European Chemicals Bureau

Institute for Health and Consumer Protection

European Chemicals Bureau

Existing Substances

European Union Risk Assessment Report

| CAS No: 91-20-3 | EINECS No: 202-049-5 |

naphthalene

European Union Risk Assessment Report naphthalene

CAS: 91-20-3
EC: 202-049-5

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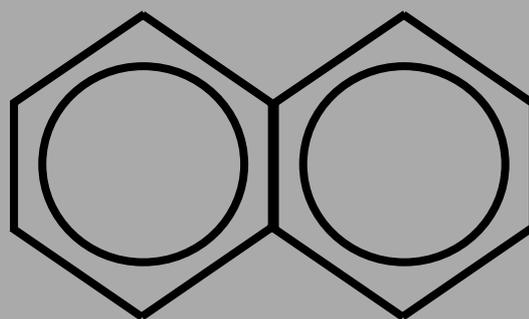
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European Union Risk Assessment Report

NAPHTHALENE

CAS No: 91-20-3

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RISK ASSESSMENT

NAPHTHALENE

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RISK ASSESSMENT

Final Report, 2003

United Kingdom

This document has been prepared by the UK rapporteur on behalf of the European Union. The scientific work on the environmental part was prepared by the Building Research Establishment Ltd (BRE), under contract to the rapporteur.

Contact (human health): Health & Safety Executive
Industrial Chemicals Unit
Magdalen House, Stanley Precinct
Bootle, Merseyside L20 3QZ
e-mail: ukesrhh@hse.gsi.gov.uk
Tel: + 44 151 951 3086
Fax: + 44 151 951 3308

Contact (environment) Environment Agency
Chemicals Assessment Section
Ecotoxicology and Hazardous Substances National Centre
Isis House, Howbery Park
Wallingford, Oxfordshire, OX10 8BD
Fax: +44 (0)1491 828 559

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Review of report by MS Technical Experts finalised: 2001
Final report: 2003

Foreword

We are pleased to present this Risk Assessment Report which is the result of in-depth work carried out by experts in one Member State, working in co-operation with their counterparts in the other Member States, the Commission Services, Industry and public interest groups.

The Risk Assessment was carried out in accordance with Council Regulation (EEC) 793/93[1] on the evaluation and control of the risks of "existing" substances. "Existing" substances are chemical substances in use within the European Community before September 1981 and listed in the European Inventory of Existing Commercial Chemical Substances. Regulation 793/93 provides a systematic framework for the evaluation of the risks to human health and the environment of these substances if they are produced or imported into the Community in volumes above 10 tonnes per year.

There are four overall stages in the Regulation for reducing the risks: data collection, priority setting, risk assessment and risk reduction. Data provided by Industry are used by Member States and the Commission services to determine the priority of the substances which need to be assessed. For each substance on a priority list, a Member State volunteers to act as "Rapporteur", undertaking the in-depth Risk Assessment and recommending a strategy to limit the risks of exposure to the substance, if necessary.

The methods for carrying out an in-depth Risk Assessment at Community level are laid down in Commission Regulation (EC) 1488/94[2], which is supported by a technical guidance document[3]. Normally, the "Rapporteur" and individual companies producing, importing and/or using the chemicals work closely together to develop a draft Risk Assessment Report, which is then presented at a Meeting of Member State technical experts for endorsement. The Risk Assessment Report is then peer-reviewed by the Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) which gives its opinion to the European Commission on the quality of the risk assessment.

If a Risk Assessment Report concludes that measures to reduce the risks of exposure to the substances are needed, beyond any measures which may already be in place, the next step in the process is for the "Rapporteur" to develop a proposal for a strategy to limit those risks.

The Risk Assessment Report is also presented to the Organisation for Economic Co-operation and Development as a contribution to the Chapter 19, Agenda 21 goals for evaluating chemicals, agreed at the United Nations Conference on Environment and Development, held in Rio de Janeiro in 1992.

This Risk Assessment improves our knowledge about the risks to human health and the environment from exposure to chemicals. We hope you will agree that the results of this in-depth study and intensive co-operation will make a worthwhile contribution to the Community objective of reducing the overall risks from exposure to chemicals.
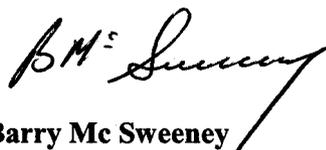
Barry Mc Sweeney
Director-General
DG Joint Research Centre

Catherine Day
Director-General
DG Environment

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[1] O.J. No L 084, 05/04/199 p.0001 – 0075

[2] O.J. No L 161, 29/06/1994 p. 0003 – 0011

[3] Technical Guidance Document, Part I – V, ISBN 92-827-801 [1234]

0 OVERALL RESULTS OF THE RISK ASSESSMENT

CAS-No.:   91-20-3
EINECS-No.:   202-049-5
IUPAC name: Naphthalene

Environment

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion is based on site-specific data from one plant using naphthalene in the manufacture of grinding wheels. The calculations indicate that the use of naphthalene in this process is likely to cause adverse effects in water and sediment, to microorganisms in the wastewater treatment plant and in the soil compartment. Information from another plant using naphthalene in the manufacture of grinding wheels has indicated that there should be no adverse effects arising from its use at this location.

It is recognised that for both sediment and soil the PNEC used is derived from the surface water PNEC using the equilibrium partitioning method, and so in both of these cases the PNEC could be revised through toxicity testing. However, risks have only been identified for one site using naphthalene for this purpose. This site is developing plans to reduce releases to water, air and sludge so the rapporteur does not think it necessary to require such testing to be carried out. The planned risk management strategy will be based on the above conclusions to ensure that emissions to water (and hence sediment) and sludge are limited.

Conclusion (ii)   There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to release of naphthalene to the aquatic (including sediment) and terrestrial compartments from naphthalene production and its use as an intermediate, in pyrotechnics and in mothballs. There is also no risk to microorganisms from production or any of these uses.

Human health

Human health (toxicity)

Workers

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

The endpoints for which there are concerns are haemolytic anaemia, local effects on the respiratory tract following repeated inhalation exposure and carcinogenicity. In relation to haemolytic anaemia, the available data do not allow the identification of a NOAEL nor the dose-response characteristics for this endpoint. In the absence of such information, it is considered that any significant body burden (values in the mg/kg range) arising from inhalation and/or dermal exposure gives rise to concern. Thus, conclusion (iii) is reached for this endpoint for all

occupational exposure scenarios, except the professional use of coal tar soaps and shampoos. In relation to local effects on the respiratory tract following repeated inhalation exposure, and carcinogenicity, there are concerns for human health for all occupational scenarios, except the professional use of coal tar soaps and shampoos. Thus, conclusion (iii) is reached for this endpoint for all occupational exposure scenarios, except the professional use of coal tar soaps and shampoos.

Conclusion (ii)   There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

There is no concern for inhalation effects or for haemolytic anaemia for workers during the professional use of coal tar soaps and shampoos, hence conclusion (ii) is reached for this exposure scenario.

Consumers

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

In relation to haemolytic anaemia, the available data do not allow the identification of a NOAEL nor the dose-response characteristics for this endpoint. In the absence of such information, any significant body burden (values in the mg/kg range) is considered to give rise to concerns for human health. Exposure of infants to textiles (clothing/bedding) which have been stored for long periods with naphthalene moth repellent raises significant concern. There is documented evidence for the development of severe haemolytic anaemia resulting from such use, although there is no quantitative information available on the level or duration of exposure to naphthalene in these cases. There are no consumer exposure scenarios for which both inhalation and dermal exposures are considered to be negligible, particularly when considering body burdens for infants, and therefore conclusion (iii) applies for all consumer exposure scenarios for this endpoint.

In relation to local effects on the respiratory tract following repeated inhalation exposure, and carcinogenicity, all scenarios for which there is the potential for repeated inhalation exposure to naphthalene are considered to give rise to concern. Thus, conclusion (iii) applies to the consumer use of mothballs and to exposures arising after damp-proof laying.

Conclusion (ii)   There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

There are no concerns for local effects on the respiratory tract, or carcinogenicity, for those exposure scenarios that are single, rare events, nor for exposure scenarios that result in negligible inhalation exposure. Thus, conclusion (ii) is reached for creosote application, for damp-proof laying and for the use of coal tar soaps and shampoos.

Humans exposed via the environment

Conclusion (i)   There is need for further information and/or testing.

Exposure in the locality of grinding wheel plants is estimated to result in a much higher daily intake (0.25 mg/kg/day) than that for regional exposure and it is not possible to conclude there is no concern for human health. Since a conclusion (iii) has been reached for environmental exposure for this use, it is anticipated that exposures will be reduced as a result of environmental risk reduction measures. It is therefore proposed that measured exposure data for this scenario be

obtained following environmental risk reduction activity and these data used to reconsider the risk to humans via local environmental exposure. Therefore conclusion (i) is reached.

Conclusion (ii)   There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

For regional exposure, the environmental airborne levels and resultant estimated total daily human intake of naphthalene are very low. Although it is not possible to quantitatively assess the risks for haemolytic anaemia, given the extremely low level of exposure for the regional scenario, this exposure does not give rise to concern. Similarly, in relation to local effects on the respiratory tract and carcinogenicity, these very low exposures do not give rise to concern. Therefore conclusion (ii) applies.

Combined exposure

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

In relation to haemolytic anaemia, the available data do not allow the identification of a NOAEL nor the dose-response characteristics for this endpoint. In the absence of such information, any significant body burden (values in the mg/kg range) is considered to give rise to concerns for human health. The potential body burden arising from combined exposure is too high to be considered negligible and thus there are concerns. Although further information on the exposures in the locality of grinding wheel plants has been requested, which may result in refinement of the risk characterisation for this scenario, nevertheless, the combined exposures arising from occupational and consumer exposure give rise to concern. Thus, conclusion (iii) is reached. There are also concerns for most occupational and some consumer exposures for repeated inhalation toxicity and carcinogenicity. Therefore, there are concerns for these endpoints for combined exposure and hence the same conclusions as for workers and consumers apply.

Human health (risks from physicochemical properties)

Conclusion (ii)   There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

There are no significant risks from physicochemical properties. Therefore, conclusion (ii) is reached.

CONTENTS

Euses Calculations can be viewed as part of the report at the website of the European Chemicals Bureau: http://ecb.jrc.it

TABLES

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS-No.: 91-20-3
EINECS-No.: 202-049-5
IUPAC name: naphthalene
Synonyms: antimite, naphthalin, naphthene, tar camphor
Molecular weight: 128.18
Molecular formula: $C_{10}H_8$
Structural formula:
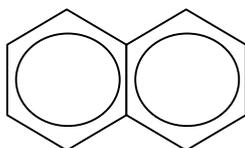
1.2 PURITY / IMPURITIES, ADDITIVES

1.2.1 Purity

The purity from the suppliers varies but is generally > 95%. Impurities present (% w/w) include some or all of the following:-

Benzo[*b*]thiophene   < 2 %
Indan   0.2 %
Indene   < 2 %
Methylnaphthalene   < 2 %

1.2.2 Additives

There were no stated additives used with this product.

1.3 PHYSICO-CHEMICAL PROPERTIES

The data on the physico-chemical properties of naphthalene have been obtained from many sources, including the IUCLID data, handbooks and in many instances original literature references to confirm the values.

1.3.1 Physical state (at ntp)

Naphthalene is a colourless to brown solid depending on manufacture and purity. It has a characteristic, readily detectable odour (threshold circa 0.08 ppm; Amoore and Hautala, 1983) and sublimes slowly at room temperature. It is available as flakes, powder, cakes or balls.

1.3.2 Melting point

The melting point of *pure* naphthalene is 80.2-80.3^{o}C and this value is consistent within the handbooks of chemical data and is supported by the literature (McCullough et al., 1957; Zwolinski, 1986).

Commercial naphthalene typically has a melting point of 78-80°C (quoted in the IUCLID data set) depending on the purity which is usually greater than 95%.

1.3.3 Boiling point

The boiling point of *pure* naphthalene is 217.9-218°C at 1,013 hPa (760 mm Hg) and this is supported by the chemical handbooks and the literature (McCulloch et al., 1957; Zwolinski et al., 1986) and can be accepted as valid.

The IUCLID data on boiling point has been carried out to EEC Directive 84/449/EEC.A.2 and the results accurately reflect the measured literature values.

1.3.4 Density

The relative density of naphthalene is quoted as 1.025 at 20°C (CRC Handbook, 1995), 1.175 at "25°C" (Kirk-Othmer, 1991) although the original reference (Zwolinski et al., 1986) states this value is applicable at 20°C. The Merck Index (1989) quotes 1.162 at 20°C.

The IUCLID data set quotes 1.025 at 20°C (CRC, 1995) and 1.05 from a safety data sheet (Rutgers, date unknown). Other figures quoted from the IUCLID data include 1.145-1.179, from various secondary sources.

1.3.5 Vapour pressure

The vapour pressure of naphthalene at room temperature is relatively low (circa 0.01 kPa). Sources of vapour pressure data on naphthalene include the data published by the Coal Tar Research Association (1965) and the work of Fowler et al. (1968), using 99.99% pure naphthalene, and Camini and Rossini (1955), although these are at elevated temperatures.

The CRC Handbook (1995) quoting data from the Data Institute for Physical Properties Research (DIPPR, 1987) indicates vapour pressures of 0.011 kPa at 25°C, 0.768 kPa at 75°C, 2.5 kPa at 100°C, 6.84 kPa at 125°C and 16.2 kPa at 150°C. Also quoted is a range of vapour pressures for solid naphthalene. Where vapour pressure is quoted below the melting point, this is in effect the *sublimation* pressure - see **Table 1.1**.

Table 1.1   Sublimation pressure values for naphthalene at various temperatures below the melting point

Temperature/°C (°C= K-273.14)	Vapour pressure (Pa) *
-23.14	0.04
-3.14	0.51
6.86	1.66
16.86	4.92
26.86	13.43
36.86	34.15
46.86	182.9
80.29	999.6

(CRC Handbook, 1995)

A graph of log vapour (sublimation) pressure versus 1/T where the temperature is in K is illustrated in **Figure 1.1**. The plot is a straight line as expected and the data can be considered as reliable.

The IUCLID data set quotes 0.008 kPa at 20°C for naphthalene *pure* (Rutgers, date unknown) and values ~0.01 kPa at 25°C (Bradley and Cleasby, 1953; Macknick and Prausnitz, 1979; Wasik et al., 1983) with further values of 0.11 kPa at 50°C (Auer, 1988).

A paper by deKruif et al. (1982) looked closely at the low and medium temperature vapour pressure of 99.97% pure naphthalene and the experimental data can be considered very reliable. This gives a vapour pressure of 7.29 Pa at 20.69°C and 10.42 Pa at 24.27°C. These values are probably the most accurate available. The vapour pressure of naphthalene for modelling purposes will be taken as 7.2 Pa at 20°C and 10.5 Pa at 25°C.

Figure 1.1   Log sublimation pressure (Pa) v 1/ T (K)

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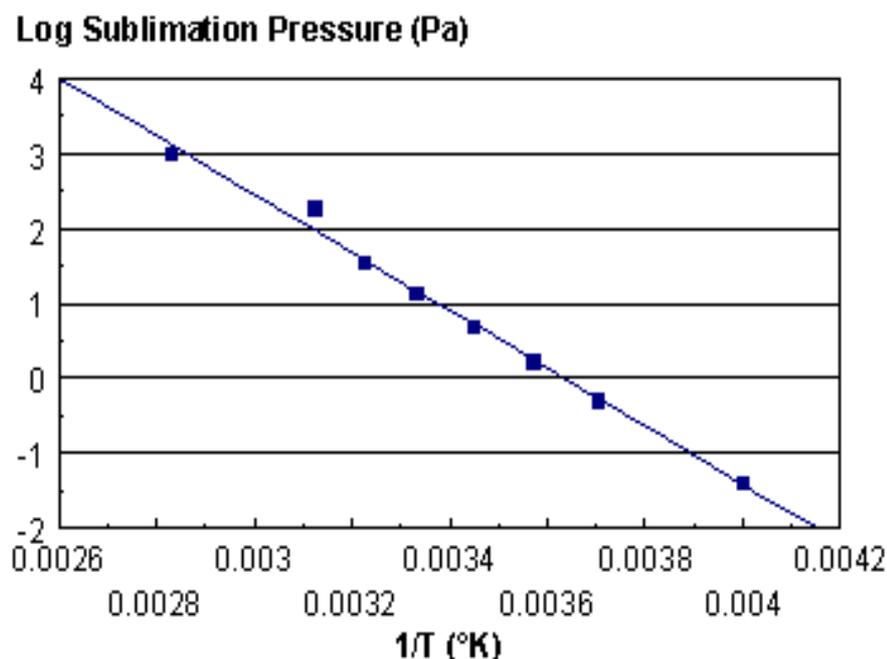
1.3.6   Solubility

Naphthalene is very slightly soluble in water but is appreciably soluble in many organic solvents - it is soluble in alcohol, benzene and very soluble in ether and carbon tetrachloride (Kirk-Othmer, 1991; Faradays encyclopaedia, 1960 (quoting original refs.); Heric and Posey, 1964; Heric and Yeh, 1970; and Ward, 1932).

The Merck Index (1989) and the CRC handbook (1987) describe naphthalene as "insoluble" in water, however the CRC handbook also quotes a water solubility value of 0.03 g/l (30 mg/l) for naphthalene based on the IUPAC solubility data series.

The IUCLID data set quotes naphthalene as being of low solubility and quotes water solubility values of 0.022-0.034 g/l (22-34 mg/l) using a fluorescence method (Schwartz and Wasik, 1976 and 1977; Mackay and Shiu, 1981 etc.) for *pure* naphthalene. These have not been carried out to GLP although the accuracy of the method is claimed to be high - with detection limits of $0.03 \cdot 10^{-6}$ g/l.

Studies using HPLC methods (Wasik et al., 1983) also quote a similar value (30.64 mg/l). Studies by May et al. (1978) using modified chromatographic methods give a value of 31.69 mg/l (accuracy > 3%).

Earlier references in the literature quote values in this range (Mackay and Shui (1981) gives 31.7 mg/l; Gordon and Thorne (1967) gives 33.6 mg/l; and Bohon and Claussen (1951) gives 34.4 mg/l). Some of the early references quoting a value of 0.030 g/l date back to work done in the earlier part of the century (Hilpert, 1916).

These figures represent the accepted literature values. However, a more recent study (Landis, 1995) using radiolabelled naphthalene has assigned a water solubility of $32 \cdot 10^{-6}$ g/l (32 μg/l). This study indicates a solubility 10^3 less than the accepted literature values. The study, according to GLP guidelines, was conducted for scrutiny by the US FDA. This result which is at variance with the other data in the literature is not used in the assessment.

1.3.7   N-octanol/water partition coefficient (log Kow)

The partition coefficient of naphthalene has been assessed both experimentally and theoretically by a number of authors. Sangster (1989) has summarised literature data on the partition coefficients of a variety of organic compounds including naphthalene whilst further measurements have been found in the literature post 1989 and in the IUCLID data set - see **Table 1.2**. A distinction has been made between calculated and measured values. The log values fall between 3.01 and 3.73.

The measurement by Sanemasa et al. (1994) giving a value of 3.4 is probably the best value obtainable by the shake-flask method. The difficulties with this method include the formation of emulsions during shaking, which affects the observed K_{ow} values; the aqueous phase often needs to be centrifuged to remove most of the small n-octanol droplets. The Sanemasa method involves measures to overcome this and this has led to more accurate (shake-flask) results.

A value of 3.70 obtained by co-current chromatography (Berthod et al., 1992) is also reliable as the method avoids the drawbacks associated with the shake-flask method and is claimed to be a more accurate reflection of K_{ow}.

Although the rationale behind the two most acceptable values is different it has not proved possible to choose between these two values and both can be considered correct. The higher value (3.7) has been used in the environmental modelling.

The theoretical basis for the calculated values has not been assessed.

1.3.8   Flash point

The flash point has been quoted as 79°C (open cup) and 88°C (closed cup) (Merck Index, 1989). Kirk-Othmer (1991) quotes 79°C (closed cup - National Fire Protection Association (USA), 1978). The American Petroleum Institute (1978) quotes a value (closed cup) of 80°C. Sax and Lewis (1989) quote 174°F, i.e. 79°C (open cup) although the original reference is not stated.

The IUCLID data quotes 80°C (Nabert and Schon, 1963) and 99°C for naphthalene *pure* measured to DIN 51758 (Rutgers, safety data sheet) for the closed cup method.

Although the test figures do have some variance this is likely to be due to a combination of the purity of the naphthalene used in the tests and the exact method used.

Table 1.2   Log Kow values for naphthalene

Log Kow	Method	Reference
3.01	measured (shake flask)	Rogers and Cammerata, 1969
3.04	calculated	Banerjee and Howard, 1988
3.2	Indirect- HPLC	Veith et al., 1979
3.2	calculated	Vighi and Calamari, 1987
3.28 and 3.57	calculated	Yoshida et al., 1983
3.3	shake-flask	Geyer et al., 1984
3.3	measured (shake flask)	Brooke et al., 1986
3.30-3.37	measured (shake flask)	Hansch et al., 1964, 1979, 1985
3.3	calculated	Dallas et al., 1993
3.31	shake-flask	Eadsforth and Moser, 1983
3.36	not known	Freitag et al., 1985
3.4	calculated	Kamlet et al., 1988
3.4	measured (shake flask)	Bruggerman et al., 1982
3.4	measured (shake flask)	Karickhoff et al., 1979
3.4	measured (shake flask)	Krishnamurthy and Wasik, 1978
3.4	measured (modified shake flask)	Sanemasa et al., 1994
3.4	Indirect - HPLC	Eadsforth, 1986
3.45	not known	Gossett et al., 1983
3.5	calculated	Klopman et al., 1985
3.59	n/k	Mackay et al., 1982
3.7	calculated	Campbell and Luthy, 1985
3.66-3.73	calculated	Bodor et al., 1989, 1992
3.7	measured (chromatography)	Berthod et al., 1992

The log Kow values quoted in the IUCLID data set are all within the range as above, except for one value of 4.7 (Veith et al., 1979 - taken from unpublished data dating from 1977). This value, which is at variance with all the other data, is almost certainly erroneous.

1.3.9   Autoignition

An autoignition temperature in air of 567°C has been quoted in the Merck Index (1989) and also Sax and Lewis (1989) and as 526°C in Kirk-Othmer (1991) quoting from data obtained by the National Fire Protection Association (USA), 1978. The American Petroleum Institute quote a value of 587°C measured (using reagent grade naphthalene; melting point = 79-81°C) by the US Bureau of Mines (Jones and Scott, 1946). The IUCLID data give a value of 540°C when measured to DIN 51794 (Nabert and Schon, 1963).

Although the test figures do have some variance this is likely to be due to a combination of the purity of the naphthalene used in the tests and the exact method used.

1.3.10   Explosivity

The flammability (explosive limits by volume of fuel at 25°C and 760 mm Hg) for naphthalene have been quoted at 0.9 (lower) to 5.9 (higher) in Lange's Handbook (1992) and Kirk-Othmer (1991) where the original reference is to data obtained by the US Bureau of Mines (Jones and Scott, 1946) using reagent grade naphthalene (melting point = 79-81°C). This range is also quoted in the American Petroleum Institute monograph on naphthalene (1978). The IUCLID data set quotes the same range for explosive limits (Nabert and Schon, 1963).

Although the test figures do have some variance this is likely to be due to a combination of the purity of the naphthalene used in the tests and the exact method used.

Naphthalene can be considered as capable of forming explosive mixtures with air in particulate or vapour form.

1.3.11   Oxidising properties

Although not an oxidising agent itself naphthalene can be readily oxidised by other oxidising agents and undergoes a violent reaction with chromic oxide, CrO_3.

1.3.12   Summary

The physico-chemical data for naphthalene appear to be well established and the data can be considered as reliable, the original literature references having been examined and comparisons made with early observations.

Any variation would relate to the origin and purity of the naphthalene and in some cases the test method employed, particularly so in the case of the n-octanol/water partition coefficient.

The basic physical parameters can be accepted as reliable.

Vapour pressure data have been established at a variety of temperatures and it can be seen that at room temperature naphthalene does not have a large vapour pressure. However, it does have a low odour threshold, which accounts for the characteristic smell of "mothballs". Naphthalene "flakes" would volatilise faster than "mothballs" because of the greater surface area.

The vapour pressure data from a variety of sources have been shown to be reasonably consistent and can be accepted as reliable.

Although there appears to be some confusion over the exact flash point and autoignition temperatures this is likely to be due to a combination of the purity of the naphthalene used in the tests and the exact method used.

A summary of the physico-chemical properties of naphthalene is presented in **Table 1.3**. The values used in the risk assessment are summarised in **Table 1.4**.

Table 1.3   Summary of the physico-chemical properties of naphthalene

Parameter	Value	Comments
CAS No.	91-20-3	
Physical state at ntp	colourless brown solid	Physical characteristics of solid depend on manufacture
Melting point	80.0-80.3°C	Refers to pure compound – technical material (>95% pure) will have melting point circa 78-80°C
Boiling point	217.9-218°C	Refers to pure compound – technical material will have some variation.
Density	1.175 at 25°C	Refers to pure compound – technical material will have some variation.
Vapour (sublimation) pressure	circa 7.2 Pa at 20°C 10.5 Pa at 25°C	Vapour density is 4.42
Solubility in water	0.03 g/l	Practically insoluble
Octanol-water partition coefficient	3.40 (modified shake flask) 3.70 (modified chromatographic method)	Both values have been accurately measured but the rationale behind the methods is different.
Flash point	79°C	Open cup National Fire Protection Association (USA)
	80-88 and 99°C	Closed cup, value of 99°C using method to DIN 51794
Autoignition	526 and 567°C 587°C (per Am. Pet. Inst.)	National Fire Protection Association (USA) gives 526°C
Explosive limits in air % by volume	l=0.9 h=5.9	Jones & Scott (1946)
Oxidising properties	Not an oxidising agent	Can be easily oxidised itself, burns readily in air. Violent reaction with CrO_3
Conversion factor	1 ppm = 5.24 $\mu g/m^3$	At 25°C and 101.3 kPa

Table 1.4   Values for physico-chemical parameters used in the risk assessment

Parameter	Value used in assessment
Melting point	80°C
Boiling point	218°C
Density	1.175
Vapour (sublimation) pressure at 25°C	10.5 Pa
Solubility in water	0.03 g/l
Octanol-water partition coefficient	3.70

1.4   CLASSIFICATION

The classification and labelling of naphthalene has been agreed at technical levels to be listed in Annex I to Directive 67/548/EEC following the adoption of the 29th Adaptation to Technical Progress, as follows:

Classification	Carc. Cat. 3; R40
	Xn; R22
	N; R50/53
Labelling	R: 22-40-50/53
	S-(2-)36/37-(46-)60-61
R40 states:	Possible risk of irreversible effects
	Category 3 is for substances which cause concern for humans owing to possible carcinogenic effects but in respect of which the available information is not adequate for making a satisfactory assessment. There is some evidence from appropriate animal studies but this is insufficient to place the substance in Category 2.
R22 states:	Harmful if swallowed
R50 states:	Very toxic to aquatic organisms.
R53 states	May cause long-term adverse effects in the aquatic environment.
S2 states:	Harmful if swallowed
S46 states:	If swallowed, seek medical advice immediately and show this container or label
S36/37 states:	Wear suitable protective clothing/gloves
S60 states:	This material and/or its container must be disposed of as hazardous waste
S61 states:	Avoid release to the environment. Refer to special instructions/safety data sheet

2 GENERAL INFORMATION ON EXPOSURE

2.1 PRODUCTION

There are two sources for the manufacture of naphthalene; coal tar (which accounts for the majority of the production) and petroleum.

2.1.1 Production processes

2.1.1.1 Production from coal tar

Naphthalene is produced from coal tar fractions by crystallisation and distillation (Chem-Facts, 1991). Coal tar that has been condensed and separated from coke oven gases during high temperature carbonisation of bituminous coal in coke plants is used as a source for naphthalene. Approximately 10 gallons of coal tar are recovered from one ton of coal (or 38 l from one metric tonne) and naphthalene makes up approximately 10% of the coal tar weight (Faith et al., 1975).

Distillation of coal tar produces several fractions including the middle fraction (naphthalene oil) which is the most abundant source of naphthalene and contains about 50% of the naphthalene available from coal tar. The oil fractions obtained during distillation are detailed in **Table 2.1** and a schematic of tar distillation and typical naphthalene mass balance is given in **Figure 2.1**. The middle fraction is allowed to cool in shallow pans and the naphthalene crystallises. The crude naphthalene produced may then be distilled further. The yield of crude naphthalene is 4.8 kg/100 litres of coal tar. The naphthalene oil fraction is then further processed to produce naphthalene. This processing can involve the distillation of the naphthalene oil to produce a crude grade with a crystallisation point of $74^{o}C$ to $78^{o}C$. This crude grade is suitable for applications such as the manufacture of phthalic anhydride. A purer grade can be produced by treating the naphthalene oil fraction with sulphuric acid followed by neutralisation and re-distillation to give a product with a crystallising point of over $79^{o}C$. However this method does not completely remove thionaphthalene which is the main impurity in the crude naphthalene. Alternatively, the more commonly adopted method is to carry out a crystallisation of the naphthalene oil to produce a pure grade that does not contain thionaphthalene and other impurities. The pure grades produced by these methods can be used for applications such as insecticides and mothballs. Drained oils remaining from this purification of the naphthalene oil may be blended for use in creosote oils or if not suitable they can be used in the manufacture of carbon black.

As well as the naphthalene oil, various other fractions are also produced which can contain naphthalene. These oil fractions are further processed to separate commercially viable chemicals such as anthracene from anthracene oil. Alternatively they are used in blends, for example in base oil for road tar production. Drained oils remaining from this further processing are then blended to produce creosote, which may contain up to 25% naphthalene. Any remaining oils (these may contain about 4% naphthalene) may be sold for the manufacture of carbon black. It is also understood that some producers may supply heating oils containing up to 10% naphthalene.

Table 2.1   Typical concentrations of naphthalene in oil fractions obtained during the distillation of coal tar

Substance	Naphthalene content (%)	Boiling point (ºC)
Tar	10	-
Liquor	0	-
Light oil	0	80-150
Carbolic oil	13	160-190
Naphthalene oil	70	210-225
Wash oil	18	235-300
Anthracene oil	4	270-370
Base oil	2	310-400
Pitch	0	-

Figure 2.1   Typical schematic of tar distillation
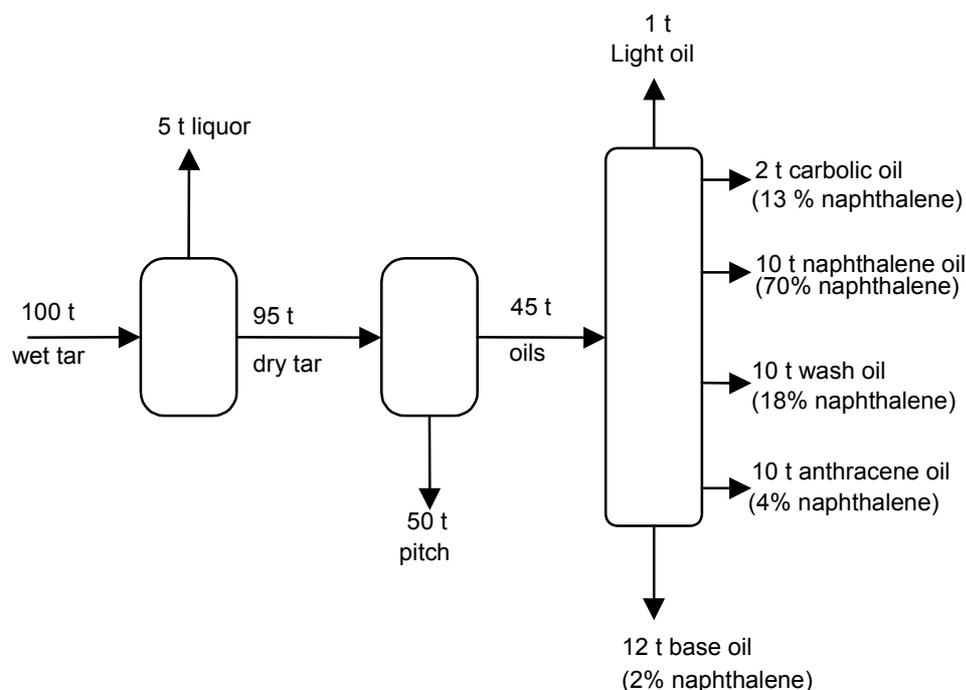
2.1.1.2   Production from petroleum

Until 1961 coal was the only source of naphthalene but it can also be produced from petroleum fractions high in methylnaphthalenes (Faith et al., 1975). Dealkylation is carried out at high temperature and pressure in the presence of hydrogen to produce naphthalene that is 99% pure and low in sulphur. This method is not thought to be used in the UK but one European producer uses this method for part of its naphthalene production.

It was not possible to determine the manufacturing method adopted by the only company producing naphthalene in the European Union (EU) from petroleum. The literature details several methods that involve two principal steps. The first is the production of an aromatic oil in

the naphthalene - alkylnaphthalene boiling range by hydroaromatization or cyclisation. The second step is the dealkylation of such oils either thermally or catalytically. The naphthalene that is produced, usually by crystallisation, is recovered as a high quality product, usually by fractional distillation.

Naphthalene is also recovered from the stream of methyl naphthalenes formed in cracking of heavy liquids (naphthas and gas oils) for ethylene production (Faith et al., 1975).

2.1.2 Production volumes

Western European production capacity for naphthalene in 1985 was 282,000 tonnes (SRI, 1985). Information from IUCLID states that 217,000 tonnes of naphthalene were produced in Western Europe in 1987 and that production is between 100,000 and 500,000 tonnes annually with a number of producers in Europe. The total annual production capacity of all facilities in Western Europe in 1986 was estimated as 281,000 tonnes, which is very similar to that estimated by SRI (BUA, 1989). More recent figures suggest that the total annual production in Western Europe is lower than these figures. 1.5 million tonnes of coal tar are used in Western Europe per annum. Of this a maximum of 10% can be used in naphthalene production, giving a production figure of 150,000 tonnes per annum (Industry Sources). However, available site-specific information suggests that the annual production of naphthalene is between 199,000 and 204,000 tonnes per annum. This figure includes a production tonnage of 20,000 tonnes per annum of 'naphthalene oil'. This material is believed to be at least 90% pure. Lower grade naphthalene oil, containing about 60% naphthalene, has a separate CAS-number and has not been considered in the assessment. In the assessment the total annual production of naphthalene in the EU has been taken to be 200,000 tonnes.

Companies producing naphthalene are located in the UK, Belgium, France, Italy, Netherlands, Denmark, Germany, Austria and Spain. One company uses both coal tar and petroleum as sources for naphthalene and the other companies use coal tar as their only source. Production figures from individual producers ranged from 4,000 to 70,000 tonnes per annum. In the UK there is only one company distilling naphthalene from coal tar using plants at two of their sites. Purification of the naphthalene oil is carried out at only one of these sites. It was estimated in 1985 that 12,000 tonnes would be produced in the UK in 1990 (SRI, 1985). The total volume of naphthalene entering the UK market from production and imports is about 16,000 tonnes/annum.

These figures do not include naphthalene present in distillates used for the production of creosote, which represents a further 10,000 tonnes/annum and 2,500 tonnes/annum in the EU and UK, respectively. The volume of naphthalene present in the EU as constituents of oils used for carbon black manufacture or in heating oils was not established. There are also a number of distributors throughout the EU who act as agents for naphthalene produced in the EU or from elsewhere in the world.

2.2 USE

Figures for the amount of naphthalene used within the EU vary. 160,000 tonnes per annum were used in Western Europe in 1986 (BUA, 1989). More recent figures suggest that current demand in Western Europe does not exceed 127,000 tonnes per annum (Industry sources). Only small amounts of naphthalene are imported into the EU and up to 25% of production is exported. If 25% of the total production tonnage were exported the amount used within the European Union

would be 150,000 tonnes per annum based on a production tonnage of 200,000 tonnes per annum. For the purposes of this assessment the amount of naphthalene used within the European Union has been taken as 140,000 tonnes per annum. This is derived from the most recent information available for the specific uses as given below.

2.2.1 Use as an intermediate

2.2.1.1 Phthalic anhydride

Naphthalene is used as feedstock in the manufacture of phthalic anhydride. However, the amount of naphthalene used for this purpose depends on its market price relative to that of o-xylene (which is also used in phthalic anhydride production) and on the demand for naphthalene for use in other processes. The use of naphthalene in phthalic anhydride production has consequently fallen over recent years. Approximately 70% of naphthalene produced in 1976 was used in the production of phthalic anhydride (Collins and Richey, 1992) although more recent figures show that production of phthalic anhydride from naphthalene has subsequently fallen. 59,000 tonnes per annum were used in 1982 in phthalic anhydride production (Posthumus and Canton, 1995). In 1986 64,000 tonnes of phthalic anhydride were produced from naphthalene (BUA, 1989). The 1989 figure was 50,000 tonnes per annum (Weissermel and Arpe, 1993). Industry sources indicate that the amount of naphthalene used in the production of phthalic anhydride in the EU is currently about 40,000 tonnes per annum and this figure has been assumed for the purposes of the risk assessment.

There are three plants using naphthalene to manufacture phthalic anhydride in the EU, one in the UK, one in Belgium and one in Italy. Two of these companies use xylene as a co-feed stock, although naphthalene is the main starting material.

2.2.1.2 Dyestuffs

Naphthalene is used in the production of azo dyes, via the intermediates 2-naphthol and naphthalene sulphonic acids. In 1986 about 46,000 tonnes of naphthalene were used in the production of azo dyes (BUA, 1989). No more up to date information is available and this figure has therefore been used in the assessment. The number of companies using naphthalene for this application was not established.

2.2.1.3 Naphthalene sulphonic acids

Naphthalene is used to produce naphthalene sulphonic acids by reaction with formaldehyde and sulphuric acid and subsequent neutralisation with sodium hydroxide and ammonia. The principal use for naphthalene sulphonic acids is for the manufacture of plasticisers for concrete. They are also used in the manufacture of an ingredient for plasterboard, as dispersants in synthetic and natural rubbers and in tanning agents for the leather industries. There is understood to be only negligible residual naphthalene remaining in the naphthalene sulphonic acids after reaction.

24,000 tonnes per annum of naphthalene were used in the production of naphthalene sulphonic acids in the EU in 1986 in addition to that used in the production of azo dyes (BUA, 1989). This figure could be an underestimate of current use. The use of naphthalene in the production of

naphthalene sulphonic acids is growing in the USA where the tonnage used in this process is expected to increase at 7-9% per annum (Kirk-Othmer, 1991). However, as no more up to date information is available for the EU the 1986 value has been used in the assessment.

