however, since prokaryotic cells do not phagocytize particles as do eukaryotic cells.

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

II.C.1. Summary of Risk Estimates

Inhalation Unit Risk — 2.3E-1 per (f/mL)

Extrapolation Method — Additive risk of lung cancer and mesothelioma, using relative risk model for lung cancer and absolute risk model for mesothelioma

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-4 f/mL
E-5 (1 in 100,000)	4E-5 f/mL
E-6 (1 in 1,000,000)	4E-6 f/mL

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

Human Data Occupational Group	Fiber Type	Reported Average Exposure (fiber- yr/mL)	% Increase in Cancer per fiber-yr/mL	Reference
Lung Cancer:				
Textile Products	Predominantly Chrysotile	44	2.8	Dement et al., 1983b
Textile Products	Chrysotile	31	2.5	McDonald et al., 1983a
Textile Products	Chrysotile	200	1.1	Peto, 1980
Textile Products	Chrysotile	51	1.4	McDonald et al.,

Human Data Occupational Group	Fiber Type	Reported Average Exposure (fiber- yr/mL)	% Increase in Cancer per fiber-yr/mL	Reference
				1983b
Friction Products	Chrysotile	32	0.058	Berry and Newhouse, 1983
Friction Products	Chrysotile	31	0.010	McDonald et al., 1984
Insulation Products	Amosite	67	4.3	Seidman, 1984
Insulation Workers	Mixed (Chrysotile, al., 1979 Crocidolite and Amosite)	300	0.75	Selikoff et
Asbestos Products		374	0.49	Henderson and Enterline, 1979
Cement Products		89	0.53	Weill et al., 1979
		112	6.7	Finkelstein, 1983
Mesothelioma:				
Insulation workers	Mixed	375	1.5E-6	Selikoff et al., 1979; Peto et al., 1982
Insulation Products	Amosite	400	1.0E-6	Seidman et al., 1979
Textile Products Manufacturer	Chrysotile	67	3.2E-6	Peto, 1980; Peto et al., 1982

Human Data Occupational Group	Fiber Type	Reported Average Exposure (fiber- yr/mL)	% Increase in Cancer per fiber-yr/mL	Reference
Cement Products	Mixed	108	1.2E-5	Finkelstein, 1983

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

Risks have been calculated for males and females according to smoking habits for a variety of exposure scenarios (U.S. EPA, 1986). The unit risk value is calculated for the additive combined risk of lung cancer and mesothelioma, and is calculated as a composite value for males and females. The epidemiological data show that cigarette smoking and asbestos exposure interact synergistically for production of lung cancer and do not interact with regard to mesothelioma. The unit risk value is based on risks calculated using U.S. general population cancer rates and mortality patterns without consideration of smoking habits. The risks associated with occupational exposure were adjusted to continuous exposure by applying a factor of 140 cu.m/50 cu.m based on the assumption of 20 cu.m/day for total ventilation and 10 cu.m/8-hour workday in the occupational setting.

The unit risk is based on fiber counts made by phase contrast microscopy (PCM) and should not be applied directly to measurements made by other analytical techniques. The unit risk uses PCM fibers because the measurements made in the occupational environment use this method. Many environmental monitoring measurements are reported in terms of fiber counts or mass as determined by transmission electron microscopy (TEM). PCM detects only fibers longer than 5 um and >0.4 um in diameter, while TEM can detect much smaller fibers. TEM mass units are derived from TEM fiber counts. The correlation between PCM fiber counts and TEM mass measurements is very poor. Six data sets which include both measurements show a conversion between TEM mass and PCM fiber count that range from 5-150 (ug/cu.m)/(f/mL). The geometric mean of these results, 30 (ug/cu.m)/(f/mL), was adopted as a conversion factor (U.S. EPA, 1986), but it should be realized that this value is highly uncertain. Likewise, the correlation between PCM fiber counts and TEM fiber counts is very uncertain and no generally applicable conversion factor exists for these two measurements.

In some cases TEM results are reported as numbers of fibers <5 um long and of fibers longer than 5 um. Comparison of PCM fiber counts and TEM counts of fibers >5 um show that the fraction of fibers detected by TEM that are also >0.4 um in diameter (and detectable by PCM) varies from 22-53% (U.S. EPA, 1986).

It should be understood that while TEM can be specific for asbestos, PCM is a nonspecific technique and will measure any fibrous material. Measurements by PCM which are made in conditions where other types of fibers may be present may not be reliable.

