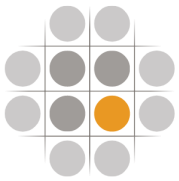
Supported by

SoBRA

The Society of Brownfield Risk Assessment



Category 4 Screening Levels: Naphthalene

CL:AIRE

ISBN: 978-1-905046-46-1

Published by CL:AIRE, Reading Business Centre, Fountain House, Queens Walk, Reading, RG1 7QF.
Web: www.claire.co.uk Email: enquiries@claire.co.uk

Report Citation
It is recommended citation to this report is made as follows:
CL:AIRE, 2024. Category 4 Screening Levels: Naphthalene. CL:AIRE, Reading. ISBN 978-1-905046-46-1. Download at www.claire.co.uk/c4sl

Disclaimer
This and other documents in this C4SL Phase 2 project have been developed for the Soil and Groundwater Technology Association (SAGTA) by the following:

- C4SL Phase 2 Project Team – see page ii where the team members are listed.
- C4SL Phase 2 Steering Group – see page ii where the participants are listed.

The work reported herein together with other related documents was carried out on an agreed basis by the companies and organisations listed on page ii. However, any views expressed are not necessarily those of the members of the Phase 2 Project Team, SAGTA as the client, the Steering Group member organisations nor any individual's personal view.

Documents are intended to provide information on the risk that may be posed by particular potentially contaminative substances in soil, which readers may find relevant to the assessment of risk to human health by land affected by contamination.

However, it is emphasised that users must not refer to the Category 4 Screening Levels in isolation. The values are based on detailed exposure elements and toxicological opinions. As such, in referring to the documents it is emphasised that users:

- Must satisfy themselves that they fully understand their derivation and limitations as are described in the text
- Should undertake their own checks on accuracy to again satisfy themselves that the contents are appropriate for their intended use
- Take appropriate specific professional advice as may be necessary to fulfil these criteria

SAGTA is making outputs freely available to industry via downloading from the CL:AIRE website (www.claire.co.uk). As such, they may be reproduced free of charge in any format or medium. This is subject to them being reproduced accurately and not in a misleading context, as well as them being fully and appropriately referenced.

In making the documents available, it is on the basis that SAGTA, the Steering Group and the Project Team are not engaged in providing a specific professional service.

Whilst reasonable skill and care has been made to ensure the accuracy and completeness of the work and the content of the documents, no warranty as to fitness for purpose is provided or implied.

CL:AIRE, SAGTA, the Project Team or the Steering Group neither accept nor assume any responsibility for any loss or damage howsoever arising from the interpretation or use of the information within the documents, or reliance upon views as may have been included.

Foreword by Frank Evans, Chair of SAGTA

Looking back, the original Defra work from 2014 that developed the Category 4 Screening Levels (C4SL) was important in establishing the level at which risk from land contamination was considered to be acceptably low. It also provided a useful scientific framework for making this assessment of risk. I was also impressed by the delivery model used to create the Soil Generic Assessment Criteria in 2010 and in particular the strength that comes from the collective efforts of a group of experts and peers.

This report presents an output from a phase 2 project to develop a further set of C4SL. It is the result of a cross-industry collaboration brought together by seed funding from SAGTA, project management from CL:AIRE and a project team made up of a number of toxicologists and exposure modellers' who have given considerable time and expertise. This guidance document would not have been possible without everyone's collaborative working, determination, and enthusiasm. My deepest thanks go to them, and to the members of the Steering Group who have overseen the development of this guidance document.

I would also acknowledge the effort and commitment of Doug Laidler who was the long-standing secretary of SAGTA and who played an important role in initiating and coordinating the project. Sadly, Doug died in the autumn of 2019 and as with so many other matters in his life, was unable to see this work brought to conclusion. May he rest in peace.

Frank Evans
Chair of SAGTA

Acknowledgements

Project Management Team

Naomi Earl	
Simon Firth	Firth Consultants Ltd
Nicola Harries	CL:AIRE

Project Team

Camilla Alexander-White	MK Tox & Co Ltd
Laura Aspinall	RSK
Kate Baker	Antea Group UK
Gareth Barns	Geosyntec
Dave Brooks	Sirius
Sarah Bull	TARA Consulting
Lucy Burn	Worley
Catherine Cussell	RSK
Simon Cole	Hydrock
Melinda Evans	Soilfix
Natasha Glynn	AtkinsRéalis
Duncan Grew	Worley
George Kowalczyk	GK Toxicology Consulting
James Lymer	
Barry Mitcheson	WSP
Rob Reuter	Wardell Armstrong LLP
Adam Symonds	Worley
Gareth Wills	WSP
Joanna Wilding	Freelance

Steering Group Members and Nominated Contact

AGS	Mike Plimmer
Brookbanks Consulting	Richard Boyle
Defra	Harriet Cooper and Rachel Boulderstone
Environment Agency	Ian Martin and Angela Haslam
EIC	Richard Puttock
EPUK	Karen Thornton
HBF	Frances Gregory
Newport City Council	Steve Manning
NHBC	Steve Moreby
NRW	Matthew Llewhellin
UK Health Security Agency	Sarah Dack and Kerry Foxall
Public Health Wales	Andrew Kibble
Rochdale Borough Council (MAPAC)	Michael Moore
SAGTA	Daniel May and Hannah White
SoBRA	Rachel Dewhurst
Welsh Contaminated Land Group	Rachael Davies
Welsh Government	Andrew Williams and Richard Clark
Wyre Council	David Johnson
YALPAG	Lucie Watson

CONTENTS

ABBREVIATIONS

ADE	Average Daily Exposure
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark Dose
BMDS	Benchmark Dose Software
C4SL	Category Four Screening Level
CAS	Chemical Abstracts Service
CL:AIRE	Contaminated Land: Applications in Real Environments
CLEA	Contaminated Land Exposure Assessment
CSAF	Chemical-Specific Adjustment Factor
Defra	Department for Environment, Food and Rural Affairs
DW	Dry Weight
ECHA	European Chemicals Agency
EFSA	European Food Safety Authority
ELCR	Excess Lifetime Cancer Risk
ETS	Environmental Tobacco Smoke
HBGV	Health Based Guidance Value
HCV	Health Criteria Value
IRIS	Integrated Risk Information System
LLTC	Low Levels of Toxicological Concern
$LLTC_{inhal}$	Low Levels of Toxicological Concern - Inhalation
$LLTC_{oral}$	Low Levels of Toxicological Concern - Oral
LOAEC	Lowest Observed Adverse Effect Concentration
LOAEL	Lowest Observed Adverse Effect Level
MDI	Mean Daily Intake
MRL	Minimal Risk Level
NOAEC	No Observed Adverse Effect Concentration
NOAEL	No Observed Adverse Effect Level
PAH	Polycyclic Aromatic Hydrocarbon
POD	Point of Departure
POS	Public Open Space
POS_{park}	Public Open Space - Park
POS_{resi}	Public Open Space – Residential
RIVM	National Institute of Public Health and the Environment (Netherlands)
SOM	Soil Organic Matter
SR	Science Report
UF	Uncertainty Factor
UK	United Kingdom
US EPA	United States Environmental Protection Agency

1. INTRODUCTION

This report presents Category 4 Screening Levels (C4SLs) for naphthalene based on the methodology described in Section 5 of CL:AIRE (2014) "SP1010 – Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination". Section 1.1 provides brief background information on naphthalene while Section 2 summarises the toxicological review from which Low Levels of Toxicological Concern (LLTCs) are identified. Section 3 presents the exposure modelling aspects for the generic land-uses under consideration (Step 3), while Section 4 presents the C4SLs.

1.1 BACKGROUND TO NAPHTHALENE

Naphthalene (CAS No. 91-20-3) is the structurally simplest polycyclic aromatic hydrocarbon (PAH) and has the chemical formula $C_{10}H_8$. Naphthalene is a white crystalline solid with a strong tar-like odour at room temperature, and when mixed with air its vapours easily burn (ATSDR, 2005). Naphthalene odours can be smelled by humans at low concentrations beginning at 84 ppb (440 µg m^{-3}) in air and 21 ppb (≈21 µg L^{-1}) in water (ATSDR, 2005).

Naphthalene is formed naturally by the incomplete combustion or pyrolysis of organic materials and most commonly enters the environment from the burning of wood and use of fossil fuels. Other sources include tobacco use (ATSDR, 2005).

Naphthalene is a volatile compound (vapour pressure of approximately 2.31 Pa at 10˚C) and is sparingly soluble in water (19 mg L^{-1} at 10˚C) (Environment Agency, 2008).

Naphthalene is expected to volatilise readily from soils and surface waters, and to sorb weakly to soil and sediments (ATSDR, 2005). Once in the atmosphere naphthalene is removed through reactions with photochemically-produced hydroxyl radicals, with an estimated atmospheric half-life of <1 day (ATSDR, 2005).

Biodegradation of naphthalene in soils occurs via aerobic microorganisms, with degradation rates observed to decline when soils become anaerobic (Klecka *et al.,* 1990, as referenced in ATSDR, 2005). There is considerable variability in reported naphthalene soil half-lives from circa 2 days to 3.5 months (ATSDR, 2005).

Naphthalene is degraded in water by photolysis and biological processes (ATSDR, 2005) with half-life estimates ranging from 12 hours to 20 days for surface water and 24 hours to 258 days for groundwater (Howard*,* 1991).

2. DERIVATION OF LOW LEVEL OF TOXICOLOGICAL CONCERN FOR NAPHTHALENE

A framework for evaluating chemical-specific toxicology data for the purposes of LLTC derivation is presented in the form of a flowchart in Figure 2.2 of the SP1010 report (CL:AIRE, 2014) and reproduced below as Figure 2.1. The remainder of this section demonstrates the application of this framework to naphthalene. A proforma summarising the pertinent information referred to in this section is included as Appendix A.

As indicated in Figure 2.1, the first task is to perform a review of existing health based guidance values (HBGV) for all routes of exposure, collating information from authoritative bodies, as per the process in SR2 (Environment Agency, 2009a).

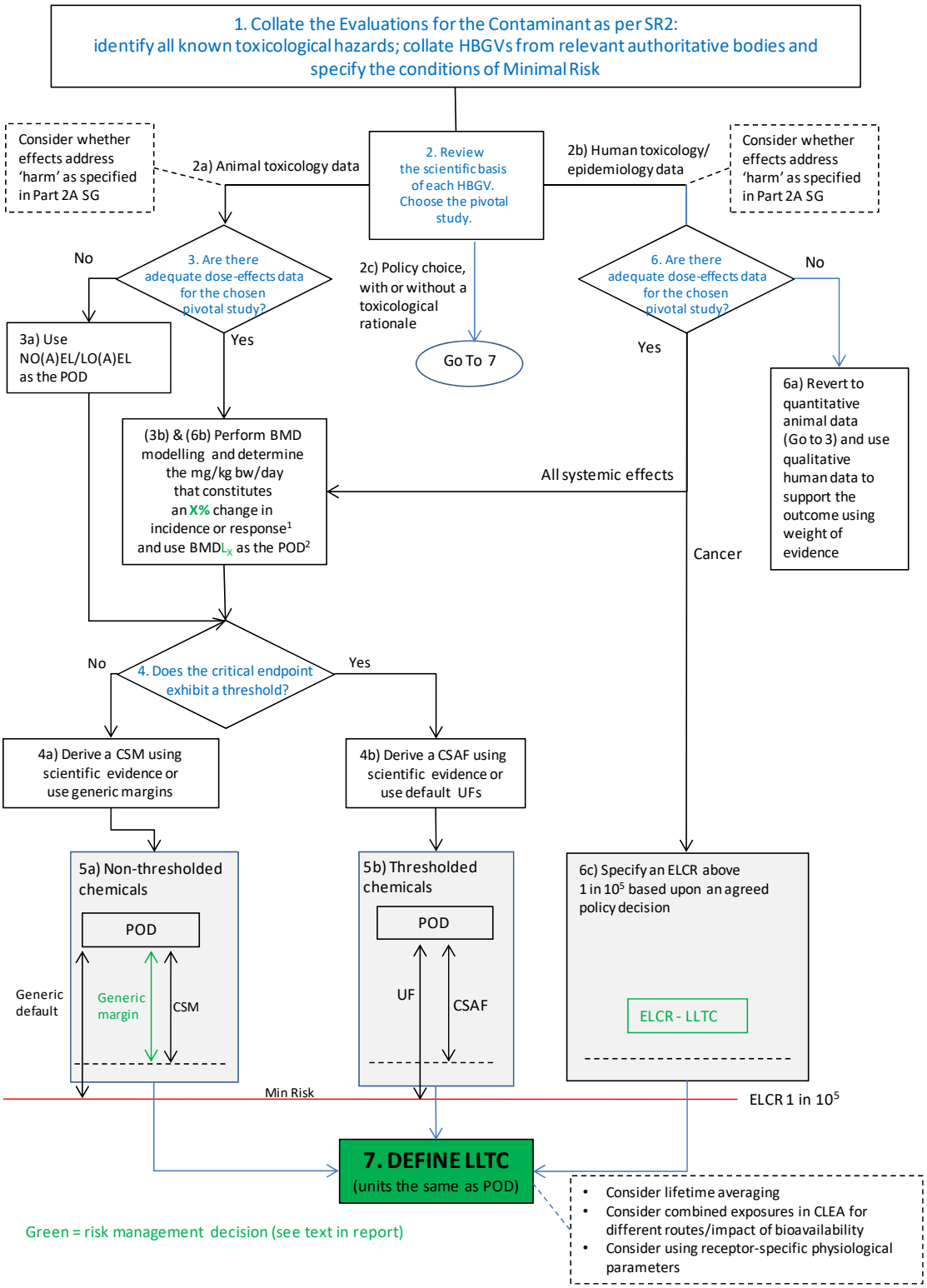


Figure 2.1: A framework for evaluating chemical-specific toxicology data for the purposes of LLTC derivation (reproduced from Figure 2.2 of SP1010 (CLAIRE, 2014a)).