There are only two companies within the UK using naphthalene for this purpose, with a further 10-15 companies in other member states. The total volume used in the UK for the manufacture of naphthalene sulphonic acids is about 4,000 tonnes/annum.

2.2.1.4   Alkylated naphthalene solvents

There is one company in the EU using naphthalene to manufacture alkylated naphthalene solvents. This company is located in Germany. It is estimated that up to 15,000 tonnes/annum are used in the manufacture of these solvents.

2.2.1.5   2-Naphthol

One company is listed in IUCLID as a manufacturer of 2-naphthol from naphthalene. It has been assumed that about 12,000 tonnes of naphthalene per annum are used in this process. However, as 2-naphthol is used as an intermediate in the manufacture of azo dyes, there may be some double counting in assigning tonnages to these uses.

2.2.1.6   Others

Naphthalene is also used as a feed stock in the synthesis of a number of miscellaneous chemicals and pharmaceuticals. The extent of its use in these applications has not been established although the 1989 BUA report estimated that 4,000 tonnes of naphthalene were used in various "miscellaneous" applications in 1986. Naphthalene is used in the manufacture of the insecticide 1-naphthyl-N-methylcarbamate (trade names Carbaryl, or Sevin, although this substance is not believed to be produced in significant quantities within the EU).

2.2.2   Other uses

2.2.2.1   Mothballs

15,000 tonnes of naphthalene were used in the manufacture of fumigants in the EU in 1986 (BUA, 1989). However, the use of naphthalene as a moth repellent and insecticide has decreased since the introduction of chlorinated compounds such as p-dichlorobenzene (Merck, 1989). Naphthalene is still used in the manufacture of mothballs. In the EU this is predominantly carried out by a company located in Belgium, which distributes world-wide. This may be to companies who re-package the mothballs for re-distribution. About 1,000 tonnes/annum are used for the manufacture of mothballs throughout the EU. Naphthalene is also used in museums in order to protect articles preserved in storage drawers/cupboards from attack by pests.

2.2.2.2   Pyrotechnics

Naphthalene is used in special effects for the film industry. It is either used with other components to make an ignitable pellet for the generation of black smoke or in a container with a charge for the simulation of a explosion.

A number of companies in the EU are known to use naphthalene in the manufacture of pyrotechnics. Four are in the UK, two in Germany, and one each in France and Italy. One UK site uses between 1 and 1.5 tonnes of naphthalene per annum in pyrotechnics. This is believed to account for at least 25% of the UK market. It has been assumed that about 15 tonnes/annum of naphthalene is used for pyrotechnic manufacture in the EU.

2.2.2.3   Grinding wheels

Naphthalene is used as an artificial pore former in the manufacture of grinding wheels to give a high porosity product. At least 3 companies in the EU use naphthalene in the manufacture of grinding wheels. A total of 350 tonnes/annum are used at these plants. Naphthalene is purchased as sized granules and put into the wheel during the manufacturing stage. The materials are cold-pressed to give the required shape and dried to remove excess moisture. Naphthalene is then driven off at about 120°C and collected over water in a recovery oven. The wheel is then kiln fired to vitrify the ceramic materials at temperatures of up to 1,250°C. Naphthalene may be used in the manufacture of grinding wheels in other EU countries, although para-dichlorobenzene is also used. However, information is not currently available regarding the total number of plants in the EU.

2.3   OTHER PRODUCTS CONTAINING NAPHTHALENE

2.3.1   Creosote

Creosote which is used for timber treatment consists of blends of distillates of coal tar. It comprises over 200 components, the major constituents being naphthalene and its alkyl homologues. In the EU the tar distillers who produce naphthalene use tar distillates to blend creosote. In addition to the nine tar distillers, there is one further producer of creosote in the UK who uses coal tar produced from low temperature coking. There are also understood to be a number of other smaller companies who purchase distillates to blend creosote, primarily for brush application. These small blenders may use distillates not containing naphthalene and may not manufacture to the standards below.

In the UK creosote is produced to BS 144: Part 1: 1990. This describes three specifications for creosote, Type 1 for pressure impregnation of timber (predominantly for railway sleepers), Type 2 which has a more closely defined distillation range for impregnation of timber (predominantly for telegraph poles) and Type 3 for timber treatment by immersion, spraying or brushing.

The concentration of naphthalene is only specified for Type 2 at 8-25%. This is understood to be important where bleeding of the creosote may be a problem. Although the level of naphthalene is not specified in Type 1 and 3 it is still likely to be present at concentrations between 5 and 10%.

In the EU creosote is manufactured to grades specified by the Western European Institute for Wood Preservation. Two grades are described, A for railway sleepers and B for telegraph poles.

The concentration of naphthalene is not specified, although similar ranges to those for BS144: Part 1: 1990 are likely.

It is estimated that about 100,000 tonnes/annum of creosote are used in the EU, 25,000 tonnes of which are used in the UK market. About 10% of these figures represent the level of naphthalene in this industry. The level of use of creosote may vary between member states depending on their own national policies. The effects of the requirements for the control of volatile organic compounds (VOCs) under the UK Environmental Protection Act (EPA) 1990 may result in manufacturers reducing the naphthalene content of creosote. Directive 99/13/EC on the limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations (the Solvent Emissions Directive), which will require EU member states to control emissions of VOCs, may also result in companies reducing the naphthalene content of creosote.

It is understood that about 60% of the above figures represent the amount of creosote used by bulk impregnators of timber, with the remaining 40% being packaged for brush applications (predominantly for domestic use). It is estimated that about 50% of creosote used for brush applications does not contain naphthalene. There are understood to be 4 bulk impregnation plants in the UK, with a further 5 elsewhere in the EU. There are about 10 packaging plants in the UK. The total number of packaging plants in the EU was not established.

2.3.2 Tar paints, waterproof membranes, etc.

Tar containing naphthalene is used in some specialist paints and waterproof membranes. It is understood that Germany does not use tar paints and the Scandinavian countries are moving away from them. The size of the market for these was not established, although one producer reported that about 600,000 litres of waterproof membrane are used each year in the UK. The membranes contain about 1% naphthalene. Coal tar paints contain about 1-2%, coal tar epoxy paints contain less than 0.1% and coal tar polyurethane sealers less than 1%. These paints and membranes are generally used by the building trade. Waterproof membranes are supplied in 2.5 litre containers up to 200 litre drums. These are generally used to retrospectively waterproof floors and walls, and can be applied to wet surfaces. These systems are estimated to account for about 10% of the waterproofing market.

2.4 ENVIRONMENTAL RELEASES

Releases of naphthalene may occur during production and during use as an intermediate in the production of phthalic anhydride, dyes and other chemicals, as described above. However the major release of naphthalene is from combustion processes, in particular from vehicle exhausts. Naphthalene is released during the treatment of wood with preservative and from treated wood. Release of naphthalene also occurs from other sources, such as the production of aluminium (it volatilises from the electrodes used), from oil refineries and from offshore drilling activities. Section 3 considers releases from these "indirect" sources as well as those from the production and use of naphthalene as a substance and products in which it is contained.

2.5   LEGISLATIVE CONTROLS

Naphthalene is included in the European Community priority candidate list (List of 129) which indicates substances that are likely candidates for EC-wide control and in the meantime should be treated as List II substances under the EEC Dangerous Substances Directive 76/464/EEC. Naphthalene is included in the reference list of substances identified for consideration at the Fourth North Sea Conference (North Sea Conference, The Hague, 1990). Naphthalene, as a hydrocarbon, may be classified as a List I substance under the EEC Directive 80/68/EEC on ground water protection. Naphthalene, as an organic compound, is a prescribed substance for release to air under the UK Environmental Protection (Prescribed Processes and Substances) Regulations, 1991.

The majority of EU Member States have adopted an Occupational Exposure Limit for naphthalene of 50 mg/m^3 (10 ppm) 8-hour TWA. The German MAK limit of 50 mg/m^3 was removed from their MAK list in 1995. The UK has an additional Short Term Exposure Limit of 75 mg/m^3 (15 ppm) 15-minute reference period.

Occupational exposure limits should be looked at in the context of the measurement and enforcement regimes used to ensure that the limits are met. Limits viewed in isolation may be misleading in terms of the levels of exposure and consequently of risk that they imply.

Naphthalene is an active ingredient in two products registered with the Approvals Group of Pesticides Safety Directorate in the UK for non-professional use as animal repellent (Pesticides, 1996). Eight amateur products and 1 professional product containing naphthalene are registered under the UK Food and Environment Protection Act 1985 and the Control of Pesticides Regulations 1986 (Pesticides, 1996).

3 ENVIRONMENT

3.1 EXPOSURE ASSESSMENT

3.1.1 Environmental releases

Naphthalene has several different uses, although most of the naphthalene produced is used in the chemical industry. However, releases of naphthalene from the chemical industry, from combustion processes and from use as mothballs are likely to be small in comparison to release from vehicle exhausts. The majority of naphthalene release will be to the atmosphere with smaller releases to water and soil.

3.1.1.1 Release during production of naphthalene

For the purposes of the assessment it has been assumed that the total annual production of naphthalene in the EU is 200,000 tonnes. This figure is based on the available site-specific information for production plants as discussed in Section 2.2. Site-specific data for 85% and 90% of the assumed total production are available for releases to air and water , respectively. Releases to air and water for sites producing naphthalene were provided. No information regarding releases to soil is available.

The production of naphthalene (and of phthalic anhydride, isopropylated naphthalene and naphthalene sulphonic acid) does not include process water and there is no wastewater from production. However, industry representatives have stated that during naphthalene production, tanks of the coal tar feedstock and the naphthalene produced are vented to the atmosphere. Therefore, naphthalene is likely to be released to the atmosphere during storage.

Releases to air

Process emissions during naphthalene production have been estimated based on particulate organic matter emissions which are 87% naphthalene (US EPA, 1988). The emission factor to air was estimated to be 0.239 kg/tonne naphthalene produced. An emission factor for release to air from storage has been estimated to be 0.0227 kg/tonne naphthalene produced (US EPA, 1988). The default value in the Technical Guidance Document for the emission factor to air during production, based on a solubility for naphthalene of 30 mg/l and a vapour pressure of 10.5 Pa and assuming that naphthalene is in category Ic (intermediates stored off-site), is 0.0001 (0.1 kg/tonne).

Site-specific information for naphthalene release to air is available. The information generally gives the total emissions arising from all activities (production, processing, storage and handling) carried out at each site. These emissions mainly arise as a result of storage, leakage and open handling rather than from the production processes themselves. Emission factors for release to air derived from this data are in the range 0.001-0.026 kg/tonne. These emission factors cover 85% of the total EU production and are assumed to be representative of the industry as a whole. The highest of these figures has therefore been used in estimating "worst-case" emissions for the remaining production sites. Releases to air for all production sites are summarised in **Table 3.1** together with the regional and continental releases from production. In calculating regional and continental releases from naphthalene production it has been assumed

that there is only one production site in any region. Releases at the regional and continental levels have therefore been respectively derived from the highest local release and the total of the releases from all sites. Regional and continental releases have been averaged over 365 days.

Table 3.1   Release of naphthalene to air during production

Environment/Site	Release (kg/day)
A	0.58
B	0.13
C	1.30
D	0.95
E	0.52
F	0.35
G	0.33
H	2.08
I	0.97
J	0.07
Regional	1.71 (over 365 days)
Continental	4.27 (over 365 days)

Releases to water

Default emission factors for the release of naphthalene to water during the production process can be derived from the Technical Guidance Document (EC, 1996a). Assuming that naphthalene is in category Ic (intermediates stored off-site), the emission factor for a wet process is 0.003. For a dry process the value is 0 (Emission Scenario Document, Technical Guidance Document Chapter 7).

However, site-specific information on release to water accounting for 90% of the total production of naphthalene has been provided. Emission factors for release of naphthalene to water after on-site wastewater treatment are in the range 0.001-0.002 kg/tonne. Emission factors based on concentrations in water prior to any treatment range up to 0.114 kg/tonne. These releases are reported to arise from crude tar processing rather than naphthalene production itself. This information is assumed to be representative of the industry as a whole and has therefore been used in the risk assessment.

The site-specific information shows that wastewater is subject to different treatments at different plants. The treatments used include:

- treatment of wastewater by adsorption which removes most polycyclic aromatic hydrocarbons prior to treatment in a biological treatment works;
- treatment of emissions by resin adsorption before release;
- storage of effluents in a tank before being sent to an effluent treatment plant (no further information on treatment is available);
- release to a municipal treatment plant with no on-site treatment.

For sites where specific information is not available releases to water have been estimated using a worst-case emission factor of 0.114 kg/tonne prior to treatment. Releases have been assumed to occur to wastewater treatment plants that have the characteristics specified in the Technical Guidance Document[4] unless the available information indicates otherwise. This assumption has been made irrespective of whether the release occurs to an on-site or a municipal treatment plant and gives releases that are higher than those obtained using an emission factor of 0.002 kg/tonne (the highest factor for emissions after on-site wastewater treatment).

Local, regional and continental releases to water are summarised in **Table 3.2**. In calculating regional and continental releases it has been assumed that there is only one production site in any region. Releases from naphthalene production at the regional and continental levels have therefore been derived from the highest local release and the total of the releases from all sites. Regional and continental releases have been averaged over 365 days.

Table 3.2 Local, regional and continental releases of naphthalene to water during production (after wastewater treatment)

Environment	Release (kg/day)
A	0.43
B	0.021
C	$1 \cdot 10^{-5}$
D	1.46
E	0.79
F	$1.8 \cdot 10^{-5}$
G	0.0015
H	$5 \cdot 10^{-4}$
I	$1.5 \cdot 10^{-4}$
J	0.0067
Regional	1.2 (over 365 days)
Continental	1.03 (over 365 days)

Release to soil

No site-specific information is available regarding releases to soil from naphthalene production. The default value given in the Technical Guidance Document for naphthalene production, assuming that naphthalene is in category Ic (intermediates stored off-site), is 0.0001. Releases calculated for the regional and continental levels using the default value from the Technical Guidance Document are given in **Table 3.3**.

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4   It has been assumed that naphthalene is inherently biodegradable (see Section 3.1.1.2). From the EUSES model the fate of naphthalene in the wastewater treatment plant is: 27.4% to air; 11.2% adsorbed to sludge; 26.6% degraded; and 34.8% released to water.

Table 3.3   Release of naphthalene to soil during production

Environment	Emission factor	Production volume (t/a)	Release (kg/day)
Regional	0.01%	70,000	19.2
Continental	0.01%	130,000	35.6

3.1.1.2   Release from use in the chemical industry

Naphthalene is predominantly used as an intermediate in the production of phthalic anhydride, azo dyes, naphthalene sulphonic acid, alkylated naphthalene solvents and in the synthesis of a number of miscellaneous chemicals and pharmaceuticals as discussed in Section 2.3. Releases may potentially occur from all these processes. The tonnages of naphthalene assumed to be used in the various processes within the chemical industry are summarised in **Table 3.4**.

Table 3.4   Tonnages of naphthalene used as intermediates

Process	Annual continental tonnages used in assessment
Phthalic anhydride production	40,000
Manufacture of dyestuffs	46,000
Naphthalene sulphonic acid manufacture	24,000
Alkylated naphthalene solvent production	15,000
2-Naphthol	12,000
Total	137,000

None of the manufacturing processes considered here includes process water and the processes have therefore been considered to be dry. For a chemical (vapour pressure 10.5 Pa, water solubility 30 mg/l, category Ic) used as an intermediate in a dry process, default emission factors to air, water and soil are 0.001%, 0% and 0.01%, respectively (Emission Scenario Document, Technical Guidance Document, Chapter 7).

US EPA data from the 1980s showed that for each tonne of phthalic anhydride produced from naphthalene 25-34 kg of volatile organic compounds are emitted (US EPA, 1986). However, it was not known how much of this was naphthalene. Other data have shown that emission factors for release of naphthalene from storage tanks used in phthalic anhydride production from naphthalene ranged from 0.6 g/kg produced (uncontrolled) to 0.006 g/kg produced (controlled) (US EPA, 1988).

Site-specific information is available for approximately 75% of the total phthalic anhydride production and for about 30% of total intermediate use assumed in this assessment. All the emissions included are reported to occur during the loading and storage stages. There are no reported releases from the production process itself. Based on the available data a worst-case emission factor to air of 0.1 kg/tonne can be derived for the process as a whole. A small amount is also released to water, although the production processes themselves are dry and a worst-case emission factor of 0.0045 kg/tonne can be derived from site-specific data for releases to water. No information is given for releases to soil.

For the purpose of this assessment, the releases of naphthalene during use as an intermediate have been calculated using emission factors of 0.1 kg/tonne to air and 0.0045 kg/tonne to water (taken from site-specific data), and 0.001% to soil (taken from the Technical Guidance Document). These values have been assumed for all of the processes considered here. The releases are given in **Table 3.5** together with the regional and continental releases.

There are three plants known to be using naphthalene in the production of phthalic anhydride in the EU. One plant is known to produce alkylated naphthalene solvents and one known to produce 2-naphthol. Naphthalene sulphonic acid is known to be produced at up to 17 plants within the EU. The number of plants using naphthalene in the production of dyestuffs is unknown but, given the wide range of products, is likely to be fairly large. If use of naphthalene as an intermediate were consistent throughout the EU the regional use would be about 14,000 tonnes per annum (as 10% of the total). The Technical Guidance Document gives a value for $f_{mainsource}$ of 0.25 for this tonnage (IC = 2: Chemical Industry, chemicals used in synthesis), suggesting that the largest plant in any region uses about 3,500 tonnes of naphthalene per year. Larger plants are known to exist however. The largest plant using naphthalene as an intermediate uses 20,000 tonnes of naphthalene per annum. However, the largest site for which no site-specific data on releases are available uses about 12,000 tonnes of naphthalene per annum. This site has therefore been used as the basis for a generic calculation for releases on the local level.

In estimating releases from naphthalene use in the chemical industry at the regional level a regional production of 20,000 tonnes per annum has been assumed. The continental tonnage is therefore 117,000 tonnes per annum. Regional and continental releases have been averaged over 365 days. Local, regional and continental releases are summarised in **Table 3.5**.

Naphthalene may also be present in waste streams from washing and purification processes in the production of 1-nitronaphthalene (US EPA, 1977). Wastewater from the production of chloronaphthalenes and bromonaphthalenes may contain naphthalene (US EPA, 1977). The air vent streams from production of Tetralin and Decalin may contain naphthalene as may the air vent and overhead gases from the concentrator in 1-naphthalenesulphonic acid production (US EPA, 1977). Emission factors for naphthalene for these processes and the amount of naphthalene used in each process have not been found, but they are considered to be covered by the estimates above.

Table 3.5   Release of naphthalene during use as an intermediate

Environment	Emission factor	Volume produced (t/a)	Release (kg/day)
Local (generic)	air 0.1 kg/tonne water 0.0045 kg/tonne	12,000	air 4.0 water 0.18
Local (worst case site-specific)			air 3.33 water 0.15
Regional	air 0.1 kg/tonne water 0.0045 kg/tonne soil 0.01%	20,000	air 5.48 water 0.25 soil 5.48
Continental	air 0.1 kg/tonne water 0.0045 kg/tonne soil 0.01%	117,000	air 32.1 water 1.44 soil 32.1

3.1.1.3   Release from use in pyrotechnics

There are a number of companies in the EU using naphthalene in the production of pyrotechnics; four are in the UK, two in Germany, and one each in France and Italy. One UK site uses between 1 and 1.5 tonnes of naphthalene per annum as a pyrotechnic. This is believed to account for at least 25% of the UK market. Some of this is used as a component in the formulation of other products. Much of it, however, is used neat to produce black smokes. All processes are water free. No other site-specific information is available concerning the volumes produced or emissions.

It has been assumed that about 15 tonnes/annum of naphthalene is used in the EU (based on ~10 plants using about 1.5 tonnes per annum) and that the largest plant uses 2 tonnes/annum with up to 2 plants within one region. It has also been assumed, as a worst case, that naphthalene is used solely as a component in the formulation of other products.

The default values for emission factors to air, water and soil given in the Technical Guidance Document for a chemical in Industry Category 0 (other) with a vapour pressure of 10.5 Pa and water solubility of 30 mg/l in main category Ic for formulation are 0.005, 0.02 and 0.0001, respectively. These default values have been used for the purposes of the assessment to represent a worst-case scenario. However, the formulation process is reported to be water-free and the default value for release to water from the use of naphthalene in pyrotechnics is therefore likely to be higher than in practice. Local, regional and continental emissions are given in **Table 3.6**.

Releases during the use of pyrotechnics have been estimated by treating this as a processing stage. The emission factors to air, water and soil given in the Technical Guidance Document are 0.01, 0.01 and 0.005, respectively. Use has been assumed to be consistent throughout the EU. Regional and continental emissions are given in **Table 3.7**.

Table 3.6   Releases of naphthalene during the formulation stage for pyrotechnics

	Tonnage	Emission factors		Releases (kg/day)	
Local	2 tonnes/annum	air water	0.005 0.02	air water	0.033 0.13
Regional	4 tonnes/annum	air water soil	0.005 0.02 0.0001	air water soil	0.055 0.22 0.0011
Continental	11 tonnes/annum	air water soil	0.005 0.02 0.0001	air water soil	0.151 0.603 0.003

Table 3.7   Releases of naphthalene during use for pyrotechnics

	Tonnage	Emission factors		Releases (mg/day)	
Regional	1.5 tonnes/annum	air water soil	0.01 0.01 0.005	air water soil	0.041 0.041 0.021
Continental	13.5 tonnes/annum	air water soil	0.01 0.01 0.005	air water soil	0.37 0.37 0.185

3.1.1.4   Release from mothballs

Naphthalene is still used in the manufacture of mothballs. In the EU this is predominantly carried out by a company located in Belgium, which distributes world-wide. This may be to companies who re-package the mothballs for re-distribution. About 1,000 tonnes/annum are used for the manufacture of mothballs throughout the EU.

Specific data on releases of naphthalene to air and water arising from the manufacture of mothballs are not available. Releases of naphthalene during the manufacture of mothballs can be estimated using the default values given in the Technical Guidance Document (Industry Category = Personal/domestic). For a formulation stage using dedicated equipment with infrequent cleaning these are 0.001 to air, 0.003 to water and 0.0001 to soil. Based on these values a plant using 1,000 tonnes of naphthalene over 300 days a year to produce mothballs would release 3.3 kg/day to air and 10 kg/day to wastewater. However, information from the manufacturer indicates that the process is dry with no contact with water at any stage and no releases to wastewater. Naphthalene powder is compressed in a 'tabletting' machine. The tablets are stored in a stainless steel bin and then packed into 25 kg cartons. The process is fully automated using dedicated equipment. A plant using 1,000 tonnes of naphthalene over 300 days a year to produce mothballs would therefore release 3.3 kg/day to air and 0 kg/day to wastewater.

Naphthalene is also released during the use of mothballs. Release of this kind is really from multi point sources as only a small amount is likely to be used in many places. However, it is likely that all naphthalene used in mothballs will be released to the air during use. Therefore, releases have been estimated assuming that all mothballs produced in Europe are used in Europe and all the naphthalene is released during use. Regional and continental releases are given in **Table 3.8**.

Table 3.8   Release of naphthalene to air from the use of mothballs

Environment	Emission factor	Volume used (t/a)	Release (kg/day)
Regional	100%	100	273.9
Continental	100%	900	2,466

Release of naphthalene to water and soil from mothballs is not likely to be significant although release of naphthalene to water may occur during washing of clothes that have been stored in mothballs.

3.1.1.5   Releases from the manufacture of grinding wheels

Naphthalene is used as an artificial pore former in the manufacture of grinding wheels to give a high porosity product. At least three companies in the EU use naphthalene in the manufacture of grinding wheels. A total of 350 tonnes/annum are used at these plants. Naphthalene may be used in the manufacture of grinding wheels in other EU countries, although para-dichlorobenzene is also used. However, information is not currently available regarding the total number of plants, the processes involved or the consequent releases to the environment.

Site-specific data are available for two of the three plants. This information does not permit daily releases or emission factors to be determined accurately. For one of the plants (Site A) the

concentration in wastewater is less than 1 µg/l. The total organic carbon released to air at this plant is 1-2 ppm. Emissions from the second plant (Site B) are appreciably higher, however. The following values have been estimated as a worst-case release scenario for this site and have been used in the assessment:

Total emission to water	16.9 kg/day
Total emission to air	6.7 kg/day.

Note: data for emissions to sewers indicated that the naphthalene content of wastewater exceeded the solubility limit.

The worst-case plant emissions are also used as the regional emissions.

3.1.1.6   Indirect releases

Naphthalene is also released to the environment from a number of materials and sources which are not part of the life cycle of naphthalene as a substance. This section considers the possible releases from these and estimates emissions on the regional and continental scales for use in modelling the background concentrations. These estimates also provide a context for the emissions from the naphthalene industry proper.

3.1.1.6.1   Releases from products containing naphthalene

Release from wood-treating processes and treated wood products

In the EU, there are 9 tar distillers who use tar distillate to blend creosote, one company who use coal tar produced from low temperature coking (which does not produce coal tar containing naphthalene) to blend creosote and a number of smaller companies who purchase distillates to blend creosote.

Approximately 40,000 tonnes of creosote was produced in the UK in 1986 (Consultants in Environmental Sciences Ltd., 1988). A quarter of this was exported, a quarter was used in industrial pressure treatment plants and the rest was divided between immersion treatment processes and retail sale for domestic use. It is estimated that about 100,000 tonnes/annum of creosote are used in the EU and it is thought that about 10% of this represents the level of naphthalene.

Release from the use of creosote

Bulk impregnators of timber use about 60% of all creosote, with the remaining 40% being packaged for brush applications. It is estimated that 50% of creosote used for brush applications does not contain naphthalene. There are understood to be four bulk impregnation plants in the UK and a further five elsewhere in the EU.

Various emission factors for naphthalene have been estimated for wood-preserving and log-treating processes using creosote and creosote and pentachlorophenol together (US EPA, 1988) and are shown in **Table 3.9**. A local release can be calculated from the figures for log-treating. The total emission factor for log treating is 3.08 kg/hour which gives a release of 24.7 kg/day assuming operation for 8 hours per day.

Naphthalene concentrations have been measured at a plant that carries out bulk impregnation of timber with creosote (Industry communication, 1995). The naphthalene concentrations at various locations on the site were all less than 1 mg/m^3.

It is not possible to calculate a regional or continental release using the available site-specific information. However, regional and continental emissions can be estimated using the default values given in the Technical Guidance Document. Emission factors in the Technical Guidance Document (Industry Category = Paints, laquers and varnish industry, Use category = 39) for a processing stage for a solvent-based material are 0.001 to air, 0.005 to water and 0.005 to soil. It has been assumed that 60,000 tonnes of creosote (containing 6,000 tonnes of naphthalene) are used in the EU for the bulk impregnation of timber. Regional and continental releases are given in **Table 3.10**.

Table 3.9 Emission factors for naphthalene for wood-preserving and log-treating processes   (US EPA, 1988)

Emission Source	Emission factors for naphthalene
WOOD PRESERVING Thermal evaporation of pentachlorophenol wastewater Thermal evaporation of creosote wastewater Incineration of waste Pentachlorophenol treating cylinder fugitives Creosote treating cylinder fugitives LOG TREATING Creosote conditioning Creosote cool-down Pentachlorophenol conditioning Pentachlorophenol cool-down Pentachlorophenol treating fugitives	 2,000 mg/sm^3 gas vented 2,500 mg/sm^3 gas vented 290 mg/sm^3 stack gas 2.6 mg/sm^3 gas leaked 2.8 mg/sm^3 gas leaked 2.2 lb/hour 1.06 lb/hour 2.29 lb/hour 0.99 lb/hour 0.24 lb/hour

Note: sm^3 refers to volumes at standard temperature and pressure.

Table 3.10 Regional and continental releases of naphthalene during bulk impregnation of timber

	Naphthalene in creosote	Emission factors		Releases (kg/day)	
Regional	600 tonnes/annum	air water soil	0.001 0.005 0.005	air water soil	1.64 8.22 8.22
Continental	5400 tonnes/annum	air water soil	0.001 0.005 0.005	air water soil	14.8 74.0 74.0

Releases during domestic use of creosote

The releases of naphthalene during domestic use have been estimated using the emission factors given in the Technical Guidance Document. The default values for emission factors to air, water and soil given in the Technical Guidance Document for Industry Category 14 (Paints, lacquers and varnish industry, use category 39) with a vapour pressure of 10.5 Pa for private use are 0.001, 0.005 and 0.005, respectively. Regional and continental emissions are given in **Table 3.11**. In this assessment it has been assumed that 20,000 tonnes/annum of creosote containing 10% naphthalene is used in domestic applications in the EU.

Table 3.11 Releases of naphthalene during private use of creosote

	Naphthalene in creosote	Emission factors		Releases (kg/day)	
Regional	200 tonnes/annum	air water soil	0.001 0.005 0.005	air water soil	0.548 2.74 2.74
Continental	1800 tonnes/annum	air water soil	0.001 0.005 0.005	air water soil	4.93 24.7 24.7

Release from treated wood products

Release of naphthalene to air from treated products is likely to occur. It has been estimated (Petrowitz and Becker, 1964) that less than 2% of naphthalene applied in creosote is released each year from treated railway sleepers.

The migration of creosote and its components from treated piling sections in a marine environment was studied by Ingram et al. (1982). The amount of creosote released from aged and newly treated pilings was determined in both seawater and distilled water. The amount of polycyclic aromatic hydrocarbons that migrate from creosote-treated pilings into water was estimated to be approximately 77-147 g annually for a piling with a surface area of 15 m^2, of which naphthalene accounted for between 16.4 and 22.8%. The release of naphthalene from treated wood cannot be quantified as no information on the amount of wood treated in the EU by these processes has been found.

The amount of naphthalene released to air in the Netherlands is reported to be approximately 380 tonnes per annum. This corresponds to a release of about 1,040 kg/day. If this is treated as a regional emission and is typical of the EU as a whole then a continental emission of 10,400 kg/day can be estimated.

No emission factors are available for release of naphthalene from wood treatment and treated wood products to soil.

Elevated levels of naphthalene have been found in soil and groundwater at sites of disused wood treatment plants and in the atmosphere near wood treatment plants (see Sections 3.1.3.2, 3.1.4.2 and 3.1.5.2) so release from this source is likely.

Paints, waterproof membranes and bituminous materials

Naphthalene may be released to the atmosphere from tar and bitumen processes (HMIP, 1995). These processes include the distillation of tar or bitumen and the heating of tar or bitumen for the manufacture of electrodes or carbon-based refractory materials. Naphthalene may be released during storage, atmospheric distillation, vacuum distillation or heat treatment of pitch. However, no emission factors have been found for naphthalene for these processes.

Bituminous material containing coal tar which is commonly used for damp proofing floors in the UK was found to be a source of naphthalene (Brown et al., 1990). The damp proof membrane is a cold applied bituminous emulsion containing natural rubber latex and coal tar. Tar containing naphthalene is also used in some specialist paints. About 600,000 litres of waterproof membrane are reported to be used each year in the UK. The membranes contain about 1% naphthalene and coal tar paints contain about 1-2%, coal tar epoxy paints contain less than 0.1% and coal tar polyurethane sealers less than 1%. These paints and membranes are generally used by the

building trade. Naphthalene concentrations up to 0.97 µg/l (mg/m³) were found in homes having an objectionable smell, where the damp proof membrane had been applied, compared with < 0.3 µg/l (mg/m³) for control homes.

In this assessment it has been assumed that the UK use is representative of the EU as a whole and that, assuming that the UK population is about 55 million the regional and continental uses of naphthalene-containing tars and paints are 218,000 litres/annum and 2,180,000 litres/annum, respectively. If the naphthalene content is assumed to be 1% the amounts of naphthalene present in tars and paints on the regional and continental scales are 2.6 and 26 tonnes, respectively, assuming that the density of naphthalene is 1.175.

The releases of naphthalene during use of these products have been estimated using the emission factors given in the Technical Guidance Document. The default values for emission factors to air, water and soil given in the Technical Guidance Document for Industry Category 14 (Paints, lacquers and varnish industry) with a vapour pressure of 10.5 Pa for private use are 0.001, 0.005 and 0.005, respectively. Regional and continental emissions are given in **Table 3.12**. It has been assumed that naphthalene is present as a constituent of the tar used in the paints and is not added as a separate component. The releases to the environment from the use of these paints are therefore indirect and have not therefore been considered at the local level.

Table 3.12 Releases of naphthalene during private use for paints and waterproof membranes

	Naphthalene in paint	Emission factors		Releases (kg/day)	
Regional	2.6 tonnes/annum	air water soil	0.001 0.005 0.001	air water soil	0.0071 0.036 0.0071
Continental	26 tonnes/annum	air water soil	0.001 0.005 0.001	air water soil	0.060 0.356 0.071

3.1.1.6.2 Releases from production of other substances

Phenol production

Naphthalene is emitted during the production of phenol from cumene. Emissions from the cumene peroxidation vent account for approximately 51% of the mass of emissions from this process. All plants in the US use some form of emission control with an average efficiency of 81% but it is not known whether these controls are used in Europe. An emission factor of <0.0001 g/kg phenol produced was given for the outlet from the control device with an emission factor at the inlet to the control device of <0.0005 g/kg phenol produced (US EPA, 1979a). Another estimate for the emission factor of naphthalene during phenol production is similar at 0.0001 g/kg phenol produced (US EPA, 1988). The production of phenol in the EU is between 762,000 and 2,360,000 tonnes per annum. 93% of this phenol is produced from cumene (IUCLID). Western European consumption of phenol is 1,240,000 tonnes per annum (ECN, 1992).

The release of naphthalene from production of phenol has been estimated assuming that the same control systems are used as in the US. The releases to the environment from the production of phenol are indirect and have not therefore been considered at the local level. If the total annual production of phenol from cumene is taken to be 2,195,000 tonnes the regional and continental

releases to air, averaged over 365 days per year, are 0.060 and 0.541 kg/day , respectively. The production of phenol is a wet process and release of naphthalene to water is therefore possible. However, no information is available regarding release of naphthalene to water and soil during production of phenol.

Propylene oxide production

Propylene oxide production by the chlorohydrin process has an emission factor to air for naphthalene of 0.08 g/kg produced (US EPA, 1988). The amount of propylene oxide produced in Europe by the chlorohydrin process has been estimated to be 766,000 t/a (Propylene oxide RAR). Averaged over 365 days per year, the regional and continental releases are 16.8 kg/day and 151 kg/day, respectively.

Production of aluminium

Polynuclear aromatic hydrocarbons, including naphthalene, can be released from the production of aluminium. They evaporate from the electrode paste, which consists of coal tar pitch, and coke (the Søderberg electrode). No site-specific information on releases of naphthalene from aluminium production plants in Europe has been found. However, emission factors of 27.5 g/tonne aluminium produced and 63.2 g/tonne aluminium produced have been calculated for the Søderberg potline for the primary aluminium production process (US EPA, 1988).

Production of aluminium by the primary production process was approximately 244,200 tonnes/annum in the UK (CSO, 1994). Total European aluminium production in 1991 was approximately 4.4 million tonnes (UN, 1995). The production capacity of one plant in the UK is 40,000 tonnes per annum (Lane, 1992) but it is not known if this is typical of aluminium production plants in Europe. The release of naphthalene from the production of aluminium is indirect and has not been estimated on the local scale. A regional release for the UK can be estimated using an emission factor of 63.2 g/tonne aluminium produced and is 51.44 kg/day. The regional and continental releases (averaged over 365 days per year) for Europe can be calculated by assuming production figures of 0.44 million tonnes/year and 4.4 million tonnes/year, respectively. The same emission factor can be used to estimate daily regional releases of 76.2 kg/day and continental releases of 686 kg/day. Elevated levels of naphthalene have been measured in the vicinity of aluminium production sites in Sweden and Norway (see Section 3.1.5.2). No information is available regarding release of naphthalene to water and soil during aluminium production.

3.1.1.6.3   Release during waste incineration

Particulate emissions from incinerators have been found to contain naphthalene (Williams et al., 1994). Stack particles from a municipal waste incinerator burning municipal and up to 5% medical waste contained naphthalene at a level equivalent to 12.3 ng/kg fuel burned. Naphthalene has been found in samples of flue gas from a hazardous waste incinerator (Wienecke et al., 1995). Samples taken from the second combustion line of an incinerator in Germany in 1992 contained 53.24 and 30.91 ng/m^3 of naphthalene.

Incinerators in the UK operate with a throughput of 6-10 tonnes per hour (Rae, 1994). A local release of naphthalene to air has been estimated based on an emission factor of 12.3 ng/kg fuel burned and a throughput of 10 tonnes fuel burned per hour and operation for 24 hours per day, 300 days per year. This gives a local release of 0.003 g/day. No information is available about

the amount of waste incinerated in Europe but this is not likely to be a significant source of naphthalene. Release of naphthalene to water from waste incineration is not likely to occur. Naphthalene may be released to soil from disposal of ash from waste incineration but no information is available to quantify this release.

3.1.1.6.4 Release from oil production

Hydrocarbons may be released to the environment from a variety of petroleum processes (HMIP, 1992a). Naphthalene is a component of formation water which is discharged from offshore production operations (Sauer, 1981a). Formation water is produced with oil and gas and on average 0.6 litre of formation water is produced per litre of oil. Formation water samples were taken from a producing platform in the Gulf of Mexico and were found to contain 170 µg/l of naphthalene. In 1994, 114,383,000 tonnes of crude oil were produced offshore by the UK (DTi, 1995). Assuming that 0.6 litres of formation water is produced per litre of oil (and 1kg of oil is equivalent to 1litre), $6.86 \cdot 10^{10}$ litres of formation water were produced which gives a release of 11.67 tonnes of naphthalene per year or 31.96 kg/day (assuming operation for 365 days per year). No more information has been found to allow calculation of a local release. Regional and continental releases have been estimated by assuming that 50% of offshore production in the EU occurs within the UK. A regional release of 11.6 kg/day, assuming the UK population is 55 million, and a continental release of 52.3 kg/day have therefore been derived.

Naphthalene is emitted from oil industries and refineries (Bouscaren et al., 1986). Fugitives from petroleum refineries have an emission factor for naphthalene of 0.33 lb/hour (US EPA, 1988). This figure gives a local release of 3.6 kg/day based on operation for 24 hours per day. It is not possible to estimate regional and continental releases or releases to water and soil from the available information.

3.1.1.6.5 Release of naphthalene from traffic

Naphthalene is a component of fuels and oils. It is emitted from traffic/gasoline/lubricants (Bouscaren et al., 1986). Williams et al. (1986) analysed samples of diesel fuel from five places in the UK, together with gas oil and kerosene samples. The mean naphthalene content of the diesel samples was 220 ppm (mg/l), with 300 ppm (mg/l) in the gas oil and 1,500 ppm (mg/l) in the kerosene.