In addition to the studies cited above, there were three studies of asbestos workers in mining and milling which showed an increase in lung cancer (McDonald et al., 1980, Nicholson et al., 1979; Rubino et al., 1979). The slope factor calculated from these studies was lower than the other studies, possibly because of a substantially different fiber size distribution, and they were not included in the calculation. The slope factor was calculated by life table methods for lung cancer using a relative risk model, and for mesothelioma using a absolute risk model. The final slope factor for lung cancer was calculated as the weighted geometric mean of estimates from the 11 studies cited in section II.C.2. The final slope factor for mesothelioma is based on the calculated values from the studies of Selikoff et al. (1979), Peto et al. (1982), Seidman et al. (1979), Peto (1980) and Finkelstein (1983) adjusted for the mesothelioma incidence from several additional studies cited previously.

There is some evidence which suggests that the different types of asbestos fibers vary in carcinogenic potency relative to one another and site specificity. It appears, for example, that the risk of mesothelioma is greater with exposure to crocidolite than with amosite or chrysotile exposure alone. This evidence is limited by the lack of information on fiber exposure by mineral type. Other data indicates that differences in fiber size distribution and other process differences may contribute at least as much to the observed variation in risk as does the fiber type itself.

The unit risk should not be used if the air concentration exceeds 4E-2 fibers/ml, since above this concentration the slope factor may differ from that stated.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

A large number of studies of occupationally-exposed workers have conclusively demonstrated the relationship between asbestos exposure and lung cancer or mesothelioma. These results have been corroborated by animal studies using adequate numbers of animals. The quantitative estimate is limited by uncertainty in the exposure estimates, which results from a lack of data on early exposure in the occupational studies and the uncertainty of conversions between various analytical measurements for asbestos.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Asbestos and the 1986 Airborne Asbestos Health Assessment Update have received Agency Review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 09/15/1987, 12/02/1987

Verification Date — 12/02/1987

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Asbestos
CASRN — 1332-21-4

VI.A. Oral RfD References

None

VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

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VII. Revision History

Substance Name — Asbestos
CASRN — 1332-21-4

Date	Section	Description
09/26/1988	II.	Carcinogen summary on-line

VIII. Synonyms

Substance Name — Asbestos
CASRN — 1332-21-4
Last Revised — 09/26/1988

- 1332-21-4
- Asbestos
- calidria-asbestos

30. WA, 2021. Guidelines for the Assessment, Remediation and Management of Asbestos Contaminated Sites in Western Australia

Full Reference: WA, 2021. Guidelines for the Assessment, Remediation and Management of Asbestos Contaminated Sites in Western Australia, Government of Western Australia, Department of Health, 2021.

[Extract: pages 22-23, 25-26, 42-44, 64]

Government of **Western Australia**
Department of **Health**

Guidelines for the Assessment, Remediation and Management of Asbestos Contaminated Sites in Western Australia

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3.8 Screening levels for asbestos in soil

In establishing screening criteria, consideration has been given to the following principles and assumptions used for regulating and controlling asbestos at a National level:

- screening criteria do not distinguish between commercial asbestos mineral fibre types
- the reporting, assessment and management of asbestos contamination under the CS Act provides a mechanism to inform and protect persons from potential future exposure to asbestos-contamination
- fibres are more likely to be released from both FA and AF than from bonded ACM
- bonded ACM in soil is assumed to eventually (over a long period) degrade to asbestos fines as a result of damage or destruction over time
- exposure to asbestos, which has no evidence of a threshold level for mesothelioma risk, should be kept as low as reasonably practicable.

The criteria are summarised in Table 2 and remain the same as in previous Department of Health guidance. A background in establishing screening criteria is provided in Appendix One.

Table 3 Asbestos in soil screening levels

Site uses[4]	Soil asbestos screening criteria
All site uses – FA	10 mg/kg (0.001 %) w/w asbestos
All site uses – AF	10 mg/kg (0.001%) w/w asbestos
Residential A – bonded ACM	100 mg/kg (0.01 %) w/w asbestos
Residential B – bonded ACM	400 mg/kg (0.04 %) w/w asbestos
Recreational C – bonded ACM	200 mg/kg (0.02%) w/w asbestos
Commercial/Industrial D – bonded ACM	500 mg/kg (0.05%) w/w asbestos

3.8.1 Application of investigation and screening levels

The criteria for FA and AF remain fixed for all site uses as there is high uncertainty associated with quantifying asbestos concentrations below 0.01% w/w asbestos. As such, the sampling error and lack of analytical sensitivity in establishing concentration differences between 0.001% w/w and 0.01% w/w would make any adjustment at this order of magnitude meaningless. Example calculations for estimating asbestos in soil concentrations are provided in Appendix Two.