2.1 ORAL ROUTE

2.1.1 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

A review of toxicological hazards and available HBGVs presented by authoritative bodies for the oral route of exposure has been undertaken and is provided in Appendix A. This review indicates that decreased body weight and kidney lesions are the most sensitive[1] toxicological effects following exposure to naphthalene by the oral route. These are threshold effects and therefore a thresholded $LLTC_{oral}$ will be derived for naphthalene.

2.1.2 FLOWCHART ELEMENT 2: Review the scientific basis of each HBGV. Choose the pivotal study

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).

2a) Animal Toxicology Data

A range of toxic endpoints are seen in animal studies following exposure to naphthalene, including decreased body and organ weights, kidney lesions, and thyroid hypertrophy. Cataracts and haemolytic anaemia have also been observed, but not consistently.

Due to the limited chronic oral data available, an unpublished sub-chronic toxicity study in F344 rats, undertaken by Battelle's Colombus Laboratories as part of the National Toxicology Program (NTP) has been selected as the pivotal study (NTP, 1980[2]).

In the 90-day NTP study, doses of 0, 25, 50, 100, 200 and 400 mg kg^{-1} bw day^{-1} naphthalene were administered by gavage in corn oil to groups of 10 male and 10 female F344 rats, 5 days per week for 13 weeks. There was a low incidence of kidney lesions in male rats at 200 and 400 mg kg^{-1} bw day^{-1}. Body weights were found to decrease with increased dose, with the most marked changes occurring at the highest dose group of 400 mg kg^{-1} bw day^{-1} in both males and females (NTP, 1980).

A no observed adverse effect level (NOAEL) of 100 mg kg^{-1} bw day^{-1} and a lowest observed adverse effect level (LOAEL) of 200 mg kg^{-1} bw day^{-1} were selected from this study based on a >10% decrease in mean terminal body weight in males compared to the controls and kidney lesions at 200 mg kg^{-1} bw day^{-1}. The NOAEL was adjusted to 71 mg kg^{-1} bw day^{-1} to account for continuous exposure (5 days per week / 7 days per week) and the LOAEL similarly adjusted to 143 mg kg^{-1} bw day^{-1}.

The United States Agency for Toxic Substances and Disease Registry (ATSDR, 2005) identified 200 mg kg^{-1} bw day^{-1} as a "less serious" LOAEL and identified a "serious" LOAEL of 400 mg kg^{-1} bw day^{-1} based on a 28% decrease in mean terminal body weights in male rats; however only minimal renal lesions occurred in 10% of male rats at this dose. NTP (1980) was also selected as the critical study by the United States Environmental Protection Agency (US EPA, 1998) and Defra and Environment Agency (2003). The ATSDR also selected the study to derive an intermediate-duration minimal risk level (MRL), although it

---

[1] In defining minimal/tolerable risk, it is only necessary to focus on the most sensitive of all effects in defining the HBGV. In order to choose a point on the dose-response curve that is higher than minimal/tolerable risk, it is important to note that the dose-responses for the most sensitive effects may overlap with other effects. Therefore, in setting the LLTC, ALL endpoints must be borne in mind. This is an important principle in any of the toxicological evaluations where there are overlapping toxicological effects data, and is an important departure from the principles of evaluation of minimal/tolerable risk as described in SR2.

[2] Battelle's Columbus Laboratories study referenced by ATSDR (2005) as NTP (1980). "Subchronic toxicity study: Naphthalene (C52904), F344 rats. Research Triangle, Park, NC: U.S. Department of Health and Human Services, National Toxicology Program." ATSDR (2005) and US EPA (1998) have described the study in sufficient detail to be used as the pivotal study without the original report.

was concluded that there were no appropriate data from which to derive the chronic MRL (ATSDR, 2005).

The data from NTP (1980) were considered by the US EPA to be suitable for benchmark dose (BMD) modelling (US EPA, 1998). However, it subsequently chose not to use the results of the modelling on the basis that these did not markedly reduce uncertainty or provide a significant advantage in deriving a reference dose for naphthalene. Insufficient data are presented in the IRIS Appendix to re-run the calculations through the US EPA's BMD modelling software (BMDS).

Adverse health effects have been reported in other studies at doses lower than the LOAEL of 200 mg kg^{-1} bw day^{-1}. A 90-day gavage study in CD-1 mice (Shopp *et al.,* 1984) identified organ weight decreases (brain, liver and spleen) at 133 mg kg^{-1} bw day^{-1}. However, this dose was administered 7 days per week, as opposed to 5 days per week for the NTP (1980) study. Adjusting the LOAEL from the NTP (1980) study for continuous exposure (7 days per week) gives an adjusted LOAEL of 143 mg kg^{-1} bw day^{-1} (200 mg kg^{-1} bw day^{-1} x 5/7), which is similar to the LOAEL from Shopp *et al*.

GO TO FLOWCHART ELEMENT 3

2b) Human Toxicology/Epidemiology Data

Although there are reported cases of haemolytic anaemia in humans (ATSDR, 2005), the data are inadequate to draw any conclusions about the dose-response relationship. Human toxicological or epidemiological data are not applicable to the derivation of an oral LLTC for naphthalene.

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

There is no UK drinking water standard for naphthalene and so this is not applicable to the derivation of an oral LLTC.

2.1.3 FLOWCHART ELEMENT 3: Are there adequate dose-effects data for the chosen pivotal study to perform BMD modelling – animal data?

Yes	No	Not applicable
	X	

Body weight decreases and kidney lesions in F344 rats, reported in an unpublished sub-chronic 90-day study (NTP, 1980), are considered to be the critical effects on which to base the $LLTC_{oral}$.

GO TO FLOWCHART ELEMENT 3a/b

2.1.4 FLOWCHART ELEMENT 3a: Use NOAEL/LOAEL as POD

The NOAEL of 100 mg kg^{-1} bw day^{-1} (71 mg kg^{-1} bw day^{-1} adjusted) was selected as the point of departure (POD) as effects were noted in other studies between the NOAEL and the LOAEL of 200 mg kg^{-1} bw day^{-1} (143 mg kg^{-1} bw day^{-1} adjusted).

2.1.5 FLOWCHART ELEMENT 3b: Perform BMD modelling

As discussed above, whilst US EPA (1998) considered the data to be suitable for benchmark modelling, it subsequently chose the NOAEL (as opposed to the BMD modelling results) as the POD for derivation of the reference dose. Insufficient data are presented in the IRIS Appendix to carry out BMD modelling.

GO TO FLOWCHART ELEMENT 4a/b

2.1.6 FLOWCHART ELEMENT 4: Does the critical endpoint exhibit a threshold?

Yes	No	Not applicable
X		

2.1.7 FLOWCHART ELEMENT 4a: Define a suitable chemical-specific margin

Not applicable for threshold effects.

2.1.8 FLOWCHART ELEMENT 4b: Derive a chemical-specific assessment factor (CSAF) using scientific evidence or use default UFs

For the derivation of an $LLTC_{oral}$, the default uncertainty factors (UFs) are proposed as per the following:

- Intraspecies variability: 10 (to account for toxicokinetic and toxicodynamic variability within the human population);
- Interspecies variability: 10 (to account for toxicokinetic and toxicodynamic variability between humans and rats);
- Short duration study: 3 (to account for extrapolation from sub-chronic to chronic exposure). This is within the range of uncertainty factors typically applied for use of short duration studies noting that US EPA (1998) applied a factor of 10, whilst the European Chemicals Agency (ECHA, 2012) recommends a default uncertainty factor of 2 for extrapolation from sub-chronic to chronic; and
- Database deficiencies: 3 (to account for lack of reproductive toxicity data).

As with other C4SLs, where two UFs of 3 are combined, these are rounded up from 9 to 10. Therefore a total UF of 1000 is proposed.

GO TO FLOWCHART ELEMENT 5b

2.1.9 FLOWCHART ELEMENT 5b: Calculate the LLTC for thresholded chemicals

For thresholded chemicals, the POD is divided by the total UF to derive the LLTC:

POD/UF = LLTC (units as per POD)

Therefore, for this evaluation:

NOAEL/1000 = LLTC

71 mg kg^{-1} bw day^{-1}/1000 = 0.071 mg kg^{-1} bw day^{-1} = 71.0 µg kg^{-1} bw day^{-1}

GO TO FLOWCHART ELEMENT 7

2.1.10 FLOWCHART ELEMENT 7: Assess LLTC for naphthalene

Based upon a scientific evaluation of decreased body weights and kidney lesions in F344 rats, an oral LLTC of **71 µg kg^{-1} bw day^{-1}** is proposed, based on a NOAEL of 71 mg kg^{-1} bw day^{-1} and a total UF of 1000. This LLTC value is 3.5 times higher than the minimal risk oral HBGV derived by Defra and Environment Agency (2003) of 20 µg kg^{-1} bw day^{-1}.

2.2 INHALATION ROUTE

2.2.1 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

A review of toxicological hazards and available HBGVs presented by authoritative bodies for the inhalation route of exposure has been undertaken and is provided in Appendix A. This review indicates that respiratory and nasal epithelium effects are the most sensitive[3] toxicological effect following exposure to naphthalene by the inhalation route.

2.2.2 FLOWCHART ELEMENT 2: Review the scientific basis of each HBGV. Choose the pivotal study

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).

2a) Animal Toxicology Data

The pivotal animal study chosen for the inhalation effects of naphthalene is that of Dodd *et al.* (2012) in which mild to minimal hyperplasia of the nasal transitional/respiratory epithelial was identified as the critical endpoint.

In the Dodd *et al.* (2012) study, F344 rats (10/sex/group) were exposed in whole body chambers to naphthalene vapour for 6 hours/day, 5 days/week for 13 weeks at concentrations of 0, 0.1, 1, 10 or 30 ppm (0, 0.524, 5.24, 52.4 or 157.2 mg m^{-3}). Mild decreases in body weight (<10%) and food/water consumption were observed in the rats exposed to 30 ppm. There were no naphthalene-related clinical observations at any concentration.

Nasal transitional/respiratory epithelial hyperplasia and olfactory epithelial degeneration, all considered to be local, site of action effects, were observed at 1 ppm and were described by the authors as 'minimal'. Such minimal effects were reported in 10/10 males (histopathology score of 1 = minimal) (female data are not presented but were said to be similar).

At 10 ppm, hyperplastic lesions of the respiratory and of the olfactory epithelium in males were scored 1.4 and 1.6, respectively. These effects were higher than the "minimal" criterion score of 1 but did not reach the "slight/mild" criterion score of 2. Mild hyperplasia and minimal squamous metaplasia were observed in the respiratory epithelium of rats exposed at either 10 or 30 ppm.

Systemically, only minimal effects were noted in this study. Statistically significant decreases in relative (although not absolute) organ weights were noted for spleen, testis, heart and thymus at 10 ppm and above, but without gross observations at necropsy (no histopathology was performed). Absolute liver and heart weights were significantly reduced in females exposed to 0.1 ppm, and also significantly reduced at some, but not all, of the higher exposure levels (male data not provided in the paper). However, all systemic effects diminished in magnitude and were not statistically significant after a 4-week recovery period.

The study by Dodd *et al.* (2012) is more recent than the authoritative reviews conducted by ATSDR (2005), US EPA (1998), WHO (2010), Health Canada (2008) and RIVM (2007). All

[3] In defining minimal/tolerable risk, it is only necessary to focus on the most sensitive of all effects in defining the HBGV. In order to choose a point on the dose-response curve that is higher than minimal risk, it is important to note that the dose-responses for the most sensitive effects may overlap with other effects. Therefore, in setting the LLTC, ALL endpoints must be borne in mind. This is an important principle in any of the toxicological evaluations where there are overlapping toxicological effects data, and is an important departure from the principles of how SR2 and minimal risk evaluations are implemented more simply.

the pivotal studies selected by these authoritative bodies identified nasal and respiratory epithelium effects similar to those identified in Dodd *et al.* (2012).

ATSDR selected two pivotal studies; an NTP (1992) 104 week study in mice, and an NTP (2000, Abdo *et al.,* 2001) 105 week study in F344 rats. For NTP (1992), B6C3F1 mice were exposed by inhalation at concentrations of 0, 10, and 30 ppm for 6 hours/day, 5 days/week. For the later NTP (2000) study, F344 rats were exposed by inhalation at concentrations of 0, 10, 30 and 60 ppm for 6 hours/day, 5 days/week. Both studies identified a lowest observed adverse effect concentration (LOAEC) of 10 ppm in both sexes based on non-neoplastic lesions in nasal epithelium and respiratory epithelium.

US EPA (1998) and Defra and Environment Agency (2003) selected NTP (1992) as their pivotal study and Health Canada (2008) and WHO (2010) selected NTP (2000, Abdo *et al.,* 2001). RIVM selected a 4-week rat study where signs of proliferative repair in the nasal olfactory epithelium were observed at all doses (0, 1, 3, 10, 29 and 71 ppm). RIVM considered that had the study been extended to 2 years the mild nasal effects would have progressed in severity and determined a LOAEC of 1 ppm on that basis (RIVM, 2007).

GO TO FLOWCHART ELEMENT 3

2b) Human Toxicology/Epidemiology Data

Although human data from naphthalene exposure are available, reliable dose-response information is lacking and so the data are insufficient for the purposes of determining an LLTC.