Tancell et al. (1995) found 300 ppm naphthalene in commercially available diesel fuel. However, they also added ^{14}C-labelled naphthalene to diesel fuel that was combusted in a 2 litre direct injection diesel engine and found that only 0.47% of the naphthalene added survived combustion.

Coleman et al. (1984) measured the composition of gasoline, kerosene and No. 2 fuel oil and that of the water-soluble fraction of each of the fuels. The naphthalene contents in the fuel and in the water soluble fraction respectively were 1.0% and 6.0% in unleaded gasoline, 5.7% and 44.8% in kerosene and 32.4% and 79.7% in No. 2 fuel oil. However naphthalene was not included in a list of detectable hydrocarbons found in US finished gasolines at a concentration of 1% by weight or more (WHO, 1989).

Road traffic

Emission rates of 22 gas-phase hydrocarbons, including naphthalene, from motor vehicles in highway operation were determined in a mountain tunnel of the Pennsylvania Turnpike (Hampton et al., 1983). Airborne concentrations of naphthalene in the intake air and at stations just inside the exit of the tunnel were determined and the tunnel airflow was measured. Traffic composition was determined, and in 1979, heavy-duty diesel trucks and light duty spark ignition vehicles accounted for 96.8% of all vehicles using the tunnel. Emission rates for these types of vehicles were estimated with and without filtering to evaluate the contribution of condensed-phase material captured in the sampling process and are given in **Table 3.13**. These emission rates would be appropriate for highway driving conditions at approximately 80 km/hour on a straight, nearly level highway.

Table 3.13 Emission rate of naphthalene from diesel trucks and light duty gasoline powered vehicles   (Hampton et al. 1983)

	Gasoline vehicles mg/km	Diesel trucks mg/km
Filtered	8.6 ± 0.2	7.4 ± 0.7
Not filtered	8.1 ± 0.9	9.2 ± 1.7

Larssen (1984) measured polyaromatic hydrocarbon emission factors from traffic in Oslo by an indirect method. Two sites were monitored, one exposed to traffic and one exposed to a similar background but shielded from traffic emissions. Carbon monoxide was used as a reference pollutant to allow conversion of pollutant concentrations to emission factors. The site chosen had average daily traffic of approximately 13,000 cars, with speeds varying between 15 km/h and 35 km/h (24- hour weighted average 30 km/h). Light duty gasoline powered cars accounted for 95% of the vehicles, with 3% light duty diesels and 2% diesel trucks. The fraction of cars in the warming up phase was estimated as 15%. Much lower emission factors for naphthalene were obtained than in the US study above, with values of 95 μg/km in winter and 90 μg/km in summer. The author commented that lighter compounds were not thought to be fully retained on the sampling system and so the real emission factors are likely to be higher.

Williams et al. (1986) measured average emissions from diesel vehicles of 0.2 mg/kg fuel burned (averaged over low, mid and high load).

Nelson (1989) measured the naphthalene in diesel fuel and in diesel exhaust emissions. Exhaust samples were collected from a 3-litre indirect injection engine diesel truck driven on a chassis dynamometer at 40 km/h. (This test did not represent the urban driving cycle and the absolute magnitude of the emissions may not be representative of overall conditions.) Naphthalene was not detected in the fuel but 620 μg of naphthalene per gram of fuel burnt was detected in the exhaust emission.

Zielinska and Fung (1994) collected air samples from the Caldecott road tunnel in San Fransisco. The average daily traffic was estimated in 1981 to be approximately 110,000 vehicles per day. The range of naphthalene concentrations found during 6 two-hour sampling periods over two days was 9.42-28.55 ppbC (~ 8.4-25.3 μg/m^3).

RIVM data give an emission factor of 2.5 mg/km for all vehicle types.

For this assessment, the emission factors calculated by Hampton et al. for filtered samples of 8.6 mg/km for gasoline vehicles and 7.4 mg/km for diesel trucks will be used as a worst case.

The mileage for automobiles running on gasoline has been estimated to be $336.8 \cdot 10^9$ km/year in the UK (Transport Statistics for Great Britain, 1994) which would give a release of naphthalene based on an emission factor of 8.6 mg/km of 2,896 tonnes/year (7,936 kg/day). Total mileage for diesel vehicles (including buses, light vans and goods vehicles) in the UK was estimated to be $69.3 \cdot 10^9$ km/year (Transport Statistics for Great Britain, 1994). This figure gives a naphthalene release based on an emission factor of 7.4 mg/km of 513 tonnes/year (1,405 kg/day).

Mileage figures for Europe are not known but the total number of cars and taxis in Europe is 155,144,000 (Transport Statistics for Great Britain, 1994) and the average annual mileage for cars is 19,300 km (Draft Use Category Document on Lubricants and Lubricant Additives, 1996). Assuming all these vehicles run on gasoline, the release of naphthalene to air can be calculated using the emission factor of 8.6 mg/km. The regional and continental releases are shown in **Table 3.14**.

Table 3.14 Release of naphthalene to air from gasoline powered vehicles

Environment	Emission factor	Mileage (km)	Release (kg/day)
Regional	8.6 mg/km	$2.99 \cdot 10^{11}$	7,045
Continental	"	$2.69 \cdot 10^{12}$	63,404

The combined number of goods vehicles and buses in Europe is 17,611,500 (Transport Statistics for Great Britain, 1994) and the average annual mileage for goods vehicles is 96,600 km (Draft Use Category Document - Lubricants and Lubricant Additives, 1996). Assuming the mileage is the same for buses and goods vehicles and that all these vehicles run on diesel, the emission factor of 7.4 mg/km can be used to estimate the release of naphthalene to air. The regional and continental releases are shown in **Table 3.15**.

Table 3.15 Release of naphthalene to air from diesel powered vehicles

Environment	Emission factor	Mileage (km)	Release (kg/day)
Regional	7.4 mg/km	$1.7 \cdot 10^{11}$	3,447
Continental	"	$1.53 \cdot 10^{12}$	31,020

The regional release calculated for the UK (population 55 million) is slightly larger than for a typical European region (population 20 million) for gasoline-powered vehicles and approximately half those for a typical region for diesel powered vehicles. This discrepancy is thought to be due to the different methods of calculation used for the UK and the European region. The regional release has been used in the assessment.

The releases from gasoline powered vehicles are likely to be overestimates because a significant proportion of cars run on diesel. However, these figures represent a worst-case situation. New technologies, such as lean burn engines and three-way catalytic converters, are being used to reduce emissions from vehicles. Eggleston (1987) has estimated that future emissions of VOCs from petrol engine vehicle exhausts will decrease from the 1980 values by about 12% and 15% in 2000 and 2010, respectively. However, no emission factors specifically for vehicles using

these technologies have been found. Therefore, these release estimates will be used as representing worst-case conditions for vehicles without catalytic converters or lean burn engines.

No information is available concerning release of naphthalene to water and soil from vehicle exhaust emissions. Direct release to water and soil is not likely but the naphthalene released to the atmosphere will undergo deposition to soil and water. Elevated levels of naphthalene have been found in soils near roads and in roadside dusts (see Section 3.1.5.2).

Emission factors for other forms of transport have not been found.

Motor boats

Increased naphthalene levels have been found in surface water samples where there is a significant amount of motor boat traffic (Jüttner, 1988) and in surface water samples in harbours (Ehrhardt and Burns, 1990). Jüttner (1994) found that 13 mg of naphthalene was introduced into an 800 l tank of water during 10 minutes of operation of a four-stoke outboard motor. A similar experiment using a two-stroke engine found that 1,400 mg of naphthalene was introduced into an 800 l tank during 10 minutes operation (Jüttner et al., 1995). When a catalyst was used with the two-stroke engine, the naphthalene released in the first 2.5 minutes of operation decreased from 120 mg to 55 mg and in the first ten minutes from 460 mg to 63 mg (Jüttner et al., 1995). Naphthalene has been found in the water-soluble fractions of both new and used motor oils (Chen et al., 1994). No information on the volume of motor boat traffic in Europe is available so it is not possible to quantify release of naphthalene to surface water from this source.

3.1.1.6.6   Release of naphthalene from coal combustion

A four-year study of samples of stack gas and fly, grate and stack ashes from the combustion of coal and mixtures of coal and refuse-derived fuel was carried out (Junk et al., 1986). The amounts of naphthalene in the vapour phase varied according to firing conditions and stack temperature. Naphthalene in respirable particles ranged from below the detection limit (0.05 ng/g) to 18 ng/g and in non-respirable particles from 0.5 to 23 ng/g. The concentration range of naphthalene in the vapour phase was between 10 and 1,800 ng/m^3.

Emissions of naphthalene from combustion of pulverised coal in a small-scale combustor were measured in a study conducted by the US EPA's Air and Energy Engineering Research Laboratory (Miller, 1994). The combustor was operated under different conditions to simulate baseline, high excess air firing and nitrogen oxide controls by combustion modification. Measurements were taken at the exit of the primary combustion zone and emission factors were calculated. Calculated emission factors range from $2.371 \cdot 10^{-8}$ lb/10^6 BTU to $1.364 \cdot 10^{-7}$ lb/10^6 BTU. Although these factors were calculated from a small-scale combustor they can be used to estimate naphthalene release during coal combustion. In the UK in 1994, 81.72 million tonnes of coal were combusted (CSO, 1994). US coals typically have heating values between 8,000 and 15,000 BTU/lb (Khan et al., 1992). Assuming a heating value of 10,000 BTU/lb for coal used in the UK, the total heating value of all coal combusted in one year is $2.023 \cdot 10^{15}$ BTU. This would give release estimates for the UK under best and worst-case conditions of 19.2 kg/year (0.053 kg/day) and 110.4 kg/year (0.302 kg/day), respectively.

No information has been found regarding the amount of coal combusted in Europe. However, figures for the production of coal in Europe are available. European production of hard coal, lignite, peat, briquettes and cokes was 874.7 million tonnes in 1993 (UN, 1995). Continental and regional releases have been calculated using this figure assuming that the levels of imports and

exports are equivalent and all coal produced in Europe is combusted here. Khan et al. (1992) have described the processes of coal combustion in the US and provided figures for the amount of coal combusted in these processes. Therefore, assuming the same processes are used in Europe, local, regional and continental releases can be calculated. In the US, 78-80% of coal is used in power stations for commercial electricity production. These stations burn between 1,000 and 10,000 tonnes coal per day using the pulverised fuel process. For the local scenario it has been assumed that 10,000 tonnes of coal a day are combusted with a heating value of 10,000 BTU/lb. The total heating value for the coal is therefore $2.2 \cdot 10^{11}$ BTU. Using an emission factor of $1.364 \cdot 10^{-7}$ lb/10^6 BTU the local release is 0.014 kg/day. Khan et al. (1992) also report that up to 100 tonnes per day is used in plants which burn coal for industrial use and up to 0.05 tonnes per day is burned in domestic and commercial situations for space heating. Local releases calculated for these two processes are 0.14 g/day for burning of coal for industrial use and 0.07 mg/day for burning of coal for domestic or commercial use. For the regional and continental scenarios the releases are 0.327 kg/day and 2.94 kg/day, respectively (assuming that burning of coal takes place continuously 365 days per year).

No information is available concerning release of naphthalene to water and soil as a result of coal combustion, but the major emissions are considered likely to be to air.

3.1.1.6.7   Release of naphthalene from coal carbonisation and gasification processes

Naphthalene may be formed from a wide variety of coal carbonisation and gasification processes. Both types of process involve heating coal to high temperatures in order to produce other products. Carbonisation processes are used to convert coal to coke, tars, chemicals and industrial gases whereas gasification processes are designed to produce a gaseous product for use in heating and chemical production. During coal carbonisation, several categories of by-products are produced including liquid effluent containing naphthalene (Richards et al., 1993). Details of several processes are given below. Although no emission factors have been found for naphthalene from most of these processes, they all involve heating coal at high temperatures and so there is a potential for naphthalene to be formed.

<u>Coke production</u>

1992 coke production for the EU was estimated to be 52,350,000 tonnes per year (EC, 1996b) at 52 coke oven plants. Germany has the most coke oven plants followed by the UK, Italy and France. However, it was planned to take one plant in France, two plants in Germany and one plant in the UK out of operation in the near future.

There are several process steps involved in coke making (EC, 1996b). First, the charge is prepared by blending coals with different properties and by pulverisation. The coking blend is then charged into the hot coke oven where it is carbonised. Generation of steam, organic vapours and gas starts immediately after charging and they are exhausted via ascension pipes into a crude gas collecting main. Depending on the oven width and heating conditions the process is finished after 14-24 hours. The oven doors are then opened and the coke is pushed into a quenching car, moved to a quenching tower and cooled by water. Up to 500 l water per tonne of coke is vaporised and is clarified by sedimentation and fed back to the quenching tower. The coke produced is then screened and crushed. Approximately 70-75% of the charged coal is yielded as coke. During carbonisation, 300-400 m^3 of gas per tonne of coal is generated as well as 2-4.5% tar and 0.5-1.5% benzol (mainly benzene, toluene and xylenes). The crude coke oven gas is cooled by ammonia-liquor spray to approximately 80°C. The liquid phase is directed to tar/water

separators and the gas is cooled in primary coolers to $< 20^{o}C$ where most higher boiling compounds and water condense. In modern gas treatment plants hydrogen sulphide, ammonia, BTX (benzene, toluene, xylenes) and in some cases naphthalene are recovered. Gas washing and by-product recovery is done by absorption of chemical reaction. Wastewater from benzol and naphthalene recovery is led to a tar/water separator.

A survey of 52 coke oven plants in the EU was carried out and replies were received from 25 (EC, 1996b). At 19 of the plants wastewater is treated in a biological treatment plant and at 6 plants chemical/physical treatment is used. For both treatment methods the PAH levels after treatment range from 0.003 to 0.2 mg/l.

In the UK, coke production follows these stages (HMIP, 1992b). Carbonisation is carried out at temperatures of $1,250\text{-}1,350^{o}C$ and can take up to 14 hours. The gas evolved is cooled to approximately $80^{o}C$ which causes most of the tar to condense and then passes to a primary cooler which reduces the temperature to approximately $30^{o}C$ causing water vapour and further tar to condense. The liquors pass to settling tanks from which the clarified liquor overflows. Excess liquor ("virgin liquor") is formed during the process and this is treated before discharge. Tar is recovered for sale. Further treatment of the gas includes processes to remove and recover impurities. Naphthalene can be recovered by scrubbing with a suitable solvent, which can then be treated to regenerate it. Naphthalene vapours can be scrubbed from the gas using wash oils of coal or petroleum origin and recovered by steam stripping. Water used for coke quenching is recycled after removal of solid particles. Wastewater resulting from collection of gases is usually treated before being discharged (e.g. activated sludge plant, activated carbon filters, sand bed filtration or reed bed).

An emission factor has been estimated for a coke by-products plant based on the capacity and the percentage weight of naphthalene to be 0.00592 kg per tonne of coke (US EPA, 1988). In the UK in 1994, 8,595,000 tonnes of coal were used to produce 6,164,000 tonnes of coke. Using the emission factor of 0.00592 kg per tonne of coke produced and assuming production is carried out for 300 days per year gives a regional release for the UK of 99.9 kg/day (averaged over 365 days). Assuming that coke production is roughly spread between coke oven plants a local production volume of approximately 1,000,000 tonnes per year can be used to estimate a local release of naphthalene. Using the emission factor of 0.00592 kg/tonne of coke produced and assuming production is carried out for 300 days per year gives a local release of 19.7 kg/day. A regional release of 84.9 kg/day and continental release of 764 kg/day have also been calculated assuming a total production of 52,350,000 tonnes per year and 10% of total production in each region.

Coal gasification

Gasification involves the reaction of a carbon source such as coal with a source of hydrogen and/or oxygen to yield a gas rich in methane, carbon monoxide, carbon dioxide and hydrogen in proportions dependent on the ratio of the reactants utilised and the reaction conditions. The feedstock is usually coal, lignite or liquid hydrocarbons. There are three main processes used: catalytic gasification with steam, thermal hydrogenation and gasification with oxygen and/or steam (HMIP, 1992c).

No emission factors are available for naphthalene from these processes but high levels of naphthalene have been found in groundwater near gasification sites in the USA (see Section 3.1.3.2.2).

Currently there are no commercially operated gasification units in the UK. However, no information has been found regarding gasification processes in the rest of Europe.

3.1.1.7 Other releases

Naphthalene is one of a complicated mixture of compounds produced during the combustion of propane (Eklund et al., 1987). Naphthalene is produced when propane is burned in an atmosphere deficient in oxygen in the presence and absence of hydrochloric acid.

Naphthalene is released from burning of different fuels in small stoves (Nielsen et al., 1992). An emission factor for release of naphthalene from uncontrolled waste oil fired burners (<150 million BTU/hour) or space heaters was 0.018 g/litre of oil burned (US EPA, 1988). In 1985 1.87 million tonnes of burning oil were used in the UK mainly for domestic use (Eggleston and McInnes, 1987). Assuming the density of oil is approximately the same as water, $1.87 \cdot 10^9$ litres of oil per year are burned. Therefore 33,660 kg/year of naphthalene (92.2 kg/day) is released to air in the UK from combustion of oil. No information is available on the amount of oil burned as fuel in Europe so releases from this source could not be quantified. However, regional and continental releases have been estimated by assuming that use is consistent throughout the EU and that the population of the UK is 55 million. Regional and continental releases are therefore approximately 33.5 kg/day and 620.3 kg/day, respectively.

Naphthalene is released to air from shale oil wastewater treatment plants (Hawthorne and Sievers, 1984). Groundwater samples from the sites of disused underground coal gasification plants have been found to contain high levels of naphthalene (Turney and Goerlitz, 1990; Mattox and Humenick, 1980; Steurmer et al., 1982) as have samples of soil (Turney and Goerlitz, 1990). Landfill leachates also contain elevated levels of naphthalene (Barker, 1987). Concentrations of naphthalene in groundwater were found to decrease with increasing distance from a petrol storage facility (Tester and Harker, 1981).

In addition to these human-related sources, naphthalene is produced by natural combustion sources, such as forest fires.

3.1.1.8 Summary of releases

Releases of naphthalene from major sources under worst-case conditions are summarised for the local, regional and continental environments in **Tables 3.16**, **3.17** and **3.18**. Releases from specific production sites have been used in the assessment to calculate PECs. The values given in the table for production on a local level summarise the worst-case releases for air and water.

By far the most significant release on the continental and regional scales is from combustion of gasoline and diesel by vehicles although this figure may be an overestimate as the influence of catalytic converters has not been considered.

Table 3.16 Local environmental releases of naphthalene   (see text for details)

Process	Release factor	Release (kg/day)
Naphthalene production site-specific releases (worst case)		air 2.08* water 4.18*
Phthalic anhydride production - site-specific (worst case)		air 3.33 water 0.15
Intermediate production	air 0.1 kg/tonne water 0.0045 kg/tonne	air 4.0 water 0.18
Pyrotechnics manufacture (worst case)	air 0.005 water 0.02	air 0.033 water 0.13
Mothballs manufacture	air 0.001 water 0	air 3.3 water 0
Grinding wheels manufacture		air 6.7 water 16.9

*   Worst case releases from naphthalene production. The releases to air and water refer to different sites. The water release is pre-wastewater treatment plant, rather than the post-wwtp value in **Table 3.2**, so that it is on the same basis as the other values.

Table 3.17 Regional environmental releases of naphthalene

Process	Type	Release factors	Release (kg/day)
Naphthalene production	Point	site-specific, soil 0.01%-70,000 tonnes/annum	air 1.71 water 1.2 soil 19.2
Use as intermediate	Point	air 0.1 kg/tonne water 0.006 kg/tonne soil 0.01%	air 5.48 water 0.25 soil 5.48
Pyrotechnics manufacture (worst case)	Point	air 0.005 water 0.02 soil 0.0001	air 0.055 water 0.22 soil 0.0011
Pyrotechnics use	Point	air 0.01 water 0.01 soil 0.005	air 0.041 water 0.041 soil 0.021
Mothballs manufacture	Point	air 0.005 water 0 soil 0.001	air 2.7 water 0 soil 0.27
Mothballs	Disperse	air 100%	air 273.9
Grinding wheels manufacture	Point		air 6.2 water 16.2
Timber impregnation	Point	air 0.001 water 0.005 soil 0.005	air 1.64 water 8.22 soil 8.22
Creosote - private use	Disperse	air 0.001 water 0.005 soil 0.005	air 0.548 water 2.74 soil 2.74
Treated wood products	Disperse		air 1040

Table 3.17 continued overleaf

Table 3.17 continued Regional environmental releases of naphthalene

Process	Type	Release factors	Release (kg/day)
Use of paints & membranes	Disperse	air 0.001 water 0.005 soil 0.001	air 0.0071 water 0.036 soil 0.0071
Phenol production (from cumene)	Point	air 0.0001 g/kg phenol produced	air 0.060
Propylene oxide production	Point	air 0.8 g/kg propylene oxide produced	air 16.8
Aluminium production	Point	air 63.2 g/tonne produced	air 76.2
Oil production	Point	water 170 µg/l formation water	water 31.96 (UK) water 11.6 (regional)
Gasoline vehicles	Disperse	air 8.6 mg/km	air 7,045
Diesel vehicles	Disperse	air 7.4 mg/km	air 3,447
Coal combustion	Point	air 1.364 x 10^{-7} lb/106 BTU	air 0.327
Coke production	Point	air 0.00592 kg/tonne coke produced	air 84.9
Oil burning	Point	air 0.018 g/l oil	air 92.2 (UK) air 33.5 (regional)
TOTAL			air 12,037 water 40.51 soil 35.94

Table 3.18 Continental environmental releases of naphthalene

Process	Type	Release factor		Release (kg/day)	
Naphthalene production	Point	air water soil	0.239 kg/tonne 0.003 0.01%	air water soil	4.27 1.03 35.6
Use as intermediate	Point	air water soil	0.1 kg/tonne 0.006 kg/tonne 0.01%	air water soil	32.1 1.44 32.1
Pyrotechnics manufacture	Point	air water soil	0.005 0.02 0.0001	air water soil	0.151 kg/day 0.603 kg/day 0.0030 kg/day
Pyrotechnics use	Point	air water soil	0.01 0.01 0.005	air water soil	0.37 kg/day 0.37 kg/day 0.185 kg/day
Mothballs	Multi-point	air	100%	air	2,466
Timber impregnation	Point	air water soil	0.001 0.005 0.005	air water soil	4.8 74.0 74.0
Creosote - private use	Disperse	air water soil	0.001 0.005 0.005	air water soil	4.93 24.7 24.7
Treated wood products	Disperse			air	10,400

Table 3.18 continued overleaf

Table 3.18 continued Continental environmental releases of naphthalene

Process	Type	Release factor		Release (kg/day)	
Use of paints & membranes	Disperse	air water soil	0.001 0.005 0.001	air water oil	0.060 kg/day 0.356 kg/day 0.071 kg/day
Phenol production (from cumene)	Point	air	0.0001 g/kg phenol produced	air	0.541
Propylene oxide production (by chlorohydrination)	Point	air	0.8 g/kg propylene oxide produced	air	151
Aluminium production	Point	air	63.2 g/tonne produced	air	686
Oil production	Point	170 µg/l formation water		water	52.3
Gasoline powered vehicles	Disperse	air	8.6 mg/km	air	63,404
Diesel powered vehicles	Disperse	air	7.4 mg/km	air	31,020
Coal combustion	Point	air	1.364×10^{-7} lb/106 BTU	air	2.94
Coke production	Point	air	0.00592 kg/tonne coke produced	air	764
Oil burning		air	0.018 g/l oil	air	620
TOTAL				air water soil	109,571 154.4 159.7

3.1.2   Environmental fate

3.1.2.1   Degradation

3.1.2.1.1   Abiotic degradation

Photolysis

Naphthalene (10 µM stock solution in acetonitrile) was added to distilled water or artificial seawater (1 l) in a reaction flask and irradiated (Fukuda et al., 1988). A high-pressure mercury lamp was used to irradiate the flask for 1-96 hours. The half-life of naphthalene in distilled water was 25 hours and in artificial seawater the rate of photolysis was found to increase several times over.

Zepp and Schlotzhauer (1979) studied the direct photolysis of naphthalene in pure water. They calculated a near surface half-life of 71 hours, corresponding to midsummer sunlight at midday, latitude 40°N. They also calculated a half-life of 550 hours for a 5-metre deep-water body with 20 mg/l sediment - the effect of partitioning to the sediment on the half-life was considered negligible.

Photolysis rates of naphthalene in three different surface water-sediment systems followed first order kinetics (Saleh et al., 1984). Half lives for naphthalene in the three systems ranged from 48.1 to 335 hours.

The photolytic degradation of ^{14}C-naphthalene at pH 7 was studied by Landis International (private communication, 1995). The experiment summary states that after irradiation under an artificial sunlight source for 15 days, 99.5% of the ^{14}C-naphthalene was degraded to polar materials (the complete test report has not been seen).

Tuhkanen and Beltrán (1995) studied the products of naphthalene decomposition by UV and UV/H_2O_2 treatment. A mercury arc lamp was used to irradiate solutions prepared in distilled deionised water. Treatment with H_2O_2 alone caused no decrease in naphthalene concentration. Naphthalene could be decomposed by UV irradiation but the addition of H_2O_2 accelerated the decomposition. Naphthalene was oxidised to naphthol then successively to naphthoquinone and benzaldehyde and phthalic acid and benzoic acids.

However, photodecomposition, oxidation and hydrolysis are not considered to be significant pathways for polynuclear aromatic hydrocarbon degradation in the soil environment (Sims and Overcash, 1983).

Hydrolysis

Naphthalene does not contain groups amenable to hydrolysis (US EPA, 1979b).

Summary

The half-life for photolysis in water lies in the range 25-550 hours depending on the experimental conditions used. The highest value in this range, which corresponds to photolysis in a 5-metre deep body of water has been used as a worst-case half-life in the assessment.

Atmospheric oxidation

Atmospheric oxidation of naphthalene occurs by reaction with the hydroxyl radical and reactions may also occur with ozone and nitrogen oxides. The rate constants for the gas phase reactions of hydroxyl radicals and ozone with naphthalene were determined under atmospheric conditions at 294 ± 1 K (Atkinson et al., 1984). The rate constant for the reaction of naphthalene with hydroxyl radicals was $(2.42 \pm 0.19) \cdot 10^{-11}$ $cm^3 \cdot molecule^{-1} \cdot s^{-1}$. Assuming an average daytime atmospheric hydroxyl radical concentration of approximately $1 \cdot 10^6$ $molecule \cdot cm^{-3}$, the lifetime of naphthalene due to reaction with hydroxyl radicals can be estimated to be approximately 1 day. The rate constant for reaction of naphthalene with ozone was measured and no decay of naphthalene by ozone was observed in the dark. Naphthalene was also found to react with NO_3 radicals which indicates that this may be an additional sink for naphthalene during nighttime hours in a polluted urban atmosphere.

Klöpffer et al. (1986) measured the rate of reaction of naphthalene with OH radicals in a smog chamber. At 300 K and $1.05 \cdot 10^5$ Pa the rate constant was $2 \cdot 10^{-11}$ $cm^3 \cdot molec^{-1} \cdot sec^{-1}$.

Masclet and Mouvier (1988) also measured the reaction rate of naphthalene with hydroxyl radicals. The rate constant was measured as $2.4 \cdot 10^{-11}$ $cm^3 \cdot molecule^{-1} \cdot sec^{-1}$ and, based on a hydroxyl radical concentration of $1 \cdot 10^6$ $molecule/cm^3$, the lifetime was calculated to be 12 hours.

Biermann et al. (1985) measured the rate constant for reaction of naphthalene with hydroxyl radicals to be $2.35 \cdot 10^{-11}$ $cm^3 \cdot molecule^{-1} \cdot sec^{-1}$ and calculated the half-life to be approximately 12 hours assuming a hydroxyl radical concentration of $1 \cdot 10^6$ $molecule/cm^3$.

The major products of the reaction of naphthalene with hydroxyl radicals were found to be 1- and 2-naphthols and 1- and 2-nitronaphthalenes (Atkinson et al., 1987). The reaction products and kinetics of the reaction of naphthalene with N_2O_5 were also studied. The rate constant was determined to be $(1.4\pm0.2)\cdot 10^{-17}$ $cm^3\cdot molecule^{-1}\cdot s^{-1}$ and the major products were 1- and 2-nitronaphthalene. The atmospheric lifetime of naphthalene due to reaction with N_2O_5 was calculated to be approximately 80 days for an estimated ambient N_2O_5 concentration of $2\cdot 10^6$ $molecules\cdot cm^{-3}$ during 12-hour night-time periods.

Summary

A value of $2.4\cdot 10^{-11}$ $cm^3\cdot molecule^{-1}\cdot sec^{-1}$ for the specific degradation rate constant with OH radicals, k_{OH}, has been used in the risk assessment. Combined with the standard OH concentration of $5\cdot 10^5$ $molecules\cdot cm^{-3}$ this gives a half-life of 16 hours.

3.1.2.1.2   Biodegradation

Aerobic degradation

Standard tests

The biodegradation of naphthalene by microorganisms has been investigated (CITI, 1992). The method used corresponds to OECD 302C: Inherent Biodegradability: Modified MITI test (II). Sludge sampling was carried out at 10 locations in Japan. Cultivation was carried out at $25\pm1^{\circ}C$ for 14 or 28 days. It was found that only 2% (by BOD) of naphthalene had degraded after 4 weeks.

Wastewater treatment plants and simulations

The removal of polycyclic aromatic hydrocarbons including naphthalene in a municipal treatment plant was studied (Melcer et al., 1995). The Hamilton Woodward plant in the Great Lakes area is a conventional activated sludge treatment facility designed for a dry weather flow of 409 Ml/day consisting of 17% residential, 7% commercial, 10% industrial and 65% unaccounted for (probably inflow and infiltration). The plant discharges to Hamilton harbour and receives coke plant wastewaters from a steel mill containing significant amounts of PAH. Influent, primary effluent and secondary effluent were collected over three sampling periods: April-May 1988, September 1988 and August 1989. The average liquid and solid concentrations of naphthalene in the raw sewage, primary effluent and secondary effluent were 5.1, 3.7 and 0.2 µg/l, respectively. The Hamilton plant removed 96.8% of the influent naphthalene.

A batch reactor filled with acclimated sludge, mineral salts and naphthalene was used to follow the degradation of naphthalene under aerobic conditions (Buitrón and Capdeville, 1993). Depending on the initial concentration (range 5-25 mg/l) up to 99% of naphthalene was degraded in 2.8-8 hours. Naphthalene was transformed to CO_2 (27.2%) and metabolites (4.0%).

Aquatic mesocosms (water and sediment)

Various oil spill scenarios were simulated in four mesocosm experiments to study the environmental factors that control the bacterial response in cold seawater (Siron et al., 1995). The half-lives calculated for degradation of naphthalene ranged from less than 1.5 days when the naphthalene concentration was 44.3 µg/l to 1.7 days when the naphthalene concentration was

0.34 µg/l. The experimental results indicated that the temperature threshold for observing significant oil degradation is approximately 0°C and that crude oil spilled in intertidal zones is expected to be slowly degraded. However, the potential capability of the community to adapt and biodegrade spilled oil within a few days was also demonstrated.

Batch experiments to investigate biodegradation of naphthalene in three freshwater lakes with varying degrees of pollution were carried out (Cooney et al., 1985). Lake water, sediment slurry and a mixture of four hydrocarbons including naphthalene were incubated at the seasonal in situ temperature and photo period. After 21 days naphthalene was not detected in the sample from the polluted lake but it was less degraded in samples from less polluted lakes.

Rates of microbial transformation of naphthalene were measured in stream water and sediment samples collected in the vicinity of a coal-coking wastewater discharge (Herbes, 1981). Radio-labelled naphthalene was incubated with sediment and water samples and the transformation products were isolated and quantified. The mean rate constant (at 20^oC) for transformation of naphthalene in sediment collected downstream from the effluent outfall was $7.8 \cdot 10^{-2}$ hour^{-1}, which corresponds to a turnover time of 13 hours. Transformation in water samples was slower with a rate constant of $3.2 \cdot 10^{-3}$ hour^{-1}, which corresponds to a turnover time of 310 hours (13 days).

Flow-through microcosm test systems were used to monitor the biodegradation rates of naphthalene in freshwater and estuarine ecosystems (Heitkamp and Cerniglia, 1987). Microcosms containing 200 ml of a 1:10 mixture of sediment and water from three ecosystems were exposed to 500 ng/g of ^{14}C-labelled naphthalene for 8 weeks under aerobic conditions at 22^oC. Evolved $^{14}CO_2$ was trapped and measured using scintillation counting. Estimated half lives for naphthalene in the three sites ranged from 2.4 to 4.4 weeks. Volatile ^{14}C residues detected in trapping solutions accounted for approximately 12-15% of total radioactivity.

Sediment contaminated with hydrocarbons (naphthalene concentration 7 µg/g wet weight) was incubated with ^{14}C-labelled naphthalene in spring water (Herbes and Schwall, 1978). The $^{14}CO_2$, extractable ^{14}C and bound ^{14}C were determined. The initial decrease in naphthalene followed first order kinetics but after 60% of initial naphthalene had been degraded the rate decreased. The decrease in naphthalene was accompanied without lag by increases in $^{14}CO_2$ and bound ^{14}C. Parent ^{14}C-labelled naphthalene was nearly completely degraded within 24 hours.

The degradation of naphthalene in sediment was investigated under oxidising conditions where aerobic microorganisms predominate (DeLaune et al., 1980). ^{14}C-labelled naphthalene (5 µg/g sediment) was added to a flask containing wet sediment with a water-to-sediment ratio of 6:1 and a redox potential of +500 mV. The naphthalene had a residence time of 70 days in this oxidised sediment.

Aerobic degradation of naphthalene in intertidal marine sediments was investigated in laboratory studies (Bauer and Capone, 1985). Sediment was taken from the top 0-2 mm of an intertidal mudflat and diluted 1:2 (wet weight: volume) with filtered seawater from the same area. Unlabelled naphthalene (1-1,000 µg/g) was added to the sediment slurry in incubation vials and >99% was removed by day 6. ^{14}C-labelled naphthalene was added to sediment slurry and $^{14}CO_2$ production was monitored; 42% of the substrate was mineralised by day 12 after a 2-day lag period.

Groundwater

Samples of groundwater from three sites along a gradient from heavily polluted to unpolluted were taken from a site which had been polluted by a leak of fuel oil two years earlier (Aamand et al., 1989). A mixture of naphthalene (190 µg/ml) and seven other compounds regarded as representative of fuel oil was added to the water samples which were then kept in the dark at 12°C. The naphthalene concentration decreased rapidly with lag times of 1.2 days for heavily polluted groundwater, 1.9 days for slightly polluted groundwater and 12 days for unpolluted groundwater.

Naphthalene added to groundwater drawn from a shallow well was found to degrade completely within eight days (Delfino and Miles, 1985). There was a lag period of six days followed by complete degradation within the next two days. There was no change in the naphthalene concentration of a sterile control during the experimental period.

The aerobic degradation of naphthalene was studied for 149 days in replicate batch experiments with groundwater and sediment from 8 locations from a 15 m·30 m section of an aerobic aquifer (Nielsen and Christensen, 1994). Microcosms containing groundwater, sediment and a mixture of aromatic hydrocarbons including naphthalene were incubated in the dark at 10°C for 149 days. Samples were taken 24 times during the incubation period and two microcosms were poisoned and used as controls. Naphthalene was 99.9% degraded in 15.2±8.4 days following a lag period of 4.5±2.3 days.

A study was carried out to measure and compare degradation of organic chemicals in groundwater 12 km from Lake Superior, Lester river water and Superior harbour water (Vaishnav and Babeu, 1987). Naphthalene was incubated at concentrations of 0.0, 0.8, 1.6 and 3.2 mg/l with water samples in 300 ml biochemical oxygen demand flasks. Naphthalene was found to be resistant to degradation in all three water types except when acclimated microbes or acclimated microbes and nutrients were added.

An incubation test was performed by adding a microbial soil suspension prepared from creosote contaminated soil to filtered groundwater and monitoring the naphthalene concentration with time (Mueller et al., 1991). The mixture was incubated at 30°C, with shaking, in the dark for 14 days. Naphthalene was readily degraded from an initial concentration of 28.7 µg/ml to 0.1 µg/ml after incubation for three days by the microorganisms and was extensively degraded after 5 days. Analysis of killed-cell controls showed that losses due to abiotic processes were minimal.

Soils

Degradation of naphthalene in 2 sandy loam soils (organic carbon content 0.5 and 1.1%) was studied (Park et al., 1990). The soils were incubated with naphthalene for up to 196 days and the half-life of naphthalene in both soils was found to be approximately 2 days.

Silty loam soils were incubated with naphthalene (20 mg/kg) at 30°C and after 10 days almost 90% of the naphthalene was lost (Ashok et al., 1995). Within 60 days naphthalene had disappeared completely from the soil.

A clay loam soil was dosed with ^{14}C-naphthalene and evolved CO_2, volatiles and radioactivity remaining in the soil were studied (Landis International, private communication, 1995). The recovery from the soil was poor (43.9-47.9%) but the summary states that the half-life of

naphthalene in clay loam soil was less than 30 days. Loss of naphthalene from the soil was thought to be primarily due to volatilisation rather than to degradation.

The degradation of naphthalene in a soil-water system was monitored by batch experiments (Mihelcic and Luthy, 1988). Mineral medium was mixed with soil (1 g dry weight) and a stock solution of naphthalene. Under aerobic conditions, naphthalene was degraded microbially from approximately 7 mg/l to non-detectable levels (detection limit 0.01 mg/l) in 10 days with an acclimation period of approximately 2 days.

Incubation tests were performed by adding a mixture of 8 polycyclic aromatic hydrocarbons (levels of 5 and 50 mg/kg for each compound) to unacclimated soils and monitoring the concentrations with time (Bulman et al., 1987). Naphthalene disappeared to non-detectable levels (detection limit 0.01 mg/kg) in 12 days with no apparent lag period.

Microcosms were constructed using various combinations of contaminated aquifer soil and groundwater samples with ^{14}C-labelled naphthalene (Klecka et al., 1990). The samples were incubated at 10°C and analysed periodically. The initial naphthalene concentration was approximately 0.5 mg/l and 50% was degraded in 11-18 days.

In-situ microcosms and laboratory batch microcosms were used for the determination of first order degradation rate constants of 19 compounds including naphthalene (Nielsen et al., 1996). The study was performed in two locations in a shallow, glaciofluvial unconfined sandy aquifer in Denmark. The degradation rate for naphthalene ranged from 0.2 to 0.9 day^{-1} with lag phases between 6 and 12 days.