Inadequate sampling strategies rather than lack of accuracy in the adopted analytical methods characteristically limit the effective evaluation of sites contaminated by asbestos.

As for other contaminants, the results from soil analysis must be interpreted in the context of the information obtained from the broader site investigation and applying professional judgement on whether the criteria have been exceeded. More information is provided in Appendix B1 (external site), Section 3 of the ASC NEPM.

[4] Classification of site uses as per the ASC NEPM.

The use of statistics may be appropriate in some circumstances. However, justification for the use of statistics, along with a description of any limitations and assumptions and compliance with DWER Contaminated Sites Guidelines and ASC NEPM, must be provided.

The final decision regarding assessment against criteria should include multiple lines of evidence for which statistics may contribute but not be the sole decision-making parameter. If using statistical analysis, the following must be considered:

- where more than one distinct fill or soil stratum is impacted, separate determinations should be made for each section
- sampling strategy, including sample locations, sampling methods, and sampling density, is designed to find localised hot spots
- sampling strategy considers future subdivision plans with a sample distribution that includes each proposed lot
- where an individual sample result is equal to or greater than 0.1 % w/w asbestos soils, for any form of asbestos, the surrounding soils are subject to higher density sampling to confirm/delineate a hot spot and the impacted soils remediated.

For mixed waste materials (e.g. coarse aggregate material vs soil), a judgemental and semi-quantitative approach may be necessary to estimate the contamination concentration. The extent of the investigation and the sampling plan should consider the remediation options for the mixed waste materials present at a site. Where it is necessary to provide advice against criteria, professional judgement will be required. It is expected that sufficient justification is provided within reports to support any decisions made.

3.8.2. Surface contamination

In addition to meeting the criteria in Table 2, the accessible ground surface (as designated by site investigations or the 10 cm default) should be free of visible bonded ACM and FA at the end of the remedial site works (expected site clean-up criteria). There are two main objectives for remediating the soil surface to be visibly free of asbestos:

1. Minimises the potential for ready access to any contamination (such as isolated fragments), resulting in further deterioration or misuse.
2. Addresses aesthetic, public perception and other regulatory expectations specific to asbestos.

3.8.2.1 Accessible ground surface determined by site investigation

The accessible, readily disturbed surface layer should be differentiated from the underlying soils when describing the site's soil profile. The factors in defining the surface soil layer and the depth to which superficial asbestos contamination is observed or can be reasonably expected to be found are:

- activities undertaken at their site and their frequency (e.g. walking, driving, sports activities)
- the likely depth of soil disturbance from site activities
- any mitigating factors (e.g. ground cover, compaction, soil type and condition).

For example, shifting and sandy soils with no ground cover may have surface bonded ACM contamination extending beyond 10 cm depth that can be encountered when walking or playing in the sand.

Test pits or other sampling methods may be used to verify/justify the inspection/remediation depth of the surface impacted soils.

3.10 Air guidelines

A practical air quality control limit of 0.01 fibres per millilitre (f/mL) asbestos applies to all **removal work**, including around contaminated sites, as described by the Membrane Filter Method. [NOHSC:3003(2005) (MFM). (See Section 5.9.1)

It is important to note that the control limit **should not be used to evaluate recipient exposure risks**.

Exposure should always be minimised to as low as can be achieved by implementing effective dust control measures.

Personal sampling results for any site personnel should be below the occupational exposure standards (0.1 fibres/mL) or the site-specific action level adopted as part of the workplace risk assessment.

No ambient air guidelines have been set for asbestos. A cumulative exposure of 0.01 f/mL.yr is estimated to increase risk above 1 x 10^{-5} for mesothelioma for crocidolite and amosite fibres (Hodgson and Darnton, 2000). Exposure assessment would need to consider the duration of exposure and, potentially, the use of air monitoring reference methods that allow for a lower limit of detection.

Dust (particulate) monitoring may accompany other asbestos specific sampling during remediation activities for more immediate responses to any failures in dust management measures. The Air NEPM 24-hour guidance goal of 50 µg/m^3 for PM_{10} (particulate matter with an equivalent aerodynamic diameter of 10 µm or less) is often applied as an action level for total dust control.

3.11 Exposure assessment for public health

A quantitative or qualitative exposure assessment that assumes reasonable and probable worst-case exposure scenarios can be undertaken for asbestos contamination. Such an assessment can provide additional confidence in conclusions and recommendations, aid in health risk communication and/or provide support and justification for site-specific clean-up goals.