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

There is no UK air quality standard for naphthalene and so this is not applicable to the derivation of an inhalation LLTC.

2.2.3 FLOWCHART ELEMENT 3: Are there adequate dose-effects data for the chosen pivotal study to perform BMD modelling – animal data?

Yes	No	Not applicable
	X	

There are no adequate quantitative data available for BMD modelling from the Dodd *et al.* (2012) study.

GO TO FLOWCHART ELEMENT 3a/b

2.2.4 FLOWCHART ELEMENT 3a: Use NOAEL/LOAEL as POD

There is ongoing US EPA interest in naphthalene because the 2-year inhalation toxicity studies (NTP, 1992 and 2000) did not identify a no observed adverse effect concentration (NOAEC) and the lowest dose (10 ppm) produced serious effects in virtually all animals. Research to investigate a dose response below 10 ppm is ongoing and the 13-week study by Dodd *et al.* (2012), identified 0.1 ppm as a no effect level with possibly “less serious” effects at 1 ppm and “more serious" effects at 10 ppm. [NOTE: Dodd *et al.* (2012) do not specifically identify a “NOAEC” or a "LOAEC" in their paper.]

The inhalation toxicity of naphthalene displays a spectrum of dose-related epithelial effects ranging from inflammation, hyperplasia, metaplasia, and, ultimately, tumour formation, most probably via a non-genotoxic mechanism. The effects at 1 ppm identified by Dodd *et al.* may mark the start of the continuum of naphthalene-induced lesions. However, they were described by the authors as “minimal, reversible, and inconsistent”.

More consistent effects were seen at 10 ppm (still minimal effects but now at a higher grade) and therefore this dose could be considered to be the LOAEC, making 1 ppm the NOAEC.

1 ppm from Dodd *et al.* (2012) has been adopted as a POD for LLTC derivation.

Conversion from ppm intermittent dosing (6 hour/day; 5 days/week) to mg m^{-3} continuous dosing was carried out using the following factors:

1 ppm = 5.24 mg m^{-3} (on molecular weight basis as reported in ATSDR, 2005) and factors of 6/24 for full day exposure and 5/7 for full week exposure.

1 ppm (intermittent) = 1 x 5.24 mg m^{-3} x 6/24 x 5/7= 0.936 mg m^{-3} (continuous)

2.2.5 FLOWCHART ELEMENT 3b: Perform BMD modelling

Not applicable.

GO TO FLOWCHART ELEMENT 4a/b

2.2.6 FLOWCHART ELEMENT 4: Does the critical endpoint exhibit a threshold?

Yes	No	Not applicable
X		

2.2.7 FLOWCHART ELEMENT 4a: Define a suitable chemical-specific margin

Not applicable for threshold effects.

2.2.8 FLOWCHART ELEMENT 4b: Derive a chemical-specific assessment factor (CSAF) using scientific evidence or use default UFs

The UF is made up from the following factors:

- Intraspecies variability: 10 (to account for toxicokinetic and toxicodynamic variability within the human population); and
- Interspecies variability: 10 (to account for toxicokinetic and toxicodynamic variability between humans and rats).
- Short duration study: 3 (to account for extrapolation from sub-chronic to chronic exposure). It is noted that RIVM (2007) which used a 28 day study as its pivotal study, stated that "*Effects at the LOAEC were considered to be concentration-dependent rather than dose-dependent, and so adjustment for the duration of exposure was not necessary*", and on this basis the factor of 3 is considered to be appropriately, but not overly precautionary.

As discussed above, no further UF has been adopted to adjust from a LOAEC to a NOAEC because the effects observed in the Dodd *et al.* study at 1ppm (0.936 mg m^{-3} duration adjusted) were minimal, inconsistent and reversible, and therefore this dose was considered to be a NOAEC.

Therefore, a total UF of 300 is proposed.

GO TO FLOWCHART ELEMENT 5b

2.2.9 FLOWCHART ELEMENT 5b: Calculate the LLTC for thresholded chemicals

For thresholded chemicals, the POD is divided by the total UF to derive the LLTC:

POD/UF = LLTC (units as per POD)

Taking a duration adjusted NOAEC of 0.936 mg m^{-3} as the POD and applying a total UF of 300 results in a HBGV of 3.12 µg m^{-3}. Assuming a body weight of 70 kg and 20 m^3 of air inhaled per day gives a LLTC of 0.891 µg kg^{-1} bw d^{-1}.

GO TO FLOWCHART ELEMENT 7

2.2.10 FLOWCHART ELEMENT 7: Assess LLTC for naphthalene

Based upon a scientific evaluation of localised nasal effects in rats and mice, an inhalation LLTC of **0.891 µg kg^{-1} bw day^{-1}** is proposed, based on a NOAEC of 0.936 mg m^{-3} as the POD, and a total UF of 300. This LLTC value is:

a) Approximately three times lower than the WHO Air Quality Guideline Value (equivalent to 2.86 µg kg^{-1} bw day^{-1}) and almost identical to the ATSDR chronic MRL and US EPA Reference Concentration (equivalent to 0.86 µg kg^{-1} bw day^{-1});

b) Almost identical to the withdrawn Defra/Environment Agency Tolerable Daily Intake of 0.86 µg kg^{-1} bw day^{-1} (Defra and Environment Agency, 2003), which was based on the US EPA Reference Concentration.

The proposed inhalation LLTC is of the same order of magnitude as the tolerable risk values currently published by authoritative bodies and is based on more recent data where effects were seen at lower doses.

2.3 DERMAL ROUTE

There is insufficient evidence to suggest that naphthalene induces local effects on the skin.

In the absence of dermal toxicity data and in accordance with SR2 (Environment Agency, 2009a), dermal exposure will be compared against the $LLTC_{oral}$ for the purposes of the derivation of the C4SL for naphthalene.

2.4 MEAN DAILY INTAKE

The oral and inhalation LLTCs recommended for naphthalene are based on threshold effects. As such, in accordance with the C4SL framework (CL:AIRE, 2014), the mean daily intake (MDI) from non-soil sources is to be included in the exposure modelling.

Available oral and inhalation MDI data have been collated and reviewed and used to derive estimated adult MDIs for the oral and inhalation pathways (see Appendix B). The adult MDIs used to derive the C4SL for naphthalene are shown in Table 2.4 below.

The oral MDI of 7 µg day^{-1} is that presented in Defra and Environment Agency (2003). This is based on reported concentrations of naphthalene detected in carrots, butter, margarine, cheese and fish. Note that whilst more recent studies are available assessing concentrations of PAHs in foodstuffs (EFSA, 2008; Fernandes *et al.,* 2011) these do not include naphthalene. Defra and Environment Agency (2003) considered that exposure from drinking water was negligible. This is consistent with DWI (2023) which reports the maximum concentration of naphthalene detected in 1621 samples of raw water from public supplies taken in England in 2021 to be less than the limit of detection of 1 µg L^{-1}.

The inhalation MDI is based on a summary of personal exposure measurements of volatile organic compounds (VOCs) and particles associated with PAHs in three UK regions (urban, suburban and rural) presented by Saborit *et al.* (2009). Most personal exposures were considered to be associated with indoor sources and result from activities such as DIY, photocopying, increased heating in winter months (and reduced outdoor air flow) and environmental tobacco smoke (ETS). The mean value of 0.7 µg m^{-3} for non-ETS participants, which equates to an MDI of 14 µg day^{-1} for an average adult of 70 kg breathing 20 m^3 per day has been adopted as the inhalation MDI.

Table 2.4: Adult mean daily intake values for input to CLEA.

Adult Mean Daily Intake	Value ($\mu g\ day^{-1}$)
Oral MDI	7
Inhalation MDI	14

3. EXPOSURE MODELLING FOR NAPHTHALENE

As described in the C4SL SP1010 report (CL:AIRE, 2014), the CLEA model has been used deterministically with the above LLTCs to derive C4SLs for the following six land-uses for a sandy loam soil type:

- Residential with consumption of homegrown produce;
- Residential without consumption of homegrown produce;
- Allotments;
- Commercial;
- Public open space (POS):
    - The scenario of open space close to housing that includes tracking back of soil (POS_{resi}); and
    - A park-type scenario where the park is considered to be at a sufficient distance from the home that there is negligible tracking back of soil (POS_{park}).

3.1 CLEA PARAMETER INPUTS

CLEA derives an estimate of average daily exposure (ADE) for each exposure pathway. ADEs are then summed for some or all exposure pathways for comparison with the LLTC. The pathways considered in the summation are dependent on the critical toxicological effects that the LLTC is based on. CLEA uses iteration to find the soil concentrations at which the summed ADEs equal the respective LLTC values and these are termed 'assessment criteria'. As described in the CLEA SR2 and SR3 documents (Environment Agency, 2009a,b), the assessment criteria are normally integrated by CLEA to determine an overall value where the critical toxicological effects via both routes of exposure are systemic. Where the critical toxicological effect is localised for either the oral or inhalation routes of exposure, the assessment criteria are not integrated and the lowest of the two criteria is chosen as the overall assessment criterion.

The $LLTC_{oral}$ is based upon scientific evaluation of toxicity observed in an unpublished 90-day animal study (rats) completed by NTP (1980), in which threshold effects of decreased body weight and kidney lesions were observed. Given that the $LLTC_{oral}$ is based on systemic threshold effects, the assessment criteria based on the $LLTC_{oral}$ are the soil concentrations at which total exposure from all exposure pathways (including oral and inhalation background exposure from non-soil sources) equals the $LLTC_{oral}$.

The $LLTC_{inhal}$ is based upon scientific evaluation of toxicity observed in the pivotal animal study (rats) completed by Dodds *et al.* (2012), in which localised nasal threshold effects were observed. As the $LLTC_{inhal}$ is based on localised threshold effects, assessment criteria based on the $LLTC_{inhal}$ are the soil concentrations at which inhalation exposure (including inhalation background exposure from non-soil sources) equals the $LLTC_{inhal}$.

The C4SLs for naphthalene are the lower of the assessment criteria derived using either the $LLTC_{inhal}$ or $LLTC_{oral}$.

CLEA requires a number of contaminant and non-contaminant specific parameter values for modelling exposure. The description of these parameters is provided within the C4SL SP1010 report (CL:AIRE, 2014) and the SR3 report (Environment Agency, 2009b). Contaminant-specific parameter values used for naphthalene are shown in Table 3.1.

Table 3.1: Contaminant-specific parameter values used for derivation of C4SLs for naphthalene.

Parameter	Units	Value	Source/Justification
Air-water partition coefficient	dimensionless	6.62×10^{-3}	SR7, Environment Agency, 2008
Diffusion coefficient in air	$m^2\ s^{-1}$	6.52×10^{-6}	SR7, Environment Agency, 2008
Diffusion coefficient in water	$m^2\ s^{-1}$	5.16×10^{-10}	SR7, Environment Agency, 2008
Relative molecular mass	$g\ mol^{-1}$	128.17	SR7, Environment Agency, 2008
Vapour pressure	Pa	2.31	SR7, Environment Agency, 2008
Water solubility	$mg\ L^{-1}$	19	SR7, Environment Agency, 2008
Log K_{oc}	Log $cm^3\ g^{-1}$	2.81	SR7, Environment Agency, 2008
Log K_{ow}	dimensionless	3.34	SR7, Environment Agency, 2008
Dermal absorption fraction	dimensionless	0.13	CLEA SR3, Environment Agency 2009b
Soil-to-plant concentration factor (green vegetables)	mg g^{-1} FW plant over mg g^{-1} DW soil	5.08×10^{-3}	Environment Agency, unpublished data (Note that CLEA does not model soil-to-plant concentration factors for organic substances for shrub fruit)
Soil-to-plant concentration factor (root vegetables)		1.13×10^{-1}	
Soil-to-plant concentration factor (tuber vegetables)		4.97×10^{-2}	
Soil-to-plant concentration factor (herbaceous fruit)		3.22×10^{-2}	
Soil-to-plant concentration factor (shrub fruit)		-	
Soil-to-plant concentration factor (tree fruit)		modelled	
Soil-to-dust transport factor	g g^{-1} DW	0.5	Default value from CLEA SR3, Environment Agency 2009b
Sub-surface soil to indoor air correction factor	-	10	Environment Agency, 2009a
Relative bioavailability soil	-	1	Conservative assumption made that bioavailability of naphthalene in soil and dust is the same as bioavailability of naphthalene in critical toxicological studies used to derive the LLTC
Relative bioavailability dust	-	1	

The key contaminant-specific parameter values used for derivation of the C4SLs for naphthalene are discussed briefly below.

Dermal absorption factor

The CLEA model uses a generic default dermal absorption factor of 0.1 unless there are chemical-specific literature values available (SR3, Environment Agency, 2009b). Table 8.2 in SR3 summarises the available dermal absorption factors, which includes a value of 0.13 for 'benzo(a)pyrene and other PAHs'. This value has been selected for naphthalene.

Soil to dust transport factor

The soil to dust transport factor should be contaminant specific but where contaminant-specific data are not available, the SR3 report (Environment Agency, 2009b) recommends a default value of 0.5, meaning that the concentration of contaminant in respirable dust is assumed to be 50% of the concentration of contaminant in outdoor soil. This default value has been used to calculate the C4SL.

Soil to plant concentration factors

The Environment Agency undertook a review of the scientific literature on the plant uptake of benzo(a)pyrene and naphthalene by fruit and vegetables based on findings from literature searches conducted during November 2008 and October 2009 (Environment Agency, unpublished data). As part of this review they collated soil to plant concentration factors from available studies. These were calculated from the ratio of concentration of the contaminant in the plant ($mg^{-1}\ kg^{-1}$ fresh weight [FW]) to the concentration of the

contaminant in soil (mg^{-1} kg^{-1} DW). The summary statistics for the collated concentration factors are shown in Table 3.2. Note that soil organic matter was generally not reported from the studies.