Bioremediation experiments were carried out on a soil from a manufactured gas plant site using the fungus *Cunninghamella echinulata* var. elegans (Cutright, 1995). Contaminated soil was incubated with nutrient solution or water and acclimated fungal solution for 8 weeks. Control blanks showed that there was no activity due to indigenous microorganisms. In the untreated samples (no nutrient solution) the naphthalene weight was 11.12 g/kg soil after 8 weeks remediation but in samples with nutrient solution naphthalene was not detected.

Isolated microorganisms

Szulc et al. (1993) isolated bacterial strains from water from the Olawa River in Poland. Strains constituting the mixed populations were then used to study the biodegradation of naphthalene. The bacteria were cultured in mineral medium supplemented with naphthalene as the sole source of carbon with control samples to determine non-enzymatic loss of naphthalene. The loss of naphthalene was determined after 7, 14 and 21 days. It was found that 79.1% of the naphthalene was removed after 14 days. Therefore, the mixed population of bacteria successfully degraded naphthalene as the sole source of carbon.

Bacteria of the strain *Pseudomonas* have been shown to be capable of growing using naphthalene as the sole carbon source (Devereux and Sizemore, 1981; Kuhm et al., 1991; Stringfellow and Aitken, 1995). Sixty-two oil-degrading strains of bacteria were found in 40 samples of water and sediment. Of these 9 were able to grow on naphthalene as the sole carbon source. *Pseudomonas paucimobilis* was able to grow on agar plates or in liquid culture with naphthalene as the sole source of carbon and energy (Kuhm et al., 1991).

Two physiologically diverse bacteria, *Pseudomonas stutzeri* P-16 and *Pseudomonas saccharophila* P-15 were isolated from a creosote contaminated soil and their ability to

metabolise naphthalene was studied (Stringfellow and Aitken, 1995). Naphthalene served as a substrate for both organisms.

Naphthalene mineralisation by *Mycobacterium sp.* was monitored in a follow-through microcosm test system and 55% was degraded to CO_2 with the remainder distributed between cells and aqueous culture filtrate (Kelley et al., 1990). Two degradation pathways were proposed. The mineralisation pathway predominant in *Mycobacterium* was via initial ring oxidation to form cis 1,2-dihydroxy-1,2-dihydronaphthalene with further metabolism to ring cleavage products. Eukaryotic metabolism of naphthalene proceeds via initial epoxidation to form naphthalene-1,2-oxide which rapidly rearranges to 1-naphthol or is enzymatically hydrated to trans-naphthalene-1,2-dihydrodiol.

The biodegradation of naphthalene by an adapted strain of *Pseudomonas cepacia* under aerobic conditions at 30°C was measured (Magdaliniuk et al., 1995). Dissolved naphthalene was degraded with bacteria in the presence of polyacrylamide, montmorillonite and a complex of both.

The bioavailability of micelle-solubilised naphthalene to naphthalene degrading microorganisms comprising a mixed population isolated from contaminated waste and soils was studied (Liu et al., 1995). Experimental results showed that surfactant concentrations above the critical micelle concentration were not toxic to naphthalene degrading bacteria and the presence of surfactant micelles did not inhibit mineralisation of naphthalene. Naphthalene solubilised by micelles of two anionic surfactants in liquid media was bioavailable and degradable by the mixed culture of bacteria.

Naphthalene biodegradation was studied in culture media using a 7-day static incubation at 25°C in the dark (Tabak et al., 1981). Naphthalene undergoes significant biodissimilation in culture media dosed with 5 and 10 mg/l with rapid acclimation periods for naphthalene oxidising enzyme induction to occur.

Selected bacterial isolates from contaminated and background soil samples were incubated at 11°C in a mineral salts medium amended with a mixture of naphthalene, fluorene, anthracene and pyrene in saturated concentrations (Stetzenbach et al., 1985). Naphthalene was metabolised in culture vials and was reduced by ≥95% within 4 weeks by isolates from contaminated sites. The concentration also decreased in vials with background isolates but additional time was required.

Aerobic degradation rates for naphthalene are summarised in **Table 3.19**.

Table 3.19 Summary of aerobic degradation rates for naphthalene

Description	Degradation	Reference
301C Ready Biodegradability: Modified MITI test (I) or 302C Inherent Biodegradability: Modified MITI test (II)	2% (by BOD) after 4 weeks	Chemicals Inspection and Testing Institute, Japan, 1992
Sewage treatment plant	96.8% removal	Melcer et al., 1995
Batch reactor with acclimated sludge, mineral salts and naphthalene under aerobic conditions	99% in 2.8-8 hrs	Buitrón and Capdeville, 1993
Oil spill scenarios	$t_{\frac{1}{2}}$ = 1.5-1.7 days	Siron et al., 1995
Radiolabelled naphthalene incubated with sediment and water samples at 20°C	rate constant in water = $3.2 \cdot 10^{-3} h^{-1}$	Herbes, 1981
Microcosms containing water/sediment mixture exposed to ^{14}C-naphthalene, aerobic, 22°C	t_2=2.4-4.4 weeks	Heitkamp and Cerniglia, 1987
Aerobic degradation in intertidal marine sediments	99% in 6 days	Bauer and Capone, 1985
Naphthalene added to groundwater	100% in 2d (6d lag)	Delfino and Miles, 1985
Microcosms containing groundwater, sediment and aromatic hydrocarbons incubated in dark, 10°C	99.9% in 15.2±8.4 d lag of 4.5±2.3 d	Nielsen and Christensen, 1994
Degradation in groundwater	No degradation unless acclimated microbes added	Vaishnav and Babeu, 1987
Silty loam soils incubated with naphthalene (20 mg/kg) at 30°C	90% after 10 d 100% within 60 d	Ashok et al., 1995
Mineral medium mixed with soil and stock solution of naphthalene. Aerobic conditions	100% in 10 d, 2 d acclimation period	Mihelcic and Luthy, 1988
Aromatic hydrocarbons added to unacclimated soils	100% in 12 days	Bulman et al., 1987
Microcosms constructed using contaminated aquifer soil and groundwater samples with ^{14}C-labelled naphthalene incubated at 10°C	50% in 11-18 days	Klecka et al., 1990
In-situ and laboratory batch microcosms	rate = 0.2-0.9 d^{-1} lag of 6–12 d	Nielsen et al., 1996
Bacteria cultured in mineral medium supplemented with naphthalene	79.1% in 14 days	Szulc et al., 1993
Bacterial isolates from contaminated soil at 11°C in a mineral salts medium amended with mixture of naphthalene, fluorene, anthracene and pyrene	95% within 4 weeks	Stetzenbach et al., 1985

Anaerobic degradation

Incubation tests were performed by adding known amounts of ^{14}C-labelled naphthalene (50, 200 and 500 mg/kg) to soil slurries (9 g soil and 21 g mineral medium) in an anaerobic chamber and monitoring the naphthalene and $^{14}CO_2$ with time (Al-Bashir et al., 1990). A weathered oil-contaminated soil (4.3% organic carbon) and a pristine soil (2.1% organic carbon) with 10% contaminated soil were used. Biodegradation of naphthalene under denitrifying conditions was monitored for 160 days and mineralisation occurred after an adaptation period of approximately 18 days. When naphthalene was added at a concentration of 50 mg/kg the maximum degradation rate was 1.3 mg/kg per day after 18 days, until 80% was mineralised and then the rate decreased rapidly. After 50 days 90% naphthalene was recovered as $^{14}CO_2$.

Naphthalene was added to sediment from an intertidal mudflat (5-10 cm depth) in an incubation test (Bauer and Capone, 1985). No ^{14}C-labelled mineralisation products were produced from the

[14]C-labelled naphthalene added to the anoxic sediment. There was no difference between the naphthalene concentration of the anoxic samples and the controls.

Radioactively labelled naphthalene was added to an estuarine sediment slurry at a redox potential of -200 mV (Delaune et al., 1980). Under these conditions anaerobic bacteria are the only active species and naphthalene was not found to degrade to any extent.

The anaerobic degradation of naphthalene in groundwater drawn from approximately 150 m below ground was monitored (Delfino and Miles, 1985). After 96 days only a minimal amount of naphthalene degradation was observed.

Mineral medium was mixed with soil and a stock solution of naphthalene in batch experiments (Mihelcic and Luthy, 1988). The mixture was purged with helium to remove oxygen and anaerobic degradation was followed. A similar experiment was carried out to follow denitrification but sodium nitrate was added to the mixture so the initial nitrate concentration was approximately 75 mg/l. Under anaerobic conditions the aqueous phase naphthalene concentration did not change significantly over 50 days. Under denitrification conditions the aqueous phase naphthalene concentration decreased from 7 mg/l to non-detectable levels in 45 days with an acclimation period of approximately 10 days.

Naphthalene (approximately 4 mg/l) in a soil-water system was found to degrade under denitrification conditions to non-detectable levels in 47 days with an acclimation period of about 2 weeks (Mihelcic and Luthy, 1988b). Under nitrate limiting conditions the concentration of naphthalene in a soil-water system remained constant at 6 mg/l for 11 weeks. In tests with an acclimated soil the aqueous phase naphthalene concentration decreased from 3 mg/l to non-detectable levels (<0.01 mg/l) in approximately 16 days with no acclimation period.

Incubation tests were carried out with anoxic sediment from a polluted freshwater creek, anoxic marine sediment and anaerobic sewage sludge. Enrichment cultures with freshwater or seawater and naphthalene as substrate were inoculated with the sediment samples (Schink, 1985). No significant formation of methane was observed during 14 weeks of incubation.

Ehrlich et al. (1982) took material from an aquifer which had been contaminated by waste from coal tar distillation and a wood treatment plant in St Louis Park, Minnesota. Laboratory tests showed no evidence of anaerobic degradation of naphthalene.

Summary

The results of the only standardised screening test for inherent biodegradability for naphthalene suggest that naphthalene is not inherently biodegradable (2% degradation after 4 weeks). However, numerous other 'non-standard' biodegradation tests suggest that it is easily degraded under aerobic and denitrifying conditions, particularly where acclimated microorganisms are used, with naphthalene falling below measurable levels within 8-12 days in a number of tests. Naphthalene has therefore been considered to be inherently biodegradable in the risk assessment. Based on this, the EUSES model indicates the fate of naphthalene in a wastewater treatment plant as: 27.4% to air; 34.8% to water; 11.2% to sludge; and 26.6% degraded.

Default rate constants given in the Technical Guidance Document for inherently biodegradable substances have been used in the assessment due to the variability of the measured values given above. The resulting degradation rates in the environment are:

Biodegradation in surface water $4.62 \cdot 10^{-3}$ day^{-1}
Degradation in bulk soil $2.31 \cdot 10^{-3}$ day^{-1}
Degradation in bulk sediment $2.31 \cdot 10^{-4}$ day^{-1}

Although there are no standard tests for anaerobic degradation, the results suggest that naphthalene is resistant to biodegradation under anaerobic conditions. Some tests show no significant reduction in naphthalene levels for the duration of the test. Others showed 90-100% reduction within about 50-60 days.

3.1.2.2 Distribution

3.1.2.2.1 Adsorption

Batch equilibrium experiments were used to study the adsorption of six polycyclic aromatic hydrocarbons (PAH), including naphthalene, by two aquifer materials (Abdul and Gibson, 1986). Naphthalene was also studied separately. The soil organic carbon-water partition coefficient (K_{oc}) for naphthalene was approximately 455 for the Flint sample (organic carbon fraction of 0.0187 by weight) and approximately 650 for the Borden sample (organic carbon fraction 0.0002 by weight). However, adsorption of naphthalene as a single compound on Flint soil had a slope for the linear isotherm (K_d value) higher than for the mixture by a factor of 1.1.

Batch sorption isotherms using ^{14}C-labelled naphthalene were determined on mineral oxides and aquifer materials which had low organic carbon contents and on a surface soil substrate which had a higher organic carbon content (Stauffer and MacIntyre, 1986). Acidity and ionic strength were varied to simulate different groundwater conditions. A variation of ionic strength over three orders of magnitude had little effect on naphthalene sorption for two sorbates. The soil organic carbon-water partition coefficient for naphthalene was calculated to be 585. Under basic conditions the sorption coefficients were significantly reduced. Naphthalene sorption coefficients were positively related to surface area and to fraction organic carbon for the soil.

The sorption of naphthalene on pond and river sediments was investigated by equilibration of variable amounts of naphthalene with constant concentration of two types of sediment with organic carbon contents of approximately 3% (Karickhoff et al., 1979). The adsorption fitted well to linear isotherms over a range of water phase concentrations. The sediment organic carbon-water partition coefficient (K_{sed}) was calculated to be 1,300.

Batch adsorption experiments followed by successive desorption experiments were carried out by Kan et al. (1994). The adsorption time was varied from 1 to 30 days and soil phase naphthalene concentration was varied from 0.034 to 0.968 mg/l. Adsorption of naphthalene reached equilibrium within 1 day. Desorption experiments showed that only 30-50% of adsorbed naphthalene could be desorbed.

The adsorption of naphthalene was measured in five soils (organic carbon content 1.09-5.92%) at one initial concentration assuming a linear adsorption isotherm (Briggs, 1981). The soil organic matter-water partition coefficient (K_{om}) was calculated to be 240.

Measurements of the adsorption behaviour of naphthalene were performed on the basis of OECD test guidelines for "Adsorption/Desorption" (Rippen et al., 1982). Equilibria were determined for five different concentrations so the adsorption isotherm and adsorption coefficient could be

determined. The soil organic carbon-water partition coefficients for four soils were in good agreement (3,200, 1,600, 1,300 and 1,400).

The sorption of naphthalene for ten Danish soils was determined in laboratory studies by batch equilibrium experiments (Løkke, 1984). The adsorption significantly correlated with the organic carbon content of the soils tested. The correlations with other factors tested such as pH, cation-exchange capacity and particle-size distribution were not significant. The sorption of naphthalene was found to be partially reversible in four out of ten soils tested. A range of soil organic carbon-water partition coefficients (K_{oc}) were measured from 520-2100 (mean=1,100). These values were influenced by the uncertainty of determination of organic carbon, which is relatively large at low levels.

Batch sorption experiments were carried out with sandy soil from an agricultural area (Lindhardt and Christensen, 1994). Sorption was found to be fast with equilibrium reached within 24 hours. The soil contained 1.1% organic carbon, 4.8% clay, 3.8% silt, 17.3% fine sand and 71.9% coarse sand. The measured organic carbon-water partition coefficient was 664 cm^3/g.

Sorption isotherms for naphthalene were determined by equilibration of naphthalene solutions with soils for 24 hours (Bouchard et al., 1990). K_{oc} values for two soils were calculated as 455 and 378. Naphthalene adsorption on soils contaminated with unleaded gasoline was also studied and was found to be significantly higher on a soil contaminated with residual hydrocarbons than on a pristine soil. This indicates that naphthalene adsorption to residual hydrocarbons was greater than to natural soil organic carbon.

Sorption of naphthalene on a contaminated soil was investigated by batch experiments using initial naphthalene concentrations of approximately 0.5-10 mg/l (Klecka et al., 1990). The soil organic carbon-water partition coefficient was 989 (organic carbon content 0.18%).

Release rates of naphthalene from suspensions of freshly contaminated (days-weeks) and aged (approximately 30 years) soil were obtained using a gas purge method (Connaughton et al., 1993). For freshly contaminated soil, the sorbed mass decreased by 70-80% in 1 day and by 80-90% after three days purging. The final 10-20% showed increasing resistance to desorption. For soils exposed for long periods in the field the release rates were fast for the first 30-70% of sorbed naphthalene but release was slow for the remaining 70-30%.

Piatt et al. (1996) investigated the effect of temperature on sorption of naphthalene to low organic carbon aquifer sediments. Batch and column experiments were carried out with sediment from an uncontaminated zone of an aquifer in Minnesota. The organic carbon content was 0.00019 and the mean annual temperature of the aquifer was 12°C with pH 7.7. Equilibrium was reached within 40 hours and it was found that the dependence of the distribution coefficient and desorption rate constants on temperature was small.

Whitman et al. (1995) investigated the significance of sorption of naphthalene to microbial cells and soil with either a high or a low sorptive capacity. A mutant derivative of a naphthalene-degrading microorganism that has lost the ability to degrade naphthalene was used to determine the contribution of naphthalene biosorption relative to sorption by the soil. The experimental results and a mass balance model showed that, in soil systems with a high organic carbon content, the mass of naphthalene associated with biological solids is insignificant. However, in a soil system with non-sorptive sand, up to 10% of the initial naphthalene was associated with cells of the microorganism.

Summary

Experimental values for Koc are in reasonable agreement, ranging from 378 to 3,200. The QSAR approach given in Chapter 4 of the Technical Guidance Document in which the value for Koc is estimated from the experimentally derived octanol-water partition coefficient, Kow, gives a value of 1,250 l/kg. This value, which is consistent with, and representative of, the experimental values, has therefore been used in the assessment. The other partition coefficients estimated from this value are: sediment-water 32.1; suspended sediment - water 32.2; soil-water 37.7.

3.1.2.2.2   Volatilisation

Due to the relatively high vapour pressure and low water solubility of naphthalene, volatilisation from water bodies to the atmosphere is likely to be an important process. The half-life of naphthalene in an air-water system for a water depth of 1 m was calculated to be 7.15 hours (Mackay and Leinonen, 1975).

Schwartz and Wasik (1977) measured the water-air partition coefficient of naphthalene as 53 at $25.3^{o}C$ and 90 at $16.6^{o}C$ (both dimensionless). These values correspond to Henry's law constants of 46.8 $Pa \cdot m^{3} \cdot mol^{-1}$ and 26.75 $Pa \cdot m^{3} \cdot mol^{-1}$, respectively.

Experiments were carried out in the laboratory to simulate volatilisation from a stirred water body using well-defined wind velocities and water temperatures (Klöpffer et al., 1982). The initial concentration, airflow rate and humidity were varied within wide ranges without producing any significant influence on the half-life. For naphthalene the decrease in concentration as a function of time was exponential and the average half-life of eleven measurements at room temperature was 380 ± 52 minutes for a water depth of 22.5 cm.

Southworth (1979) calculated a Henry's law constant for naphthalene and estimated volatilisation rates from a stream 1 m deep under various conditions of wind speed and current velocity. The measured value for the (dimensionless) Henry's law constant was $2.26 \cdot 10^{-2}$. The half-life for naphthalene was predicted to be less than 100 hours under the conditions considered ($25^{o}C$, wind velocity 0-4 m/sec, current velocity 0.1-1 m/sec).

The fate of naphthalene in two sandy loam soils (organic carbon content 0.5% and 1.1%) was evaluated in the laboratory (Park et al., 1990). The soils were incubated with naphthalene at $25^{o}C$ for 48 hours and approximately 30% of naphthalene was volatilised in this time.

Henry's law constants were determined for three surface water-sediment systems and used to estimate volatilisation rates from the different types of water (Saleh et al., 1984). For a water column depth of 5.22 m the half-lives for volatilisation were estimated to be between 248 and 297 hours.

In 1968, sewage sludges containing different concentrations of polynuclear aromatic hydrocarbons were applied to soils at two experimental sites in the UK (Wild et al., 1992). At both sites the application of sludge caused significant increases in PAH concentrations. Half-lives were estimated for naphthalene to be < 2.1 years at one site (sandy loam soil) and < 2.3 years at the other site (silty loam soil).

The value of the Henry's law constant used in the assessment has been estimated from the vapour pressure and the solubility of naphthalene (see Sections 1.3.5 and 1.3.6). The value derived is 44.86 $Pa \cdot m^{3} \cdot mol^{-1}$. This value is consistent with the measured values given above.

Rain-out

Naphthalene is only slightly soluble in water (30 mg/l) and so only a small proportion will be removed from the atmosphere by precipitation. Naphthalene has been found in rain and snow samples at quite low levels (see Section 3.1.3.2.3).

3.1.2.3   Metabolism and accumulation

3.1.2.3.1   Metabolism

The metabolic fate of ^{14}C-labelled naphthalene after intraperitoneal injection into young Coho salmon (*Oncorhynchus kisutch*) was studied by Roubal et al. (1977). Coho salmon fingerlings of mixed sex were acclimatised for several weeks in aquaria with running fresh water at 14 ^{o}C. The fish received approximately 1% of their body weight per day of hatchery food containing ^{14}C-labelled benzene, naphthalene and anthracene. In a separate test fish fasted for three days and were then fed 5 µCi of naphthalene impregnated in hatchery food. Sampling was at 24, 72, 168 and 336 hours after feeding. During the period 24-168 hours after feeding, 98% of naphthalene and metabolic products were lost from the liver and brain. In a separate experiment Coho salmon fingerlings were injected intraperitoneally with 2.5 µCi of ^{14}C naphthalene dissolved in 0.05 ml ethanol. Metabolic products were found in most areas of the body with the highest percentages found in the gall bladder and the liver also containing significant amounts. A wide spectrum of metabolic products was found in various proportions in different tissues. The heart and flesh contained primarily 1-naphthol while 1-naphthol and 1-naphthyl glucuronic acid were the major metabolites in the brain, liver and gall bladder.

The metabolism of orally administered naphthalene in spawning English sole (*Purophrys vetulus*) was studied (Reichert and Varanasi, 1982). The fish were not fed for three days prior to the experiment and for the week of the experiment. They were force-fed gelatine capsules containing 140 µCi of labelled ^{3}H naphthalene. The fish were sampled at 24, 48 and 168 hours after being force-fed and the liver, muscle, bile, blood and ovaries or testes analysed separately. Detectable concentrations of naphthalene were found in English sole 24 hours after feeding. The ovaries had the highest percentage (5%) of the total administered dose. Muscle and liver in both sexes also contained considerable amounts of naphthalene. The naphthalene concentrations declined rapidly from 24-168 hours in all tissues examined. The major non-conjugated metabolite was 1,2-dihydro-1,2-dihydroxynaphthalene. Only small amounts of naphthol were detected.

A flow-through system was used to follow ^{14}C-naphthalene and naphthalene metabolite accumulation in the tissue of the oyster *Ostrea edulis* (Riley et al., 1981). Oysters were exposed to synthetic seawater with 28.5l salinity at 14°C with naphthalene concentration of 90 µg/l. The filtered seawater was glucose-enriched and contained 100 mg/l streptomycin (to control bacterial growth and reduce oxidation). The adductor muscle, "body" (digestive gland, gut, kidney, gonad and overlying mantle tissue) and 'gill tissue' (gills and overlying mantle) of the oyster were analysed for naphthalene and metabolites at the end of the 72-hour run. The body had the highest naphthalene concentration (4.03-4.40 mg/l naphthalene equivalents based on wet weight) which was significantly higher than in the adductor (1.65-3.84 mg/l) and the gill tissue (2.19-2.84 mg/l).

Male specimens of a crab (*Maia squinado*) were fed soft cores of naphthalene in lard into the foregut (Corner et al., 1973). Twelve animals received a dose of 0.19 g of naphthalene on the first day and again on days 3 and 5. Considerable losses occurred through regurgitation and defaecation. The urine was collected daily for the five-day experiment and for two days after. Unchanged naphthalene and very small amounts of metabolites were found in the urine. The metabolites were:

- 1-naphthyl glucoside,
- 1,2-dihydro-1,2-dihydroxynaphthalene,
- 1,2-dihydro-2-hydroxy-1-naphthyl glucoside and
- 1-naphthyl sulphuric acid.

Studies using acidified urine also showed that 1-naphthylmercapturic acid was present.

The metabolism of ^{14}C-labelled naphthalene in houseflies and rats was studied by Terriere et al. (1961). Flies of mixed sex were fed naphthalene in a 5% sucrose solution for 4-8 days. After this period the flies were ground and extracted with ethanol. The excreted material was also extracted. Four rats were dosed with approximately 70 µg of naphthalene intraperitoneally in peanut oil. Their urine was collected and extracted. All extracts were analysed by paper chromatography. Houseflies were found to produce all the metabolites of naphthalene that were produced by rats. These substances were identified as glucosiduranides of 1-naphthol, 1,2-dihydro-1,2-dihydroxynaphthalene and 1,2-dihydro-1-hydroxynaphthalene, 1-naphthylsulphate, mercapturic acid conjugates of 1-naphthol and 1,2-dihydro-1,2-dihydroxynaphthalene, free 1-naphthol and 1,2-dihydro-1,2-dihydroxynaphthalene.

Marine larval invertebrates were exposed to 8-12 µg/l ^{14}C-labelled naphthalene or ^{14}C-labelled naphthalene complexed with bovine serum albumin in flowing seawater (Sanborn and Malins, 1977). When in the free state and in a complex, naphthalene accumulated in stage V spot shrimp (*Pandulus platyceros*) at levels of 820 ng/g and 220 ng/g, respectively.

The metabolism of naphthalene by *Cunninghamella elegans* was studied by Cerniglia and Gibson (1977). *Cunninghamella elegans* cells were incubated with naphthalene for 24 hours and the experiment repeated using ^{14}C-labelled naphthalene. The major metabolites were 1-naphthol (67.9%) and 4-hydroxy-1-tetralone (16.7%). The minor products isolated were:

- 1,4-naphthoquinone,
- 1,2-naphthoquinone,
- 2-naphthol and
- trans-1,2-dihydroxy-1,2-dihydronaphthalene.

3.1.2.3.2 Bioaccumulation

The bioconcentration factor of naphthalene in a freshwater alga (*Chlorella fusca*) was measured using ^{14}C-labelled naphthalene (Geyer et al., 1984). The alga was exposed to naphthalene in a nutrient solution for 24 hours at room temperature under illumination and agitation. The bioaccumulation factor on a wet weight basis was measured as 130.

The bioconcentration of naphthalene in *Daphnia magna* was measured under static conditions and the bioconcentration factor was approximately 50 (Eastmond et al., 1984). Depuration studies showed that naphthalene was released very quickly.

Southworth et al. (1978) exposed *Daphnia pulex* to naphthalene in a static system. Uptake and depuration rates were measured as $197 \, h^{-1}$ and $1.7 \, h^{-1}$, respectively, giving a kinetic

bioconcentration factor as their ratio of 118. Equilibrium was reached well within 24 hours and direct measurement gave a value of 131.

The bioavailability of naphthalene from water and sediment to the marine worm *Arenicola marina* was studied (Lyes, 1979). Worms were exposed to ^{14}C-1-naphthalene in seawater and uptake of naphthalene was rapid in all tissues. The stomach wall and the oesophageal glands were important sites for accumulation with accumulation factors of approximately 300 and 160, respectively. No whole body bioaccumulation factor is given. Depuration studies showed that loss of naphthalene was also rapid with most samples exhibiting background counts within 24 hours. Uptake of naphthalene from labelled sediment was slow and accumulation factors for the stomach wall and oesophageal glands of 4.075 and 0.69, respectively.

The bioaccumulation factor of naphthalene in blue mussels (*Mytilus edulis*) was measured using ^{14}C-labelled naphthalene (Hansen et al., 1978). Mussels from an unpolluted area of Denmark were exposed to naphthalene in seawater for 8 hours then kept in clean seawater. Four naphthalene concentrations were used: 0.7, 7.1, 36 and 139 µg/l. Bioaccumulation factors were independent of naphthalene concentration and ranged from 27-38. Within 24 hours of depuration 99% of naphthalene was released from the mussels.

Oysters (*Crassostrea virginica*) were allowed to feed for 15 hours on suspensions of detrital matter in 24l seawater contaminated with naphthalene (34.6 mg/l), a PCB mixture and benzo(a)pyrene (Fortner and Sick, 1985). Samples of gill, mantle, labial pulps and digestive diverticula were excised from each oyster and analysed by liquid scintillation counting. Radioactivity of the water, suspension, tissues and faecal material was determined. The uptake of contaminants by adsorption was studied by repeating this experiment with the contaminants added to 24l seawater to give a naphthalene concentration of 83 mg/l. No food was provided in this experiment. The radioactivity of water and tissue samples was determined. More naphthalene was accumulated when oysters were exposed to naphthalene alone. The accumulation of naphthalene by oysters was tissue specific. For oysters fed contaminated detrital material the naphthalene concentration ranged from 5.4 mg/kg in the mantle tissue to 14 mg/kg in labial pulps. The concentration of naphthalene in oysters exposed to dissolved organics was generally higher than the concentration in respective tissues from oysters fed detrital material.

A flow-through system was used to study the accumulation of naphthalene and naphthalene metabolites in the oyster *Ostrea edulis* (Riley et al., 1981). Artificial seawater with a salinity of 28.5l, temperature of 14°C and a steady-state concentration of approximately 90 µg/l was used. After 72 hours the oysters were removed and dissected into adductor muscle, "body" (digestive gland, gut, kidney, gonad and overlying mantle) and 'gill tissue' (gills and overlying mantle). The body consistently had the highest bioaccumulation factors with the means for three runs ranging from 58 to 62. The mean bioaccumulation factors, on a wet weight basis, ranged from 31 to 42 for the gill tissue and from 24 to 52 for the adductor muscle.

Oysters (*Crassostrea virginica*) were exposed to naphthalene in controlled ecosystem enclosures (Lee et al., 1978). The enclosures were made of polyethylene and filled with 60,000 l of water from Saanich Inlet in western Canada. Naphthalene was dissolved in Prudhoe Bay crude oil and a dispersion was added to the enclosure. The oysters were exposed in a cage suspended at 7 m depth in the enclosure. Naphthalene was readily accumulated by the oysters with concentrations reaching 30 µg/g after two days (water concentration 5 µg/l). Naphthalene was readily released during depuration and was not detectable after 23 days.

The accumulation and release of naphthalene by clams (*Rangia cuneata*), oysters (*Crassostrea virginica*), fish (*Fundulus similus*) and shrimp (*Penaeus aztecus*) was studied by exposure to

oil-water mixtures for different lengths of time in a static system (Neff et al., 1976). After exposure the animals were returned to aquaria with continuously recirculated artificial seawater. Clams and oysters were also exposed to No. 2 fuel oil in a flow-through system for 8 hours then returned to clean filtered seawater. In the static system the oysters accumulated 6.3 mg/kg of naphthalene in four days. For the flow-through system, naphthalene accumulated by oysters and clams after 8 hours exposure was 14.7 and 3.8 mg/kg, respectively. A further experiment was carried out to assess patterns of accumulation and release of naphthalenes. Clams exposed to the water-soluble fraction of no.2 fuel oil for 24 hours accumulated 1.9 mg/kg naphthalene and the bioconcentration factor was 2.3. After 24 hours 79% of accumulated naphthalene was released.

A flow through system was used to study the accumulation of naphthalene in carp, *Cyprinus carpio* (CITI 1992). The method used corresponds to 305C, Bioaccumulation: degree of bioconcentration in fish stipulated in the OECD guidelines for testing chemicals (May 1981). The fish were acclimatised at $25\pm2^{o}C$ prior to exposure to a concentration of 0.15mg/l for 8 weeks. Bioconcentration factors of between 36.5 and 168 were measured.

Veith et al. (1979) cite a bioconcentration factor of 427 for the accumulation of naphthalene in fathead minnows (*Pimephales promelas*). The value was taken from an unpublished 28-day flow through experiment conducted by Call and Brook (1977). No further details are given concerning this test in the Veith et al. reference.

Bioconcentration factors were determined in fish, algae and activated sludge using ^{14}C-labelled naphthalene (Freitag et al., 1985). Bioconcentration was measured in golden ide (*Leuciscus idus melanotus*) using the average exposure to naphthalene dissolved in water. The fish were not fed during exposure. The bioconcentration factor was found to be 30. The bioconcentration factor for activated sludge, measured by the distribution of naphthalene between sludge and water, was 1,000; that for algae was 130, as reported above (Geyer et al., 1984).

Winter flounder (*Pseudopleuronectes americanus*) were exposed for one month to uncontaminated sediment and three types of drill cuttings from exploration wells in the Arctic and off the east coast of the USA (Payne et al., 1988). The naphthalene concentrations in the drill cuttings ranged from approximately 20-3,000 µg/g. The cuttings were added to aquaria that were supplied with air and running seawater at seasonally ambient conditions of temperature and photoperiod. Wild flounder were also analysed for comparison. The livers of the flounder were removed and analysed for hydrocarbons. The accumulation of naphthalene in exposed flounder was only approximately 5 times that of the control and 10-30 times that of wild flounder. No obvious relationship was found between the hydrocarbon concentration found in the cuttings and in the liver of exposed fish.

Black Sea Bass (*Centropristis striata*) were daily force-fed capsules containing oyster tissues (*Crassostrea virginica*) which had previously been exposed to ^{109}Cd in the presence and absence of ^{14}C-naphthalene (Fair and Sick, 1983). Fish from each group were sacrificed at 24, 72 and 336 hours and the ^{14}C- and ^{109}Cd-activity was counted in various tissues. The rates of accumulation of naphthalene and associated metabolites were not affected by the presence or absence of cadmium. The ingestion of relatively high concentrations of total naphthalene compounds for several days led to a relatively constant total naphthalene concentration over the two-week period.

White mullet (*Mugil curema*) were exposed to naphthalene concentrations of 0.01, 0.05 and 0.10 mg/l (Correa and Venables, 1985). A further group was kept in naphthalene-free seawater as a control. Increases in the mean tissue concentration for the gill, liver, spleen and muscle were measured at 24, 48, 72 and 96 hours. In a second experiment accumulation and depuration were

monitored by analysing the naphthalene concentration in tissues at 2, 4, 8, 12, 24, 48, 72, 144 and 216 hours. Water samples were taken during exposure to determine actual naphthalene concentrations. Naphthalene rapidly accumulated in *M. curema* tissues and the rate of uptake was much greater than the rate of depuration. Bioconcentration factors (BCF) were found to be highest in the liver (range 125-1054) and spleen (range 139-1158) and lower in the muscle (range 81-567) and gill (range 97-597) tissues. Bioconcentration factors in the muscle were found to be inversely related to exposure concentration.

Groups of rainbow trout (*Oncorhynchus mykiss*) were exposed to ^{14}C-labelled naphthalene in short-term tests for up to 8 hours and long-term tests for up to 4 weeks (Melancon and Lech, 1978). In the short-term tests naphthalene was rapidly taken up by the fish with levels in tissues reaching 22-340 times the initial level in water. Highest levels were found in fat tissue, followed by the liver and gill with lower levels in the blood and muscle. Half-lives for elimination of naphthalene were generally less than 24 hours. In long-term tests the liver again accumulated naphthalene to a greater extent than the blood and muscle and all tissues showed a gradual elimination of naphthalene after exposure.

Coho salmon (*Oncorhynchus kisutch*) and starry flounder (*Platichthys stellatus*) were exposed to 900 μg/l water soluble fraction of Prudhoe Bay crude oil in flowing seawater for 6 and 2 weeks, respectively (Roubal et al., 1978). The concentration of naphthalene in water was approximately 3 μg/l and the bioconcentration factor (on a dry weight basis) in muscle tissue after 6 weeks exposure was 40. After 1 week of depuration naphthalene was not found in muscle tissue (detection limit 50 μg/kg). After two weeks exposure to naphthalene, bioconcentration factors in the liver, gill and muscle of flounder were 1,100, 250 and 240, respectively.

Juvenile Atlantic cod (*Gadus morhua*) were placed in net cages at approximately 20 m depth at 4 sites from the inner end to the northern mouth of Sørfjorden in Western Norway (Goksøyr et al., 1994). After 4 weeks the fish livers were analysed for polycyclic aromatic hydrocarbons. Levels of naphthalene in livers were highest in livers of fish from the sites closest to industrialised areas at approximately 90 and 55 ng/g lipid. At the other two sites naphthalene concentrations were approximately 35 and 20 ng/g lipid.

The accumulation of naphthalene in the tissues of Redhead ducks (*Aythya americana*) was determined by force feeding the ducks for three days with crayfish artificially contaminated with ^{14}C-naphthalene (Tarshis and Rattner, 1982). The crayfish were exposed to a solution of ^{14}C-naphthalene in 5% water-soluble fraction of no. 2 fuel oil. The daily dose of naphthalene was approximately 1.25 mg/bird which, with the other petroleum hydrocarbons in the water-soluble fraction, was considered to be an environmentally realistic dose. The ^{14}C-activity was measured in six different tissues and a significant increase in activity was measured between days one and three in the brain, blood, liver and kidneys indicating an accumulation of naphthalene and metabolites in these tissues. The ^{14}C-activity of the gall bladder and fat were significantly higher than in the other tissues and remained similar throughout the three days. The study demonstrates that naphthalene, metabolites and related contaminants can be transferred by aquatic invertebrates to waterfowl and can accumulate in varying quantities in tissues of aquatic birds.

Naphthalene has been identified in South Louisiana crude oil and was detected in various tissues of Mallard drakes (*Anas platyrhynchos*) dosed with 5 ml crude oil per day for 14 days (Lawler et al., 1978). Naphthalene was found in the brain, skin, breast muscle, heart, liver, uropygial gland and blood.

Summary

Bioconcentration factors are summarised in **Table 3.20**. The values for whole fish range up to 427. Higher bioconcentration factors for individual tissues up to approximately 1,200 have been measured. A value of 279 is derived for the bioconcentration factor using the QSAR for substances with log Kow < 6 (Technical Guidance Document Chapter 4, Table 6).

Table 3.20 Bioconcentration factors

Organism	Bioconcentration factors
Fish	up to 427 (whole body) up to 1158 (individual tissues)
Algae	130
Daphnia	50-131
Other Marine worm Mussels Clams	 160 – 300 27 – 38 2.3

For this assessment the highest whole body BCF of 427 for fathead minnows has been used as a worst case even though the experimental details of this study are not known.

3.1.2.4　　　　　**Summary of fate and behaviour**

Naphthalene is considered to be inherently degradable under aerobic conditions, and can be utilised as the sole carbon source by some bacteria. Under anaerobic conditions degradation does not appear to occur but naphthalene does degrade under denitrifying conditions.

In the atmosphere, naphthalene reacts with hydroxyl radicals and has a half-life of approximately 1 day. It can also react with ozone and N_2O_5.

Naphthalene is readily volatilised from water, the half-life for volatilisation from water up to 1 m deep being approximately 7 hours. It is expected to adsorb to soils and sediments to a moderate extent. Naphthalene bioconcentration factors for the whole body and for individual tissues in fish range up to 427 and 1158, respectively. Naphthalene can be metabolised by a variety of organisms.

3.1.3　　　　　**Aquatic compartment (incl. sediment)**

3.1.3.1　　　　　**Calculation of Predicted Environmental Concentrations in water**

The predicted environmental concentrations (PECs) for naphthalene in the aquatic compartment have been calculated according to the methods in the Technical Guidance Document and the EUSES computer-modelling program for the releases given in **Tables 3.16 to 3.18**.