The magnitude of the asbestos contamination hazard depends on the potential for respirable fibres to be released from soil which is influenced by among other things:

- the type and condition of asbestos present
- the quantity/concentration of asbestos present in soil
- the depth and extent of contamination
- soil type and physical properties
- nature of surface coverings, including presence of vegetative cover
- soil moisture content.

Exposure assessment should consider reasonable worst-case seasonal variations for each of the above factors at each site. Also, the measurement of various parameters, e.g. soil moisture, can be included in the sampling plan.

It is also important to note that while the above factors are suitable for understanding the magnitude of the asbestos in soil hazard, the most significant contributing exposure factor will be the human activities/tasks that directly create and/or release dust and airborne fibres.

Exposure assessment should consider:

- future site uses
- the duration and frequency of dust-generating activities and likely levels of airborne (respirable fraction) particulates
- quantification or estimation of fibre in air concentrations during current and future site activities
- mitigation through proposed remediation and management measures.

Methods for completing an exposure assessment include:

- qualitative exposure assessment based on investigation and assessment of the site, activities undertaken, the potential for particulate/fibre release and expected air concentrations
- task/activity-based sampling for activities being undertaken at the site
- simulation of past or future tasks/activities likely to be undertaken at the site (may require additional approval from WorkSafe Division or the WorkSafe Commissioner).

It is not easy to estimate exposure for all uses of a site confidently. The feasibility of undertaking monitoring during exposure assessment should be carefully considered (See Section 5.9.2). However, in some circumstances, such as to demonstrate support for sustainable options for in situ remediation or to provide health risk information on possible past exposures, it may be justified.

If the elements of the risk determination change during the subsequent site operations, such as by uncovering unexpected additional asbestos material or as indicated by air monitoring results, the exposure assessment should be reviewed, and the CSM updated.

5.8.2 Estimating asbestos fines concentration

The same principles are used to estimate the concentration of AF in soil samples as for bonded ACM. This method provides an estimate of total AF concentration in soil (w/w).

This method allows the soil sample to be examined under laboratory-controlled conditions and can utilise stereo microscopy to identify suspect AF. The laboratory examines the entire sample and can separate, weigh and positively identify any suspect material or debris or fibrous matter found within the various size fractions, such as sub 10 mm, +7 mm, 7 mm to 2 mm and sub 2 mm.

For the estimate of concentration to be meaningful, it is important to ensure that samples submitted for analysis are representative of the asbestos contamination and not seeded with incidental finds nor diluted with uncontaminated soils (see Figure 10).

Note that where a larger fraction +2mm material is present in a soil sample, it will be the main contributor to the concentration measurement. It may be important, such as in Tier 2 or Tier 3 assessments, to have detailed observations of the AF fraction. It may also be relevant to submit a smaller, representative soil sample volume, particularly where this improves the collection of a discrete, targeted and representative area of contamination. Any variations or decisions regarding sample size should be justified by the sampling plan and data quality objectives and discussed with the laboratory undertaking the analysis.

Identifying respirable asbestos fibres in soil samples may provide important supportive information for characterising the asbestos contamination. Discretion must be used for comparing AS 4964 trace analysis results against assessment criteria. However, results may provide important qualitative data relevant to exposure assessment.

It is important to note that the laboratory sees a very small, targeted sample of soil. The origin and distribution of AF material within the investigation area may be unknown to the analyst. As such, analysts do not have the information necessary to advise whether the observed AF represents site contamination and whether the contamination should be characterised as friable, non-friable or minor contamination for legislative control or management.

There are several reference methods available internationally that can be accredited for use in Australia. These can be utilised where additional confirmatory analysis is required, such as for higher Tier assessments. Further information for laboratories is provided in Appendix Four.

5.9 Air quality monitoring

5.9.1 Air monitoring principles

The purpose of any air sampling should be clearly identified. The sampling strategy should be developed by a suitably qualified and experienced person (e.g. occupational hygienist).

The Guidance Note on the Membrane Filter Method for Estimating Airborne Asbestos Fibres NOHSC:3003 (2005) (MFM) is regularly employed for control monitoring at contaminated sites. Where real-time monitoring is required to determine the effectiveness of dust controls measures during asbestos work, a direct reading dust measurement may be used to supplement airborne fibre monitoring (see Section 5.9.3).