Table 3.2: Summary statistics for soil to plant concentration factors for naphthalene.

Produce category	Soil-to-plant concentration factors (mg g^{-1} FW plant over mg g^{-1} DW soil)				
	Geometric mean	Minimum	Maximum	Standard Deviation	Number of studies
Green vegetables	5.08×10^{-3}	1.95×10^{-3}	1.32×10^{-2}	7.95×10^{-3}	2
Root vegetables	1.13×10^{-1}	3.00×10^{-2}	5.39×10^{-1}	1.90×10^{-1}	6
Tuber vegetables	4.97×10^{-2}	7.76×10^{-3}	3.28×10^{-1}	9.75×10^{-2}	9
Herbaceous fruit	3.22×10^{-2}	1.38×10^{-2}	7.51×10^{-2}	4.33×10^{-2}	2
Shrub fruit	–	–	–	–	–
Tree fruit	–	–	–	–	–

The geometric mean values derived by the Environment Agency have been used as the soil to plant concentration factors for green vegetables, root vegetables, tuber vegetables and herbaceous fruit. There were insufficient data for the Environment Agency to calculate soil to plant concentration factors for shrub fruit and tree fruit. The CLEA model algorithms have been used to calculate a soil to plant concentration factor for tree fruit. The CLEA model does not contain algorithms for calculating soil to plant concentration factors for shrub fruit for organic substances and so exposure via shrub fruit is not considered in the C4SLs for naphthalene.

CLEA predicts the greatest exposure to naphthalene from consumption of homegrown produce to be via root vegetables and tree fruit for both the Residential and Allotments land-use scenarios. Therefore, in accordance with the “top two” approach, 90th percentile consumption rates have been used for these two produce types and mean consumption rates have been used for the remaining produce types.

Sub-surface soil to indoor air correction factor

The CLEA model assumes simple linear partitioning between soil and soil vapour concentrations and does not account for biodegradation in the unsaturated zone. As a result the CLEA model can significantly over-predict indoor air concentrations for some substances, particularly hydrocarbons (CL:AIRE, 2014). For this reason, a sub-surface soil to indoor air correction factor is included within the CLEA model to account for over-prediction.

CIRIA (2009) compared measured soil gas concentrations of petroleum hydrocarbons with concentrations predicted using simple linear partitioning and found that soil gas concentrations were over-predicted by at least a factor of 10 and generally by more than a factor of 1000. Given the likely level of over-prediction of soil-gas concentrations and the fact that naphthalene (a petroleum hydrocarbon) is readily degraded in aerobic environments, it is considered reasonable to apply a soil to indoor air correction factor of 10 for the derivation of C4SLs for naphthalene. This is the lower end of the range of over-predictions reported and therefore considered precautionary.

Relative bioavailability

There are few data available on the relative bioavailability of naphthalene and it is considered appropriately conservative to assume a relative bioavailability of 100% for the derivation of C4SLs.

4. C4SLs FOR NAPHTHALENE

4.1 C4SLS

The C4SLs for naphthalene derived using a Soil Organic Matter (SOM) content of 1%, 2.5% and 6% are presented in Table 4.1.

Table 4.1: C4SLs for naphthalene.

Land-use	C4SLs (mg.kg^{-1})		
	SOM Content		
	1.0%	2.5%	6.0%
Residential with consumption of homegrown produce	15	36	85
Residential without consumption of homegrown produce	15	36	85
Allotments	65	130	200
Commercial	1,600 *	3,700 *	8,400 *
Public Open Space (residential)	11,000 *	15,000 *	17,000 *
Public Open Space (park)	800 *	1,200 *	1,900 *

N.B. These C4SLs are based on chronic risk only. For further discussion of acute risks and other factors that should be considered when using these C4SLs see Section 4.2.
* C4SL exceeds the CLEA calculated soil saturation concentration (76 mg kg^{-1} for 1% SOM, 183 mg kg^{-1} for 2.5% SOM and 432 mg kg^{-1} for 6% SOM) – see final bullet in Section 4.2.

The ADE:HCV[4] ratios at the C4SL (6% SOM) for both the oral and inhalation LLTCs are shown in Table 4.2. The relative contribution of each exposure pathway to overall exposure at the C4SL (6% SOM) is shown for each land-use in Table 4.3.

As discussed in Section 3.1, the C4SLs for naphthalene are the lowest of the calculated assessment criteria based on the oral and inhalation LLTCs. The C4SLs for Allotments land-use are based on comparison of total exposure (from oral, dermal and inhalation pathways combined) with the oral LLTC, whereas the C4SLs for the remaining land-uses are based on comparison of inhalation exposure to the inhalation LLTC.

Table 4.2: ADE:HCV ratios at C4SLs derived at 6% SOM.

Land-use	ADE:HCV Ratio for Oral LLTC	ADE:HCV Ratio for Inhalation LLTC
Residential with consumption of homegrown produce	0.08	1.00
Residential without consumption of homegrown produce	0.02	1.00
Allotments	1.00	0.10
Commercial	0.07	1.00
Public Open Space (residential)	0.93	1.00
Public Open Space (park)	0.06	1.00

N.B. ADE:HCV ratios presented have been calculated using ratio mode in CLEA for soil with concentrations equal to the derived C4SLs (unrounded) for 6% SOM

[4] "ADE:HCV ratio" is the term used within the CLEA model, referring to the ratio between the average daily exposure and the health criteria value. Although an LLTC is used in place of the HCV the terminology has been retained, reflecting the CLEA output.

Table 4.3: Relative contributions of exposure pathways to overall exposure at 6% SOM.

Exposure pathway	Relative contribution to total exposure (%)					
	Residential		Allotments	Commercial	POS_{resi}	POS_{park}
	With home grown produce	Without home grown produce				
Direct soil & dust ingestion	9.49	26.74	0.58	67.68	90.58	60.96
Sum of consumption of homegrown produce and attached soil	64.50	0.00	97.24	0.00	0.00	0.00
Dermal contact (indoor)	0.23	0.65	0.00	5.86	3.57	0.00
Dermal contact (outdoor)	0.32	0.90	0.37	8.67	4.18	7.84
Inhalation of dust (indoor)	0.02	0.06	0.00	0.46	0.32	0.00
Inhalation of dust (outdoor)	0.00	0.00	0.00	0.00	0.00	0.02
Inhalation of vapour (indoor)	6.69	18.84	0.00	11.25	0.00	0.00
Inhalation of vapour (outdoor)	0.01	0.02	0.06	0.70	0.27	8.22
Oral background	5.94	16.73	0.55	1.80	0.36	7.28
Inhalation background	12.80	36.05	1.20	3.59	0.73	15.69

N.B. Exposure contributions presented have been calculated using ratio mode in CLEA for soil with concentrations equal to the derived C4SLs for 6% SOM

Note that the exposure contributions shown in Table 4.3 do not necessarily reflect contribution to overall risk as risk is dependent on the applicable LLTC for each pathway as well as exposure. For example, for Residential land-use with consumption of homegrown produce, inhalation of vapours indoors is the risk driving pathway despite greater exposure being predicted for consumption of homegrown produce. This is because the inhalation LLTC is two orders of magnitude lower than the oral LLTC.

Based on the information in Tables 4.2 and 4.3[5], the principal risk driving pathways for naphthalene are expected to be:

- Consumption of homegrown produce for the Allotments land-use;
- Indoor inhalation of vapours for Residential with Homegrown Produce, Residential without Homegrown Produce and Commercial land-uses;
- Ingestion of soil and soil derived dust and inhalation of indoor dust and outdoor vapour for the POS_{resi} land-use; and,
- Outdoor inhalation of vapours for the POS_{park} land-use.

[5] Note that whilst ingestion pathways are shown to be a relatively large contributor to overall exposure for Residential, Commercial and POS_{park} land-uses, they are relatively unimportant contributors to overall risk due to the relatively high oral LLTC relative to the inhalation LLTC.

4.2 OTHER CONSIDERATIONS

Other considerations that are relevant when setting the C4SLs for naphthalene include the following:

- Intake of naphthalene from non-soil sources (food, water and air) has been considered following completion of a literature review as follows:
    - According to the 2003 CLEA TOX report for naphthalene (Defra and Environment Agency, 2003) exposure to naphthalene via drinking water is assumed to be negligible. Based on exposure via food, an MDI for oral exposure of 7 µg day^{-1} was derived. For a 70 kg adult this equates to an average daily intake of 0.1 µg kg^{-1} bw day^{-1}, which is approximately 0.1% of the oral LLTC.
    - The MDI for inhalation exposure is based on the mean concentration of naphthalene from personal exposure measurements for non-smokers from three UK regions (urban, suburban and rural) reported by Saborit *et al.* (2009). The mean value of 0.7 µg m^{-3} equates to an average daily intake of 0.2 µg kg^{-1} bw day^{-1} for an average adult of 70 kg breathing 20 m^3 per day which is 22% of the inhalation LLTC.
- C4SLs have been derived on the basis of chronic exposure and risks to human health, and do not explicitly account for acute risks (e.g. due to one-off ingestion of a significant amount of soil by a young child). It is noted here that the C4SLs derived for POS_{resi} and POS_{park} are significantly higher than values for the Residential land-use, where inhalation of vapour (indoor) is the principal risk driving pathway in deriving the C4SL. Therefore, further consideration of the possibility of acute risk due to ingestion of soil at the naphthalene concentrations equal to the POS_{resi} and POS_{park} C4SLs may be necessary.
- The British Geological Survey has not derived normal background concentrations for naphthalene in soil (Defra, 2012). Various studies have reported background concentrations of naphthalene (and other PAHs) in soil in the UK. Heywood *et al.* (2006) summarised the findings of the Countryside Survey Project, which measured various PAH compounds in soils at 109 separate locations across the UK. The median concentration of naphthalene was reported to be 15.6 µg kg^{-1} (DW). Morillo *et al.* (2007) compared the PAH concentrations in urban soils from three European cities including Glasgow. The median concentration of naphthalene based on samples of surface soil (0-10 cm) collected from 20 locations across Glasgow was reported to be 68 µg kg^{-1} (DW). Jones *et al.* (1989) sampled rural and urban soils in Wales and analysed them for selected PAHs. They reported a median soil concentration of naphthalene for rural soils of 6 µg kg^{-1} (DW) and a mean concentration for urban soils of 31 µg kg^{-1} (DW). These concentrations are several orders of magnitude below the C4SLs for naphthalene.
- Tables 4.2 and 4.3 show that when the inhalation of vapour (indoor) exposure pathway is active (for both Residential and the Commercial land-use scenarios) it is the principal risk driving pathway. In applying the C4SL the risk assessor should consider that generic modelling of this pathway is based on general assumptions and published data regarding vapour partitioning of naphthalene and subsequent transport. Where exposure to soil vapour forms the principal risk driving pathway then further consideration should be given to supporting the assessment. For example, through obtaining site-specific empirical data for soil vapour concentrations. The reader is referred to CIRIA (2009) and SoBRA (2018) for further guidance on this.
- Naphthalene has an odour threshold in air of 440 µg m^{-3} (ATSDR, 2005). The inhalation LLTC for naphthalene of 0.891 µg kg^{-1} bw day^{-1} equates to a long-term average air concentration of approximately 3 µg m^{-3} for a 70kg adult breathing 20 m^3 of air per day, thus health effects could potentially occur below the odour threshold.

- The lowest derived C4SL in Table 4.1 of 15 mg kg^{-1} is above the range of typical laboratory limits of detection for naphthalene in soil (typically 0.1 mg kg^{-1} or lower).
- It should also be noted that the C4SLs for Commercial and POS land-uses exceed the CLEA calculated soil saturation concentrations of naphthalene which are 76 mg kg^{-1} for 1% SOM, 183 mg kg^{-1} for 2.5% SOM and 432 mg kg^{-1} for 6% SOM. The soil saturation concentration is the theoretical concentration in soil above which free phase contamination may be present. The assessor should be aware that the C4SLs may not be sufficiently precautionary where free phase is present and as such, where free phase is suspected, should consider the risks from this (such as direct contact and vapour inhalation) separately.

5. REFERENCES

Abdo, K.M., Grumbein, S., Chou, B.J. and Herbert, R., 2001. Toxicity and carcinogenicity study in F344 rats following 2 years of whole-body exposure to naphthalene vapors. Inhal Toxicol. 13:931-950.

Agency for Toxic Substances and Disease Registry (ATSDR), 2005. Toxicological profile for naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene. U.S. Department of Health and Human Services. Public Health Service. August 2005.

CIRIA, 2009. The VOCs Handbook. Investigating, assessing and managing risks from inhalation of VOCs at land affected by contamination. CIRIA report C682.

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

Defra and Environment Agency, 2003. Contaminants in Soil: Collation of Toxicological Data and Intake Values for Humans. Naphthalene. R & D Publication - Tox 20. December, 2003.

Defra, 2012. Normal background concentrations (NBCs) of contaminants in English soils: Final project report. Commissioned Report - CR/12/035N. NERC, 2012.

Dodd, D.E., Wong, B.A., Gross, E.A. and Miller, R.A., 2012. Nasal epithelial lesions in F344 rats following a 90-day inhalation exposure to naphthalene, Inhal Toxicol. 24:1, 70-79, DOI: 10.3109/08958378.2011.636086.

Drinking Water Inspectorate (DWI), 2023. Drinking Water 2022 Public supplies England. Indicative raw water hazard sampling data. Accessed from https://www.dwi.gov.uk/what-we-do/annual-report/drinking-water-2022/.