Local PECs arising from releases from sites producing or processing naphthalene have been calculated using site-specific information on the effluent concentrations, discharge rates and flow rates for receiving waters where this information is available. For production and processing sites where such information is not available and for releases from naphthalene use PECs have been derived in accordance with the Technical Guidance Document from the releases to water calculated in Section 3.1.1 and summarised in **Table 3.16**.

Releases have been assumed to occur to a treatment plant with the default characteristics specified in the Technical Guidance Document unless site-specific information indicates otherwise. It has been assumed that naphthalene is inherently biodegradable. From the EUSES model, the fate of naphthalene in the wastewater treatment plant is: 27.4% to air; 34.8% to water; 11.2% to sludge; and 26.6% degraded.

The EUSES model has been used to predict regional and continental environmental concentrations of naphthalene in water, based on the releases given in **Tables 3.17** and **3.18**. The EUSES printout can be viewed at the website of the European Chemicals Bureau (http://ecb.jrc.it, see Appendix 1). Based on these estimates, the continental concentration of naphthalene in surface water was estimated to be 0.00249 µg/l and the regional concentration was estimated to be 0.03 µg/l.

Local PECs for water arising from naphthalene production are given in **Table 3.21**. PECs for local releases from processing and use of naphthalene are given in **Table 3.22**.

Table 3.21 Local PECs for naphthalene production based on site-specific information

Site	Naphthalene concentration in effluent (µg/l)		PEC*local*_{water} (µg/l)
	Measured	Calculated	
A		0.5	0.036
B		1.7	0.20
C	<0.01 µg/l		0.031
D		730	0.31
E		390	0.04
F	0.22 µg/l		0.05
G		0.05	0.035
H	0.25 µg/l		0.055
I	<0.4 µg/l		<0.07
J	no wwtp		0.035

Table 3.22 Local, regional and continental PECs for water

Process	PEC (µg/l)
Production Site D (worst case site-specific release)	0.31
Phthalic anhydride production – site specific	0.04
Use as intermediate (processing)	0.042
Pyrotechnics manufacture	2.35
Mothballs manufacture	0.03
Grinding wheels manufacture - worst case	294
Regional	0.03
Continental	0.0025

3.1.3.2   Measured levels in water

3.1.3.2.1   Surface water

Naphthalene has been monitored in some surface waters in the UK (NRA communication, 1995). In one region, a mothproofing survey was carried out in 1992 and no significant levels of naphthalene were detected. In another region, sampling was carried out at 66 sites between 1993 and 1995. The maximum naphthalene concentration detected was 103 ng/l at one site and all other levels were less than 100 ng/l. Sampling was also carried out at 36 sites in another region. The maximum naphthalene concentration was less than 1,000 ng/l with most levels below 100 ng/l.

Samples of water were collected from five estuarine sampling stations and five freshwater sites at the Mersey estuary (Foundation for Water Research, 1990). Naphthalene was identified by GC/MS at four locations in the dissolved phase and two locations in the particulate phase of the freshwater inputs to the estuary and at one location in the particulate phase of the estuary samples.

A 1993 survey of PAH levels in estuaries in the UK was reported to show that in areas little affected by industry or urbanisation PAH levels were low (ENDS, 1995). In the Tweed estuary naphthalene levels were less than 15 ng/l. In the outer Mersey estuary and the Thames estuary naphthalene levels were 2 ng/l and 114 ng/l, respectively. Levels in the Thames estuary were thought to be high due to the deposition of PAHs from combustion sources in London whereas in the Mersey lower levels were found because prevailing winds carry air pollution from Liverpool and Manchester inland. The highest concentration of naphthalene found was 6,850 ng/l in the Tees estuary at Redcar close to an effluent outfall from a steelworks.

Naphthalene concentrations were measured in samples of the Rhine river at Düsseldorf (Kuhn and Clifford, 1985). The average concentration between January and September 1985 was 0.04 µg/l. Samples of Rhine river water before and after various water treatment processes were analysed for naphthalene. Naphthalene concentrations were found to decrease from 0.09 µg/l in the river water to 0.01 µg/l after bank filtration or ozonation and <0.005 µg/l after GAC adsorption.

Naphthalene concentrations in the Besós river north of Barcelona and the Llobegrat river south of Barcelona were 1,300 and 180 ng/l, respectively (Gomez-Belinchon et al., 1991). Naphthalene concentrations in samples of coastal waters in the same region were between 1.5 and 20 ng/l.

Monitoring data for napthalene in the Rhine and Meuse in the Netherlands during 1988-1990 is available (Barreveld, 1992). The maximum level in the Rhine was found to be 0.1 µg/l in 1988 and in the Meuse was 0.8 µg/l in 1989.

Naphthalene concentrations were monitored in the Adige river, Italy, in October 1989 (Benfenati et al., 1992). The values ranged from 3-2,240 ng/l with an average value of 284 ng/l. The higher values could have been associated with industrial activity although this cannot be confirmed from the data.

A summary of other measurements of levels of naphthalene in surface water samples is given in **Table 3.23**.

Table 3.23 Levels of naphthalene in surface water

Location	Level	Reference
Detroit river, USA	5-1,000 ng/kg	Platford et al., 1985
Mississippi, USA (measured 1984)	4-34 ng/l	DeLeon et al., 1986
Vineyard Sound, USA (March 1977 - June 1978).	0.5-35 ng/l	Gschwend et al., 1982
Vaal river, South Africa	detected in 3/8 samples	van Steenderen et al., 1987
Klang river basin, Malaysia	up to 6.21 µg/l	Tan et al., 1990
Brisbane river estuary, Australia	19-26 ng/l	Kayall and Connell, 1989

Water samples taken from the Delaware river in August 1976 and March 1977 were analysed for naphthalene (Sheldon and Hites, 1978). Semi-quantitative analysis gave a range of naphthalene concentrations between 0.7-0.9 µg/l in the three samples out of five where naphthalene was detected.

Samples of water were collected from marina and non-marina sites in the Washington DC area during peak boating activity (June 1990) and during low boating activity (October 1990) (Mastron et al., 1994). The average concentration of naphthalene in water samples was approximately 0.075 µg/l.

Water samples were collected from 9 sampling stations in Puget Sound including Elliott Bay, Commencement Bay, Sinclair Inlet, Budd Inlet and a reference area (Riley et al., 1980). Naphthalene concentrations in filtered seawater ranged from 0.018-0.108 µg/l. The seasonal variation of naphthalene in filtered seawater was studied at two stations by sampling in July, September and November. The naphthalene concentrations were highest in July and lowest in September.

Replicate water samples were collected from a navigation canal leading into Lake Pontchartrain in Louisiana (McFall et al., 1985a) on both the ebb and flood tides. Naphthalene was not detected in the ebb tide samples but was detected in the flood tide samples at very low concentrations. At a depth of 1.5 m the concentration was 0.02 µg/l and at 10 m the concentration was 0.1 µg/l.

Water suspected to be contaminated with fuel or lubricating oil was sampled in two harbours in Bermuda in 1988 (Ehrhardt and Burns, 1990). Naphthalene concentrations in these polluted areas were 2.92 ng/l and 0.07 ng/l and naphthalene was not found in control samples from an unpolluted area on a reef platform.

Naphthalene concentrations in surface seawater samples were determined (Marchand et al., 1988). Samples were collected from 5 areas in the Western Mediterranean in 1984. Naphthalene was found near the mouth of the river Rhône (125 ng/l), in the dispersal area of river waters in the Gulf of Lions (30-3 ng/l) and in the Cortiou area which receives urban sewage (20-300 ng/l). Levels around the coast of Corsica and at open sea were <5 ng/l.

Surface seawater samples were taken from polluted and unpolluted open ocean and coastal waters in the Gulf of Mexico (Sauer, 1981b) and analysed for naphthalene. Naphthalene was not detected (<1 ng/kg) in the unpolluted open ocean but levels of 1-4 ng/kg were found in ocean samples where an anthropogenic influence was acting. Levels of naphthalene in the coastal water samples were 2 ng/kg in the unpolluted region and in the range 2-5 ng/kg in regions with an anthropogenic influence (off-shore oil/gas production, industrial discharge activities and outflow of the Mississippi river).

A monitoring programme was carried out in the USA to evaluate the significance of priority pollutants in urban stormwater run-off (Cole et al., 1984). Samples were collected from 51 catchments in 19 cities or metropolitan areas covering a variety of climatic regimes and a range of size and population densities. The most common land uses were residential and commercial. Naphthalene was detected in 9 out of 80 samples collected (11%) and the range of concentrations detected was 0.8-2.3 µg/l.

Hoffman et al. (1984) collected urban runoff samples from 4 storm drains each serving a different land use and analysed them for selected PAH. The loading factors for residential and commercial areas were less than those in industrial and highway areas.

Maltby et al. (1995) investigated the effects of motorway run-off on several small streams receiving drainage from the M1 motorway in the UK. At each site 1 sampling station was less than 400 m upstream of the point of entrance of the run-off and 1 sampling station was less than 100 m downstream of the point of entrance. Sediment, stream water and outfall water were collected from 3 streams at 3 monthly intervals from October 1990 to July 1991. Even though motorway runoff draining into the study areas was contaminated with aromatic hydrocarbons, dilution by receiving water meant that concentrations in stream water were below the detection limit. High naphthalene concentrations were also reported for sediments taken from these sites as discussed in Section 3.1.3.5.

Naphthalene concentrations in influent and effluent of 10 wastewater treatment works in the Great Lakes basin were measured (Michael et al., 1991). At two works naphthalene was not detected but at the other works naphthalene concentrations in the influent ranged from not detected to 6 µg/l and in the effluent all concentrations were below 1 µg/l.

Influents, effluents and raw sludges from publicly owned treatment works (POTWs) were analysed for naphthalene (US EPA, 1982). Naphthalene was detected in 142 out of 287 influent samples in the range 1-50 µg/l, but was only found in 17 out of 302 effluent samples at levels between 1 and 24 µg/l. Levels in 149 out of 437 raw sludges ranged from 1 to 5,200 µg/l.

Samples of 63 effluents and 22 intake waters were collected from a wide range of chemical manufacturers in areas across the USA (US EPA, 1979c). Naphthalene was detected in 4 of the

63 effluents (naphthalene concentrations in 3 of these were <10 μg/l and in the other were between 10 and 100 μg/l).

Surface water samples were collected from four points along a stream in Louisiana (Bayou Bonfouca) which is heavily contaminated with polycyclic aromatic hydrocarbons (Catallo and Gambrell, 1987). The four sites were along an apparent gradient of creosote pollution, following a fire at a wood products treatment plant. Naphthalene was not detected in the uncontaminated site and levels in the moderately, heavily and extremely contaminated sites were 8.3 mg/l, 0.7 mg/l and 14.1 mg/l, respectively.

Naphthalene was measured in the influent and effluent of stabilisation ponds used for wastewater treatment in China (Shugui et al., 1994). Naphthalene was detected in the influent on 4 out of 9 sampling occasions but was not detected in any effluent samples.

Water samples from streams draining agricultural areas of the San Joaquin valley and from the San Joaquin river were analysed and found to contain 1.0-4.4 ng/l naphthalene (Pereira et al., 1996).

Summary

The highest concentration reported for surface water in the UK is 6.85 μg/l (the highest measured value for the Tees estuary in the vicinity of an effluent outfall from a steel works). Respective maximum concentrations of 1.3 μg/l and 2.24 μg/l have been reported in the Besós and Adige rivers in Spain and are associated with urban or industrial areas. Values of up to 2.3 μg/l have been measured in urban storm water run-off. Concentrations of up to 14.1 mg/l have been recorded for heavily contaminated sites. Levels of naphthalene in unpolluted surface water range up to 5 ng/l.

3.1.3.2.2   Groundwater

Groundwater samples were collected from the Great Ouse catchment area (UK) near a petrol storage facility (Tester and Harker, 1981). The concentration of naphthalene in the water decreased with increasing distance from the petrol storage and ranged from <0.01 μg/l (180 m from storage) to 155 μg/l (20 m from storage).

Analyses of groundwater from abandoned waste disposal sites overlying unconsolidated aquifers in Western Germany were evaluated by Kerndorff et al. (1992). Naphthalene was detected in 15 out of 124 samples. The maximum concentration was 12.6 μg/l and the mean was 2.2 μg/l.

Naphthalene levels in uncontaminated groundwater in The Netherlands were described by Zoetman (1981) as ranging from <0.005 to 0.03 μg/l. Naphthalene was amongst those compounds found most frequently in the vicinity of waste dumps and landfill sites; a value of 30 μg/l was given but without any indication if this was a maximum or a mean (Luijten and Piet, 1983).

Concentrations of naphthalene were measured in groundwater samples at five Ontario landfill sites (Barker, 1987). The concentrations ranged from <0.2-63 μg/l.

Groundwater samples from a landfill site contained levels of naphthalene up to 2.0 μg/l (Barker et al., 1988). Naphthalene concentrations in the landfill leachate ranged from 10.7-132 μg/l.

Naphthalene levels between 3 and 110 μg/l were found in samples of groundwater from 4 wells near a landfill site while naphthalene was not detected at background sites (Reinhard et al., 1994).

Groundwater quality at an abandoned creosote facility was studied over a two-year period (Bedient et al., 1984). Naphthalene was not detected in three wells. In 12 other wells naphthalene concentrations ranged from 1.4-3,490 μg/l with 3 wells nearest the waste pond having levels of naphthalene >800 μg/l.

Groundwater contamination at Gas Works Park on the site of a disused coal and oil gasification plant in Seattle was studied (Turney and Goerlitz, 1990). Naphthalene concentrations ranged from below the detection limit (0.005 mg/l) to 12 mg/l.

Groundwater from an underground coal gasification plant in Texas was sampled (Mattox and Humenick, 1980). Naphthalene concentrations before, immediately after and 1 year after gasification ceased were 2, 70 and 7 μg/l, respectively.

A town gas production site in Stroudsberg, Pennsylvania, was studied by Villaume (1984). It was operated from the mid-1800s until around 1939. Almost pure coal tar residue was found on the site, containing naphthalene at 36 g/l. The highest concentration measured in groundwater was 3.525 mg/l. The concentration of naphthalene in groundwater dropped rapidly outside the area of the free coal tar plume.

Samples of groundwater were taken before and during *in situ* coal gasification at two locations in the USA (Pellizzari et al., 1979). Levels of naphthalene were found to increase from 2.7 μg/l and 0.35 μg/l before gasification to 640 μg/l and 1,707 μg/l during gasification.

Three groundwater samples near 2 underground coal gasification sites in Wyoming 15 months after gasification ceased had naphthalene levels of 1800, 740 and 380 μg/l (Steurmer et al., 1982). A groundwater cleanup operation was carried out at this site during 1986-9 (Renk et al., 1990). Groundwater was pumped from the ground, passed through activated charcoal and reinjected into the cavity. Approximately 6.4 million gallons of contaminated groundwater was treated and samples were taken at various intervals and analysed. Naphthalene concentrations in three wells fell from 1800, 740 and 380 μg/l in January 1981 to 5, 150 and 60 μg/l in October 1989.

Naphthalene was found in groundwater samples from 6 out of 17 monitoring wells on the 5,000 acre Federal Aviation Administration Technical Centre in New Jersey (Superfund Record of Decision, EPA Report, 1989). The soil and groundwater were contaminated with volatile organic compounds apparently attributable to the jet fuel farm on site. The maximum naphthalene concentration measured was 1,000 μg/l and the average was 79.82 μg/l.

Groundwater sampled from the area near the Cliffs Dow Site in Michigan contained levels of naphthalene up to 210 μg/l (ATSDR, 1988). The site had received charcoal scraps and wood tars from the closed Cliffs-Dow charcoal manufacturing facility nearby.

Groundwater which had been contaminated by wood-preserving chemicals at Pensacola, Florida contained naphthalene levels up to 15.3 mg/l (Goerlitz, 1992).

Samples of groundwater were taken from a site that had been used for the production of charcoal and pine-tar products from 1930s to 1967 (McCreary et al., 1983). A wood-treating facility and railroad tracks also existed near this area. Naphthalene concentrations measured were semi-

quantitative since recovery efficiencies were not known. In water samples the concentration ranged from 40 to 860 µg/l.

Groundwater from 10 septic tank systems was sampled and analysed for inorganic ions, bacteria, viruses and trace level organics including naphthalene (Tomson et al., 1984). Samples from several locations throughout the USA were sampled. Naphthalene was detected at levels up to 0.833 µg/l.

Samples of groundwater contaminated by disposal of secondary sewage effluent were analysed for volatile organic compounds (Barber et al., 1988). The study site was on an air base near Falmouth, MA. Secondary treated sewage with a composition typical of municipal and domestic effluent was discharged to sand beds and percolated to the water table. Groundwater samples were collected from several wells, and naphthalene was detected in several wells with a maximum concentration of 10 ng/l.

Naphthalene has been found in groundwaters at land application waste treatment sites in Louisiana (Hutchins and Ward, 1984) at levels between 0.003 and 0.22 µg/l.

Summary

Concentrations in groundwaters in the vicinity of contaminated sites range up to 15.3 mg/l. The majority of these were not sites at which production or direct use of naphthalene took place. Levels in uncontaminated groundwater are less than 0.03 µg/l.

3.1.3.2.3   Precipitation

Surface snow samples were collected from a 3 m snowpit at the summit of the Greenland ice sheet in summer 1991 (Jaffrezo et al., 1994). The samples covered the previous 4 years of deposition. Concentrations of polycyclic aromatic hydrocarbons in the soluble fraction of the samples were all below the detection limit of a few pg/kg. However, naphthalene was detected in the insoluble fraction which is essentially the same as those present in aerosol at this location. Naphthalene was found at concentrations ranging from 42 to 498 pg/kg with maximum concentrations occurring in winter samples.

Samples of rain and snow were collected during winter, spring and summer 1985 at an urban site in Switzerland (Czuczwa et al., 1988). Levels of naphthalene in Winter rain ranged from 44-210 ng/l and in snow from below the detection limit to 39 ng/l. During Spring and Summer naphthalene concentrations ranged from 20-43 ng/l.

Snow samples were collected from two sites along a roadside where the average traffic density was approximately 9,100 motor vehicles per day (Hautala et al., 1995). Samples were collected at 10 m and 30 m from the roadside at an open field site and a forest site. Naphthalene concentrations at 10 m and 30 m from the road in the open field site were 0.24 ± 0.09 µg/m^2/month and 0.91 ± 0.28 µg/m^2/month, respectively. For the forest site, naphthalene concentrations at 10 m and 30 m were 0.13 ± 0.05 µg/m^2/month and 0.10 ± 0.01 µg/m^2/month, respectively.

Samples of Los Angeles and Portland rain were collected (Pankow et al., 1983). A desorption cartridge and an extraction cartridge were used in parallel in Oregon and levels of naphthalene measured were 46 and 30 ng/l, respectively. During a different rain event levels were 120 ng/l using the desorption cartridge and 370 ng/l using the extraction cartridge. Further rain and air sampling was carried out during 7 rain events in Oregon (February-April 1984) (Ligocki et al.,

1985). The mean dissolved rain concentration of naphthalene was 100 ± 32 ng/l (range 46-140 ng/l) and the mean gas phase concentration of naphthalene was 450 ± 220 ng/m^3 (range 280-940 ng/m^3).

Naphthalene was identified in rain and snow samples collected in Southern California in 1982 and 1983 (Kawamura and Kaplan, 1986).

Summary

Low levels of naphthalene may be present in precipitation with concentrations ranging up to 370 ng/l.

3.1.3.2.4   Drinking water

Naphthalene in Nordic tap water ranged from 1.2-8.8 ng/l (Kveseth et al., 1982).

Samples of Lake Zurich water and tap water, taken in 1973, were analysed for naphthalene (Grob and Grob, 1974). The tap water is mainly pumped from Lake Zurich (70%) with the remainder coming from groundwater streams. Naphthalene concentrations were 8 ng/l in surface lakewater, 52 ng/l in lakewater from a depth of 30 m and 8 ng/l in tap water.

LeBel et al. (1987) sampled five Great Lakes drinking water supplies, both raw and treated water. They also sampled at one site in the same area with a groundwater drinking supply to act as a control. Sampling occurred once in Summer 1982 and once in Winter 1983. Their results are in **Table 3.24**.

Table 3.24 Levels of naphthalene in Great Lakes drinking water (ng/l)   (LeBel et al. 1987)

Location	Summer		Winter	
	Raw	**Treated**	**Raw**	**Treated**
Amherstburg	3.9	5.2	23.2	20.0
Burlington	15.1	9.8	12.8	4.1
Cornwall	3.4	40.5	5.9	4.2
Fort Erie	3.5	5.6	6.6	9.9
Sault Ste Marie	48.9	101.0	95.7	23.7
Barrie (g'water)	14.5	16.8	9.4	8.4

The authors did not discuss reasons for the increase in the level of naphthalene after treatment at some of the locations.

Drinking water supplies in twelve Great Lakes municipalities were analysed for selected organics (Williams et al., 1982). Naphthalene concentrations varied from 3.4-1,271 ng/l.

Samples of drinking water from five municipalities in eastern Ontario (Canada) were collected in June and October 1978 and analysed for polycyclic aromatic hydrocarbons (Benoit et al., 1979). Levels of naphthalene ranged from 0.6 to 7.5 ng/l.

Meyer (1983) measured naphthalene levels in private drinking water wells near a chemical waste dump in Hardeman County, Tennessee, USA. Naphthalene was found in 11 out of 31 samples at levels from a trace up to 6.7 µg/l, the median result being not detected.

Naphthalene concentrations in drinking water from the Asaka water treatment plant, Japan, ranged from 0.001-0.060 µg/l between April 1988 and March 1989 (Onodera, 1991) with the higher concentrations occurring in the warmer months.

Naphthalene was found in the Kitakyusha water supply, Japan, at a level of 2.2 ng/l (Akiyama and Koga, 1983).

Summary

The highest naphthalene level reported in drinking water is 6.7 µg/l from wells near a chemical waste dump in the US. Concentrations up to 1,271 ng/l have also been reported for supplies in 12 US Great Lakes municipalities. Concentrations measured at other locations range up to about 100 ng/l.

3.1.3.2.5   Summary of measured levels in water

There are a large number of reported levels data for water. Although these do not come from detailed monitoring studies, when taken together they do give a good picture of levels in the aquatic compartment.

Levels of naphthalene in unpolluted surface waters range up to 5 ng/l and in other areas levels up to a few µg/l have been found. In one heavily contaminated area (the site of extreme contamination by creosote following a fire at a wood treatment plant) naphthalene levels up to 14.1 mg/l have been found.

Levels of naphthalene in uncontaminated groundwater range up to 0.03 µg/l. Many measurements are available for contaminated sites where levels ranged up to 15.3 mg/l.

Naphthalene levels in precipitation and drinking water range up to 370 ng/l although elevated levels have been found in drinking water samples from wells near a chemical waste dump in the US.

Some measurements of naphthalene in marine waters are available and the highest value found was 300 ng/l.

3.1.3.3   Comparison of PEC with measured levels

The continental and regional PECs for surface water are consistent with measured values in surface water that is either unpolluted or little affected by industry or urbanisation. Local PECs derived from site-specific information for production and use range up to 0.31 µg/l, which is consistent with measured levels in surface water associated with urban and industrial sources but is lower than the peak measured levels. Calculated PECs based on worst-case emissions for mothballs manufacture and grinding wheel production are higher than those for production and use but are significantly lower than measurements taken at contaminated sites.

In making comparisons with PNECs the highest measured level in background surface water for an industrial or urban area of 2.24 µg/l, the maximum level of 6.85 µg/l for the Tees estuary close to an indirect source of naphthalene and a value of 14.1 mg/l for surface water in the

vicinity of a heavily contaminated site will be used. None of the measured levels are clearly associated with the production or use of naphthalene as a substance. Most of the high levels of naphthalene reflect general urban and industrial activity in which there is likely to be significant indirect production of naphthalene (traffic, combustion etc). Site-specific and calculated PECs have therefore been taken as being more representative of naphthalene industries and will be used alongside measured values. Comparisons of PECs and measured values with the PNEC for water are discussed in Section 3.3.

3.1.3.4   Calculation of Predicted Environmental Concentration for sediment

The values for the PEClocal$_{water}$ can be used to calculate the PEClocal$_{sediment}$ using the equilibrium partitioning method in the Technical Guidance. The PEClocal$_{sed}$ values calculated for the various processes are given in **Table 3.25**. The EUSES model has been used to calculate regional and continental PECs for sediment.

3.1.3.5   Measured levels in sediment

A summary of measurements of naphthalene in sediment samples is given in **Table 3.26**.

Sediments were collected from various parts of the river Seine in the urban and industrial zone of Paris and their naphthalene content measured (Ollivon et al., 1995). In October 1991 sediment samples were collected from 13 locations along the river. Naphthalene was detected in 8 of these samples at up to 0.3 µg/g. In 1992, coring was carried out at three sites near a sewage treatment plant and suspended matter traps were used. The naphthalene concentration in the core samples ranged from 0.06 to 0.75 µg/g and in the suspended matter up to 1.47 µg/g. Suspended matter was collected in 1991 from 2 locations near storm outfalls and 1 location upstream for reference purposes. At the reference location, naphthalene was not detected before rainfall and was detected but not quantified after 4 mm rain. Naphthalene concentrations in suspended matter before rain in the other two locations were 0.16 µg/g and after rain rose to 0.23 and 0.68 µg/g, respectively.

Table 3.25 Local, regional and continental PEC's for sediment

Process	PEClocal$_{water}$ (µg/l)	PEClocal$_{sed}$ (µg/kg)
Production Site A	0.036	1.01
Production Site B	0.20	5.6
Production Site C	0.059	1.65
Production Site D	0.31	8.7
Production Site E	0.04	1.12
Production Site F	0.05	1.4
Production Site G	0.035	0.98
Production Site H	0.055	1.54
Production Site I	0.07	1.96
Production Site J	0.035	0.98
Phthalic anhydride production – site specific	0.04	1.1
Use as intermediate	0.042	1.2
Pyrotechnics manufacture	2.35	65.8
Mothballs manufacture	0.03	0.83
Grinding wheels manufacture – worst case	294	8232
Regional	0.03	1.0
Continental	0.025	0.095

Table 3.26 Levels of naphthalene in sediments

Location	Level	Reference
North-Eastern Irish Sea	6.4-91 ng/g (dry weight)	Law et al., 1989
Rhône delta and Western Mediterranean	delta 4.3-13.5 ng/g open sea 0.5-10.9 ng/g (dry weight)	Lipiatou and Saliot, 1991
Detroit River, USA	detected in 15/31 sites range 0.13-1.80 mg/kg	Fallon and Horvath, 1985
Mouths of 3 passes of Lake Pontchartrain, USA	Inner Harbour 0.9 ng/g Chef Menteur 3.4 ng/g Rigolets 3.0 ng/g	McFall et al., 1985b
South Texas, USA	trace amounts < 10 ng/g	Bedinger and Nulton, 1982
Maryland waters of Chesapeake Bay, USA	0.16-3.61 µg/g	Foster and Wright, 1988
Puget Sound, USA	0.02-0.42 µg/g	Riley et al., 1980
Brisbane river estuary	0.04-0.16 µg/g	Kayal and Connell, 1989
Mina al Fahal coast	31.36-69.30 ng/g	Badawy et al., 1993
Arabian Gulf	0.01-7.14 ng/g	Al-Saad, 1987
Marsh sediments, Iraq	0.01-0.09 ng/g	Al-Saad and Al-Timari, 1989
River Rhône, France	50 -720 µg/kg	Larbaight, 1997

Maltby et al. (1995) investigated the effects of motorway run-off on several small streams in the North of England. The study examined the "worst-case" scenarios so sampling stations were immediately downstream of discharges into small watercourses. Seven streams receiving drainage from the M1 motorway were sampled with 1 sampling station less than 400 m upstream of the point of entrance of the run-off and 1 sampling station < 100 m downstream of the point of entrance. Sediment samples were collected from all 14 sampling stations during July and August 1990. Sediment, stream water and outfall water were collected from 3 streams at 3 monthly intervals from October 1990 to July 1991. Sediment samples were also collected from these 3 sites in April 1993. Results of the study indicated that the concentration of aromatic hydrocarbons in sediments was elevated at all sites receiving motorway drainage. At the most contaminated site, which was examined in more detail, the concentration of naphthalene in sediment upstream was 0.017 µg/g wet weight and downstream was 0.52 µg/g wet weight. Naphthalene was found at 2.28 µg/g wet weight and 2.89 µg/g wet weight in sediments from 2 other sites examined in more detail.

Sediments from four contaminated Great Lakes tributaries and a reference site were analysed for naphthalene (Fabacher et al., 1991). The naphthalene concentration in the reference site was 0.12 µg/g and in the industrial sites ranged from 0.08-3.8 µg/g on a dry weight basis.

Surface sediment samples from a contaminated stream (Bayou Bonfouca in the USA) were analysed for polycyclic aromatic hydrocarbons (Catallo and Gambrell, 1987). The stream was contaminated following a fire at a wood treatment plant nearby. Four sites with different degrees of contamination were sampled and the naphthalene concentrations were up to 7,720 µg/g.

Naphthalene was found in sediments of a discharge stream near the site of creosote contamination (Elder and Dresler, 1988). Levels of naphthalene at two sites were 300 µg/kg and 200 µg/kg. Naphthalene was not detected at two sites in Pensacola Bay, USA.

Baumann et al. (1982) took sediment samples from the vicinity of a coke plant outfall. The sample was a non-homogeneous mixture of clay soil, sand and a black tar substance. Naphthalene was present at 31 µg/g.

Sediments were collected from the North Sea and Norwegian and Swedish fjords (Sporstøl et al., 1983) and analysed for naphthalene. Concentrations, on a dry weight basis, in the North Sea were 4.32 ng/g at a distance of 10 km from an oil field and 31.6 ng/g at a distance of 500 m from the oil field. Concentrations in fjord sediment samples ranged from 2,870 ng/g near a ferro alloy plant to 41.5 ng/g at 15-20 km from an industrial plant.

Sediment samples were taken near the Iona Island sewage outfall (Rogers and Hall, 1987). At 0.5 km from the outfall a trace (<15 ng/g) of naphthalene was found and at 1.0 km the naphthalene concentration was 20 ng/g (dry weight).

Naphthalene was measured in surface sediments at 21 locations in Commencement Bay and the Tacoma Waterways in Washington (Schults et al., 1987). Naphthalene concentrations on a dry weight basis were 44-81 µg/kg in the waterways, 90-546 µg/kg at the entrance of the waterways and 67-128 µg/kg in the bay. At a reference site naphthalene was not detected (detection limit approximately 10 µg/kg).

Pruell and Quinn (1985) collected surface sediment samples (top 1 cm) from three sites along a pollution gradient in Naragansett Bay, USA. The concentrations found were 72.3 ng/g at the Providence River (head of the bay), 13.3 ng/g in mid-bay and 4.4 ng/g in Rhode Island Sound.

Sediment samples were collected from various sites in the Washington DC area during periods of high and low boating activity (Mastron et al., 1994). More sites were found to contain naphthalene in sediments collected during peak boating activity than during low boating activity. The average concentration of naphthalene in all sediment samples was approximately 750 µg/kg.

Sediment samples from 23 beaches located along shipping lanes in the Strait of Juan de Fuca, San Juan Islands and Northern Puget Sound were analysed for naphthalene (US EPA, 1979d). Sediments from 18 sites contained naphthalene during spring and/or summer with the highest concentrations being 4 ng/g (dry weight). Naphthalene was not detected in fall and winter samples.

Naphthalene was only detected in sediment from one station out of 30 sampled in Casco Bay, Maine in 1980 (Larsen et al., 1983). The concentration of naphthalene measured was 113 ng/g on a wet weight basis.

Naphthalene was found to be a major compound in surface sediments of the Elizabeth River sub estuary (Bieri et al., 1986).

Naphthalene was not detected (detection limit 10 ng/g) in surficial sediment samples from the continental shelf of Tabasco state, Mexico (Botello et al., 1991).

Naphthalene levels of 2.3-15 ng/l were found in suspended sediments from streams draining the San Joaquin valley and from the San Joaquin river (Pereira et al., 1996).

Summary

Measured levels of naphthalene in sediments are available from a wide range of locations. Naphthalene was widely detected in surface water sediments with maximum levels exceeding 100 µg/kg in many of these studies. Levels in sediments from estuarine and coastal sites were generally lower, although peak values again exceed 100 µg/kg. There are no widespread uses of naphthalene to account for its widespread occurrence and the high values were not associated with sites where naphthalene itself is produced or used. The majority of the high values (up to 7,720 mg/kg) found are related to contamination, for example by creosote, where naphthalene is a component in the mixture. Naphthalene is also found in the sediment from motorway run-offs and near sites of boating activity as a result of the combustion processes in car and boat engines. The highest value reported for urban areas is 1.47 mg/kg for suspended matter at a site near a sewage treatment plant. Other values for urban areas ranged up to 720 µg/kg and are likely to arise from combustion processes. Values of up to 520 µg/kg have been reported for motorway run-offs.

3.1.3.6   Comparison of PEC with measured levels for sediment

The highest PEC calculated using site-specific information for production and use of naphthalene is 8.7 µg/kg. This is consistent with the range of measured values although none of the locations studied are known to be close to sites where naphthalene is produced or used. Measured levels of naphthalene in sediment range up to 7.72 mg/kg at a contaminated site. However, these values are not considered to be representative of levels in the vicinity of producers and users of naphthalene itself so the calculated PNECs will be used in the risk characterisation.

3.1.4 Terrestrial compartment

3.1.4.1 Calculation of Predicted Environmental Concentration in soil

3.1.4.1.1 PEC$_{soil}$

Direct release of naphthalene to soil from point sources is small and has been estimated for naphthalene production and use of naphthalene as an intermediate and in the production of phthalic anhydride. Release to soil may occur from treated wood products although this release has not been quantified. Naphthalene is expected to absorb to soil and sediment to a moderate extent. The PEClocal has been calculated for soil using the equation in the Technical Guidance Document.

On the local scale, concentrations in soil and groundwater are principally estimated as long-term steady state concentrations due to atmospheric deposition and/or application of sludge from a sewage treatment plant. Direct application of chemicals is not taken into account. The calculation also takes into account removal of the substance by biodegradation, volatilisation and leaching to deeper soil layers. The releases of naphthalene to air and water were used to estimate the concentration due to deposition and sludge application. Concentrations of naphthalene in agricultural soil have been estimated for naphthalene production and use as an intermediate and are given in **Table 3.27**. Unless information is given to the contrary it has been assumed that wastewater is treated in an STP with the default characteristics specified in the Technical Guidance Document and that sludge is applied to soil. However, this is unlikely to be the case in dedicated plants and the values given in **Table 3.27** are likely in some cases to be overestimates.

The EUSES model and parameters described earlier have been used to calculate the regional and continental PECs for the terrestrial compartment (see Appendix 1). Based on these estimates, the following regional and continental concentrations were calculated:

PECregional (μg/kg):	Natural soil	0.122
	Agricultural soil	0.296
PECcontinental (μg/kg):	Natural soil	0.025
	Agricultural soil	0.023

3.1.4.1.2 PEC$_{soil, porewater}$

The PEC for soil porewater has been calculated using the equation in the Technical Guidance document. PECs have been calculated using the site-specific information on the releases to air and water where this is available. Unless information is given to the contrary it has been assumed that wastewater is treated in an STP with the default characteristics specified in the Technical Guidance Document and that sludge is applied to soil. However, this is unlikely to be the case in dedicated plants and the values given in some cases are likely to be overestimates.

The EUSES model and parameters described earlier have been used to calculate regional and continental PECs for the soil porewater. The regional and continental concentrations estimated for soil porewater are given in **Table 3.27** along with the calculated local PECs for soil.

Table 3.27 PECs calculated for agricultural soil

Process	PEClocal$_{soil}$ (µg/kg)	PEC$_{soil, porewater}$ (µg/l)
Site A	1.7	0.078
Site B	0.15	0.007
Site C	0.45	0.02
Site D	0.55	0.025
Site E	0.35	0.016
Site F	0.19	0.009
Site G*	0.29	0.012
Site H*	0.96	0.039
Site I	0.32	0.014
Site J	0.14	0.006
Phthalic anhydride production (site specific)	1.0	0.047
Use as intermediate (generic)	50	1.8
Pyrotechnics manufacture	36	1.3
Mothballs manufacture	1.0	0.046
Grinding wheels manufacture	4,600	161

* No information is available regarding the fate of sludge from wastewater treatment plants servicing these sites. However, preliminary assessments for these sites indicated that the application of sludge from these plants to soil would not lead to adverse effects (see Section 3.3.2) and so steps to obtain further information were not taken. It is unlikely that such solid waste is applied to soil and the PECs calculated for these sites are likely to have been overestimated. For all other production sites solid waste arising from wastewater treatment plants is known to be either incinerated or sent to landfill.

3.1.4.2   **Measured levels in soil**

Archived soil samples from the Rothamstead Experimental Station were analysed for naphthalene (Jones et al., 1987). From 1846 to 1914 there was an increase from 39 to 53 µg/kg dry weight. The naphthalene levels in the samples from 1956 and 1980 were 28 and 23 µg/kg dry weight, respectively. Samples of soil from 27 sites in Wales were also analysed for naphthalene. Naphthalene levels ranged from 0-28 µg/kg dry weight with a mean of 4.9 µg/kg and means of 2.4, 3.8 and 11.3 µg/kg for rural, remote and urban areas, respectively.

Surface soil samples from 49 sites in Wales had a mean naphthalene concentration of 35 µg/kg dry weight with a range of <1.0-1,000 µg/kg (Jones et al., 1989a). Typical values (excluding selected sites in coal-mining and remote areas) ranged from <1.0-131 µg/kg with a mean of 8.7 µg/kg.

Concentrations of naphthalene in soil samples from an experimental plot at Rothamstead were measured (Jones et al., 1989b). The plot had received no fertilisers or amendments since 1843 and samples were taken from various depths. Archived samples from 1893 and 1944 were compared with samples collected in 1987 and naphthalene concentrations in the top 23 cm were 49, not detected and 20 µg/kg dry weight, respectively. For soils from 23-46 cm depth naphthalene was present at 9.7 µg/kg dry weight in the 1893 sample and was not detected in the 1944 and 1987 samples.

Surface soils were collected from forested areas of Southern and Central Norway away from local sources and naphthalene concentrations were found to range from 4 to 110 µg/kg dry weight (Aamot et al., 1996).

Samples of soil from a reference site and the site of a garage and a waste disposal site in the Netherlands were found to have naphthalene levels of 11 mg/kg at the reference site, 15 and 150 mg/kg at the garage site and 45 mg/kg at the waste disposal site (Kliest et al., 1989).

Soil samples were collected from several hazardous waste dumping sites located in the Besós and Llobegrat basin in Catalonia, Spain (Navarro et al., 1991). Naphthalene was only found at one site in the Besós basin at a depth of 2.4 m at a level of 53 µg/g.