Control monitoring is used to confirm that control measures have effectively prevented the release of fibres during remediation or site works. Where best practice dust control measures are used, it is expected that a sampling plan will be in place that outlines the number and position of samples and that the action level is the limit of reporting of 0.01 f/mL.

Personal air monitoring provides an index or estimate of exposure to respirable fibres in air. The air monitoring program must consider the need for exposure monitoring of workers undertaking tasks that may expose them to elevated levels of particulate emissions (including sample collection and remediation works). Personal monitoring of asbestos in air to assess and control workplace exposure is an occupational health and safety issue; however, results may also be used in site investigation reports to show that controls have been effective in minimising fibre release and, in effect, protecting public health. The WorkSafe Division, DMIRS, is the lead agency with regard to employee exposure.

5.9.2 Air monitoring for public health exposure assessment

A specific sampling plan should be considered for exposure assessment undertaken as part of the contaminated sites assessments, i.e. in Tier 2 or 3 assessments where more information is required to characterise exposure risks consider monitoring during activities that have the potential to release fibres. Undertaking simulated activities (activity-based sampling) for exposure assessment may require approval from DMIRS. (See Section 3.9.1).

In low-level exposure settings, the MFM may significantly underestimate and sometimes overestimate the fibre concentration in air. For example, fibre counts may include other background fibres present in the environment (organic and other mineral fibres), and MCE filters may also contribute to the fibre count. In addition to ensuring that field blank filters (which are a requirement) are included for all sampling events, a methodology that allows a lower limit of reporting (LOR) should be considered. The analytical sensitivity of MFM may be improved for individual samples and/or relevant international methods can be used that allow a lower LOR and identification of fibre type. Laboratories may seek NATA accreditation for relevant methods.

Exposure assessments have been completed in Western Australia based on a modification of *ISO 14966:2002 Ambient air — Determination of numerical concentration of inorganic fibrous particles — Scanning electron microscopy method* which has a limit of reporting of 0.002 f/mL.

Air sampling results taken during periods of no activity or when effective remediation controls are in place should not be used to conclude that there has been no asbestos fibre release from soils or to justify the use of less stringent site management measures.

5.9.3 Dust/Particulate monitoring

Dust monitoring does provide a useful surrogate for assessing the effectiveness of overall dust control measures at a site for the following reasons:

- real-time dust sampling can be undertaken with alarms/action levels set that provide immediate feedback regarding the effectiveness of dust control measures or changes in conditions that may lead to elevated dust levels
- dust monitoring is commonly used, well known and does not require specific asbestos monitoring expertise
- results are immediately available and easy to interpret, and data logging provides evidence that adequate dust management has been employed during the entire period of remedial/site works.

Dust monitoring equipment should demonstrate that particulate levels are kept as low as reasonably possible. The site dust management plan will need to identify triggers used for control actions. Dust monitoring should be considered as an adjunct, not as a substitute for fibre monitoring.

Equipment should be located along the site perimeter at “background” upwind and downwind locations, taking into account local site features and topography. Where there is a well-defined diurnal and seasonal variation in the dominant wind direction, monitoring stations should be located along the key axes. Generally, regional meteorological data will be sufficient to aid the planning of fixed dust monitoring stations, and portable devices may be repositioned depending on daily conditions. For fixed stations (e.g. Tapered Element Oscillating Microbalance), a detailed log of atypical meteorological conditions may be useful for interpreting results or addressing complaints.

Dust/particulate control monitoring cannot be used as a surrogate for asbestos exposure monitoring.

5.10 Quality assurance/Quality control

Quality assurance and quality control (QA/QC) practices should be consistent with guidance provided by the NEPM, which also provides information on the development of Data Quality Objectives (DQO) and on quality control samples.

Relevant considerations particular to asbestos include:

- investigators should have adequate asbestos experience and breadth of knowledge to ensure the quality of recommended visual detection and quantitation methodologies
- sampling and analytical procedures should be justified as to their appropriateness and effectiveness
- GHS labelling and safe sample packaging and transport requirements are to be met
- analytical methods should be consistent and allow results to be reproducible within and between laboratories. Importantly, fibre-counting criteria should be consistent for all sample analyses
- National Association of Testing Authorities (NATA) asbestos accreditation is a standard QA/QC requirement
- wherever there is analytical uncertainty regarding whether fibres in a sample are asbestos, the fibres should be assumed to be asbestos. Re-sampling should be considered to clarify the presence of asbestos at a site
- Australian Standard Method for the Qualitative Identification of asbestos in bulk samples (AS4964-2004) or relevant, validated international method can be used to identify asbestos in bulk materials (including soil).