Environment Agency, 2008. Complication of data for priority organic pollutants for derivation of Soil Guideline Values – SC050021/SR7. Environment Agency, Bristol, UK.

Environment Agency, 2009a. Human health toxicological assessment of contaminants in soil. Science Report – SC050021/SR2. Environment Agency, Bristol, UK.

Environment Agency, 2009b. Updated technical background to the CLEA model. Science Report – SC050021/SR3. Environment Agency, Bristol, UK.

European Chemicals Agency (ECHA), 2012. Guidance on information requirements and chemical safety assessment. Chapter R.8: Characterisation of dose [concentration]-response for human health. Reference: ECHA-2010-G-19-EN. November 2012.

European Food Safety Authority (EFSA), 2008. Scientific Opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on Polycyclic Aromatic Hydrocarbons in Food. The EFSA Journal (2008) 724, 1-114.

Fernandes, A., Holland, J., Miller, M., Petch, R., Carlisle, S., Stewart, J. and Rose, M., 2011. Survey for polycyclic aromatic hydrocarbons (PAHs) in cereals, cereal products, vegetables, vegetable products and traditionally-smoked foods. Report Number: FD 10/04.

Health Canada, 2008. Screening Assessment for Naphthalene (91-20-3). July, 2008.

Heywood, E., Wright, J., Wienburg, C.L., Black, H.I.J., Long, S.M., Osborn, D. and Spurgeon, D.J., 2006. Factors influencing the national distribution of polycyclic aromatic hydrocarbons and polychlorinated biphenyls in British soils. Environ. Sci. Techn. 40, 7629-7635.

Howard, P.H., 1991. Handbook of environmental degradation rates. Lewis Publishers.

Jones, K.C., Stratford, J.A., Waterhouse, K.S. and Vogt, N.B., 1989. Organic contaminants in Welsh soils: poly-nuclear aromatic hydrocarbons. Environ. Sci. Techn. 23, 540-550.

Morillo, E., Romero, A.S., Maqueda, C., Madrid, L., Ajmone-Marsan, F., Grcman, H., Davidson, C.M., Hursthouse, A.S. and Villaverde, J., 2007. Soil pollution by PAHs in urban

soils: a comparison of three European cities. Journal of Environmental Monitoring, 9, 1001-1008.

National Toxicology Program (NTP), 1980. Battelle's Columbus Laboratories, Columbus, OH. Subchronic Toxicity Study: Naphthalene (C52904), Fischer 344 Rats, Report to U.S. Department of Health and Human Services.

NTP, 1992. NTP technical report on the Toxicology and Carcinogenesis Studies of Naphthalene (CAS no. 91-20-3) in B6C3F mice (inhalation studies). U.S. Department of Health and Human Services. Public Health Service. National Toxicology Program. April, 1992.

NTP, 2000. NTP technical report on the Toxicology and Carcinogenesis Studies of Naphthalene (CAS no. 91-20-3) in F344/N rats (inhalation studies). U.S. Department of Health and Human Services. Public Health Service. National Toxicology Program. December, 2000.

RIVM, 2007. Health-based guideline values for the indoor environment. RIVM report 609021044/2007. Dusseldorp, A., van Bruggen, M., Douwes, J., Janssen, P.J.C.M., Kelfkens, G.

Saborit, J.M.D, Aquilina, N.J., Meddings, C., Baker, S., Vardoulakis, S. and Harrison, R.M., 2009. Measurement of personal exposure to volatile organic compounds and particle associated PAH in three UK regions. Environ. Sci. Technol. 2009, 43, 4582-4588.

Shopp, G.M., White, K.L. Jr, Holsapple, M.P., Barnes, D.W., Duke, S.S., Anderson, A.C., Condie, L.W. Jr, Hayes, J.R. and Borzelleca, J.F., 1984. Naphthalene toxicity in CD-1 mice: General toxicology and immunotoxicology. Fundam Appl Toxicol. 4:406-419.

United States Environmental Protection Agency (US EPA), 1998. Toxicological Review of Naphthalene (CAS No. 91-20-3). In Support of Summary Information on the Integrated Risk Information System (IRIS). August, 1998.

Society of Brownfield Risk Assessment (SoBRA), 2018. Vapour Intrusion to Support Sustainable Risk Based Decision Making. Summer Workshop 2017.

World Health Organization (WHO), 2010. WHO guidelines for indoor air quality: selected pollutants. ISBN 978 92 890 0213 4. 2010.

APPENDIX A
HUMAN TOXICOLOGICAL DATA SHEET FOR NAPHTHALENE

Human Toxicological Data Sheet for C4SL derivation: Reference checklist for sources of authoritative information

Chemical: **Naphthalene**

Human Health Hazard Profile - References

Authoritative bodies	Website	Checked (Y/N)	References
Environment Agency	http://www.environment-agency.gov.uk/	Y	Defra/Environment Agency, 2003,TOX20: Contaminants in Soil: Collation of Toxicological Data and Intake Values for Humans. Naphthalene http://webarchive.nationalarchives.gov.uk/20140328111046/http://www.environment-agency.gov.uk/research/planning/64002.aspx
Foods Standards Agency	http://www.food.gov.uk/	Y	No relevant information found
Public Health England	https://www.gov.uk/government/organisations/public-health-england	Y	Chemical Hazards Compendium https://www.gov.uk/government/collections/chemical-hazards-compendium#chemicals-m-to-o
Committee on Carcinogenicity	http://www.iacoc.org.uk/	Y	No relevant information found
Committee on Mutagenicity	http://www.iacom.org.uk/	Y	No relevant information found
Committee on Toxicity	http://cot.food.gov.uk/	Y	No relevant information found
ECHA REACH - is there a dossier?	http://echa.europa.eu/information-on-chemicals	Y	REACH dossier submitted and included in CLP Regulation Harmonised Classification and Labelling Inventory https://echa.europa.eu/substance-information/-/substanceinfo/100.127.910
EFSA - is there an opinion?	http://www.efsa.europa.eu/	Y	No relevant information found. EC Scientific Committee on Toxicity, Ecotoxicity and the Environment Opinion, 2002 http://ec.europa.eu/health/ph_risk/committees/sct/documents/out145_en.pdf
JECFA	http://www.fao.org/food/food-safety-quality/scientific-advice/jecfa/en/?	Y	No relevant information found
WHO	http://www.who.int/en/	Y	WHO Guidelines for Indoor Air Quality: Selected Pollutants, 2010 https://www.ncbi.nlm.nih.gov/books/NBK138704/
WHO IPCS	http://www.who.int/ipcs/en/	Y	Poisons Information Monograph 363 http://www.inchem.org/documents/pims/chemical/pim363.htm
WHO EHC	http://www.who.int/ipcs/publications/ehc/en/	Y	EHC 202 http://www.inchem.org/documents/ehc/ehc/ehc202.htm
RIVM	http://www.rivm.nl/English	Y	https://www.rivm.nl/dsresource?objectid=37c132ff-274e-4d21-b969-2090d2dab1df&type=org&disposition=inline
US ATDSR	http://www.atsdr.cdc.gov/	Y	Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene, 2005 https://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=240&tid=43
US EPA	http://www.epa.gov/	Y	IRIS toxicological review, 1998 https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=436
US National Toxicology Program	http://ntp.niehs.nih.gov/	Y	NTP 14th edition report on carcinogens (https://ntp.niehs.nih.gov/ntp/roc/content/profiles/naphthalene.pdf) and Technical Report on the Toxicology and Carcinogenesis Studies of Naphthalene, 2000 (https://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr500.pdf)
Health Canada	http://www.hc-sc.gc.ca/index-eng.php	Y	https://www.canada.ca/en/health-canada/services/publications/healthy-living/residential-indoor-air-quality-guideline-naphthalene.html
Australia NICNAS	http://www.nicnas.gov.au/	Y	Human Health Tier II assessment, https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessment-details?assessment_id=1701#cas-A_91-20-3
Risk Assessment Information System	http://rais.ornl.gov	Y	Toxicity profile, 1993 https://rais.ornl.gov/tox/profiles/naphthalene_c_V1.html
Other scientific reviews	**Check for key reviews on pubmed**	Y	
Other key sources:			
IPCS INCHEM OECD	http://www.inchem.org/	Y	PIM 363 and monograph 82 http://inchemsearch.ccohs.ca/inchem/jsp/search/search.jsp?inchemcasreg=1&Coll=inchemall&serverSpec=charlie.ccohs.ca%3A9900&QueryText1=&QueryText2=naphthalene&Search.x=48&Search.y=12
IARC	http://monographs.iarc.fr/	Y	https://monographs.iarc.fr/ENG/Monographs/vol82/mono82.pdf
EC SCOEL	http://ec.europa.eu/social/main.jsp?catId=148&intPageId=684&langId=en	Y	Recommendation from the Scientific Committee on Occupational Exposure Limits for naphthalene SCOEL/SUM/90 March 2010 (http://ec.europa.eu/social/search.jsp?advSearchKey=naphthalene&mode=advancedSubmit&langId=en&x=9&y=9)
Health Council of the Netherlands	https://www.gezondheidsraad.nl/en/home	Y	Naphthalene; Evaluation of the carcinogenicity and genotoxicity, 2012 (https://www.gezondheidsraad.nl/en/task-and-procedure/areas-of-activity/healthy-working-conditions/naphthalene-evaluation-of-the)

NB. These weblinks were checked on 14 March 2018, and may be subject to change at source.

Human Toxicological Data Sheet for C4SL derivation: Toxicological Evidence, HBGVs, MDIs and LLTC derivation

Chemical: **Naphthalene**

I) Human Health Hazard Profile - Toxicological Evidence

Most sensitive health effects:

Sensitive endpoints	Other information	Source of evidence
Carcinogenicity	*non-neoplastic diffuse epithelial hyperplasia*	*NTP 2008*
Haematotoxicity	Haemolytic anaemia	Human
Other	Cataracts	Human
Other	Cytotoxicity, hyperplasia and metaplasia in respiratory and olfactory epithelium ; kidney lesions	NTP 1992,2000 and Abdo 2001

II) Health Based Guidance Values (HBGVs) from Authoritative Bodies (in descending order of magnitude)

A) Oral route

Authoritative body (date) and HBGV type	HBGV value	Unit	UF used	PoD	POD value	Unit	Endpoint	Pivotal data used & Comments
EXAMPLE: Draft USEPA 2010 RfD	*0.9*	*µg/kg bw/day*	*100*	*BMDL10*	*0.09*	*mg/kg bw/day*	*Epithelial hyperplasia*	*Based on epithelial hyperplasia in female mice (NTP 2008). NTP classified focal epithelial hyperplasia as a preneoplastic lesion so diffuse epithelial hyperplasia may also represent a preneoplastic lesion. However, although this lesion may progress to cancer (adenoma), EPA considered this to be a non-cancer endpoint because definitive data on the progression of this lesion does not exist. UF of 100 was applied (10 for inter and intraspecies differences; 1 to account for database deficiencies).*
USEPA (1998) RfD	20	µg/kg bw/day	3000	NOAEL	71	mg/kg bw/day	Decreased mean terminal bodyweights in males	**Pivotal study - Unpublished sub-chronic oral rat study, Battelle Colombus Laboratories (BCL) 1980.** Doses of 0, 25, 50, 100, 200 and 400 mg/kg were administered by gavage to groups of 10 male and 10 female Fischer 344 rats for 5 days a week for 13 weeks. A NOAEL (based on a >10% decrease in mean terminal body weight) of 100mg/kg bw/day was identified and adjusted to 71 mg/kg bw/day for continuous exposure. A total UF of 3000 (10 for interspecies differences, 10 for intraspecies variability, 10 for sub-chronic to chronic extrapolation, and 3 for database deficiencies) was applied to the NOAEL to give the RfD.
USEPA (1998) RfD	None derived			BMDL	93	mg/kg bw/day	BMR = 10% decrease in terminal mean body weight	**Pivotal study - Unpublished sub-chronic oral rat study, Battelle Colombus Laboratories (BCL) 1980**. There is an Appendix to the USEPA IRIS document which summarises the BMD modelling approaches used on the oral toxicity data. Calculations were conducted prior to introduction of US EPA BMDS. While the Appendix concludes (p111) that the "BMD calculated here does appear to be sufficently reliable for use in the derivation of the RfD" EPA chose not the use the modelling in their RfD derivation. No explanation is given in the IRIS document for this decision. BMD modelling used only Polynomial amd Power models and BMDs of 171 and 172 mg/kg bw/day (duration adjusted = 122 and 123 mg/kg bw/day) and BMDLs of 130 and 135 mg/kg bw/day (duration adjusted 93 and 96 mg/kg bw/day) were calculated. There are insufficient data presented in the IRIS Appendix to re-run the calculations through EPA BMDS.
ATSDR (2005) Chronic MRL	None derived							No appropriate data were located by ATSDR to derive a chronic MRL. A potential intermediate-duration MRL of 0.7 mg/kg bw/day (700 µg/kg bw/day) was derived based on the duration-adjusted NOAEL of 71 mg/kg bw/day based on decreased body weight in male and female rats administered naphthalene by gavage for 5 days/week for 13 weeks in the BCL 1980 study. The MRL was calculated by applying a total UF of 100 (10 for interspecies and 10 for intraspecies differences). This intermediate MRL value was close to the acute duration MRL of 0.6 mg/kg bw/day (600 µg/kg bw/day) derived from a 10 day developmental toxicity study in CD rats based on a minimal "less serious" LOAEL of 50 mg/kg bw/day; the intermediate duration MRL was therefore reduced to 0.6 mg/kg bw/day (600 A123) for consistency.