Naphthalene was measured in rice paddy soil where clean water and wastewater had been used for irrigation (Ou et al., 1992). Where clean water was used, 0.014 mg/kg naphthalene was found in soil and where wastewater was used 0.045 mg/kg naphthalene was found.

Naphthalene was not detected in any samples of soil from a waste pit for produced water in the Duncan oil field, New Mexico (Davani et al., 1986).

Soil samples from a wood preserving plant contained naphthalene at a maximum of approximately 400 mg/kg dry weight at a depth of 14 feet below the surface (Ball, 1987).

Soil from an abandoned creosote facility, in Texas, had levels of naphthalene of 3.74 mg/kg at 0.7-1.8 feet below the surface and 0.003 mg/kg at 26.5 feet below the surface (Bedient et al., 1984).

Soil samples were collected from 12 locations in Norway six of which were within 1,000 m of an aluminium plant or a ferrosilicon works (Vogt et al., 1987). Average concentrations of naphthalene in polluted, unpolluted and bog soil were 48.3, 46.2 and 57.7 ng/g.

Four soil samples representing different levels of pollution from the site of a former gasworks site in Denmark were found to contain between 0.4 and 3,120 µg/g (dry weight) of naphthalene (Lindhardt et al., 1996).

Soil samples were collected from 8 agricultural fields receiving effluent from an oil refinery in India were found to contain 1.9-250 µg/kg of naphthalene (Ashok et al., 1995).

Ten soil samples were collected from Gas Works Park, Seattle which was built on the site of a disused coal and oil gasification plant (Turney and Goerlitz, 1990). Naphthalene was detected in 5 of these samples at concentrations ranging from 0.037-46 mg/kg.

Samples of soil from the site of the disused West Melbourne Gas Works, Australia, contained naphthalene at levels of 0.6-32.9 mg/kg (Parker and Wolfe, 1990).

Soil samples from a site in Washington used for processing and handling of animal by-products, solvents, acids, industrial wastes and storage of hazardous wastes were collected (Aldis et al., 1983). Naphthalene was found in 3 out of 30 soil samples and the highest level found was 5.2 mg/kg.

Four contaminated soils from samples collected from former manufactured gas plant sites were analysed using batch extraction, soxhlet extraction and sonication extraction followed by GC analysis (Chen et al., 1996). The level of naphthalene in these soils ranged from approximately 5 mg/kg to approximately 4,500 mg/kg (levels read from graphs).

Soil samples from the site of a blow-out at an oil field were analysed for naphthalene (Kaplan et al., 1996). Naphthalene levels in surface soil samples from the control, lightly oiled and highly oiled sites were 0.7 ng/g, 3.5 ng/g and 10.2 ng/g, respectively.

Naphthalene has been detected in two sewage sludge samples from the UK (Crathorne et al., 1989). One treatment plant receives approximately 80% industrial waste and the other treats mainly (90%) domestic waste. Levels of naphthalene were not quantified.

Sewage sludge samples were collected monthly from two sewage treatment plants in the UK for seven months, one receiving mainly domestic effluent and one receiving mainly industrial effluent (Sweetman et al., 1992). Naphthalene concentrations in digested sludge from the treatment plant receiving mainly domestic effluent range from 0.77 to 2.97 mg/kg dry weight (mean 1.78 mg/kg dry weight) and in sludge from the treatment plant receiving industrial effluent the range of naphthalene concentrations was 0.49-14.88 mg/kg dry weight (mean 7.42 mg/kg dry weight).

Naphthalene was found in 60% of 15 Canadian sludges from various regions (Webber and Lesage, 1989). The concentrations found range from a trace to 5.8 mg/kg (dry weight).

Sampling of influent, effluent and sludge streams was carried out for 6 days at 20 publicly owned treatment plants in the USA (Naylor and Loehr, 1982). Naphthalene was detected 9 times in combined sludge and the concentration range was 0.9-70 mg/kg dry weight (median 7.5 mg/kg dry weight).

Summary

Although there are no detailed monitoring studies of naphthalene levels in soil, there are several reported levels that give a good indication of levels in the terrestrial compartment. In uncontaminated soils, mean naphthalene levels are around 10-20 µg/kg (with peak values up to 131 µg/kg). Much higher levels have been found on contaminated sites, the highest found being 400 mg/kg. The vast majority of the sites are contaminated with naphthalene indirectly, for example on gasworks sites or hazardous waste dumps. No values are available for locations close to the production and use sites of naphthalene itself.

3.1.4.3   Comparison of PEC with measured levels

The regional and continental PECs calculated for naphthalene are lower than measured levels. The local PECs calculated for production and use of naphthalene based on site-specific data also tend to be lower than most of the measured levels although the highest local PECs are within the range of measured values.

For this assessment, the mean naphthalene concentration measured in soil of 20 µg/kg will be taken as representative of non-contaminated sites. This value will be used together with the calculated local PEC's of 55.3 µg/kg for use as an intermediate based on default data and 2.29 µg/kg for a site for which specific data are available as given in **Table 3.27**.

3.1.5 Atmosphere

3.1.5.1 Calculation of Predicted Environmental Concentration in air

Regional and continental PECs calculated using the EUSES model (see Appendix 1) are given in **Table 3.28** together with PECs for local releases.

Table 3.28 Local PECs calculated for the atmospheric environment

Process	$PEC_{local(air)}$ ($\mu g/m^3$)
Site A	0.30
Site B	0.19
Site C	0.46
Site D	0.38
Site E	0.28
Site F	0.25
Site G	0.24
Site H	0.64
Site I	0.39
Site J	0.18
Phthalic anhydride production - site specific	0.90
Use as an intermediate (worst case)	1.1
Pyrotechnics manufacture	0.15
Mothballs manufacture	0.90
Grinding wheels manufacture	1.9
Regional	0.14
Continental	0.028

3.1.5.2 Measured levels in air

Results from a number of studies reporting levels of naphthalene in air at remote and urban sites are shown in **Table 3.29**.

Table 3.29 Levels of naphthalene in air

Location	Level (ng/m³)	Comments	Reference
Kiel Bight	2.2-26.0	remote site	Bouchertall, 1986
US-Canada border	2.4 + 1.6 3.2 + 1.2	September January	Hoff and Chan, 1987
US various: rural urban site dominated	ND 120-940 380-8,400	First-third quartile range	US EPA, 1983
Oslo: traffic affected traffic free	63 31 51 14	winter summer winter summer	Larssen, 1984
Los Angeles basin	2,800 3,300	Night day	Arey et al., 1987

Measurements of volatile organic compounds in outdoor air in the Grenoble area were carried out (Foster et al., 1991). During one week in winter and one week in summer average naphthalene concentrations were 2.65 and 4.40 $\mu g/m^3$, respectively. Measurements carried out over one year had an average naphthalene concentration of 5.99 $\mu g/m^3$.

Naphthalene was detected in air samples collected in a 45-year-old heavily damaged spruce forest (Helmig et al., 1989). Samples of air collected from the Black Forest also contained naphthalene (Jüttner, 1986).

Alfheim et al. (1985) sampled urban air close to busy roads in Oslo in 1981. From four sites the mean concentrations measured were 473, 160, 350 and 1,130 ng/m^3.

Schröder and Dannecker (1994) measured naphthalene concentrations in four areas of Germany. Naphthalene was found at a much higher level at an industrial site near a busy highway than at sites in a medium and a small town. Naphthalene was not detected at a rural site.

Soil and wind-blown surface and roadside dusts were taken at various locations in Birmingham (UK) and Lahore (Pakistan) and analysed for polynuclear aromatic hydrocarbons (Smith et al., 1995). In Birmingham, the highest concentration of naphthalene was 720 $\mu g/kg$ detected in roadside dust from a road tunnel in central Birmingham. Roadside dust from a busy spine road in Birmingham and a road on the university campus contained 300 $\mu g/kg$ naphthalene while soil from the university campus contained 27.2 $\mu g/kg$ naphthalene. In various locations in Lahore, concentrations of naphthalene in roadside dust and soil samples ranged from 6.04 to 36.2 $\mu g/kg$. The results suggest that vehicular emissions are a major source of polycyclic aromatic hydrocarbons in urban areas.

Particulate and vapour phase PAHs were collected in winter (February) and summer (August) from an urban location in Birmingham, UK (Harrison et al., 1996). The total naphthalene found in the winter sample was 13.24 ng/m^3 and in the summer sample was 1.87 ng/m^3.

Raymond and Guiochon (1974) measured hydrocarbon levels in the air above the roof of their laboratory in Paris. They found naphthalene levels of 3.8 to 11.2 $\mu g/m^3$.

Naphthalene levels in air at 10 locations in the Netherlands during 1992 have been reported (RIVM, 1993). The mean levels of naphthalene range from 0.04 to 0.61 $\mu g/m^3$ in rural and street samples, respectively.

Concentrations of volatile organic compounds in indoor and outdoor air samples were measured (DeBortoli et al., 1984). The range of naphthalene concentrations in 14 homes and one office building in Northern Italy was <1-70 $\mu g/m^3$. Outdoor concentrations ranged from <1-11 $\mu g/m^3$. Another investigation of 6 indoor locations found naphthalene concentrations up to 80 $\mu g/m^3$.

Flue gas samples from small wood stoves using different fuels were collected and analysed for naphthalene (Nielsen et al., 1992). Lowest levels of naphthalene were emitted during burning of virgin beech wood (1.98 mg/m^3), sorted domestic waste (4.26 mg/m^3) and rolled up newspapers (10.7 mg/m^3). Much higher emission levels were observed during burning of briquettes made of wood chips (34.1 mg/m^3), scrap wood (72.1 mg/m^3) and pine wood preserved with pentachlorophenol (71.6 mg/m^3).

Flue gas samples from a solid grate incinerator were analysed for various PAH including naphthalene (Li et al., 1995). The wastes were burned in batches (15 kg/run) and seven runs were investigated. The waste burned was oily sludge samples blended with polyethylene plastic at 0%, 20%, 33.3%, 50%, 66.7%, 80% and 100%. During incineration, the auxiliary fuel (liquid diesel) was added automatically to keep the incineration temperature constant. Naphthalene concentrations in the gas phase of the flue gas ranged from 219 to 853 $\mu g/m^3$ (mean 495 $\mu g/m^3$) and in the particulate phase concentrations ranged from 0.64 to 12.8 $\mu g/m^3$ (mean 5.18 $\mu g/m^3$). Ambient air samples were also analysed for naphthalene with concentrations in the gas phase between 459 and 1821 ng/m^3 (mean 804 ng/m^3) and in the particulate phase between 3.57 and 25.1 ng/m^3 (mean 10.4 ng/m^3).

Naphthalene concentrations in air samples inside and outside a shale oil wastewater treatment plant were measured and compared with concentrations in urban and rural air (Hawthorne and Sievers, 1984). Naphthalene concentrations inside and outside the plant and in urban air were 18 and 7 and 0.1 $\mu g/m^3$, respectively. Naphthalene was not detected in the rural air sample.

Hawthorne and Sievers (1984) also simulated volatilisation of organic compounds from wastewaters in a sealed holding tank and in open treatment ponds and measured naphthalene emissions. In the sealed system, naphthalene concentrations in the air above the water were all <0.6 mg/l. Naphthalene emissions from various wastewaters in open treatment plants ranged from 1.4 to 0.15 μg emitted/ml wastewater.

Area air samples were taken at a pilot plant for coal liquefaction in Washington and analysed for polycyclic aromatic hydrocarbons (Gammage, 1983). Naphthalene concentrations in the coal preparation area ranged from 0.30-3.69 $\mu g/m^3$ and in other areas ranged from 0.8 $\mu g/m^3$ in the product solidification area to 10.96 $\mu g/m^3$ in the solvent recovery area. Personal air samples were also taken in the coal preparation area and levels up to 76.07 $\mu g/m^3$ were found (for a welder). Levels of naphthalene in other synthetic fuel plants were also measured (Gammage, 1983). Concentrations of naphthalene around tar storage tanks and manifolds of a low BTU-gasifier plant were between 26 and 160 $\mu g/m^3$. In a coal liquefaction plant naphthalene concentrations were between 52 and 340 $\mu g/m^3$ and a concentration of 104 $\mu g/m^3$ was measured in a small-scale coal hydrocarbonisation plant.

Osborn et al. (1984) analysed naphthalene on particulates trapped by a 0.2 μm filter inside and outside a fixed bed coal gasification pilot plant. For the two indoor sampling dates the levels

were 3.3 μg/m³ and a trace, while for the two outdoor samples they were 0.2 μg/m³ and 2.1 μg/m³. The authors were unable to measure levels in the gas phase due to problems with their sampling medium.

Air sampling at the site of a company that carries out bulk impregnation of timber with creosote found that the naphthalene concentration at various points on the site was less than 1 mg/m³ (Personal communication).

Thrane (Thrane and Wikstrom 1983, Thrane 1987, 1988) measured levels of naphthalene at locations in the vicinity of aluminium production sites in Scandinavia. The results are presented in **Table 3.30**. The authors correlated the levels of PAHs measured with those of fluoride as an aluminium production tracer and demonstrated that they had the same source.

Table 3.30 Levels of naphthalene in air in vicinity of aluminium production plants in Scandinavia (Thrane & Wikstrom 1983, Thrane 1987, 1988)

Location	Season	Level (ng/m³) Range (mean)	Comments
Sandsvall	Winter Summer	97.7-401 3.0-43.3	4 sites in area arithmetic means
Hoyanger Mosjean Ovre Arda Ardalstangen	Range over 4 seasons	12-45 19-107 9-107 26-117	Sites near 4 different sources
Sunndalsora	Summer Fall Winter	3.5-22.5 (9.1) 1.4-20.1 (10.6) 19.1-24.3 (28.6)	Residential area

Randomly selected 24-hour air samples near an aluminium reduction plant in Norway had an average naphthalene concentration of 11.3 ng/m³ (Vogt et al., 1987).

Naphthalene levels in air were measured at the boundary fence of a company producing phthalic anhydride (Industry communication, 1995). The average naphthalene concentration was 0.143 mg/m³ and the maximum was 0.415 mg/m³.

Andersson et al. (1983) determined the naphthalene concentration in workplace air of three different industries. Personal samplers were used in a coke plant, an aluminium plant and a creosote impregnating plant. At the coke-oven battery top the total naphthalene concentration was 650 μg/m³. Samples taken from the handling area for creosote-impregnated railroad ties contained 650 μg/m³ naphthalene. Air samples were taken from the pot-room of an aluminium plant that uses the Søderberg process and were found to contain less than 1 μg/m³ of naphthalene.

Bjørseth et al. (1978a) investigated the polycyclic aromatic hydrocarbon content of air and airborne particulate matter in a primary aluminium smelting plant. Air samples were collected at a number of places in an anode plant, a vertical pin Søderberg plant and a closed prebaked anode plant. The same authors also carried out air sampling in the work atmosphere of a coke plant (Bjørseth et al., 1978b). The results of these two investigations are given in **Table 3.31**. Personal sampling for particulates was also carried out at the aluminium plant and naphthalene was only detected in the sampler of the coke packer.

Personal exposure measurements were carried out for 15 job groups involved in the manufacture and distribution of gasoline (CONCAWE, 1987). Measurements were carried out in 13 countries

during 1984-1985 and naphthalene was only found in personal air samples of people in 4 areas of work. Average exposures for tanker drivers filling their own vehicles by a top loading procedure were 0.020 mg/m^3 and 0.008 mg/m^3 for loading and full shifts, respectively. For workers involved in on site operations at refineries the average exposure was 0.011 mg/m^3 and for those involved in ancillary operations at refineries the average exposure was 0.005 mg/m^3.

Table 3.31 Naphthalene concentrations in an aluminium smelting plant and a coke plant

Location	Naphthalene concentration in particulates (µg/m³)	Naphthalene concentration in gaseous samples (µg/m³)
Aluminium smelting plant - stationary sampling		
Anode plant	ND	ND – 147
Prebaked plant	ND	ND – 12.04
Vertical pin Søderberg plant	ND - 4.0	0.72 – 311.3
Coke plant - stationery sampling		
Battery top	0-4.4	300-1,002
Atmosphere of plant	ND	11.35-1,119

Summary

As for water and soil, there are a large number of reported levels for naphthalene in air, which give a good overall picture of naphthalene levels in the atmosphere. In remote and urban sites levels of naphthalene in the atmosphere ranged up to 11.2 µg/m^3 but at source dominated locations levels up to 415 µg/m^3 were found. Most of the measurements relate to indirect sources of naphthalene and not to the activities which are the subject of this assessment.

3.1.5.3 Comparison of PEC with measured levels in air

The PECs calculated for the regional and continental environments are consistent with measured levels found in remote and some urban areas. The PECs calculated for the local environment are higher and are similar to the high concentrations measured at urban and site dominated locations. For this assessment the highest calculated concentrations; that derived using default emission factors for grinding wheel manufacture (1.9 µg/m^3) and the highest derived from site-specific data for naphthalene production and use (0.64 µg/m^3) will be used, along with the highest measured concentration (average 143 µg/m^3).

3.1.6 Secondary poisoning

Naphthalene has a log K_{ow} of 3.7, and some bioaccumulation factors greater than 100 have been measured. However, naphthalene does not carry the risk phrases T or T+, R47, R48 or R60-63. Therefore, it is not necessary to carry out a risk characterisation of secondary poisoning.

3.1.6.1 Predicted environmental levels in biota

The predicted levels of naphthalene in biota and foodstuffs calculated using EUSES are shown in **Table 3.32** and the estimated daily doses in **Table 3.33**. In EUSES the concentration arising in biota are for biota exposed to naphthalene in contaminated areas.

For fish the uptake of naphthalene is modelled using bioconcentration factors. The fish is assumed to be exposed to the dissolved surface water concentration at 1,000 m from the wastewater treatment plant effluent.

Table 3.32 Concentration in human intake

	Regional	Production - composite site	Processing - generic	Pyrotechnics	Mothballs manufacture	Grinding wheels manufacture
Air	0.137 μg/m³	0.61 μg/m³	1.05 μg/m³	0.145μg/m³	0.90 μg/m³	1.92 μg/m³
Drinking water	0.03 μg/l	0.26 μg/l	1.8 μg/l	1.9μg/l	0.046 μg/l	0.28 mg/l
Fish	13 μg/kg	111 μg/kg	17 μg/l	0.83 mg/kg	13 μg/kg	120 mg/kg
Leaves of plants	0.34 μg/kg	1.5 μg/kg	2.7 μg/l	0.37μg/kg	2.2 μg/kg	5.0 μg/kg
Root of plants	0.64 μg/kg	2.1 μg/kg	84 μg/kg	0.061 mg/kg	2.2 μg/kg	7.7 mg/kg
Meat	0.0053 μg/kg	0.024 μg/kg	0.052 μg/l	0.019μg/kg	0.034 μg/kg	2.1 μg/kg
Milk	0.0017 μg/kg	0.0077 μg/kg	0.016 μg/l	0.006μg/kg	0.011μg/kg	0.66 μg/kg

Table 3.33 Daily human dose via indirect exposure (mg/kg (body weight)/day)

	Regional	Production (site D)	Processing	Pyrotechnics	Mothballs manufacture	Grinding wheels manufacture
Air	$2.9 \cdot 10^{-5}$	$1.3 \cdot 10^{-4}$	$2.3 \cdot 10^{-4}$	$3.1 \cdot 10^{-5}$	$1.9 \cdot 10^{-4}$	$4.1 \cdot 10^{-4}$
Drinking water	$8.5 \cdot 10^{-7}$	$7.4 \cdot 10^{-6}$	$5.1 \cdot 10^{-5}$	$5.5 \cdot 10^{-5}$	$1.3 \cdot 10^{-6}$	$8.1 \cdot 10^{-3}$
Fish	$2.1 \cdot 10^{-5}$	$1.8 \cdot 10^{-4}$	$2.8 \cdot 10^{-5}$	$1.4 \cdot 10^{-3}$	$2.1 \cdot 10^{-5}$	0.198
Leaves of plants	$5.9 \cdot 10^{-6}$	$2.6 \cdot 10^{-5}$	$4.5 \cdot 10^{-5}$	$6.3 \cdot 10^{-6}$	$3.9 \cdot 10^{-5}$	$8.6 \cdot 10^{-5}$
Root of plants	$3.5 \cdot 10^{-6}$	$1.2 \cdot 10^{-5}$	$4.6 \cdot 10^{-4}$	$3.3 \cdot 10^{-4}$	$1.2 \cdot 10^{-5}$	$4.2 \cdot 10^{-2}$
Meat	$2.3 \cdot 10^{-8}$	$1.1 \cdot 10^{-7}$	$2.2 \cdot 10^{-7}$	$8.3 \cdot 10^{-8}$	$1.4 \cdot 10^{-7}$	$9.0 \cdot 10^{-6}$
Milk	$1.3 \cdot 10^{-8}$	$6.2 \cdot 10^{-8}$	$1.3 \cdot 10^{-7}$	$4.9 \cdot 10^{-8}$	$8.5 \cdot 10^{-8}$	$5.3 \cdot 10^{-6}$
Total	$6.0 \cdot 10^{-5}$	$3.6 \cdot 10^{-4}$	$8.1 \cdot 10^{-4}$	$1.8 \cdot 10^{-3}$	$2.7 \cdot 10^{-4}$	0.25

The uptake of naphthalene by plants is modelled on plants growing on agricultural soil on which sludge from a wastewater treatment plant has been applied and aerial deposition is occurring. For plant stems uptake of the chemical is considered from soil and aerial deposition, while for plant roots uptake from soil only is considered.

The uptake of naphthalene by cattle results in concentrations in meat and milk. These are modelled by biotransfer factors, which are the steady state concentrations in either meat or milk

divided by the animal's daily contaminant intake. The cattle are assumed to derive their total consumption of soil and grass from contaminated soil and grass only.

The daily human intake of naphthalene has been estimated using EUSES at the regional and local levels. The estimation based upon typical human consumption and inhalation rates at the regional level is 0.06 μg/kg/day (see Appendix 1 for EUSES printout). **Tables 3.32** and **3.33** give the concentrations in human intake and the daily human doses arising from releases from production (based on Site D), processing and the manufacture of creosote and mothballs, and for releases at the regional level.

3.1.6.2   **Measured levels in biota and foodstuffs**

Samples of mussels from 27 locations around the Scottish coastline contained levels of naphthalene ranging from a trace to 196 ng/g-wet weight (Mackie et al., 1979).

Average levels of naphthalene in cod and haddock from around three platforms (19 samples) and three reference stations (13 samples) in the North Sea were 0.06 μg/g (range 0-0.23) and 0.01 μg/g (range 0-0.02), respectively (Vogt et al., 1988).

Mussels and fish from the Finnish Archipelago Sea were analysed for naphthalene (Rainio et al., 1986) and the levels found are shown in **Table 3.34**.

Table 3.34 Naphthalene levels in Biota from Finnish Archipelago Sea   (Rainio et al., 1986)

Species	Level μg/kg wet weight
Blue mussel (*Mytilus edulis*)	detected in 4/7 sites range 5-41
Baltic herring (*Clupea harengus*)	muscle < 0.5
Pike-perch (*Stizostedian lucioperca*)	muscle < 0.5 gallbladder < 0.5 liver 45
Burbot (*Lota lota*)	muscle < 0.5 liver 72 gallbladder 215

Samples of fish and shellfish were collected from the Gulf of Naples where there is a high concentration of industries (Cocchieri et al., 1990) and the levels of naphthalene are shown in **Table 3.35**. Naphthalene was not detected in the common mussel (*Mytilus edulis)*, the edible cockle (*Cardium edule)* and 11 other species (bogue, brill, cleaver wrasse, common sole, cuckoo wrasse, horse mackerel, pandora, piper, rainbow wrasse, scorpion fish and spotted weaver).

Table 3.35 Levels of naphthalene in biota from the Gulf of Naples (Cocchieri et al., 1990)

Species	Level µg/kg (wet weight)
Razor fish (*Ensis siliqua*)	20
Shortnecked clam (*Amigdala degussata*)	32
Wart venus (*Venus verrucosa*)	25
Anchovy (*Engraulis enchrasicholus*)	63
Comber (*Serranus cabrilla*)	4
Rock goby (*Gobius paganellus*)	20

Baltic clams (*Macoma balthica*) and clam worms (*Nereis succinea*) were collected from 7 locations in the Maryland waters of Chesapeake Bay (Foster and Wright, 1988). Naphthalene was only detected in Baltic clams from two locations at concentrations of 3.75 and 7.20 µg/g (normalised to lipid content). Naphthalene was detected in clam worms from five locations at levels between 1.56 and 8.57 µg/g (normalised to lipid content).

Snails (*Thais haemostoma*) were collected from two areas offshore in Pensacola Bay near an onshore hazardous waste site (Rostad and Pereira, 1987). Levels of naphthalene in snails from the two sites were 14.5 and 33.9 µg/kg-wet weight.

Naphthalene levels in brown bullhead catfish (*Ictalurus nebulosus*) from the Black River and striped bass (*Morone saxatillus*) from the Sacramento River were 140 and 4 ng/g wet weight, respectively (Vassilaros et al., 1982).

Samples of oysters (*Crassostrea virginica*) and clams (*Rangia cuneata*) were collected from the mouths of three passes at Lake Pontchartrain, USA (McFall et al., 1985b). Oysters from the Inner Harbour Navigation Canal had an average naphthalene concentration of 35 ng/g wet weight. Clams from Chef Menteur pass and the Rigolets had naphthalene levels of 120 and 51 ng/g wet weight, respectively.

Samples of coquina clams (*Donax variabilis*), white shrimp (*Penaeus setiferus*), blue crab (*Callinectes sapidus*) and silver seatrout (*Cynoscian arenarius*) were collected 8 months after oil from the Ixtoc-1 blow-out had disappeared from South Texas beaches (Bedinger and Nulton, 1982). A trace amount of naphthalene was found in the clam samples but the level was below the detection limit (10 ng/g).

Bioaccumulation of polycyclic aromatic hydrocarbons from a Pensacola (Florida) creosote contamination site was investigated by analysis of two mollusc species (Elder and Dresler, 1988). Adult oysters (*Crassostrea virginica*) were kept in cages at three sites and one control site for six weeks. A gastropod, (*Thais haemostoma*), which is native to the study area was collected from two sites and a control site. Water and sediment samples were also collected. Naphthalene was not found at higher levels in the oysters or snails from the test site (oysters not detected-2 µg/kg, snails 15-35 µg/kg) than in those from the control site (oysters 8 µg/kg, snails 25 µg/kg).

Gossett et al. (1983) sampled fish and other organisms from a point 6 km north east of the Los Angeles County wastewater treatment plant discharge zone at Palo Verdes. Levels of naphthalene in fish liver ranged from <20 µg/kg wet weight (detection limit) to 42 µg/kg wet weight in white croaker (*Genyonemus lineatus*). Naphthalene was not detected in crab digestive

gland, shrimp mussel, nor in whole invertebrates sampled from just above the bottom sediments at 60 m depth.

Juvenile Chinook salmon (*Oncorhynchus tshawytscha*) were collected from two sites in the Duwamish Waterway which passes through the industrial section of Seattle and two reference sites in the Nisqually river which flows through a predominantly rural area (McCain et al., 1990). Samples of liver, stomach contents and bile were analysed for 17 aromatic hydrocarbons. Naphthalene was found at levels of 2.4 and 1.7 µg/g dry weight in the stomachs of fish from the contaminated sites. In contrast only 0.09 µg/g dry weight was found in the stomachs of fish from the reference site.

Composite samples of 14 fish species were collected from the North West Arabian Gulf (DouAbdul et al., 1987) and the levels of naphthalene found are given in **Table 3.36**.

Table 3.36 Naphthalene levels in fish from the Arabian Gulf   (DouAbdul et al., 1987)

Species	Level (µg/kg) wet weight average (range)
Sea catfish (*Arius thalassinus*)	14 (9-19)
Fourfingered threadfin (*Eleutherinema tetradactnum*)	not detected
Croaker (*Johnieops sina*)	31 (19-43)
Large scale tongue sole (*Cynoglosus arel*)	49 (28-59)
Indian flathead (*Platycephalus indicus*)	28 (11-38)
Silvery grunt (*Pomadasys argenteus*)	61 (43-78)
Elongate illisha (*Ilisha elongata*)	64 (40-78)
Forktail needlefish (*Tylosurus strongylurus*)	106 (89-117)
Threadfish shad (*Nematalosa nasus*)	6 (not detected-10)
Hamilton thryssa (*Thryssa hamiltonii*)	20 (10-27)
Yellowfin sea bream (*Acanthopagrus luteus*)	30 (22-45)
Mullet (*Liza dussumeiri*)	27 (18-33)
Silverbanded crocker (*Otoliths argenteus*)	54 (40-61)
Indian shad (*Tenualosa ilisha*)	80 (66-91)

Soil fauna and leaf litter were collected from the site of a major road 10 km from Brisbane (Pathirana et al., 1994). Levels measured on a wet weight basis were 55 ng/g for leaf litter, 11 ng/g for the slug and 4 ng/g for the millipede. Naphthalene was not detected in two species of beetle (*Laxta granicollis* and *Platyzosteria nitida*) or earthworms.

Various parts of carrots were analysed for polycyclic aromatic hydrocarbons (Wild and Jones, 1991). Naphthalene levels in the peel, inner peel, outer core and core were 23.3, 9.1, 8.6 and 4.9 µg/kg dry weight, respectively. Naphthalene levels were determined in uncooked, cooked, tinned and frozen carrots to be 7.8, 7.1, 12.4 and 7.6 µg/kg dry weight.

Naphthalene was found in grains of barley when three different types of fertiliser were used (Kirchmann and Tengsved, 1991). Levels of naphthalene when nitrogen fertiliser, pig slurry and sewage sludge were used as fertiliser were 4.3, 2.8 and 3.3 µg/kg dried barley grain.

Naphthalene in untreated soil was found at 0.01 mg/kg dry matter. This indicates that root uptake was not the primary transport path to above ground plant parts.

Ou et al. (1992) found naphthalene in rice grown in paddy fields where wastewater was used for irrigation. Naphthalene was found at 18.26 µg/l and 0.48 µg/l in the influent and effluent of a paddy rice system and at 0.028 mg/kg in rice grown in this system. In rice with clean water irrigation 0.010 mg/kg naphthalene was found.

Summary

Most of the measured concentrations of naphthalene in biota, from various locations, are below 50 µg/kg although levels up to 215 µg/kg have been measured in the gallbladder of fish from the Finnish Archipelago Sea and 2.4 mg/kg naphthalene was found in the stomach of fish from the Duwamish Waterway. Levels in food up to 28 µg/kg have been measured. The highest level was for rice where wastewater was used for irrigation.

3.1.7 Summary of PECs for naphthalene

PECs for naphthalene are summarised in **Table 3.37**.

Table 3.37 Summary of PECs for naphthalene

Water (µg/l)				
Local		Regional	Continental	Measured
Production (worst case)	0.31	0.03	0.0025	2.24 (freshwater)
Use as intermediate (site specific)	0.031			0.3 (marine water)
Use as intermediate	0.042			
Pyrotechnics manufacture	2.35			
Mothballs manufacture	0.03			
Grinding wheels manufacture	294			
Sediment (µg/kg)				
Local		Regional	Continental	Measured
Production (worst case)	8.7	0.83	0.07	up to 91 (marine)
Use as intermediate (site specific)	0.87			up to 750
Use as intermediate	1.2			contaminated site up to 7,720 mg/kg
Pyrotechnics manufacture	66			
Mothballs manufacture	0.83			
Grinding wheels manufacture	8232			

Table 3.37 (continued) Summary of PECs for naphthalene

Soil (µg/kg)				
Local		**Regional**	**Continental**	**Measured**
Production (worst case)	1.7	0.3 (agri)	0.023 (agri)	up to 131 µg/kg
Use as intermediate (site specific)	1.0	0.12 (nat)	0.025 (nat)	contaminated site up to 400 mg/kg
Use as intermediate	50			
Pyrotechnics manufacture	36			
Mothballs manufacture	1.0			
Grinding wheels manufacture	4600			
Air (µg/m^3)				
Local		**Regional**	**Continental**	**Measured**
Production (worst case)	0.64	0.14	0.028	remote/urban-up to 11.2
Use as intermediate (site specific)	0.9			site dominated-up to 415
Use as intermediate	1.1			
Pyrotechnics manufacture	0.15			
Mothballs manufacture	0.9			
Grinding wheels manufacture	1.9			

3.2   EFFECTS ASSESSMENT: HAZARD IDENTIFICATION AND DOSE (CONCENTRATION) - RESPONSE (EFFECT) ASSESSMENT

3.2.1   Aquatic compartment (incl. sediment)

3.2.1.1   Toxicity test results

3.2.1.1.1   Fish

Acute toxicity

Data on the acute toxicity of naphthalene to fish are summarised in **Table 3.38**. 96-hour LC_{50}s range from 1.6 mg/l for rainbow trout to 150 mg/l for mosquito fish. Care must be taken when interpreting data from tests based on nominal concentrations because naphthalene can be rapidly lost from solution (see Section 3.2.1.1.3).

Landis International (personal communication, 1995) measured a 96-hour LC_{50} for Bluegill sunfish of 31 mg/l but the complete test report has not been seen.

Rice and Thomas (1989) studied the acute toxicity of naphthalene to pink salmon fry (*Oncorhynchus gorbuscha*) and calculated a 96-hour LC_{50} of 0.96 mg/l. Two-day pre-treatment exposures of between 52% and 87% of the control LC_{50} concentrations significantly increased the tolerance of pink salmon fry to naphthalene. Even 12-hour pre-treatment exposures of naphthalene (85% of LC_{50} concentration) significantly increased the tolerance of the fry to naphthalene. Korn and Rice (1981) found that the toxicity of naphthalene to Coho salmon (*Oncorhynchus kisutch*) increased from eggs through early, mid and late alevins to emergent fry. 96-hour LC_{50}s ranged from >11.8 mg/l to 5.6 mg/l in static renewal tests.

Chronic toxicity

Freshwater fish

Black et al. (1983) studied the effects of naphthalene on the embryo-larval stages of rainbow trout (*Oncorhynchus mykiss*) and largemouth bass (*Micropterus salmoides*). Exposure to naphthalene was initiated 20 minutes after fertilisation for trout and 2 to 4 hours after spawning for bass, and maintained until 4 days after hatching. Average hatching times were 23 days for trout and 3 days for bass. For trout, survival 4 days post hatch fell from 97% at 0.008 mg/l to 32% at 0.230 mg/l. Survival of bass at 4 days post hatch ranged from 93% at 0.028 mg/l to 65% at 0.239 mg/l. LC_{50} values calculated from these results after exposure to naphthalene until 4 days post hatch were 0.11 mg/l for rainbow trout and 0.51 mg/l for largemouth bass.

Table 3.38 Toxicity of naphthalene to fish

Organism	size/age	Stat/ Flow	temp (°C)	hardness (mg/l)	pH	parameter	concentration (mg/l)	Reference
Freshwater								
Pink salmon *Onchorhynchus gorbuscha*	Fry 1-2 g	flow	10-12 12			48h-LC$_{50}$ 24h-LC$_{50}$	0.9-1.01 m 0.92	Rice and Thomas, 1989 Thomas and Rice, 1979
Coho salmon *Onchorhynchus kisutch*	1 g	flow	8-10			96h-LC$_{50}$	2.1 m	Moles et al., 1981
Rainbow trout *Onchorhynchus mykiss*	3.9 g	flow	15			96h-LC$_{50}$	1.6 m	DeGraeve et al., 1982
Fathead minnow *Pimephales promelas*	0.9 g 0.27 g 31-35 days " " 34 days	flow stat flow " " " flow	15 20±0.5 24.6±0.6 " " 24.5	43.9	6.9-7.7 " " " 7.4	96h-LC$_{50}$ 96h-LC$_{50}$ 24h-LC$_{50}$ 48h-LC$_{50}$ 72h-LC$_{50}$ 96h-LC$_{50}$ 96h-LC$_{50}$	7.8 m 1.99 7.76 6.35 6.08 6.08 6.14 m	DeGraeve et al., 1982 Millemann et al., 1984 Holcombe et al., 1984 Holcombe et al., 1984 Holcombe et al., 1984 Holcombe et al., 1984 Geiger et al., 1985
Tilapia *Oreochromis mossambicus*		stat				96h-LC$_{50}$	7.9	Dange, 1986b
Mosquito fish *Gambusia affinis*	Adult	stat	22-24		8.1-8.5	96h-LC$_{50}$	150 n	Wallen et al., 1957
Estuarine and marine								
Sheepshead minnow Cyprinodon variegatus						96h-LC$_{50}$	2.4	Anderson et al., 1974

Notes: stat - static conditions (water unchanged for duration of test)
flow - flow-through conditions (naphthalene concentration continuously maintained)
n - nominal concentration; m - measured concentration

Milleman et al. (1984) also exposed the embryo-larval stages of rainbow trout (*Oncorhynchus mykiss*) and largemouth bass (*Micropterus salmoides*) to naphthalene. Flow-through conditions were used and naphthalene concentration, temperature, dissolved oxygen, conductivity, hardness and pH were monitored. Rainbow trout eggs were exposed 20 minutes after fertilisation until 4 days post-hatch (average hatching time was 23 days). Largemouth bass were exposed from 2-4 hours post-spawning until 4 days post-hatch (average hatching time was 3 days). The eggs and larvae were examined daily and LC_{50} values were determined to be 0.12 mg/l for rainbow trout and 0.68 mg/l for largemouth bass. Although the final values are slightly different these exposures are almost certainly the same as those reported by Black et al. (1983) as described above.

DeGraeve et al. (1982) carried out embryo-larval tests on fathead minnow (*Pimephales promelas*) under flow-through conditions. Naphthalene concentrations of 0.85 mg/l or more were found to significantly decrease hatchability, mean length and weight of fry at 30 days. No fry survived to 30 days at naphthalene concentrations of 4.38 or 8.51 mg/l. Based upon these results the authors concluded that the maximum acceptable toxicant concentration (MATC) was >0.45 and <0.85 mg/l.

Moles et al. (1981) exposed Coho salmon fry (*Oncorhynchus kisutch*) to naphthalene concentrations of 0.2, 0.4, 0.7 and 1.4 mg/l for 40 days under flow-through conditions. Dry weight, wet weight and length of fry were significantly less than controls at the two highest exposure concentrations. Moles and Rice (1983) exposed juvenile pink salmon (*Oncorhynchus gorbuscha*) to naphthalene concentrations ranging from 0.12 to 0.80 mg/l for 40-days. At the end of the experiment wet weight was significantly lower in fish exposed to 0.38 mg/l or more (NOEC 0.12 mg/l); length of fish, and the caloric and fat content were significantly reduced in fish at the highest dose of 0.80 mg/l.