Figure 10 Buried waste FA found during site works.

The use of duplicates during sampling for asbestos is not a mandatory requirement. However, there may be situations, for instance, the potential for legal challenge, where a duplicate or triplicate sample may be useful. In such a case, it may be a division of a single asbestos material sample (e.g. division of a suspect ACM fragment) rather than an attempt to collect equivalent samples.

Appendix One:

Background information on the development of screening criteria

DOH established screening criteria in 2009 mainly based on international research by Swartjes and Tromp in The Netherlands (2008).

The determination of asbestos in soil has some differences with chemical contaminants, such as:

- fibres are physical structures of various sizes and dimensions, rather than a chemical molecule
- the available analytical methods provide semi-quantitative (estimate of) concentrations and depend on adequate representative sampling and consideration of other supporting information to characterise contamination
- concentration in soil does not consider the potential for release of airborne fibres, and there is a poor correlation between the two;

Asbestos is a banned and controlled substance, and contaminated sites management must consider other legislation (see Section 2.1) that applies to the handling, removal and disposal of asbestos contaminated soils, e.g. restriction on sale and supply, notification, labelling. This is a consideration for any remediation objective. The basis for the screening criteria is two-fold.

1. For all asbestos types, the concentration of 100 mg/kg or 0.01% w/w asbestos in soil is expected to keep outdoor airborne fibre levels below 0.001 f/mL and probably around 0.0001 f/mL.

DOH applied this criterion to the less hazardous bonded ACM, depending on on-site use. These mirror the Assessment of Contaminated Sites (ASC) National Environmental Protection Measure (NEPM) (1999) site uses and associated default exposure ratios.

A lower criterion has been applied to both FA and AF as activity and disturbance may result in the suspension of smaller particles from FA and AF in air. The 100mg/kg was divided by a factor of 10 to account for greater dryness and dust-generating potential of local soils and the fact that current exposure standards (external site) treat the mineralogical forms of asbestos as equivalent.

Note: For low concentration exposures (cumulative exposure of less than 0.01 f/mL.yr), the risk of mesothelioma, the most applicable health outcome from crocidolite fibre (the most potent fibre), is low. There are generally accepted quantitative estimates of disease, extrapolated from dose-response relationships established for higher occupational exposures. These are those presented by WHO (2000) and Hodgson and Darnton (2000). The estimates suggest that asbestos exposure below 0.0002 f/mL.yr is likely to be less than the lifetime risk of 1×10^{-5} and possibly less than 1×10^{-6} (WHO 2000 and Hodgson and Darnton, 2000). These are lifetime cancer risks estimates that are broadly acceptable for environmental contaminant hazards.

31. WHO, 2000. Air Quality Guidelines for Europe

Full Reference: WHO, 2000. Air Quality Guidelines for Europe, second edition. World Health Organisation Regional Office for Europe, Copenhagen, 2000.

[Extract: pages 128-135]

World Health Organization
Regional Office for Europe
Copenhagen

Air Quality Guidelines

for Europe

Second Edition

WHO Regional Publications, European Series, No. 91

6.2 Asbestos

Exposure evaluation

Actual indoor and outdoor concentrations in air range from below one hundred to several thousand fibres per m^3.

Health risk evaluation

On the basis of the evidence from both experimental and epidemiological studies, it is clear that asbestos inhalation can cause asbestosis, lung cancer and mesothelioma. The evidence that ingested asbestos causes gastrointestinal or other cancers is insufficient. Furthermore, the carcinogenic properties of asbestos are most probably due to its fibre geometry and remarkable integrity; other fibres with the same characteristics may also be carcinogenic.

Current environmental concentrations of asbestos are not considered a hazard with respect to asbestosis. However, a risk of mesothelioma and lung cancer from the current concentrations cannot be excluded.

In 1986 a WHO Task Group expressed reservations about the reliability of risk assessment models applied to asbestos risk. Its members suggested that such models can only be used to obtain a broad approximation of the lung cancer risk of environmental exposures to asbestos and "that any number generated will carry a variation over many orders of magnitude". The same was found to be true for estimates of the risk of mesothelioma. The same document stated: "In the general population the risks of mesothelioma and lung cancer attributable to asbestos cannot be quantified reliably and probably are undetectably low." *(1)*.

The following estimates of risk are based on the relatively large amount of evidence from epidemiological studies concerning occupational exposure. Data from these studies have been conservatively extrapolated to the much lower concentrations found in the general environment. Although there is evidence that chrysotile is less potent than amphiboles, as a precaution chrysotile has been attributed the same risk in these estimates.