COT/COC Opinion

None

Current UK oral HCV

Authoritative body (date) and HBGV type	HBGV value	Unit	UF used	PoD	POD value	Unit	Endpoint	Pivotal data used & Comments
EA/Defra 2003 TDI	**20**	**µg/kg BW/day**	**3000**	NOAEL	71	mg/kg BW/day	decreased mean terminal bodyweights in males	**Pivotal study - Unpublished sub-chronic oral rat study, Battelle Colombus Laboratories 1980.** Doses of 0, 25, 50, 100, 200 and 400 mg/kgbw/day were administered via gavage to groups of 10 male and 10 female Fischer 344 rats for 5 days a week for 13 weeks. The NOAEL of 100 mg/kg bw/day (based on a >10% decrease in mean terminal body weight) was duration-adjusted to 71 mg/kg bw/day for continuous exposure. There was a low incidence of kidney lesions in males at 200 mg/kg bw/day.

B) Inhalation Route

Authoritative body (date) and HBGV type	Converted HBGVinh	Unit	HBGVinh	Unit	UF used	POD	POD value	Unit	Endpoint	Pivotal Study used & Comments

EXAMPLE: ATSDR 2010 MRL	*1.43*	*ng/kg bw/day*	*5*	*ng/m³*	*100*	*NOAEL*	*0.5*	*µg/m³*	*Nasal toxicity*	For chromium aerosols and mists. Based on occupational data from workers exposed to chromic acid (Lindberg & Hedenstierna 1983). LOAEL of 2 µg m-3 adjusted for continuous exposure (0.5 µg m-3), and UF of 10 used for interspecies variation and 10 for extrapolating from a LOAEL.
ATSDR (2005) chronic MRL	0.86	µg/kg bw/day	3	µg/m³	300	LOAELhec	0.2 (duration adjusted)	ppm	respiratory and nasal epithelium effects	**Pivotal Study - Chronic 105 week rat study Abdo et al 2001.** There are two chronic studies that were considered by ATSDR a) NTP 1992 104 week study in mice, and b) NTP 2000 105 week study in F344 rats. This second study was also reported in Abdo et al 2001. a) NTP 1992 study - B6C3F1 mice of each sex were exposed by inhalation at concentrations of 0, 10, or 30 ppm, for 6 hours/day, 5 days/week for 104 weeks. b) NTP 2000/Abdo 2001 - 49 male and 49 female F344/N rats were exposed to naphthalene at concentrations of 0, 10, 30 or 60 ppm, for 6 hours/day, 5 days/week for 105 weeks. Both studies identified a LOAEC of 10 ppm in both sexes for non-neoplastic lesions in nasal epithelium and respiratory epithelium. The ATSDR HEC calculation considers naphthalene as a Category 1 gas (i.e. directly acting on respiratory epithelium) and produces HECs of 0.2 ppm for rat and 0.3 ppm for mouse (both duration adjusted values). The lower HEC of 0.2 ppm (duration adjusted) in the rat is equivalent to 1 mg/m³ (duration adjusted - assuming a conversion factor of 5) and this was was chosen as the PoD for the chronic MRL derivation. As a HEC is chosen as the PoD, a total UF of 300 (3-for interspecies-differences, 10 for human variability and 10 for use of a LOAEC rather than a NOAEC) was applied to the HEC to produce a Chronic MRL of 0.0007ppm calculated by ATSDR to be 3.0 (rounded down from 3.3 µg/m³). This is converted to 0.86 µg/kg bw/day assuming a 70 kg adult inhales 20m³ of air per day. Note that the margin between the LOAEC (9.3 mg/m³ duration adjusted) and the HBGV is around 3000 which is a similar margin to the total UF of 3000 used by USEPA 1998 in their RfC derivation, despite different methodologies being used to derive the resulting HBGV values.
USEPA (1998) RfC	0.86	µg/kg bw/day	3	µg/m³	3000	LOAELhec	9.3	mg/m³	respiratory and nasal epithelium effects	**Pivotal study - Chronic 104 week mouse study NTP 1992**. Concentrations of 0, 10 and 30 ppm (converted for continous exposure to 0, 9.3, 28 mg/m³) were administered to groups of B6C3F1 mice (75/sex/group) for 6 hours per day, 5 days per week for 104 weeks. A LOAEC of 9.3 mg/m³ (duration adjusted) was identified. For HEC calculation, naphthalene was considered as a USEPA category 3 gas (i.e. capable of extra respiratory effects) and the ratio of blood/gas partition coefficients was used to extrapolate from the animal dose to a human equivalent concentration. In the absence of partition coefficient data, a default value of 1 was used to derive the HEC of 9.3 mg/m³. A total UF of 3000 (10 for interspecies and intraspecies differences, 10 for use of LOAEC and 3 for data base deficiencies) was applied to the HEC to derive the RfC. EPA commented that the data were not sufficient for BMD modelling.
WHO (2010) Indoor AQ Guideline	2.86	µg/kg bw/day	10	µg/m³	1000	LOAEL	10	mg/m³	respiratory and nasal epithelium effects	**Pivotal Study - Chronic 105 week rat study Abdo et al 2001.** **A LOAEL of 10 ppm was identified based on non-neoplastic lesions in nasal epithelium and respiratory epithelium in both sexes. A** chronic inhalation study with 49 male and 49 female F344/N rats exposed 0, 10, 30 or 60 ppm naphthalene, 6 hours/day, 5 days/week for 105 weeks. 10 ppm was converted to "*about 10 mg/m³*". An UF of 1000 (10 for interspecies, 10 for intra-individual variability and 10 for use of LOAEC) was applied to the LOAEC to derive the guideline value . Assuming that a 70kg adult inhales 20m³ of air per day converts the guideline value of 10 µg/m³ to a dose of 2.86 µg/kg bw/d.
Health Canada (2008) Residential Maximum Exposure Limit	2.86	µg/kg bw/day	10	µg/m³	1000	LOAEL	10	mg/m³	respiratory and nasal epithelium effects	**Pivotal Study - Chronic 105 week study in rats Abdo et al 2001.** Based on a chronic inhalation study with 49 male and 49 female F344/N rats exposed to 0, 10, 30 or 60 ppm naphthalene , 6 hours/day, 5 days/week for 105 weeks, a LOAEC of 10 ppm was identified based on nonneoplastic lesions in nasal epithelium and respiratory epithelium in both sexes. The LOAEC of 10 ppm duration adjusted to a value 1.8ppm, and then converted to a mass concentration in air of 10 mg/m³. Note: other conversions from 10 ppm have produced a value of 9.3 mg/m³ as a duration-adjusted, mass in air concentration. An UF of 1000 (10 for interspecies, 10 for intra-individual variability and 10 for use of LOAEC) was applied to the LOAEC to derive the guideline value . Assuming that a 70kg adult inhales 20m³ of air per day converts the guideline value of 10 ug/m³ to a dose of 2.86 ug/kg bw/d.
RIVM (2012) indoor air guideline value	7.14	µg/kg bw/day	25	µg/m³	200	LOAEL	5	mg/m³	Local damage to the nasal epithelium	**Pivotal study - 4 weeek study in rats HRC 1993 reported in the EU RAR which is referenced by RIVM (although full details of the study are not reproduced by RIVM).** Groups of 5 male and 5 female rats were exposed to naphthalene at concentrations of 0, 1, 3, 10, 29 and 71 ppm for 6 hours per day, 5 days a week for 4 weeks. Signs of proliferative repair in the nasal olfactory epithelium were observed at all doses. RIVM considered that had the study been extended to 2 years, the mild nasal effects observed at 4 weeks would have progressed to more severe effects. Therefore a LOAEC of 5 mg/m³ (converted from 1 ppm) was adopted for repeated exposure inhalation toxicity including carcinogenicity. RIVM also concluded that the LOAEC of 5mg/m³ marks the starting point of the dose-response curve leading ultimately to more serious effects on the respiratory and olfactory epithelium. Effects at the LOAEC were considered to be concentration-dependent rather than dose-dependent, and so adjustment for the duration of exposure was not necessary. Likewise, only a limited extrapolation factor was used when extrapolating from the LOAEC to the NOAEC and a factor 2 was used for this (resulting in an estimated NOAEC of 2.5 mg/m³). Uncertainty factors for species differences (10) and human variability (10) were also applied giving a total UF on the LOAEC = 200, resulting in a guideline value of 25 µg/m³. Assuming that a 70kg adult inhales 20m³ of air per day converts the guideline value of 25 µg/m³ to a dose of 7.14 µg/kg bw/day.

COT/COC Opinion	None

Current UK inhalation HCV

Authoritative body (date) and HBGV type	HBGV value	Unit	UF used	PoD	POD value	Unit	Endpoint	Pivotal data used & Comments
EA/Defra 2003 TDI	**0.86**	µg/kg bw/day	3000	LOEL	9.3	mg/m^3	nasal effects	**Pivotal study - Chronic mouse study (NTP 1992).** Experimental concentrations of 0, 10, and 30 ppm converted for continuous exposure and human equivalent concentrations of 0, 9.3 28 mg/m^3. As the blood/gas partition coefficients for naphthalene were not known, a default value of 1 was used for calculation of HEC from continuous exposure concentrations. Hyperplasia and metaplasia in respiratory and olfactory epithelium was observed at at both doses and 9.3mg/m^3 was identified as the LOAEC. The review noted that data were not sufficient for BMD modelling. A total UF of 3000 was used based on the same uncertainty factors as were used for the US EPA RfC derivation. Note: The NTP 2000 / Abdo et al 2001 chronic study in rats was not considered as pivotal study.

C) Dermal Route

Authoratative body (date) and HBGV type	HBGV value	Unit	UF used	POD	POD value	Unit	Endpoint	Pivotal Study used & Comments
None								

III) Current UK (WHO) regulatory values

	Value	Units	Refs
UK drinking water standard	none		
WHO drinking water standard	none		
UK air quality standard	none		
WHO air quality standard	10	$µg/m^3$	WHO (2010) Guidelines for Indoor Air Quality, Selected Pollutants, WHO Regional Office for Europe
PHE indoor air quality guidelines	3	ug/m3	PHE (2019) Indoor Air Quality Guidelines for selected Volatile Organic Compounds (VOCs) in the UK

IV) Mean Daily Intakes from Other Sources (e.g. Diet)

	Pathways	Units	Adults	Children	Refs
Food (average)	Oral				
Food (average)	Oral				
Water	Oral				
Air	Inhalation				
Smoking	Inhalation				

V) LLTC derivation

A) ORAL

Choice of Pivotal Data	Dosing vehicle	Doses	Units	Species	Study Type	Comments
NTP 2008	*Drinking water*	*0.38, 0.91, 2.4 or 5.9 (m/m); 0.38, 1.4, 3.1 or 8.7 (f/m)*	*mg/kg bw/day*	*Mouse*	*2 year drinking water study*	*Endpoints based on non-neoplasic epithelial hyperplasia in female mice via a threshold MOA (BMDL 0.09) or oral carcinoma in male mice mg kg (BMDL 1.2) (IPCS 2011) .*

Unpublished study Battelle Colombus Laboratories 1980	**Gavage**	**0, 25, 50, 100, 200, 400**	**mg/kg bw/day**	**Rat**	**13 wk sub-chronic gavage study**	The ATSDR review of the 13 week BCL 1980 study in F344 rats identified a NOAEL of 100 mg/kg bw/day (71 mg/kg bw/day duration-adjusted) based on a less than 10% decrease in terminal mean bodyweights and absence of other signs of toxicity. A "less serious" LOAEL of 200 mg/kg bw/day (142 mg/kg bw/day duration-adjusted) was identified by ATSDR being based on a 12% depression of mean terminal body weight in males but not females (6% depression) and mild kidney lesions in 2/10 male rats (focal cortical lymphocytic infiltration or renal tubular regeneration) (nb only minimal renal lesions occurred in 1/10 male rats -diffuse renal tubular degradation- at 400 mg/kg bw/day.) Consideration was given to selecting the ATSDR "less serious" LOAEL of 200 mg/kg bw/day (142 mg/kg bw/day duration-adjusted) as a PoD for the oral LLTC derivation. However a review of other repeated dose oral toxicity studies indicated that there were also some effects evident at 200 mg/kg bw/day and below, and therefore the **NOAEL of 71 mg/kg bw/day (duration-adjusted) was selected as the PoD.** A total UF of 900 is proposed (3 for use of subchronic study, 10 for interspecies differences, 10 for intraspecies differences and 3 for database deficiencies) to be applied to the NOAEL of 71 mg/kg bw/day to derive the LLTC of 78.9 µg/kg bw/day. A factor of 3 was chosen for the adjustment between subchronic and chronic exposure on the basis that this is consistent with the UFs selected to adjust between subchronic and chronic exposure for other C4SL substances .