Estuarine and marine

Falk-Peterson et al. (1982) exposed cod eggs (*Gadus morhua*) to naphthalene concentrations of 0.4, 1.2 and 3.8 mg/l for up to 4 days. The development of cod eggs was unaffected by concentrations of 0.4 or 1.2 mg/l. At 3.8 mg/l there was a slight increase in the number of abnormal eggs after four days. Saethre et al. (1984) exposed fertilised cod eggs to naphthalene concentrations of between 0.90 and 2.78 mg/l for 4 days. No effect on survival was found; in duplicate tests at the highest concentration, percentage abnormalities were 24% at the blastula stage and 38% at the gastrula stage in one test but no abnormal cells were recorded in the other. The authors noted that naphthalene was lost from solution very rapidly.

3.2.1.1.2   Amphibians

Edmisten and Bantle (1982) studied the effects of naphthalene on larvae of the South African clawed toad *Xenopus laevis* in flow-through toxicity tests. A 6-hour EC_{50} based on depigmentation (larvae change colour in response to naphthalene exposure) was 3.7 mg/l and the 96-hour LC_{50} was calculated to be 2.1 mg/l.

Schultz and Dawson (1995) used an *in vitro* test system, the Frog Embryo Teratogenesis Assay-Xenopus (FETAX) to determine the development hazard of exposure to naphthalene and its known metabolites (1,2-dihydroxynaphthalene, 2-hydroxybenzaldehyde, 2-hydroxybenzoic acid, 2,5-dihydroxybenzoic acid and 1,2-dihydroxybenzene). Embryos in groups of 25 were exposed to a series of concentrations for each test chemical dissolved in FETAX solution. Embryos in

FETAX without the test chemical served as control. The test was a 96-hour static renewal test and was carried out at 23 ± 1°C. The endpoints determined included LC_{50} (mortality) and EC_{50} (malformation). No effects were seen for naphthalene at saturation. However, the investigation showed that aerobic microbial metabolism of naphthalene may produce metabolites that pose potentially significant developmental harm.

3.2.1.1.3 Aquatic invertebrates

Acute toxicity

Data on the acute toxicity of naphthalene to aquatic invertebrates are summarised in **Table 3.39**. 48-hour LC_{50}s for daphnids range from 2.16 to 24.1 mg/l. 96-hour LC_{50}s range from 0.8 to 17 mg/l for a range of estuarine and marine crustacea. All of the data appear to be within the solubility of naphthalene. However, those based on nominal values do not take into account loss of the chemical during the test. These can be very variable depending on the test system employed. Saethre et al. (1984) noted that naphthalene was rapidly lost from solution with 80 to 90% lost over a 4-day period. The authors pointed out that this was probably due to evaporation from poorly sealed test beakers. Caldwell et al. (1977) found significant losses of 25 to 50% in flow-through systems. However, Ott et al. (1978) reported losses of less than 8% in a closed static system. Similar losses of 2 to 7% were found in flow-through systems over periods of up to 40 days (Moles et al., 1981; Edmisten and Bantle, 1982).

Freshwater invertebrates

LeBlanc (1980) exposed daphnids (*Daphnia magna*) to nominal naphthalene concentrations for up to 48 hours. A no-discernible effect concentration of 0.60 mg/l was determined. Landis International (personal communication, 1995) measured a 48-hour LC_{50} for Daphnia of 17.2 mg/l but no further details of the test are known (complete test report not seen).

Estuarine and marine invertebrates

Deshmukh et al. (1985a) exposed the speckled prawn (*Metapenaeus monoceros*) to naphthalene at salinities ranging from 3.5 $^{o}/_{oo}$ to 35 $^{o}/_{oo}$. The 48- and 96-hour LC_{50}s ranged from 5.5 to 6.5 mg/l and 4.4 to 5.5 mg/l, respectively.

Naphthalene showed less toxicity at salinities of 17.7 and 26.25‰ than at 3.5, 8.75 or 35‰. In a similar experiment, prawns were exposed to naphthalene at temperatures of 21°C, 25°C or 30°C. Ninety-six hour LC_{50}s were 5.7, 5.5 and 4.2 mg/l, respectively (Deshmukh et al., 1985b).

Rice and Thomas (1989) studied the acute toxicity of naphthalene to the kelp shrimp (*Eualis suckleyi*) and calculated a 96-hour LC_{50} of 1.39 mg/l. Pre-treatment exposure to 32% of the LC_{50} concentration decreased the subsequently measured 96-hour LC_{50}. However, this was only significant if the pre-treatment period was in excess of 8 days.

Table 3.39 Acute toxicity of naphthalene to aquatic invertebrates

Organism	Size/age	Stat/flow	Temp (°C)	Hardness (mg/l)	PH	Parameter	Concentration (mg/l)	Reference
Freshwater								
Water flea *Daphnia magna*	< 24 hour	stat		173	7.4-9.4	24h-LC$_{50}$	17 n	LeBlanc, 1980
	< 24 hour	stat		173	7.4-9.4	48h-LC$_{50}$	8,6 n	LeBlanc, 1980
		stat	22-26		8-8.6	24h-LC$_{50}$	13.2 m	Crider et al., 1982
		stat	22-26		8-8.6	48h-LC$_{50}$	3.4 m	Crider et al., 1982
		stat	19	134	7.6	48h-LC$_{50}$	22.6 n	Eastmond et al., 1984
	4-6 days	stat	21-25			48h-LC$_{50}$	4.7	Abernethy et al., 1986
	24 hour	stat	19.5			48h-LC$_{50}$	24.1	Parkhurst, 1982
		stat			~6.5	48h-LC$_{50}$	2.16 n	Millemann et al., 1984
	4-6 days	stat	23"2			48h-LC$_{50}$	16.7 n	Bobra et al., 1983
Water flea *Daphnia pulex*		stat	15	43	7.2	48h-LC$_{50}$	2.92-3.89 n	Geiger and Buikema, 1982
	1.9-2.1mm	stat			7.5	96h-LC$_{50}$	1.0 n	Trucco et al., 1983
Snail *Physa gyrina*	0.057 g	stat	19.5-20.5			48h-LC$_{50}$	5.02 n	Millemann et al., 1984
Amphipod *Gammarus minus*	adult	stat	21-24			48h-LC$_{50}$	3.93 n	Millemann et al., 1984
Midge *Chironomus tentans*	4th instar	stat	23-26			48h-LC$_{50}$	2.81 n	Millemann et al., 1984
Estuarine and marine								
Estuarine copepod *Eurytemora affinis*	adult	stat	15	20 s		24h-LC$_{50}$	3.798 n	Ott et al., 1978
Kelp shrimp *Eualis suckleyi*	1 g	flow	6-6.9	seawater		96h-LC$_{50}$	1.39 m	Rice and Thomas, 1989
Grass shrimp *Palaemonetes pugio*	adult	stat	19-21	15 s	8.1	96h-LC$_{50}$	2.6	Anderson et al., 1974
						96h-LC$_{50}$	2.35 n	Tatem, 1975

Table 3.39 continued overleaf

Table 3.39 continued Acute toxicity of naphthalene to aquatic invertebrates

Organism	Size/age	Stat/flow	Temp (EC)	Hardness (mg/l)	PH	Parameter	Concentration (mg/l)	Reference
Brown shrimp *Penaeus aztecus*						96h-LC$_{50}$	2.5	Anderson et al., 1974
Artemia	larvae nauplii	stat stat	19-21		8.5-8.7	24h-LC$_{50}$ 24h-LC$_{50}$	10.6 n 3.19 m	Abernethy et al., 1986 Foster and Tullis, 1984
Mysid *Neomysis americana*		stat$ stat$ flow flow	15 25 15 25	seawater " " "		96h-LC$_{50}$ 96h-LC$_{50}$ 96h-LC$_{50}$ 96h-LC$_{50}$	1.42 m 0.80 m 1.28 m 0.85 m	Hargreaves et al., 1982 Hargreaves et al., 1982 Smith and Hargreaves, 1983 Smith and Hargreaves, 1983
Speckled prawn *Metapenaeus monoceros*	juvenile juvenile	stat stat	30 30	17.5 17.5		48h-LC$_{50}$ 96h-LC$_{50}$	4.9 n 4.2 n	Deshmukh et al., 1985b Deshmukh et al., 1985b
Dungeness crab *Cancer magister*	1st instar	stat$	13	30 s		96h-LC$_{50}$	> 2.0 n	Caldwell et al., 1977
Crab *Scylla serrata*	intermoult juvenile intermoult juvenile	stat stat	27-29 27-29	30-31 s 30-31 s	7.6-8.0 7.6-8.0	48h-LC$_{50}$ 96h-LC$_{50}$	20 n 17 n	Kulkarni and Masurekar, 1983 Kulkarni and Masurekar, 1983
Blue crab *Callinectes sapidus*	adult adult	flow flow	22-25 22-25	10-30 s 10-30 s		24h-LC$_{50}$ 48h-LC$_{50}$	2.4 m 2.3 m	Sabourin, 1982 Sabourin, 1982

Notes: stat - static conditions (water unchanged for duration of test)
stat$ - static renewal conditions (water changed periodically)
flow - flow-through conditions (naphthalene concentration continuously maintained)
n - nominal concentration;   m - measured concentration
s - salinity (‰)

Sanborn and Malins (1977) found that naphthalene at 8 to 12 µg/l in flowing seawater produced 100% mortality in 24 to 36 hours in newly hatched Dungeness crab (*Cancer magister*) zoea and stage I and IV spot shrimp (*Pandalus platyceros*).

Le Gore (1974) exposed developing larvae of the Pacific oyster (*Crassostrea gigas*) to naphthalene in 48-hour toxicity tests. An EC_{50}, based on abnormality and mortality and termed 'ecological mortality' by the author, was calculated to be 110 mg/l.

Chronic toxicity

Freshwater invertebrates

A 28-day No Observed Effect Concentration (NOEC) for *Daphnia magna* was quoted as 3 mg/l by Parkhurst (1982). However, no information about test conditions or procedure was given.

Geiger and Buikema (1982) exposed *Daphnia pulex* to naphthalene concentrations of 0.33 or 0.6 mg/l in chronic toxicity tests. No significant effect was noted on moulting frequency, growth rate, production of total and live young, number of non-viable eggs, partial and full abortions, and whether or not abortions occurred prior to completion of embryonic development. However, at both exposure concentrations daphnids lived significantly longer than did the controls.

Estuarine and marine invertebrates

Saethre et al. (1984) exposed fertilised sea urchin (*Strongylocentrotus droebachiensis*) eggs to naphthalene concentrations of between 0.90 and 2.78 mg/l for 4 days. The lower exposure concentration had no effect on the survival of the eggs. The higher concentration killed all the eggs within 2 to 4 days: 60% to 98% of the cells were abnormal within 6 hours of exposure. The authors note that naphthalene was lost from solution very rapidly. Falk-Peterson et al. (1982) exposed both sea urchin eggs (*Strongylocentrotus droebachiensis*) and copepod adults (*Calanus finmarchicus*) to naphthalene concentrations of 0.4, 1.2 and 3.8 mg/l for up to 4 days. The normal development of sea urchin eggs and copepod survival were unaffected by concentrations of 0.4 or 1.2 mg/l. However, at 3.8 mg/l all sea urchin eggs showed abnormal development within one day and copepod survival was reduced within four days.

Laughlin and Neff (1979) exposed zoeae of the mud crab *Rhithropanopeus harrisii* to factorial combinations of salinity (5, 15 and 25‰), temperature (20, 25 and 30°C) and naphthalene concentrations (125, 250 and 500 µg/l) throughout larval development. No significant effect of naphthalene exposure was observed at any combination.

Berdugo et al. (1977) exposed copepods (*Eurytemora affinis*) to naphthalene concentrations of 1 or 2 mg/l for 24 hours and found a significant reduction in ingestion rate. An exposure of 1 mg/l significantly reduced the total number of eggs produced by each female. However, mean brood size, rate of egg production and length of life were not significantly different from controls. A 10-day exposure to ^{14}C naphthalene concentrations of 10 or 50 µg/l increased mortality, egg production and ingestion rates, although the latter two parameters were not significantly different from controls at the 5% level.

Ott et al. (1978) found significant reductions in the length of life, total numbers of nauplii produced and mean brood size of female copepods (*Eurytemora affinis*) exposed to 14 µg/l naphthalene for the duration of their adult life (up to 29 days).

3.2.1.1.4   Aquatic plants

Soto et al. (1975a,c) exposed the green flagellate *Chlamydomonas angulosa* to a medium saturated with naphthalene (solubility 34 mg/l). In open systems, 61% of the cells were killed in the presence of naphthalene. However, the generation time of survivors was the same as controls. In closed systems, up to 98% of the cells were killed within 3 to 7 days but the generation time of survivors was much shorter than controls. In general, saturated naphthalene exposure caused a prolonged lag growth phase. Soto et al. (1975b,c) found that exposure to saturated naphthalene in closed systems caused more than a 90% reduction in photosynthetic rate with no recovery during an 8-day exposure. In open systems, similar reductions in photosynthetic rate were observed upon exposure to naphthalene. However, rapid recovery accompanied the evaporation of naphthalene especially when the media was aerated. The photosynthetic rate had reached 60% to 70% of controls within 20 hours. Morphological changes in *Chlamydomonas* exposed to 50% saturated naphthalene were as follows: inhibited motility, loss of flagella, an increase in the activity of contractile vacuoles and the appearance of cytoplasmic granulation. Other changes were cell wall thickening, increased space between the cell walls and the plasmalemma, abnormal chloroplast lamellae, and an increase in starch grains and electron dense material deposited in the cytoplasmic vacuoles (Soto et al., 1975c, 1979). Soto et al. (1977) incubated *C. angulosa* in a closed system with 50% saturated naphthalene for 7 days and observed that pigments and total cellular carbon remained constant, total protein decreased significantly and carbohydrate and lipids increased significantly.

Gaur (1988) carried out assays to determine the effects of naphthalene on *Selenastrum capricornutum* over a 14-day test period. An IC_{50} value, the concentration of naphthalene that caused a 50% reduction in the growth of the final standing crop of test algae, of 25 mg/l was calculated.

Kauss and Hutchinson (1975) studied the effect of naphthalene on the growth of *Chlorella vulgaris*. Algae were exposed to naphthalene concentrations of 3.3, 8.3, 16 and 30 mg/l for up to 10 days. All exposure concentrations caused a significant inhibition of growth after one day. An LD_{50} based on the reduction of *Chlorella* cell numbers was calculated to be 33 mg/l. Naphthalene concentrations of 3 to 24 mg/l in closed systems caused a significant dose related decrease in photosynthesis (as measured by bicarbonate uptake) within 30 minutes. After 120 minutes, photosynthesis was reduced to 2% of controls at all concentrations. In open systems, 30 mg/l naphthalene caused a significant decrease in photosynthesis (Kauss et al., 1973).

Østgaard et al. (1984) studied the effect of naphthalene on the growth of the alga *Skeletonema costatum*. The growth was reduced by 30-50% when the algae were exposed to 400 µg/l and when the naphthalene concentration was increased to 1,100 µg/l growth stopped in three days.

Milleman et al. (1984) exposed the green algae *Selenastrum capricornutum* and the diatom *Nitzschia palae* to a series of naphthalene concentrations for four hours. The concentration of naphthalene causing 50% reduction in the rate of ^{14}C assimilation was used to measure the toxic effects. Four-hour EC_{50} values were 2.96 mg/l for the green algae and 2.82 mg/l for the diatom.

Ren et al. (1994) investigated the effect of light on the phytotoxicity of polycylic aromatic hydrocarbons to the Duckweed *Lemna gibba* L. G-3. The toxicity of naphthalene was found to increase when plants and chemicals were incubated together in the presence of light and when chemicals were pre-treated in light and then incubated with plant tissue. UV was the only region of the solar spectrum that effectively enhanced toxicity.

3.2.1.1.5   Microorganisms

Blum and Speece (1991) studied the effect of naphthalene on bacterial populations using IC_{50} tests (the concentration that inhibits the culture by 50%). The inhibition of ammonia consumption was used as the criterion for the toxic inhibition of *Nitrosomonas* and an IC_{50} of 29 mg/l was reported. For aerobic heterotrophs, inhibition of oxygen uptake was used as the criterion for toxic inhibition and an IC_{50} of 670 mg/l was reported.

Kiene and Capone (1984) studied the effect of naphthalene (1,000 mg/kg) on methanogenesis, sulphate reduction and carbon dioxide evolution of anaerobic salt marsh sediment over a 7- to 9-day period. Initially methanogenesis was significantly inhibited but towards the end of the experiment there was significant stimulation of methanogenesis. Sulphate reduction was significantly inhibited and carbon dioxide evolution was only significantly inhibited in one of three experiments.

Bauer and Capone (1985b) studied the metabolism of ^{14}C-glucose and the incorporation of [methyl-^{3}H]thymidine by aerobic and anaerobic marine sediment microbes exposed to naphthalene concentrations of 1, 10, 100 and 1,000 mg/l. Naphthalene concentrations of 100 and 1,000 mg/l significantly inhibited glucose metabolism and thymidine incorporation in both aerobic and anaerobic sediment.

Vaishnav (1986) studied the effect of naphthalene on the biodegradation of primary alcohols by a mixed microbial culture. An IC_{50}, based on maximum observed biodegradation rate, was calculated to be 1,154 mg/l.

Schultz et al. (1983) exposed the microorganism *Tetrahymena pyriformis* to naphthalene and studied the effect on biological activity monitored as population growth. A graded series of 4 naphthalene concentrations were tested and cultures without toxicant were used as controls. Incubation of cultures at each concentration was carried out in the dark for 60 hours. The concentration that inhibits 50% growth of population following 60-hour exposure was calculated to be 188.85 mg/l.

3.2.1.2   Calculation of Predicted No Effect Concentration (PNEC)

3.2.1.2.1   Calculation of PNEC for water

Toxicity data for naphthalene were available for microorganisms, aquatic plants, aquatic invertebrates and fish under acute conditions together with some chronic data. The reported results are from both static and flow-through tests with some based on nominal concentrations and some on measured concentrations.

The test results available for algae appear to show short-term effects at lower levels than the results from longer term tests. Thus growth was affected at 400 µg/l over three days (Østgaard et al., 1984) and a 4-hour EC_{50} for CO_2 assimilation of 2.9 mg/l was determined (Milleman et al., 1984), whereas 10-day and 14-day tests had LC_{50}s for biomass production of 33 and 25 mg/l (Kauss and Hutchinson, 1975 and Gaur, 1988, respectively). This may indicate acclimation to the chemical with time.

There are a lot of data available on fish and invertebrates, and a wide range of species has been tested. The majority of the results from short-term tests lie in the range 1-10 mg/l. All of the

organisms tested appear to show similar sensitivity in the short-term tests. There is some evidence to suggest that naphthalene exerts its toxic effect by narcosis. Longer-term studies on fish (over 40 days) give similar values for the LC_{50} to tests over short time scales. Acute toxicity values have been predicted by Bol et al. (1993) using QSAR equations for chemicals that act by narcosis. The predicted values were 7.8 mg/l (LC_{50} for fish), 6.1 mg/l (LC_{50} for daphnia) and 3.8 mg/l (EC_{50} for algae), all of which fit closely the range of measured values whilst being towards the high end.

Longer-term studies are also available. As mentioned above, 40-day tests on Coho and pink salmon (Moles et al., 1981 and Rice and Thomas, 1989, respectively) gave similar LC_{50}s to the short-term tests. The NOEC for weight gain in a 40-day test with coho salmon fry was 0.12 mg/l (Moles and Rice, 1983). An embryo-larval study on fathead minnows had a NOEC for survival of 1.84 mg/l and a NOEC for hatchability and fish length/weight of 0.45 mg/l (de Graeve et al., 1982). The NOEC for daphnia is 0.6 mg/l (Geiger and Buikema, 1982) and for sea urchin eggs 0.22 mg/l (Falk-Petersen et al., 1982). The available data cover two trophic levels and, as all organisms appear to have similar sensitivity in the short-term tests, these can be considered to cover the most sensitive species. Hence a factor of 50 is applied to the lowest value (0.12 mg/l) to give a PNEC of 2.4 μg/l.

There are a small number of results that cannot be fitted easily into this scheme. One study derived LC_{50}s for fish exposed from egg fertilisation through to 4 days post hatching of 0.11 mg/l and 0.51 mg/l (Black et al., 1983). The method uses mortality in assessing toxicity, determined through tests carried out during early life stages. The test substance can act much earlier and more intensively on the early development stages as the egg jelly is very thin and permeable to water soluble substances immediately after fertilisation. The results derived in these studies (LC_{50}s of 0.11 mg/l and 0.51 mg/l) are consequently low in comparison with those of other studies. Similar trends are observed with other substances used in this technique. However, it is difficult to use these results in a conventional acute or chronic framework as used when deciding on assessment factors. The German UBA has carried out a Probit analysis using the data from this study to calculate an LC10. This has been treated as a NOEC and has been used to derive a PNEC of 1.9μg/l. This PNEC value is similar to that of 2.4 used in the assessment. The results obtained by Black et al. have also been called into question due to problems in reproducing the results in other laboratories. The WRc, in attempting to repeat the toluene toxicity study, obtained results that were consistent with those of other studies rather than with those obtained by Black et al., although there may have been differences regarding the handling of eggs immediately after fertilisation.

Sanborn and Malins (1977) found that 8-12 μg/l naphthalene affected marine larval invertebrates (although Caldwell et al. (1977) determined an LC_{50} for the same crab species as >2 mg/l). Ott et al. (1978) found that 14 μg/l naphthalene caused an effect on female copepods exposed for their adult life (up to 29 days). None of these studies allows a NOEC value to be determined. They are also all considered as 'use with care' studies and so should be used to support the PNEC derivation rather than act as the basis for it. The value of 2.4 μg/l derived above is lower than the effect levels and so should be sufficiently protective.

Provisional ecotoxicological assessment criteria for naphthalene in seawater and sediment were agreed in November 1993 (Oslo and Paris Commissions, 1994). For seawater, the ecotoxicological assessment criteria was provisionally set as 1-10 μg/l, based on a NOEC of 40 μg/l and an assessment factor of 10. Concentrations in sediment were calculated by applying the equilibrium partitioning approach and the provisional assessment criteria for sediment is 10-100 μg/kg dry weight.

An environmental quality standard (EQS) of 1.0 μg/l has also been recommended by the Scientific Advisory Committee who advises the European Commission (EUR 15674 reported in ENDS, 1994).

3.2.1.2.2   Calculation of PNEC for microorganisms in wastewater treatment plants

Chemicals can have an adverse effect on microbial activity in WWTPs. Therefore, a $PNEC_{microorganisms}$ is derived. The PNEC should be calculated as the concentration at which significant effects occur, in particular short-term measurements equivalent to the retention time of the chemical in the WWTP are preferable. The assessment factor to be used depends upon the microbial effect data available. If the test has been performed on nitrifying bacteria, the effect concentration may be compared directly with the effluent concentration. For other tests assessment factors in the range of 10 to 100 may be applied.

For naphthalene, toxic effects upon nitrifying microorganisms are observed at 29 mg/l (IC_{50} for *Nitrosomonas*). An assessment factor of 10 should be applied to the EC_{50} result from tests performed with specific bacterial populations. Thus the PNEC for microorganisms is 2.9 mg/l.

3.2.1.2.3   Calculation of PNEC for sediment dwelling organisms

In the absence of data on the toxic effects of naphthalene on sediment dwelling organisms it is proposed that a sediment partition method is used to calculate a $PNEC_{sediment}$. The $PNEC_{sed}$ has been calculated from the PNEC for water using the equation:

$$PNEC_{sed} = \frac{K_{suspwater}}{RHO_{susp}} \cdot PNEC_{water} \cdot 1000$$

where:
$K_{susp-water}$ =   suspended matter-water partition coefficient
RHO_{susp} =   bulk density of suspended matter

Note: This equation differs from that given in the Technical Guidance Document. It uses the suspended matter - water coefficient rather than that for sediment - water and so is consistent with the equation used to calculate PEC_{sed} from PEC_{water}. The value of $K_{suspwater}$ is 32.2, from Section 3.1.2.2.1.

For naphthalene, $PNEC_{water}$ is 2.4 μg/l. This gives a $PNEC_{sediment}$ of 67.2 μg/kg for freshwater.

3.2.2   Terrestrial compartment

3.2.2.1   Toxicity to terrestrial organisms

Laboratory tests of the growth rate of basidomycete fungi in the presence of naphthalene vapour were carried out (Newell et al., 1987). Nine different basidomycete fungi were grown on malt extract agar at 11°C in the presence of naphthalene vapour for 6 days. All but one of the test fungi showed significant reduction in radial growth and sparse, irregular growth in the presence

of naphthalene vapour. The mean reduction in radial growth compared with the controls varied between 31% and 70%.

Neuhauser et al. (1986) studied the toxicity of organic compounds to earthworms in a 48-hour contact test. A 48-hour LC_{50} of 4,670 $\mu g/cm^2$ was calculated for the earthworm *Eisenia foetida*. The LC_{50} value refers to μg of naphthalene applied per cm^2 of filter paper in contact with the earthworm.

Walton (1980) exposed early instar crickets (*Acheta domesticus*) to naphthalene in both acute and chronic toxicity studies. The LD_{50} was found to be >15,000 mg/kg in food and >580 mg/kg body weight via topical application. In chronic tests, crickets were exposed to 0.1% naphthalene in feed and an LT_{50} of 12 days was calculated. In topical toxicity tests, naphthalene was applied every other day at 0.1% and an LT_{50} of >18 days was found. However, this information cannot be used in the derivation of a PNEC.

3.2.2.2   Calculation of PNEC

Results of toxicity testing for terrestrial organisms are limited and only short-term tests have been carried out. The PNEC for the terrestrial compartment has therefore been calculated using the equilibrium partitioning method in the Technical Guidance document. The PNEC for soil is 53.3 $\mu g/kg$ using the PNEC calculated for freshwater. This value will be used in the assessment.

It has been suggested that naphthalene might be able to have an effect on organisms exposed through the air in soil as well as through the water. The equilibrium partition approach assumes that exposure occurs through the water alone and so would not address this. In the absence of data on naphthalene toxicity to terrestrial organisms, information on 1,4-dichlorobenzene has been examined (from the risk assessment for that substance). Although there are structural differences the physical properties are similar, with 1,4-dichlorbenzene having a lower boiling point and higher vapour pressure. It is used in mothballs in the same way as naphthalene. A set of short-term terrestrial toxicity tests are available for 1,4-dichlorobenzene, which give a PNEC of 85 $\mu g/kg$ wet weight using a factor of 1,000. The equilibrium partitioning approach gives a value of 160 $\mu g/kg$ from the aquatic PNEC, which is derived using a factor of ten on a data set of three long-term tests. Longer term testing on soil organisms might be expected to result in an increase in the PNEC from this route. On the basis of these data, there is no indication of extra toxicity to soil organisms through uptake from solids or air. The same can be expected for naphthalene, and therefore the equilibrium partitioning PNEC will be used in the risk characterisation.

3.2.3   Atmosphere

3.2.3.1   Calculation of PNEC

It is not possible to calculate a PNEC for naphthalene in the atmosphere using the methods of the Technical Guidance Document. Information on inhalation toxicity is presented in the human health sections of the report, and this will be referred to in the risk characterisation.

Naphthalene absorbs strongly in the solar UV region and is not expected to contribute to global warming.

The lifetime of naphthalene in the atmosphere is thought to be approximately 1 day based on measurements of reactions with hydroxyl radicals. Since the atmospheric lifetime is short and naphthalene does not contain chlorine or bromine substituents, naphthalene will not contribute significantly to stratospheric ozone depletion.

Naphthalene has a photochemical ozone creation potential (POCP) of 35 relative to ethylene which has a PCOP of 100 (Derwent, 1991, personal communication). Therefore, naphthalene may contribute to the formation of tropospheric ozone.

Naphthalene is not likely to contribute to acidification in the atmosphere.

3.2.4   Secondary poisoning

It is assumed that the available mammalian toxicity data can give an indication of possible hazards to higher organisms in the environment. This is done by considering whether the substance has certain classifications: Toxic or Very Toxic, or potential for harm over prolonged exposure (R48), effects on fertility (R60 or R62), on the unborn child (R61, R63) or harm to breast-fed babies (R64). Naphthalene has none of the indicated classifications and so does not require a secondary poisoning assessment.

3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment (incl. sediment)

3.3.1.1 Water

Comparison of the predicted no effect concentrations with those measured in the environment can be used to identify those areas, if any, where the chemical might have effects.

Using 2.4 µg/l as the PNEC and 2.24 µg/l as the highest measured concentration in European surface water in industrial/urban areas gives a PEC/PNEC ratio of 0.93, which indicates no concern. The maximum levels measured in other river waters (6.85 µg/l) gives a ratio of 2.85. This could indicate some effects in the rivers most affected by naphthalene release. The highest level reported (14.1 mg/l), from a heavily contaminated stream, gives a ratio of 5,875, showing that spillage or gross contamination would certainly have effects on aquatic organisms. The measured levels cannot be related to specific activities involving the production or use of naphthalene as a substance and may well reflect indirect sources. Therefore the assessment will be based on the calculated PEC values. PEC/PNEC ratios derived using local PECs calculated from site-specific data or default values given in the Technical Guidance Document are given in **Table 3.40**.

Table 3.40 PEC/PNEC ratios for water

	PEC/µg/l	PEC/PNEC ratio
Production - Site A	0.036	0.015
Production - Site B	0.20	0.083
Production - Site C	0.059	0.025
Production - Site D	0.31	0.13
Production - Site E	0.04	0.017
Production - Site F	0.05	0.021
Production - Site G	0.035	0.015
Production - Site H	0.055	0.023
Production - Site I	0.07	0.029
Production - Site J	0.035	0.015
Use as intermediate (site-specific)	0.04	0.017
Use as intermediate	0.042	0.018
Pyrotechnics manufacture	2.35	0.98
Mothballs manufacture	0.03	0.013
Grinding wheels manufacture	294	123
Regional	0.03	0.013
Continental	0.0025	0.001

Based on the releases calculated according to the Technical Guidance Document using emission factors derived from worst-case site-specific data for production and use as an intermediate naphthalene is unlikely to cause adverse effects on the aquatic environment. The PECs calculated for the regional and continental environments also suggest that adverse effects on the aquatic compartment will not occur. The PEC/PNEC ratios indicate that there may be adverse effects in the aqueous environment arising from the use of naphthalene in the manufacture of grinding wheels. This is based on site-specific data for one plant. Site-specific data for the use of naphthalene in the manufacture of grinding wheels at another plant have indicated that there should be no adverse effects arising from its use at this location.

The PEC/PNEC ratio for the use of naphthalene in the formulation of pyrotechnics indicates that there will not be any adverse effects in the aqueous environment. However, the comparatively high value of 0.95 reflects the fact that the calculation was based on a worst-case release scenario. The release to water was calculated using the default emission factor in the Technical Guidance Document. As the process is reported to be dry the calculated release is likely to be an overestimate. Much of the naphthalene used in pyrotechnics is known to be used "neat" rather than being used in a formulation. However, the tonnage used in the calculation assumed that all of is subject to a formulation stage and this assumption will also lead to the actual release being overestimated.

If the recommended EQS value for naphthalene is met then adverse effects would also not be expected.

Conclusions for surface water

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the use of naphthalene as a pore former in the production of grinding wheels.

Conclusion (ii)   There is at present no need for further information and/or testing, and no need for risk reduction measures beyond those which are already being applied.

This conclusion applies to the production of naphthalene and its use as an intermediate, in pyrotechnics and in mothballs.

3.3.1.2   Sediment

The PNEC for sediment from freshwater areas was calculated based on the equilibrium partitioning to be 67.2 μg/kg. This can be compared with measured levels of naphthalene in sediments.

The majority of measurements of naphthalene in sediments have been carried out at contaminated sites. Maximum levels exceed 100 μg/kg in many studies and levels of naphthalene up to 7,720 mg/kg have been found. This value gives a PEC/PNEC ratio of 124,000. Values up to 1.47 mg/kg have also been measured in urban areas, giving a PEC/PNEC ratio of 23.6. The measured levels cannot be related to specific activities involving the production or use of naphthalene as a substance and may well reflect indirect sources. Therefore the assessment will be based on the calculated PEC values. PEC/PNEC ratios for sediment derived using local

PEC's, calculated from site-specific data or default values given in the Technical Guidance Document, are given in **Table 3.41**.

Based on the releases calculated according to the Technical Guidance Document using emission factors based on worst-case site-specific data for production and use as an intermediate naphthalene is unlikely to cause adverse effects. The PECs calculated for the regional and continental environments also suggest that adverse effects will not occur.

Table 3.41 PEC/PNEC ratios for sediment

	PEC/μg/kg	PEC/PNEC ratio
Production - Site A	1.0	0.015
Production - Site B	5.6	0.083
Production - Site C	1.7	0.025
Production - Site D	8.7	0.13
Production - Site E	1.1	0.017
Production - Site F	1.4	0.021
Production - Site G	0.98	0.015
Production - Site H	1.5	0.023
Production - Site I	2.0	0.029
Production - Site J	0.98	0.015
Use as intermediate (site-specific)	1.1	0.016
Use as intermediate	1.2	0.018
Pyrotechnics manufacture	66	0.98
Mothballs manufacture	0.83	0.012
Grinding wheels manufacture	8232	123
Regional	0.83	0.012
Continental	0.07	0.001

The PEC/PNEC ratio for the use of naphthalene in the manufacture of grinding wheels indicates that there may be adverse effects in the aqueous environment. This is based on site-specific data for one plant. Site-specific data for the use of naphthalene in the manufacture of grinding wheels at another plant have indicated that there should be no adverse effects arising from its use at this location.

The PEC/PNEC ratio for the use of naphthalene in the formulation of pyrotechnics indicates that there will not be any adverse effects in the aqueous environment. However, the comparatively high value of 0.98 reflects the fact that the calculation was based on a worst-case release scenario. The release to water was calculated using the default emission factor in the Technical Guidance Document. As the process is reported to be dry the calculated release is likely to be an overestimate. Much of the naphthalene used in pyrotechnics is known to be used "neat" rather than being used in a formulation. However, the tonnage used in the calculation assumed that all of is subject to a formulation stage and this assumption will also lead to the actual release being overestimated.

Measured levels in urban and motorway run-offs, and at a number of contaminated sites are high enough to cause concern based on the PNEC derived. However, the production and use of naphthalene itself does not contribute significantly to these levels. Combustion related sources account for about 88% of total naphthalene releases on a regional scale (see **Table 3.17**).

Conclusions for sediment

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the use of naphthalene as a pore former in the production of grinding wheels. It is recognised that for sediment the PNEC used is derived from the surface water PNEC using the equilibrium partitioning method, and so could be revised through toxicity testing. However, risks have only been identified for one site using naphthalene for this purpose. This site is developing plans to reduce releases to water, air and sludge so the rapporteur does not think it necessary to require such testing to be carried out. The planned risk management strategy will be based on the above conclusions to ensure that emissions to water (and hence sediment) are limited.

Conclusion (ii)   There is at present no need for further information and/or testing, and no need for risk reduction measures beyond those which are already being applied.

This conclusion applies to the production of naphthalene and its use as an intermediate, in pyrotechnics and in mothballs.

3.3.1.3   **Microorganisms**

The PNEC for microorganisms is 2.9 mg/l and this can be compared with $PEC_{effluent}$. The highest measured concentration in effluent included in the site-specific information for sites at which naphthalene is produced and used) is 20 μg/l. The highest calculated level in effluent is 730 μg/l. The ratio PEC/PNEC is 0.007 or 0.25 suggesting that naphthalene is unlikely to cause any adverse effects in a treatment plant. For the use of naphthalene in the manufacture of grinding wheels the value for $PEC_{effluent}$ is 2.9 mg/l. The PEC/PNEC ratio is therefore 1 indicating that naphthalene may cause adverse effects in a treatment plant as a result of this use.

Conclusion for microorganisms in wastewater treatment plants

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the use of naphthalene as a pore former in the production of grinding wheels.

Conclusion (ii)   There is at present no need for further information and/or testing, and no need for risk reduction measures beyond those which are already being applied.

This conclusion applies to the production of naphthalene and its use as an intermediate, in pyrotechnics and in mothballs.

3.3.2 Terrestrial compartment

A PNEC for the terrestrial compartment has been calculated based on the PNEC for aquatic organisms - this gives 53.3 µg/kg. Naphthalene levels in various soils have been measured. In uncontaminated soils, mean naphthalene levels are around 10-20 µg/kg (with peak values up to 131 µg/kg). The mean values give a PEC/PNEC ratio of 0.35. Much higher levels have been found on contaminated sites, the highest found being 400 mg/kg. The vast majority of the sites are contaminated with naphthalene indirectly, for example on gasworks sites or hazardous waste dumps. No values are available for locations close to the production and use sites of naphthalene itself. Therefore the calculated values for PEC will be used in the assessment. PEC/PNEC ratios derived from measured values in soil and the calculated PECs for specific uses of naphthalene are given in **Table 3.42**.

Table 3.42 PEC/PNEC ratios for soil

	PEC/µg/kg	PEC/PNEC ratio
Production Site A	1.7	0.032
Production Site B	0.15	0.003
Production Site C	0.45	0.008
Production Site D	0.55	0.010
Production Site E	0.35	0.007
Production Site F	0.19	0.004
Production Site G	0.29	0.005
Production Site H	0.96	0.018
Production Site I	0.32	0.006
Production Site J	0.14	0.003
Use as intermediate (site-specific)	1.0	0.019
Use as intermediate	50	0.94
Pyrotechnics manufacture	36	0.68
Mothballs manufacture	1.0	0.019
Grinding wheels manufacture	4600	86
Regional	0.3	0.006
Continental	0.023	0.0004

PEC's for soil calculated using site-specific data for production and processing sites give PEC/PNEC ratios below 1 in all cases. The PEC/PNEC ratio for the generic calculation for emissions from use as an intermediate is high, but not sufficiently so to indicate that there may be adverse effects. However, the PEC for this release has been calculated according to the Technical Guidance Document, with sludge from treatment plants assumed to be applied to soil. The evidence from production and processing sites indicates that this is unlikely to be the case and the PEC/PNEC ratio for such a site would therefore be expected to be considerably lower. PEC/PNEC ratios for sites for which specific information is available are all below 0.02.

The PEC/PNEC ratio for the use of naphthalene in the manufacture of grinding wheels indicates that it may cause adverse effects if the sludge from treatment plants is applied to soil. This is based on site-specific data for one plant. Site-specific data for the use of naphthalene in the manufacture of grinding wheels at another plant have indicated that there should be no adverse effects arising from its use at this location.

The PECs calculated for the regional and continental environments suggest that adverse effects will not occur at these levels.