Mesothelioma

A formula by which the excess incidence of mesothelioma can be approximated has been derived by Peto *(2)*. Fibre concentration, duration of exposure and time since first exposure are parameters incorporated in this model, which assumes a linear dose–response relationship. Peto verified this model

from data on an urban population exposed for its whole life and on workers exposed for many decades. In both cases, duration of exposure is assumed to be equal or close to time since first exposure. The data show that the incidence of mesothelioma is proportional to the fibre concentration to which the workers were exposed and to time since first exposure for both workers and the general population. Starting from this relationship, one may calculate the risk of lifetime exposure to environmental concentrations from the incidence of mesothelioma in occupational populations exposed to much higher concentrations, but for a shorter time.

Apart from incomplete knowledge about the true workplace exposure, a further complication arises from the fact that workplace concentrations were measured by means of an optical microscope, counting only fibres longer than 5 µm and thicker than, say, 0.5 µm. In this chapter all fibre concentrations based on optical microscopy are marked F^*/m^3 and risk estimates will be based on F^*/m^3. If concentrations measured by optical microscopy are to be compared with environmental fibre concentrations measured by scanning electron microscopy, a conversion factor has to be used: $2\ F/m^3 = 1\ F^*/m^3$.

Several studies have been performed to calculate the risk of mesothelioma resulting from nonoccupational exposure to asbestos. Lifetime exposure to $100\ F^*/m^3$ has been estimated by various authors to carry differing degrees of mesothelioma risk (see Table 14). The risk estimates in Table 14 differ by a factor of 4. A "best" estimate may be $2 \times l0^{-5}$ for $100\ F^*/m^3$.

An independent check of this risk estimate can be made by calculating the incidence of mesothelioma in the general population, based on a hypothetical

Table 14. Estimates of mesothelioma risk resulting from lifetime exposure to asbestos

Risk of mesothelioma from 100 F^*/m^3	Values in original publication (risk for fibre concentration indicated)	Reference
1.0×10^{-5}	1.0×10^{-4} for 1000 F^*/m^3	*(3)*
$\sim 2.0 \times 10^{-5}$	1.0×10^{-4} for (130–800) F^*/m^3	*(4)*
$\sim 3.9 \times 10^{-5}$	1.56×10^{-4} for 400 F^*/m^3	*(5, 6)*
$\sim 2.4 \times 10^{-5}$	2.75×10^{-3} (females) } for 0.01 F/ml 1.92×10^{-3} (males) }	*(7)*

average asbestos exposure 30–40 years ago *(8)*. If the latter had been 200–500 F*/m^3 (corresponding to about 400–1000 F/m^3 as measured today), the resulting lifetime risk of mesothelioma would be (4–10) × 10^{-5}. With the average United States death rate of 9000 × 10^{-6} per year, this would give 0.4–0.9 mesothelioma cases each year per million persons from past environmental asbestos exposure. The reported mesothelioma incidence in the United States ranges from 1.4 × 10^{-6} per year to 2.5 × 10^{-6} per year according to various authors *(5, 8)*. Thus, the calculated risk figures would account for only part of the observed incidence. Nevertheless, other factors that may account for this discrepancy must be considered.

- Uncertainties in the risk extrapolations result from the lack of reliable exposure data in the cohort studies, errors in the medical reports, and necessary simplifications in the extrapolation model itself *(7)*. Furthermore, the amount of past ambient exposure can only be an educated guess.

- The incidence of nonoccupational mesotheliomas is calculated from the difference between the total of observed cases and the number of those probably related to occupational exposure. Neither of these two figures is exactly known. Moreover, the influence of other environmental factors in the generation of mesothelioma is unknown.

In the light of these uncertainties, the result obtained by using the risk estimate can be considered to be in relatively good agreement with the annual mesothelioma death rate based on national statistical data.

Lung cancer

Unlike mesothelioma, lung cancer is one of the most common forms of cancer. As several exogenous noxious agents can be etiologically responsible for bronchial carcinoma, the extrapolation of risk and comparison between different studies is considerably complicated. In many epidemiological studies, the crucial effect of smoking has not been properly taken into account.

Differentiation of the observed risks according to smoking habits has been carried out, however, in the cohort of North American insulation workers studied by Hammond et al. *(9)*. This study suggests that the relative risk at a given time is approximately proportional to the cumulative amount of fine asbestos dust received up to this point, for both smokers and nonsmokers. The risks for non-asbestos-exposed nonsmokers and smokers must therefore be multiplied by a factor that increases in proportion to the cumulative exposure.