Selection of POD

Published POD for ORAL LLTC:		
Are dose response data of adequate quality to derive a BMD	No	
Type of PoD	NOAEL	
Value selected	71	mg/kg bw/day (duration adjusted)

Derived POD for ORAL LLTC: (from data below)		
Type of PoD	BMD	
Value derived		
AIC value		
P value		

Comment - USEPA did not regard the modelling as sufficently robust to be used as basis of RfD. BMR = 10% body weight decrase. BMD of 93 mg/kg/d (duration adjusted) was numerically higher than NOAEL of 71 mg/kg/d (duration adjusted). Many different BMDs were calculated, some much lower than NOAEL. No BMDL was calculated - only an upper bound BMD

BMD Modelling (if answered 'Yes' to question above - see worksheet BMD modelling pivotal study)

Software used **US EPA BMDS Version [to be specified]**

	BMD1	BMD5	BMD10	BMD15
BMD modelling (value) (mg/kg bw/day)				
	BMDL1	**BMDL5**	**BMDL10**	**BMDL15**
BMD modelling (value) (mg/kg bw/day)				

Present benchmark dose graph here

Comments:

Addressing uncertainty

Thresholded effects?	Yes
If yes - use generic UF of 100 or (if data allow) calculate CSAF	1000
If no : see below for non-thresholded effects	
If animal data are used as POD (NO(A)EL or BDM) use generic margin of 5000 or (if data allows) calculate CSM	5000
If human data are used to derive a BMD use the margin that relates to a notional risk of 1 in 50000 based on the BMR (using the table opposite). The same margin can also be applied to a NO(A)EL, but not to a LO(A)EL.	
ELCR =	1 in 50000

BMR	**Margin**	**Corresponding ELCR estimate**
0.50%	250	1 in 50000
1%	500	1 in 50000
5%	2000	1 in 50000
10%	5000	1 in 50000

Chemical Specific Adjustment Factor/Chemical Specific Margin to account for uncertainties in the data

	Range	**Selected value**
Intraspecies	1 - 10	10
Interspecies	1 - 10	10
Sub-chronic to chronic	1-10	3.16

Oral LLTC calculation:

		Units	
LLTC (Thresholded chemical) using NOAEL/LOAEL		71.0	µg/kg bw/day
LLTC (Thresholded chemical) using BMD			µg/kg bw/day

Database deficiencies	1-3	3.16
Quality of study	1 - 10	1
Use of LOAEL as POD	1-10	1
Total UF/CSAF/CSM		1000

Lifetime averaging to be applied in CLEA (Yes/No)	No

LLTC (Non Thresholded chemical) using NOAEL/LOAEL		µg/kg bw/day
LLTC (Non Thresholded chemical) using BMD		µg/kg bw/day

Delete as appropriate

Sensitive Receptor	

b) INHALATION

Choice of Pivotal Data	Dosing vehicle	Doses	Units	Species	Study Type	Comments
Epidemiology study of lung cancer in workers in a chromate production (Gibb et al 2000)	*N/A*	*N/A*	*N/A*	*Human*	*Epidemiology study in chromate production workers*	*The ELCR for for lung cancer for 1, 0.1, 0.01 or 0.001 µg m-3 is equivalent to environmental exposure of 4 in 100, 4 in 1000, 4 in 10,000, or 4 in 100,000. Hence 1 in 100,000 would equate to 0.00025 mg m-3 (0.25 ng m-3).*
Sub-chronic study in rats (Dodd et al 2012)	**N/A**	**0, 0.1, 1, 10, 30**	**ppm**	**Rat**	**13 week inhalation study in male and female F334 rats**	F344 rats (10 per sex per group) were exposed in whole body chambers to naphthalene vapour for 6 h/d, 5 d/week for 13 weeks at concentrations of 0, 0.1, 1, 10 or 30 ppm (Dodd et al 2012). The heads were cross-sectioned at six levels for microscopic examination. Mild decreases in body weight (<10%) and food/water consumption were observed primarily in the rats exposed to 30 ppm. There were no naphthalene exposure-related clinical observations at any dose level. Histopathologically, there were no nasal cavity or respiratatory lesions related to naphthalene exposure in rats in the 0.1 ppm group ; The nasal respiratory epithelial and olfactory epithelial effects at 1ppm over 90 days were described by the authors as minimal. Group histopathology scores of 1 or less (1= "minimal" with 5 = severe/high) were assigned for a number of histopathological lesions at the 6 different nasal cross-sections ; these were generally observed in only a few animals of the group. The most consistent finding was nasal transitional/respiratory epithelial hyperplasia in 10/10 males (score = 1 minimal) (female data are not presented but were said to be similar). At 10ppm these hyperplasia lesions were scored 1.4 and 1.6 (males and females respectively) and so the effects at this higher dose level were more than "minimal" but not reaching the criterion of "slight/mild". Mild hyperplasia and minimal squamous metaplasia were observed in the respiratory epithelium of rats exposed at 10 or 30 ppm Systemically, only minimal effects were noted in this study. Statistically significant decreases in relative (though NOT absolute) organ weights were noted for spleen, testis, heart and thymus at 10ppm and above, but without gross observations at necropsy (no histopathology was performed). However, absolute liver and heart weights were significantly reduced in 0.1 ppm females, and also significantly reduced at some, but not all, of higher exposure levels (nb male data not provided in the paper). However, all systemic effects diminished in magnitude and were not statistically significant after a 4 week recovery period The inhalation toxicity of naphthalene displays a spectrum of dose-related epithelial effects ranging from inflammation, hyperplasia, metaplasia, and ultimately to tumour formation, most probably via a non genotoxic mechanism. If the effects at 1ppm are considered to be minimal, reversible and mark the start of the continuum of naphthalene this can be set as the PoD for LLTC derivation. 1ppm is equivalent to 0.936 mg/m3 (duration adjusted, and converted to mass in air: 1ppm x 5.24 mg/m3 per ppm x 6hrs/24hrs x 5days/7days). An uncertainty factor of 3 has been selected for a short duration study on the basis that this is consistent with the UFs selected to adjust between subchronic and chronic exposure for other C4SL substances . Standard uncertainty factors of 10 for interspecies differences and 10 for human variability have been selected, giving a total UF of 300. This produces a HBGV of 3.12 µg/m3. Assuming a body weight of 70kg and 20m3 of air inhaled per day gives a LLTC of 0.891 µg/kg bw/d (duration adjusted)

Selection of POD

Published POD for INHALATION LLTC:		
Are dose response data of adequate quality to derive a BMD	No	
Type of PoD	LOAEL	
Value selected	0.936	mg/m3

Derived POD for INHALATION LLTC: (from data below)		
Type of PoD	BMDL	
Value derived		mg/kg bw/day
AIC value		
P value		

BMD Modelling (if answered 'Yes' to question above - see worksheet BMD modelleing pivotal study)

Software used **US EPA BMDS 2.3.1**

	BMD1	BMD5	BMD10	BMD15
BMD modelling (value) (mg/kg bw/day)				
	BMDL1	BMDL5	BMDL10	BMDL15
BMD modelling (value) (mg/kg bw/day)				

Comments: BMD modelling

Present benchmark dose graph here

Thresholded effects?	Yes
If yes - use generic UF of 100 or (if data allow) calculate CSAF	100
If no : see below for non-thresholded effects	
If animal data are used as POD (NO(A)EL or BDM) use generic margin of 5000 or (if data allows) calculate CSM	5000
If human data are used to derive a BMD use the margin that relates to a notional risk of 1 in 50000 based on the BMR (using the table opposite). The same margin can also be applied to a NO(A)EL, but not to a LO(A)EL.	
ELCR =	1 in 50000

BMR	Margin	Corresopnding ELCR estimate
0.50%	250	1 in 50000
1%	500	1 in 50000
5%	2000	1 in 50000
10%	5000	1 in 50000

Chemical Specific Adjustment Factor/Chemical Specific Margin to account for uncertainties in the data		
	Range	**Selected value**
Intraspecies	1 - 10	10
Interspecies	1 - 10	10
Sub-chronic to chronic	1-10	3
Database deficiencies	1-3	1
Quality of study	1 - 10	1
Use of LOAEL as POD	1-10	1
Total CSAF/CSM		300

Lifetime averaging to be applied in CLEA (Yes/No)	No

Inhalation LLTC calculation:

			Units
LLTC (Thresholded chemical) using NOAEL/LOAEL		0.891	µg/kg bw/day
LLTC (Thresholded chemical) using BMD			µg/kg bw/day
LLTC (Non Thresholded chemical) using NOAEL/LOAEL			µg/kg bw/day
LLTC (Non Thresholded chemical) using BMD			µg/kg bw/day

Delete as appropriate

Sensitive Receptor	

ORAL LLTC - ATSDR has identified a NOAEL of 100 mg/kg bw/day, a “less serious” LOAEL of 200 mg/kg bw/day and a "serious" LOAEL of 400 mg/kg bw/day) from a 13 week study in F344 rats. The "less serious" LOAEL was based on body weight (12% decrease in males , 6% decrease in females) and mild kidney lesions, which were not replicated at the higher dose. The LOAEL was considered as a possible PoD for oral LLTC as effects could be considered as being of a "low level of toxicological concern" in relation to "serious harm" for the purposes of Part 2A. However, a review of other repeated dose studies was conducted and this indicated that in rats and other species there were some mild and "less serious" effects at below 200 mg/kg bw/day and the LOAEL of 200 mg/kg bw /d was not chosen as PoD. Instead the NOAEL of 100 mg/kg bw/day (71 mg/kg bw/day duration-adjusted) was selected as the PoD. A total of UF of 1000 was used (10 for interspecies, 10 for intraspecies differences, √10 (3.16) for use of sub chronic study and √10 (3.16) for data deficiencies) to generate the oral LLTC HBGV of 71.0 µg/kg/bw/d.

INHALATION LLTC - There are 3 main authoritative reviews (ATSDR, EPA and WHO), all based on the same PoD, i.e. the LOAEC from a 2 year rat study (no NOAEC was identified), but each using different approaches and UFs to generate 3 different HBGVs. The WHO Indoor AQ guideline 2010 (numerically the highest) is the most recent. The WHO does not make assumptions to calculate a HEC (as USEPA and ATSDR do) but derives the Guideline Value directly from an external exposure concentration.
There is ongoing EPA interest in naphthalene as the 2 year inhalation toxicity study did not identify a NOAEC, as the lowest dose (10 ppm) produced serious effects in virtually all animals. Research to investigate dose response below 10 ppm is on-going and the 13-week study of Dodd et al. (2012), identifies 0.1 ppm as a no effect level with possibly a “less serious” effects at 1ppm and more "serious" effects at 10 ppm. It has been agreed with the Steering Group that, from the Dodd et al. study, 1ppm can be considered as a PoD for LLTC derivation along with standard uncertainty factor of 3 for a short duration study and standard uncertainty factors for interspecies differences and human variability (total 300). It has been agreed with the Steering group that an additional uncertainty factor is not needed to be applied to the PoD of 1ppm. It should be noted that Dodd et al. do not specifically identify "NOAEC" or "LOAEC" in their paper and so the terms "NOAEC" or "LOAEC" do not feature in the summary of that study. To keep in line with the approach identified in the Framework, the effects at 1ppm are minimal and inconsistent (authors' description) this dose could be considered as a NOAEC - and hence this is selected as the PoD - the lower dose of 0.1ppm could be considered as a NOAEC. More consistent effects were seen at 10ppm (still minimal effects but now at a higher grade) and 10ppm could be considered to be the LOAEC. Taking 1ppm (equivalent to 0.936 mg/m3 (duration adjusted, and converted to mass in air: 1ppm x 5.24 mg/m3 per ppm x 6hrs/24hrs x 5days/7days) as the POD and applying a total UF of 300 results in an HBGV of 3.12 µg/m3. Assuming a body weight of 70kg and 20m3 of air inhaled per day gives a LLTC of 0.891 µg/kg bw/d .This value is more than three times lower than the WHO AQ Guideline Value (2.86 µg/kg bw /d), and almost identical to the ATSDR chronic MRL and USEPA RfC (0.86 ug/kg bw/d).

APPENDIX B
MEAN DAILY INTAKE DATA SHEET FOR NAPHTHALENE

Substance: **Naphthalene**

MDI Oral			**Recommended adult oral MDI**	**Units**	**Justification:** The oral MDI of 7 µg/d was calculated by the EA/DEFRA (2003) based on the concentrations reported in the food sources from the various studies reviewed and data on adult food consumption rates (Gregory et al. 1990, The dietary and nutritional Survey of British Adults: a survey of the dietary behaviour, nutritional status and blood pressure of adults aged 16 to 64 living in Great Britain. Office of Population Census and Surveys, Social Survey Division, London: HMSO) adjusted for a 70 kg UK adult. **Intake from naphthalene concentrations in drinking water was not included in the oral MDI calculation as contributions from drinking water considered to be negligible based on available data.**		
			7	ug day-1			
Organisation/Source	**Date**	**Media**	**Value**	**Units**	**Description**	**Reference**	**Web link**
Drinking Water Inspectorate	2017	Tap water	-	µg L-1	99th percentile concentrations measured in 2016 averaged across all 30 water companies in England & Wales.	Data summary tables from Drinking Water Inspectorate annual report drinking water 2016. Naphthalene not included.	http://www.dwi.gov.uk/about/annual-report/2016/index.html
Drinking Water Inspectorate	2023	Raw water	<1	µg L-1	Maximum concentration detected in 1621 samples of raw water from public water supplies in England in 2022	Drinking Water 2022 Public supplies England. Indicative raw water hazard sampling data	https://dwi-content.s3.eu-west-2.amazonaws.com/wp-content/uploads/2023/07/20160815/Raw-water-targeted-sample-data-England-2022.pdf
EU Risk Assessment	2003	Drinking water	0.03	µg L-1	Measurements of naphthalene in uncontaminated groundwater from Zurich and Osaka found up to 0.03 µg L-1.	EC-JRC, 2003. European Union risk assessment report, naphthalene. EUR 20763 EN, 1st Priority List, vol. 33, European Chemicals Bureau, European Commission Joint Research Centre and, Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a
EA / DEFRA	2003	Food (butter and cheese)	**14**	µg kg-1	Survey of samples from urban retail outlets (petrol stations, stalls, shops) next to busy roads (Food Surveillance Sheet No. 98 MAFF-UK 1996).	Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf
EA / DEFRA	2003	Food (lard and margarine)	**16**	µg kg-1	Survey of samples from urban retail outlets (petrol stations, stalls, shops) next to busy roads (Food Surveillance Sheet No. 98 MAFF-UK 1996).	Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a
EU Risk Assessment EA / DEFRA	2003	Food (fish)	**60**	µg kg-1	Average levels of naphthalene in cod and haddock from around three oil platforms (19 samples) and three reference stations (13 samples) in the North Sea were 0.06µg/g (range 0-0.23µg/g) and 0.01µg/g (range 0-0.02µg/g) respectively.	Vogt *et al.* 1988 cited in EC-JRC, 2003. European Union risk assessment report, naphthalene. EUR 20763 EN, 1st Priority List, vol. 33, European Chemicals Bureau, European Commission Joint Research Centre and, Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a
EU Risk Assessment EA / DEFRA	2003	Food (barley - representative of miscellaneous cereal)	**4.3**	µg kg-1	Testing of grains of barley when three different types of fertiliser used (nitrogen fertiliser, pig slurry and sewage sludge).	Kirchmann and Tengsved, 1991 cited in EC-JRC, 2003. European Union risk assessment report, naphthalene. EUR 20763 EN, 1st Priority List, vol. 33, European Chemicals Bureau, European Commission Joint Research Centre and, Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a
EU Risk Assessment EA / DEFRA	2003	Food (uncooked and tinned carrots)	**7.8**	µg kg-1	Testing of uncooked, cooked, tinned and frozen carrots.	Wild and Jones, 1991 cited in EC-JRC, 2003. European Union risk assessment report, naphthalene. EUR 20763 EN, 1st Priority List, vol. 33, European Chemicals Bureau, European Commission Joint Research Centre and, Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a