The dose–response relationship in the case of asbestos-induced lung cancer can be described by the following equation *(7)*.

$$I_L \text{ (age, smoking, fibre dose)} = I_L^o \text{ (age, smoking)}[1 + K_L \times C_f \times d]$$

This equation could also be written as:

$$K_L = [(I_L/I_L^o) - 1]/C_f \times d = (\text{relative risk} - 1)/(\text{cumulative exposure})$$

where:

K_L = a proportionality constant, which is a measure of the carcinogenic potency of asbestos

C_f = fibre concentration

d = duration of exposure in years

I_L = lung cancer incidence, observed or projected, in a population exposed to asbestos concentration C_f during time d

I_L^o = lung cancer incidence expected in a group without asbestos exposure but with the same age and smoking habits (this factor includes age dependence).

There are several studies that allow the calculation of K_L. Liddell *(10, 11)* has done this in an interesting and consistent manner. The results are given in Table 15.

Taking the data in Table 15 as a basis, a reasonable estimate for K_L is 1.0 per 100 F*years/ml. For a given asbestos exposure, the risk for smokers is about 10 times that for nonsmokers *(9)*. In extrapolating from workers to the general public, a factor of 4 for correction of exposure time has to be applied to K_L.

The incidence of lung cancer in the general population exposed to 100 F*/m^3 is calculated as follows:

$$I_L = I_L^o(1 + 4 \times 0.01 \times 10^{-4} \text{ F*/ml} \times 50 \text{ years})$$

or

$$I_L = I_L^o(1 + 2 \times 10^{-4} \text{ F*/ml})$$

Table 15. Increase in the relative risk of lung cancer, as shown by different studies

K_L per 100 F*year/ml	Type of activity	Reference
0.04	mining and milling	*(12)*
0.045	mining and milling	*(13)*
0.06	friction material	*(14)*
0.1	factory processes	*(15, 16)*
(M) 0.4–1.1	factory processes	
(F) 2.7 [a]	factory processes	*(17)*[b]
0.2	asbestos-cement	*(18)*
0.07	textiles (before 1951)	*(19)*
0.8 [a]	textiles (after 1950)	
6(M) 1.6 [a]	textiles	*(20)*
1.6	textiles	*(21)*[c]
1.1	insulation products	*(22)*[b]
1.5	insulation	*(23)*[b]

[a] Fewer than 10 cases of lung cancer expected (i.e. small cohort).

[b] Inadequate knowledge of actual fibre concentrations.

[c] Same factory as in *(20)*, but larger cohort.

Source: Liddell *(10)*.

The extra risk is $I_L - I_L^o$. Values for I_L^o are about 0.1 for male workers and 0.01 for male nonsmokers *(5)*.

Lifetime exposure to 100 F*/m^3 (lifetime assumed to be 50 years since, in a lifetime of 70 years, the first 20 years without smoking probably do not make a large contribution) is therefore estimated as follows.

Status	Risk of lung cancer per 100 000	Range (using the highest and lowest values of K_L from Table 15)
Smokers	2.0	0.08–3.2
Nonsmokers	0.2	0.008–0.32

This risk estimate can be compared, when adjusted to 100 F*/m^3, with estimates for male smokers made by other authors or groups:

Breslow (National Research Council) *(6)*: 7.3×10^{-5}

Schneiderman et al. *(4)*: $(14–1.4) \times 10^{-5}$

US Environmental Protection Agency *(7)*: 2.3×10^{-5}.

A fibre concentration of 100 F*/m^3 (about 200 F/m^3 as seen by scanning electron microscope) thus gives a total risk of $(2 + 2) \times 10^{-5}$ for smokers or 2.2×10^{-5} for nonsmokers.

Guidelines

Asbestos is a proven human carcinogen (IARC Group 1). No safe level can be proposed for asbestos because a threshold is not known to exist. Exposure should therefore be kept as low as possible.

Several authors and working groups have produced estimates indicating that, with a lifetime exposure to 1000 F/m^3 (0.0005 F*/ml or 500 F*/m^3, optically measured) in a population of whom 30% are smokers, the excess risk due to lung cancer would be in the order of 10^{-6}–10^{-5}. For the same lifetime exposure, the mesothelioma risk for the general population would be in the range 10^{-5}–10^{-4}. These ranges are proposed with a view to providing adequate health protection, but their validity is difficult to judge. An attempt to calculate a "best" estimate for the lung cancer and mesothelioma risk is described above.

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