MDI Inhalation			**Recommended adult inhalation MDI**	**Units**	**Justification:** Preference given to UK data from personal exposure study which excluded smokers and data which was influenced by environmental tobacco smoke. This includes data from urban, suburban and rural areas and accounts for indoor and outdoor exposure. International data influenced by use of pesticides (e.g. mothballs) are not considered representative of UK conditions. Non-environmental tobacco smoke arithmetic mean personal exposure concentration of 0.7 ug m-3 multiplied by an assumed adult respiration rate of 20 m3.d-1 to derive recommended MDI inhalation of 14 ug day-1.		
			14	ug day-1			
Organisation/Source	**Date**	**Media**	**Value**	**Units**	**Description**	**Reference**	**Web link**
Defra's UK Air Information Resource (AIR)	-	Dust in air	-	-	No data available for Naphthalene in PM10. Data only available from dust depositional samplers in rural areas. Cannot convert to air concentrations without windspeed. Data not considered to be applicable.		https://uk-air.defra.gov.uk
EU Risk Assessment EA / DEFRA	2003	Air	0.14	µg m-3	Estimated airborne levels at a regional European level based on predicted air concentrations for a number of sites. Adopted in the EA / DEFRA 2003 TOX 20 report to calculate an adult inhalation MDI.	European Union risk assessment report, naphthalene. EUR 20763 EN, 1st Priority List, vol. 33, European Chemicals Bureau, European Commission Joint Research Centre and, Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a
EU Risk Assessment EA / DEFRA	2003	Air	80	µg m-3	Maximum naphthalene concentration reported in air samples collected from 6 indoor locations.	European Union risk assessment report, naphthalene. EUR 20763 EN, 1st Priority List, vol. 33, European Chemicals Bureau, European Commission Joint Research Centre and, Environment Agency TOX 20, 2003 - Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. R&D Publication TOX 20. (Also retained in Environment Agency TOX 20, 2011 - Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20.)	https://webarchive.nationalarchives.gov.uk/20140328153859/http://www.environment-agency.gov.uk/static/documents/Research/napthal_old_approach_2028758.pdf https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a
US ATSDR	2005	Air	0.94	µg m-3	Concentrations reported in urban/ suburban air from 11 US cities found to range between 0.4-170µg m-3 with a median concentration of 0.94µg m-3.	Howard, 1989 cited in US ATSDR, 2005. Toxicological Profile for Naphthalene, 1-methylnaphthalene and 2-methylnaphthalene. U.S. Department of Health and Human Services, Public Health Service Agency for Toxic Substances and Disease Registry.	https://www.atsdr.cdc.gov/toxprofiles/tp67.pdf

MDI Inhalation			Recommended adult inhalation MDI	Units	Justification: Preference given to UK data from personal exposure study which excluded smokers and data which was influenced by environmental tobacco smoke. This includes data from urban, suburban and rural areas and accounts for indoor and outdoor exposure. International data influenced by use of pesticides (e.g. mothballs) are not considered representative of UK conditions. Non-environmental tobacco smoke arithmetic mean personal exposure concentration of 0.7 ug m-3 multiplied by an assumed adult respiration rate of 20 m3.d-1 to derive recommended MDI inhalation of 14 ug day-1.		
			14	ug day-1			
US ATSDR	2005	Air	170	µg m-3	Average naphthalene concentration reported for outdoor air in a residential area of Columbus, Ohio.	Chuang *et al.* 1991 cited in US ATSDR, 2005. Toxicological Profile for Naphthalene, 1-methylnaphthalene and 2-methylnaphthalene. U.S. Department of Health and Human Services, Public Health Service Agency for Toxic Substances and Disease Registry.	https://www.atsdr.cdc.gov/toxprofiles/tp67.pdf
US ATSDR	2005	Air	4.6	µg m-3	Maximum recorded US average concentration of naphthalene recorded at five hazardous waste/ landfill sites in New Jersey, (range 0.42- 4.6µg m-3).	LaRegina *et al.* 1986 cited in US ATSDR, 2005. Toxicological Profile for Naphthalene, 1-methylnaphthalene and 2-methylnaphthalene. U.S. Department of Health and Human Services, Public Health Service Agency for Toxic Substances and Disease Registry.	https://www.atsdr.cdc.gov/toxprofiles/tp67.pdf
US ATSDR	2005	Air	9.7	µg m-3	Maximum level of naphthalene recorded in indoor air in 24 low income homes in North Carolina US, (range 0.33-9.7 0.42- 4.6µg m-3).	Chuang *et al.* 1991 cited in US ATSDR, 2005. Toxicological Profile for Naphthalene, 1-methylnaphthalene and 2-methylnaphthalene. U.S. Department of Health and Human Services, Public Health Service Agency for	https://www.atsdr.cdc.gov/toxprofiles/tp67.pdf
Environment Canada Health Canada	2008	Air	6.78	µg m-3	A Health Canada survey of homes in Windsor Canada in 2005 and 2006 identified naphthalene concentrations in indoor air up to 158µg m-3, with a mean of 6.78µg m-3 and 90th percentile of 9.41µg m-3. Health Canada concluded that the major source of naphthalene exposure is from indoor air, with up to 99% of the total daily intake across all age groups.	Health Canada, 2008 cited in Environment Canada Health Canada, 2008. Proposed Risk Management Approach for Naphthalene, Government of Canada.	https://www.ec.gc.ca/ese-ees/B68532E6-50EB-4368-92FC-7FA0B36AD64D/batch1_91-20-3_rm_en.pdf
Health Canada	2008	Air	3.87	µg m-3	A Health Canada survey of homes in Ottawa, Canada identified naphthalene concentrations in indoor air up to 144µg m-3, with a mean of 3.87µg m-3 and 90th percentile of 4.75µg m-3.	Zhu *et al.* 2005 cited in Environment Canada Health Canada, 2008. Proposed Risk Management Approach for Naphthalene, Government of Canada.	https://www.ec.gc.ca/ese-ees/B68532E6-50EB-4368-92FC-7FA0B36AD64D/batch1_91-20-3_rm_en.pdf
International Journal of Environmental Research and Public Health	2010	Air	0.8	µg m-3	Study of indoor air quality in 12 homes in an urban area of Birmingham, UK. Measured concentrations of naphthalene reported arithmetic mean 0.8µg m-3, median 0.5µg m-3, maximum 6.0µg m-3, standard deviation 1µg m-3.	JIA, C., BATTERMAN, S., 2010. A critical review of naphthalene sources and exposures relevant to indoor and outdoor air. International Journal of Environmental Research and Public Health, 2010, 7, 2903-2939.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922736/
International Journal of Environmental Research and Public Health	2010	Air	0.44-1.2	µg m-3	Studies of indoor air quality in Europe, 39 to 201 homes or 182 to 2103 measurements (4 studies in urban areas in Germany and 2 in urban areas in Finland). Measured concentrations of naphthalene reported arithmetic mean 0.44µg m-3 to 1.2µg m-3, median 0.3µg m-3 to 0.81µg m-3, maximum 1.63µg m-3 to 40.79µg m-3, standard deviation 0.46µg m-3 to 2.8µg m-3.	JIA, C., BATTERMAN, S., 2010. A critical review of naphthalene sources and exposures relevant to indoor and outdoor air. International Journal of Environmental Research and Public Health, 2010, 7, 2903-2939.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922736/
International Journal of Environmental Research and Public Health	2010	Air	0.27-9.52	µg m-3	Study of indoor air quality in 5 to 754 international homes (Urban areas in China & Australia (one study each) Canada (4 studies). Urban, suburban and rural areas in United States (US) (8 studies)). Measured concentrations of naphthalene reported arithmetic mean 0.27µg m-3 to 9.52µg m-3, median 0.17µg m-3 to 4.59µg m-3, maximum 1.24µg m-3 to 144µg m-3, standard deviation 0.95µg m-3 to 17.25µg m-3.	JIA, C., BATTERMAN, S., 2010. A critical review of naphthalene sources and exposures relevant to indoor and outdoor air. International Journal of Environmental Research and Public Health, 2010, 7, 2903-2939.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922736/
International Journal of Environmental Research and Public Health	2010	Air	0.002-0.3	µg m-3	Two studies conducted in urban areas of Birmingham, outdoor air measured outside 12 homes in one study and 55 samples from 1 site in the other study. Measured concentrations of naphthalene reported arithmetic mean 0.002µg m-3 to 0.3µg m-3, median 0.2µg m-3, maximum 0.9µg m-3, standard deviation 0.2 µg m-3.	JIA, C., BATTERMAN, S., 2010. A critical review of naphthalene sources and exposures relevant to indoor and outdoor air. International Journal of Environmental Research and Public Health, 2010, 7, 2903-2939.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922736/
International Journal of Environmental Research and Public Health	2010	Air	0.1	µg m-3	Studies of outdoor air quality in urban areas of Europe. Outdoor air measured outside 183 homes in Finland and 47 measurements in one German study and 222 measurements in another German study. Measured concentrations of naphthalene reported arithmetic mean 0.1µg m-3, median 0.1 µg m-3, maximum 1.3µg m-3 to 1.5µg m-3, standard deviation not reported.	JIA, C., BATTERMAN, S., 2010. A critical review of naphthalene sources and exposures relevant to indoor and outdoor air. International Journal of Environmental Research and Public Health, 2010, 7, 2903-2939.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922736/
International Journal of Environmental Research and Public Health	2010	Air	<MDL-6.31	µg m-3	Study of outdoor air quality in urban areas in Australia, Korea, China (also included industrial data) and Taiwan (also included rural data) (one study each). India (2 urban area studies across multiple sites). Canada urban areas (2 studies (and rural (1 study). Urban, suburban and/or rural/remote areas in US (10 studies). Outdoor air reportedly measured outside between 1 to 159 homes/(schools/ sites) or with 80 to 11399 measurements taken across the studies. Measured concentrations of naphthalene reported arithmetic mean less than method detection limits (<MDL) to 6.31µg m-3, median <MDL to 4.15µg m-3, maximum 0.076µg m-3, to 19.83µg m-3, standard deviation 0.01µg m-3, to 6.82µg m-3.	JIA, C., BATTERMAN, S., 2010. A critical review of naphthalene sources and exposures relevant to indoor and outdoor air. International Journal of Environmental Research and Public Health, 2010, 7, 2903-2939.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922736/
Environmental Science and technology	2009	Air	**0.7**	µg m-3	UK personal exposure measurements collected from 191 people living in urban settings in London or Birmingham, 209 people living in suburban settings in Birmingham and 100 people living in rural settings in the Midlands or Wales. Measured concentrations of naphthalene reported arithmetic mean 0.78µg m-3, standard deviation 1.49µg m-3, median 0.49µg m-3, maximum 12.67µg m-3 in the urban areas, arithmetic mean 0.72µg m-3, standard deviation 0.75µg m-3, median 0.55µg m-3, maximum 6.35µg m-3 in the suburban areas arithmetic mean 0.71µg m-3, standard deviation 0.54µg m-3, median 0.58µg m-3, maximum 2.84µg m-3 in the rural areas. Arithmetic mean of 0.7µg/m3 reported from all 305 UK personal exposure measurements without environmental tobacco smoke interference in urban, sub-urban and rural settings equalled 0.7µg/m3	MARI DELGADO SABORIT, J, et al. 2009. Measurement of personal exposure to volatile organic compounds and particles associated PAH in three UK regions. Environmental Science & Technology, 2009, 43, 4582-4588.	N/A
EA	2011	Air	1.2	µg m-3	Based on a weighted average of the high end median air concentrations for ambient and indoor air reported in Jia and Batterman 2010 was calculated at 1.2 µg/m3, assuming that typical UK residents spend two-thirds of their time indoors. The weighted average of the high end air concentrations from Jia and Batterman includes data from the UK, Europe and international studies. The US and Canada data appears to be skewed by the use of naphthalene pesticides (e.g. moth balls) which have been banned in the UK and across the EU since 2008.	Environment Agency TOX 20, 2011. Unpublished Contaminants in soil: Collation of toxicological data and intake values for humans. Naphthalene. Science report - SC050021 / TOX 20 and Environment Agency, 2011. Unpublished Soil guideline values for naphthalene in soil. Science report SC050021 / Naphthalene SGV.	N/A