Conclusion for the terrestrial environment

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the use of naphthalene as a pore former in the production of grinding wheels. It is recognised that for soil the PNEC used is derived from the surface water PNEC using the equilibrium partitioning method, and so could be revised through toxicity testing. However, risks have only been identified for one site using naphthalene for this purpose. At the time of the production of the risk assessment, this site was developing plans to reduce releases to water, air and sludge so the rapporteur did not think it necessary to require such testing to be carried out. The planned risk management strategy will be based on the above conclusions to ensure that emissions to sludge (and hence the terrestrial compartment) are limited.

Conclusion (ii)   There is at present no need for further information and/or testing, and no need for risk reduction measures beyond those which are already being applied.

This conclusion applies to the production of naphthalene and its use as an intermediate, in pyrotechnics and in mothballs.

3.3.3   Atmosphere

A PNEC has not been calculated for the atmosphere. In the human health risk characterisation, the margin of safety between the air levels calculated for local emission sites and the effect level for local respiratory effects (irritation and carcinogenicity) is considered to be sufficient for there to be no risk to humans. This is also considered to be sufficiently protective of organisms in the environment exposed through inhalation.

Naphthalene is not thought to contribute to global warming, stratospheric ozone depletion or acidification. Naphthalene has a POCP of 35 so may contribute to the formation of tropospheric ozone.

3.3.4   Secondary poisoning

Naphthalene has a log K_{ow} of 3.73, and some bioaccumulation factors greater than 100 have been measured. However, naphthalene does not carry the risk phrases T or T+, R47, R48 or R60-63. Therefore, it is not necessary to carry out a risk characterisation of secondary poisoning.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 Occupational exposure

4.1.1.1.1 General introduction

Definitions and limitations

In this document, unless otherwise stated, the term exposure is used to denote personal exposure as measured or otherwise assessed without taking into account the attenuating effect of any respiratory protective equipment (RPE) which might have been worn. The effect of RPE is dealt with separately. This definition permits the effects of controls, other than RPE, to be assessed and avoids the considerable uncertainty associated with attempting to precisely quantify the attenuation of exposure brought about by the proper use of RPE.

The section entitled general discussion summarises the more important issues arising from the exposure assessments and bring together measured exposure data with that predicted from the EASE (Estimation and Assessment of Substance Exposure) model. EASE is a general purpose predictive model for workplace exposure assessments. It is an electronic, knowledge based, expert system which is used where measured exposure data is limited or not available. The model is in widespread use across the European Union for the occupational exposure assessment of new and existing substances.

All models are based upon assumptions. Their outputs are at best approximate and may be wrong. EASE is only intended to give generalised exposure data and works best in an exposure assessment when the relevance of the modelled data can be compared with and evaluated against measured data.

EASE predicts exposures as ranges in the form of conventional eight-hour time weighted averages (TWAs). It does not directly predict short-term exposures. However, because these exposures are process specific, they can be thought of as those experienced for that process either over the whole eight hours or over any shorter period. These shorter periods can be further time weighted to construct other eight-hour time weighted averages. Although this device allows short-term exposures to be dealt with by EASE, such constructs should be regarded with caution. Dermal exposure is assessed by EASE as potential exposure rate predominantly to the hands and forearms (approximately 2,000 cm^2).

Air sampling data is presented in the following sections from a number of sources and, where reported to HSE, information on the sampling methods is also presented. HSE, in general, does not have information on the validity of the techniques used and the sampling strategies adopted. There was, however, no reason to doubt the quality of the air sampling data used in this occupational hygiene exposure assessment.

4.1.1.1.2   Manufacture

It is unrealistic to attempt to estimate the total number of workers exposed to naphthalene. The number exposed during naphthalene manufacture and subsequent use is estimated to be 250 to 500 in the UK and 1,500 to 2,000 in the EU (this does not include operators handling creosote treated timber or brush applicators or users of tar paints/membranes). The number exposed as a result of incomplete combustion of organic materials is likely to be significantly higher than these figures.

Naphthalene is a solid at ambient temperature and melts at 80ºC, although the solid does readily sublime. Therefore situations where it is used as a solid (i.e. flake or powder) will give rise to exposure to both particulate and vapour. These uses include the manufacture of mothballs, its use in pyrotechnics and as a repellent in museums. Dermal exposure will also occur where workers directly handle the solid naphthalene or come into contact with surfaces contaminated with condensed vapour or particulate.

During use in chemical synthesis and as a constituent of creosote it is transported and used at 90ºC or above (i.e. greater than its melting point of 80ºC) to avoid it solidifying in pipelines and vessels. Exposure will therefore be primarily to the vapour. The only scenarios where workers in these industries come into contact with solid naphthalene are during the cleaning of solidified spillages or blocked pipelines. In these situations the naphthalene is in the form of a "solidified cake", with exposure to particulate dependent on the methods used for removing it. Removal of this "cake" is likely to result in the generation of relatively large pieces of solid naphthalene and not large amounts of inhalable particulate. Dermal exposure will occur where workers come into contact with surfaces contaminated by splashes or condensed vapour or as a result of direct splashes on to the skin. Removal of the solidified naphthalene may result in dermal exposure to the solid.

HSE has no data on occupational exposure to naphthalene during its manufacture and use and only a small amount was received from industry within the EU. With the exception of published papers this data was primarily received from companies in the UK as was information on the measures used to control exposure. Consequently the exposure assessments are based on either no results or only a few and the experience of UK companies. Professional judgement was used where there were gaps in information and it is assumed in the absence of further knowledge that these exposure assessments are representative of all EU Member States.

Occupational exposure can also occur during the incomplete combustion of organic material. This is discussed in the section titled "Occupational exposure to naphthalene from other sources". The highest exposures were found during the use of creosote and the production of mothballs and manufacture of grinding wheels.

4.1.1.1.3   Occupational exposure limits

Many EU member states have adopted an occupational exposure limit for naphthalene of 50 mg/m^3 8-hour TWA (Occupational Exposure Limits – ILO, 1991).

4.1.1.1.4   Occupational exposure during the manufacture of naphthalene

It is understood that about 10 persons (including maintenance operators) are exposed to naphthalene vapour during distillation of the coal tar to produce the naphthalene oil at each of the two UK tar

distillation plants. The total exposed during the distillation of coal tar to produce the naphthalene oil throughout the EU was not established. However, it is estimated that it is in the region of 100 to 200 employees.

During subsequent purification of the naphthalene by either distillation or crystallisation there are an estimated further 4 exposed in the UK, with a further 50 to 60 throughout the EU.

Manufacture is carried out in closed plant with occupational exposure arising during activities such as sampling, tanker filling and maintenance. The last mentioned may involve the removal of solidified naphthalene from pipe lines and vessels, for which it is understood PPE including respiratory protective equipment is worn at the UK plant. Exposure in this scenario would be to both particulate and vapour, although the latter is likely to be the greatest contributor to exposure.

Industry exposure data

Air sampling to determine occupational exposure is not carried out at the UK plants and it appears that this is the case for many EU manufacturing plants. Manufacturers have assessed the process and deemed air sampling to be unnecessary due to the exposure controls in place. Occupational exposure data from air sampling at various tar distillation plants in Europe was received from a EU producer. This occupational exposure data is presented in **Table 4.1**. Limited information was supplied with this data. They were reported as being below the German MAC value of 50 mg/m^3. It is assumed that they are from personal air sampling and represent 8-hour TWA exposure and therefore provide a reasonable prediction of exposure during tar distillation.

Table 4.1 Occupational exposure to naphthalene during tar distillation *(various plants throughout Europe)*

Plant area	Range (mg/m^3)*
Crystallisation	0.1 to 0.8
Laboratory	0.40
Tank car loading with coal tar products	0.76 to 4.8
Coal tar distillation	0.16

* The number of samples was not reported

The results in **Table 4.1** were reported as "representative" data with a further value of 6.3 mg/m^3 reported as the highest result recorded. This value will be carried forward to the risk characterisation section.

Modelled dermal exposure data

Dermal exposure can occur during the production of naphthalene, when operators come into contact with surfaces contaminated from splashing or condensed vapour, or as a result of direct contact onto the skin. As processing is in closed systems, dermal exposure will primarily occur during activities such as sampling and the uncoupling of pipes.

The best EASE scenario for this exposure is direct handling with incidental contact, where incidental refers to one significant contact in a shift, for example spilling naphthalene whilst sampling or touching a wet surface. This results in a prediction of 0 to 0.1 mg/cm^2/day, although on most days no such accidental contacts will occur and exposure will be towards the bottom of

this range. However, the upper end of the range may reflect exposure during maintenance activities where dermal contact is greater.

Operators are likely to wear gloves where the potential for skin contact exists and thus further reduce the above-predicted exposures.

4.1.1.1.5   Occupational exposure during use in chemical synthesis

<u>Manufacture of phthalic anhydride</u>

It is estimated that about 10 employees are exposed to naphthalene during its use at the UK manufacturing plant, with a total of 30 exposed throughout the EU. At the UK plant manufacture of phthalic anhydride is carried out in closed plant with occupational exposure to the vapour during activities such as material sampling and maintenance. Exposure to the vapour may also occur during delivery, where a small quantity of naphthalene is run off prior to connecting to the storage tank. This is to remove any soda that may have remained in the naphthalene after soda washing at the production plant.

Industry exposure data

Table 4.2 shows the results of a personal air sampling exercise undertaken at the UK manufacturing site in 1994 during routine work.

About every 2 to 3 years the process plant vessels are cleaned out which requires the operator to enter the vessel to scrape out solid naphthalene. This will result in exposure to both particulate and vapour, although the latter is likely to be the greater contributor to exposure to due to the nature of the solid naphthalene "cake". During this work the operator wears compressed air line breathing apparatus and gloves to reduce their exposure.

Table 4.2 Occupational exposure to naphthalene during the manufacture of phthalic anhydride
(these results represent single exposures)

Task	Result (mg/m^3)*	8-hour TWA(mg/m^3)
Process operator	0.38	0.57
Charge hand	0.22	0.33
Process operator	0.62	0.93
Charge hand	1.30	2.00

* Results represent exposure over a 12-hour shift

Modelled dermal exposure data

Dermal exposure can occur during the production and of naphthalene, where operators come into contact with surfaces contaminated from splashing or condensed vapour, or as a result of direct contact onto the skin. As processing is in closed systems, dermal exposure will primarily occur during activities such as sampling and the uncoupling of pipes.

The best EASE scenario for this exposure is direct handling with incidental contact, where incidental refers to one significant contact in a shift, for example spilling naphthalene whilst sampling or touching a wet surface. This results in a prediction of 0 to 0.1 mg/cm^2/day, although on most days no such accidental contacts will occur and exposure will be towards the bottom of this range. However, the upper end of the range may reflect exposure during maintenance activities where dermal contact is greater.

Operators are likely to wear gloves where the potential for skin contact exists and thus further reduce the above-predicted exposures.

Other chemical syntheses

Occupational exposure may also occur to naphthalene during its use in the synthesis of naphthalene sulphonic acids, alkylated naphthalene solvents, insecticides etc. Occupational exposure data was not available from these industries.

It is understood that for these applications it is used in closed plant with exposure arising during similar tasks to those during manufacture of naphthalene and phthalic anhydride. Occupational exposure via inhalation and dermal contact are likely to be similar to that during the manufacture of naphthalene and synthesis of phthalic anhydride.

4.1.1.1.6 Occupational exposure during blending and use of creosote

Occupational exposure to naphthalene in creosote may occur during blending, the treatment of timber at bulk impregnation plants, packaging and during subsequent use of the packaged product. Industry occupational exposure data was not received for creosote blending or its subsequent use. The highest occupational exposures to naphthalene are likely to occur during the bulk impregnation of timber. Exposure via inhalation will be to naphthalene vapour and not particulate, as even at ambient temperature the creosote will be a liquid.

The substances perceived to be of greatest concern by the creosote industry in respect of human health are the polycyclic aromatic hydrocarbons (PAHs) including benzo(a)pyrene (B(a)P). Naphthalene, although a PAH, is not deemed by the industry to be of significant concern in terms of human health. Concerns over these PAHs and not naphthalene appear to be the main driving force behind controlling exposure, although environmental pressures over naphthalene have also resulted in improvements in control.

Occupational exposure to naphthalene whilst blending or using creosote is discussed below for:

(a) blending;
(b) the bulk impregnation of timber;
(c) creosote packaging plants (i.e. for do-it-yourself outlets); and
(d) brush application.

At the end of these four sections dermal exposure is discussed for all uses of creosote.

Blending

Blending of creosote is generally carried out at tar distillation plants where the naphthalene is manufactured. This involves piped delivery of the tar distillates from the tar distillation plant to storage followed by delivery to closed vessels for blending. The blended creosote is then transferred to road tankers, during which the greatest potential for exposure occurs. Sampling and maintenance may also result in exposure to naphthalene vapour. Air sampling data was not received from blenders of creosote. It is estimated that occupational exposure similar to that during the manufacture naphthalene and the manufacture of phthalic anhydride will occur.

The number of workers exposed to naphthalene during creosote blending at the UK tar distillation plant was reported to be 2 to 3. Therefore it is estimated that 20 to 30 are exposed throughout the EU. These figures do not include maintenance personnel as they have previously been included in the figures for manufacture of naphthalene. The number of workers exposed at plants blending purchased distillates was not established.

Bulk impregnation plants

Air sampling data from bulk impregnation plants was not obtained as it appears that routine air sampling for naphthalene is not carried out. Operators have carried out modifications to impregnation plants primarily to reduce exposure to PAHs including B(a)P. Modifications have also been made to reduce environmental emissions of these and naphthalene. These modifications have reportedly reduced emissions to both the environment and the workplace. Air sampling has been carried out to assess environmental emissions. However, for the latter this has been subjectively assessed by impregnators on the basis of odour.

The number of workers directly exposed on an impregnation plant is 3 to 4. Therefore it is estimated that 15 to 20 are exposed throughout the UK and 25 to 50 throughout the EU. It was unrealistic to attempt to establish how many workers may be exposed during the handling of treated sleepers, telegraph poles, fences etc. The treatment of timber sleepers has declined due to the introduction of concrete sleepers.

Published exposure data

A study of occupational exposure to creosote vapours was carried out at two bulk impregnation plants in Finland in 1987 (Heikkila, P. Hemeila, M. et al.). This survey investigated a number of components including naphthalene but predominantly PAHs and B(a)P. Consequently only limited details on exposure to naphthalene were presented. At one of the impregnation plants rail road sleepers were being impregnated and in the other telegraph poles. This study primarily covered workers operating the impregnation chambers at the two plants who were potentially exposed when the chamber doors were opened. Activities also covered were those where workers handled treated timber, such as stevedores and railway workers laying sleepers or carrying out welding on track. Personal air sampling was carried out by using XAD-2 tubes with subsequent analysis by gas chromatography and mass spectrometry. The results are summarised in **Table 4.3**.

Table 4.3 Occupational exposure to naphthalene during the impregnation of timber with creosote and during subsequent work on treated sleepers

Measurement site ***	No of samples	Range (mg/m³)	Mean (mg/m³)	Percentage in range		
				<1-10	11-50	>50
Impregnation plant 1	7*	<0.5 to 51	9.00	87.50	-	12.50
Impregnation plant 2	5	<0.5 to 26	6.00	80.00	1.00	-
Switch elements room	2	2-6	4.00	100.00	-	-
Railway yard	4	<0.5**	<0.5	100.00	-	-

*	This result does not include 8-hour TWA exposure for the operator opening the chamber. A 10-minute air sample was collected during opening of the chamber which gave a result of 26 mg/m³
**	All four samples were <0.5 mg/m³
***	These results include indene and methyl naphthalene as the authors deemed their toxicological effects to be similar
NB	These results were compared by the authors against the TLV of 50 mg/m³, it is therefore assumed that they represent 8-hour TWAs

The value of 8 mg/m³ will be carried forward to the risk characterisation because the exposure over the course of a day is considered more representative than peak exposures (see Modelled exposure data below).

The authors reported that, of the total concentration of creosote vapour in the samples from the workers in the impregnation plants and from the workers handling treated timber, the percentage of naphthalene was 52% and 32%, respectively. The highest naphthalene exposure of 51 mg/m³ (this included indene and methyl naphthalene) was for an operator carrying out the annual cleaning of the impregnation chamber. The report did not provide exact details on how this cleaning was carried out. At a typical UK plant cleaning of the inside of the chamber is generally carried out by flushing through hot creosote. Should an operator need to enter the chamber to remove residues and timber debris then PPE is worn, including a respirator with an organic filter and gloves. This PPE is reported to be worn during any task giving rise to significant exposure.

The authors felt that in general exposures in plant 1 were higher than plant 2, although this was not fully borne out by the results. However, it was difficult to compare the results from the two plants directly as the surveys included different tasks. In plant 1 the chamber doors were opened manually with the operator in the proximity for 30 minutes/shift whereas in plant 2 the doors were opened automatically. In addition, extraction ventilation is fitted above the chamber openings at plant 2.

The majority of operators of impregnation plants have made improvements to prevent the worker from being in the vicinity of the chamber when it is opened. Plant 2 therefore appears to be more typical of current impregnation plants.

Modelled exposure data

Occupational exposure during the bulk impregnation of timber was modelled using EASE. The EASE scenario that best describes this is non-dispersive use with LEV, which results in a prediction of 0.5 to 3 ppm (2.6 to 16 mg/m³) 8-hour TWA for the naphthalene vapour. This assumes that the operator is only exposed to naphthalene. BS 144: Part 1: 1990. Type 2 specifies that creosote for the bulk impregnation of timber should contain 8 to 25%. However, Heikkila et al. (1987) reported that the vapour measured in bulk impregnation plants contained up to 52% naphthalene due to it being one of the more volatile components. The above prediction can therefore be

refined to take account of the naphthalene content (i.e. approximately 50% in the vapour), which results in an exposure range of 1.3 to 8 mg/m^3.

Packaging plants (for do-it-yourself outlets)

Creosote is delivered to packaging plants by road tanker and transferred to storage. This is then piped to the packaging plant, where it is delivered into small plastic containers for supply to do-it-yourself stores. This filling operation is usually automatic with the operators loading and unloading the containers. It is understood that at some small companies this may be manual filling which may result in higher occupational exposure to naphthalene. Occupational exposure to naphthalene also occurs during tasks such as tanker delivery, removal of drip trays, transfer of waste and maintenance. Air sampling data was not received for this use.

These packaging plants are operated by 3 to 4 personnel (excluding maintenance operators), therefore it is estimated that about 30 to 40 workers are exposed at packaging plants in the UK, assuming they are all working with creosote containing naphthalene. The number of workers exposed throughout the EU was not established.

Modelled exposure data

Occupational exposure during the filling of containers with creosote was predicted using EASE. The EASE scenario that best describes this is non-dispersive use with LEV, which results in a prediction of 0.5 to 3 ppm (2.6 to 16 mg/m^3) 8-hour TWA for the naphthalene vapour. A value of 52% can again be used for the content of naphthalene in the vapour (Heikkila et al., 1987). The above prediction can therefore be refined to take account of the naphthalene content in the vapour, which results in an exposure range of 1.3 to 8 mg/m^3.

The value of 8 mg/m^3 will be used in the risk characterisation.

Brush application

Occupational exposure to naphthalene may occur during brush application of creosote. The concentration of naphthalene in creosote for brush application (usually 5 to 10%) is less than that used in bulk impregnation plants. It will generally be used outdoor for treating fences, sheds etc. therefore airborne concentrations are likely to be low. This will be primarily domestic use. It is unrealistic to attempt to estimate the numbers of workers occupationally exposed to naphthalene during brush application of creosote. It is estimated that about 50% of creosote used for brush applications is blended from distillates not containing naphthalene. Workers using this creosote will therefore not be exposed to naphthalene. Air sampling data was not received for the brush application of creosote.

Modelled exposure data

The data quoted in the consumer exposure assessment of this document from a UK Health and Safety Executive (ECOS, 1995) sponsored research project is relevant. This gives levels of 0.14 mg/m^3 to 2.91 mg/m^3 and results from dermal pads of 5 to 492 µg/100 cm^2. This resulted in a potential dermal absorption of 22 mg for the two-hour exercise. This work is more fully described in the consumer exposure assessment (see Section 4.1.1.2.1). It is reasonable to assume that, for example, professional painters may paint for a full shift with an 8-hour TWA up to 2.91 mg/m^3 and a potential dermal exposure of 88 mg (i.e. 4·22 mg).

Modelled dermal exposure data for the blending and use of creosote

The extent of dermal exposure will depend on whether this is during blending, bulk impregnation, packaging or brush application. The last mentioned is likely to result in the most significant dermal exposure. Activities such as cleaning of process plant at bulk impregnators, blending plants, packing plants etc. are likely to give rise to similar dermal exposures. The best EASE scenario for this exposure is direct handling with intermittent contact, where intermittent refers to two to ten significant contacts in a shift. The results in a prediction of 0.1 to 1 mg/cm^2/day, although this will be reduced as the maximum concentration of naphthalene is 25% to 0.025 to 0.25 mg/cm^2/day.

Due to the unpleasant nature of creosote on the skin including possible irritation it is assumed that workers will takes steps to avoid contact and for most tasks will wear gloves. It was reported by blenders and users of creosote in the UK that operators generally wear gloves to reduce this dermal exposure. It is therefore likely that dermal exposure will be at the bottom of this range.

4.1.1.1.7 Occupational exposure during the manufacture of mothballs

During the manufacture of mothballs exposure to both naphthalene vapour and particulate can occur as this is carried out at ambient temperature. The principal activities giving rise to this exposure are mixing (if combined with other components), pressing into balls or rings and packaging.

Industry exposure data

At the UK plant manufacturing mothballs the naphthalene is first milled, then components are mixed and transferred into a tabulating press. The tablets are left to dry for 24 hours. Extract ventilation is fitted to the mill and mixer. The operators are also supplied with impervious overalls, gloves and respirators, which should mitigate exposure. This batch process is carried out intermittently depending on customer demand. Air sampling was carried out in 1995 to determine workers exposure to naphthalene. The results are listed in Table 4.4.

Table 4.4 Occupational exposure to naphthalene during the manufacture of mothballs - single results (8-hour TWA)

Task/location	Result (mg/m^3)
Personal – mothball prep.	10.00
Personal – packaging	3.30
Static – preparation area	8.00
Static – packaging area	3.30

These results only represent vapour as the samples were collected on charcoal tubes, with no collection of the particulate. These results therefore are likely to be an underestimate of exposure due to the presence of particulate naphthalene. The highest personal total particulate result obtained during the exercise was 36 mg/m^3 8-hour TWA. This figure includes exposure to other components which have a greater ability to generate significant airborne concentrations due to their physical characteristics. It is therefore likely that these materials are the main contributors to this particulate exposure.

The scale of production is considerably higher at the plant in Belgium, although no air sampling data was received to determine levels of exposure there. Operators at the Belgium plant wear personal protective equipment, which includes gloves. RPE is also worn when operators carry out cleaning and maintenance. Exposure may also occur at companies in the EU which distribute mothballs, if they re-package the product.

Modelled exposure data

Occupational exposure during the preparation of mothballs was modelled using EASE. The EASE scenario that best describes this is non-dispersive use with LEV, which results in a prediction of 0.5 to 3 ppm (2.6 to 16 mg/m^3) 8-hour TWAfor the naphthalene vapour. Operators will also be exposed to the dust which can also be predicted using EASE. The EASE scenario that best describes this is dry manipulation with LEV, which results in a prediction of 2 to 5 mg/m^3 8-hour TWAfor naphthalene particulate. This gives a combined exposure to the vapour and particulate of 4.6 to 21 mg/m^3.

Modelled dermal exposure data

Dermal exposure to naphthalene is likely during the manufacture of mothballs from handling the particulate and from contaminated surfaces. The best EASE scenario for this exposure is direct handling with intermittent contact, where intermittent refers to two to ten significant contact in a shift. This results in a prediction of 0.1 to 1 mg/cm^2/day, although the operators are understood to wear gloves which should reduce exposure towards the bottom of this range.

4.1.1.1.8 Occupational exposure during the manufacture and use of coal tar paints and waterproof membranes

The number of companies formulating tar paints, membranes etc. was not established, although it is not expected to be significant. These formulation processes are likely to involve mixing in closed vessels with exposure during charging of the vessels and filling of the cans. Occupational exposure data was not obtained for this, although levels similar to chemical manufacture and synthesis are anticipated.

One producer of tar paints and waterproof membranes reported that these contain only a small percentage of naphthalene (less than 0.1 to 2 percent), with the later containing about 1%. During application the degree of exposure will depend on the method of application and the location of work. It is considered unlikely that these will be spray applied due to their viscous nature. The biggest use of these types of paints/membranes is understood to be by the building trade with application by brushing. The highest exposures are likely to be during the brush application of a waterproof membrane on the floors and walls of a house. This involves two coats to give a 1 to 1.5 mm thick membrane which is subsequently covered in plaster or concrete. It is assumed that due to the unpleasant odour that the operators will leave windows and doors open to allow some natural ventilation.

Modelled exposure data

The best EASE scenario for applying waterproof membranes inside a house is uncontrolled direct handling, which results in an exposure range of 50 to 100 ppm 8-hour TWA. Taking account of the naphthalene content (i.e. 1%), this range can be refined to 0.5 to 1 ppm (2.5 to 5 mg/m^3) 8-hour TWA. The concentration of naphthalene in tar paints is understood to be higher

(1 to 2 percent), however application is generally out of doors, for marine and agricultural usage. Exposure is therefore likely to be lower than the above. Further paints such as coal tar epoxy paints and coal tar polyurethane sealers also contain naphthalene at levels of less than 0.1% and less than 1%, respectively, therefore exposures are again likely to be low.

Modelled dermal exposure data

Dermal exposure to naphthalene may also occur when handling coal tar paints and water proof membranes. The best EASE scenario for this exposure is direct handling with intermittent contact, where intermittent refers to two to ten significant contact in a shift. This results in a prediction of 0.001 to 0.02 $mg/cm^2/day$ assuming that the paint/membrane only contains up to 1 to 2 percent naphthalene. The operators, however, are likely to wear gloves due to the unpleasant nature of the material.

4.1.1.1.9   Occupational exposure to naphthalene during the professional use of consumer products

There are a number of instances where workers may be exposed to consumer products at work. Examples include professional painters using creosote on park fences (discussed in Section 3.1.1.6), professional damp-proof laying and hair salons using coal tar soaps and shampoos.

Use of creosote products: The exposure estimate is derived as for consumers (see Section 4.1.1.2.1 for details). Exposures are calculated per event for the consumer. This approach will also be taken for the professional since it is difficult to estimate how often and how frequently a professional might do this work. These factors will be taken into consideration in the risk characterisation. Maximum inhalation exposure was measured at 2.91 mg/m^3 (ECOS, 1995) and dermal exposure is calculated (from the SCIES model) to be 22 mg. These exposures might be lower for the professional if they use PPE.

Damp-proof laying: As for the use of creosote products, the exposure estimates are derived from the consumer assessment (see Section 4.1.1.2.3 for details) where an event-based approach has been used. For a single event the average inhalation exposure level was calculated to be 76 mg/m^3 (using SCIES) and the maximum estimate of dermal exposure (from the DERMAL model) is 9 mg.

The professional use of coal tar shampoos and soaps is unlikely to differ from consumer exposure (see Section 4.1.1.2.5 for details) assuming that most shampoos and soaps do not contain coal tar. The professional hair stylist/beautician is therefore unlikely to use these any more than a individual consumer who routinely uses them. The consumer exposure assessment is therefore assumed to give a reasonable prediction of this exposure. The inhalation exposure is assumed to be negligible and the dermal exposure is calculated to be 4.4 mg.

4.1.1.1.10   Occupational exposure to naphthalene during the manufacture of grinding wheels

The number of workers exposed to naphthalene during the manufacture of grinding wheels is not known, although is likely to be about 20 at any site carrying out this work. Therefore the numbers exposed in the EU during the manufacture of grinding wheels could be several hundred.

The manufacturing process typically involves the blending of sieved crystalline naphthalene with grit and binders. The blend is then transferred to a press where it is cold pressed to the required density to form the wheel. The grinding wheels are then stored on drying racks. Pore forming takes place by removal of the naphthalene through a number of recovery / drying stages. Wheels may be placed inside drying ovens at 60°C to 100°C for initial volatilisation of the naphthalene. Further naphthalene removal may then be carried out using steam recovery where the wheels are placed inside an oven and steam injected. The driven off naphthalene is carried on the steam and recovered from the subsequent condensate. The wheels are then placed inside a kiln at 1,200°C for curing.

Workers may therefore be exposed to naphthalene during:

(i) sieving;
(ii) weighing;
(iii) blending;
(iv) cold pressing;
(v) drying stages; and
(vi) steam recovery of naphthalene.

Exposure will be to both naphthalene particulate and naphthalene vapour. Exposure to vapour will occur throughout all stages, although may be more prominent where hot naphthalene exists through steam recovery or drying of the wheel. Steam recovery may involve the workers having to transfer recovered naphthalene to hessian sacks. Naphthalene vapour may also evolve into the workplace from unfinished wheels left on storage / drying racks, from containers containing blends and from residual particulate on surfaces.

Particulate exposure will be more prevalent at the early stages of the process, where the crystals are sieved, weighed, blended with grit and binders and cold pressed. Blending may involve the addition of binders that result in wetting of the blend and therefore reduce dust formation. Once added to the mix ready for blending the naphthalene concentration in the mix 30% or less. These activities may require the naphthalene blends to be transferred to and from storage / transfer containers and scooped in smaller quantities to weigh scales and moulds. Particulate exposure may also occur during cleaning activities from the routine hand brushing out of residual material from mixers and the tops of mould presses. This may also occur during more extensive plant cleaning and workshop activities. At some plants water hoses are used for washing down to aid dust suppression.

Occupational exposure may also occur during planned and unplanned maintenance. This is potentially highest when naphthalene is still present in plant, particularly if at a raised temperature. One company reported that plant is shut down and allowed to cool prior to any maintenance being carried out.

Industry exposure data

At one EU site manufacturing grinding wheels containing naphthalene personal exposure to the vapour has been measured on two separate occasions. Exposures were 2.9 and 5.4 mg/m^3 8-hour TWA for exposure to the vapour. This plant has LEV on the mixers, although mainly to control dust evolving during mixing and not during material transfer to and from vessels.

Modelled exposure

Occupational exposure during the manufacture of grinding wheels was modelled using EASE. Since some plants may use LEV and others may not two scenarios were modelled. These predictions cover in general terms all tasks in the plant. Particulate exposure will generally be similar during activities such as sieving, weighing, blending, cold pressing and cleaning. Each activity may involve the manual transfer of material and the use of hand brushes, the main difference being the duration of exposure. Since some activities may take place over a full shift or an operator may spend shorter periods of time on each of these activities but still be exposed for the full shift, the predicted exposures below can be deemed to represent typical operator exposures for the above tasks. The situation is similar for exposure to the vapour, although this will predominate where the naphthalene is at raised temperatures.

(1) Where LEV is used

Where LEV is used the EASE scenario that best describes this is non-dispersive use with LEV, which results in a prediction of 0.5 to 1 ppm (2.6 to 5.2 mg/m^3) 8-hour TWA for the naphthalene vapour. Operators will also be exposed to the particulate which can also be predicted using EASE. The EASE scenario that best describes this is dry manipulation with LEV, which results in a prediction of 2 to 5 mg/m^3 8-hour TWA for naphthalene particulate. This gives a combined exposure to the vapour and particulate of 4.6 to 10.2 mg/m^3 8-hour TWA.

(2) Where LEV is not used

Where LEV is not used the EASE scenario that best describes this is non-dispersive use with direct handling with dilution ventilation, which results in a prediction of 10 to 20 ppm (52 to 104 mg/m^3) 8-hour TWA for the naphthalene vapour. Operators will also be exposed to the particulate which can also be predicted using EASE. The EASE scenario that best describes this is dry manipulation with direct handling and dilution ventilation, which results in a prediction of 5 to 50 mg/m^3 8-hour TWA for naphthalene particulate. This gives a combined exposure to the vapour and particulate of 57 to 154 mg/m^3 8-hour TWA.

3) Further refinement of the EASE predictions

Workers are predominantly exposed to blends containing a maximum of 30% naphthalene. The workers will be exposed to airborne particulate that in simple terms will only contain 30% naphthalene, therefore the EASE predictions for particulate can be refined as follows:

> with LEV 0.6 to 1.5 mg/m^3 8-hour TWA; and
> without LEV 1.5 to 15 mg/m^3 8-hour TWA.

This approach is less valid for exposure to the vapour, since naphthalene is the only source of exposure to the vapour. However, the solid naphthalene is mixed in with the grit and binders reducing the exposed surface area. This will reduce volatilisation and therefore the exposure. In the absence of a figure for this reduction in volatilisation 30% was again used to provide a reasonable estimate. Therefore the EASE predictions for vapour can be refined as follows:

> with LEV 0.8 to 1.6 mg/m^3 8-hour TWA; and
> without LEV 16 to 31 mg/m^3 8-hour TWA.

The refined combined particulate and vapour exposures are therefore (i.e. taking account of the 30% naphthalene concentration):

> with LEV 1.4 to 3.1 mg/m^3 8-hour TWA; and
> without LEV 17.5 to 46 mg/m^3 8-hour TWA.

Recent validation studies suggest that EASE over predicts exposure to vapour from low volatility materials. Further to this is the complication of using the model for a solid that sublimes. This suggests that the above results are likely to be over predictions. The only over option for predicting this exposure is to look at the limited real measurements for vapour and combine these for the particulate results modelled using EASE. The highest measured result for vapour was 5.4 mg/m^3 8-hour   TWA for a plant with only minimal LEV. Therefore the results for "without LEV" can be refined using a combination of the EASE figures for particulate and the measured vapour result. The measured result for vapour cannot be used for "with LEV" as it represents a plant where there was only very limited use of LEV. The combined results for particulate and vapour for 'without LEV' using this measured value are therefore:

> without LEV 6.9 to 20 mg/m^3 8-hour TWA.

Using professional judgement and considering the limitations of the EASE predictions the results to be taken forward for the risk characterisation are:

> **with LEV 1.4 to 3.1 mg/m^3 8-hour TWA**; and
> **without LEV 6.9 to 20 mg/m^3 8-hour TWA**.

<u>Modelled dermal exposure data</u>

Dermal exposure to naphthalene is likely during the manufacture of grinding wheels from handling the blends and from contaminated surfaces. This work involves considerable dermal contact with the dry blends and unfinished wheels. The best EASE scenario for this exposure is direct handling with extensive contact, where extensive refers to greater than ten significant contact in a shift. This results in a prediction of 1 to 5 mg/cm^2/day, although for most of the time operators will be in contact with blends containing only 30% naphthalene reducing the dermal exposure to 0.3 to 1.5 mg/cm^2/day.

4.1.1.1.11 Occupational exposure to naphthalene from adventitious sources

Occupational exposure to naphthalene vapour can also occur whenever organic material is incompletely combusted, such as:

(a) processing of coal, crude oil and natural gas (including coal coking, coal conversion, petroleum refining, production of carbon blacks, bitumen etc.);
(b) aluminium, iron and steel production plants and foundries;
(c) power plants.

Numerous studies have been carried out to determine exposure to PAHs during these processes. Although not regarded as of one of the PAHs of most concern, exposure to naphthalene is often reported. The results of a few of these studies are detailed below.

In 1994 HSE carried out developmental work in to a method for air sampling for coal tar pitch volatiles. One survey involved taking replicate samples of fume from a coking oven using a sampling chamber. This chamber was positioned on one of the coking oven charger cars. The

sampling period was five hours and analysis for naphthalene was included. **Table 4.5** details the results.

Table 4.5   Results of static measurements for naphthalene taken above a coking oven

No of samples	Range (mg/m³)	Mean (mg/m³)
5	0.03 to 0.06	0.05

It is likely that personal exposure would have been lower than these results.

NIOSH (1985) conducted a survey at a coking plant during maintenance of a coke battery precipitator to determine occupational exposure to coal tar pitch volatiles. This included analysis of air samples for naphthalene. The results of the personal sampling ranged from 0.02 mg/m^3 to 0.11 mg/m^3 (8-hour TWA).

Lesage et al. (1987) carried out a survey to determine exposure profiles to PAHs in various industries in Canada. The PAHs chosen included naphthalene. The averages of 12 to 20 fixed air sampling measurements (the exact number of samples for naphthalene was not reported) were given. Details on the exact locations of the fixed air sampling equipment were not provided. Table 4.6 summarises the results of the measurements for naphthalene.

Table 4.6   Results of fixed location measurements for naphthalene arising from incomplete combustion of organic material in selected industries

Industry	Mean particulate* (mg/m³)	Mean gaseous (mg/m³)
Class 1	0.01	<0.01
Class 2	0.02	<0.01
Class 3	0.08	<0.01
Class 4	1.10	<0.01

* These represent naphthalene that has adsorbed onto particulate as opposed to that present in the gaseous phase
Class 1 :   Paving (mixer, loading, shipping and paving), roofing (pitch preparation and roofing), steel (covering of finished product), silicon carbide (handling & sorting product);
Class 2 :   Refractory brick (product handling and oven operation);
Class 3 :   Silicon carbide (furnace work, loader, crane);
Class 4 :   Aluminium refinery (soderberg pot room, cathode relining, anode mixing, cathode mixing).

The results of these surveys indicate that personal exposure to naphthalene from the incomplete combustion of organic material is not significant.

Workers exposed to naphthalene from adventitious sources, for example, processing of coal, crude oil and natural gas, are only exposed as a consequence of the process involving the incomplete combustion of organic material. Therefore these scenarios do not involve the supply or use of naphthalene, but exposure as a result of the adventitious formation of naphthalene. Given this, a formal risk characterisation will not be performed for these exposures.

4.1.1.1.12   Inhalation exposure (general discussion)

The industries where occupational exposure to naphthalene were found to be highest were moth ball manufacture, the manufacture of grinding wheels and the use of creosote, in particular bulk

impregnation. The blending of creosote and packaging into containers for do-it-yourself use are not considered to result in high exposures as it is contained in semi-enclosed plant with short duration exposure during specific tasks. During manufacture and use in chemical synthesis it also used in semi-enclosed plant with exposure during tasks such as sampling and maintenance. The results of exposure from tar distillation plants and during the synthesis of phthalic anhydride, although limited, reflect the levels likely in these industries. **Table 4.7** summarises the occupational exposure data used in this exposure assessment and thus the values used for risk characterisation.

Table 4.7   Summary of occupational exposure data (inhalation) used in this exposure assessment - 8-hour TWAs

Industry		Year of sampling	Range (mg/m^3)	Source
Manufacture of naphthalene		nk	0.1 to 6.3	industry
Manufacture of phthalic anhydride		1994	0.33 to 2	industry
Blending and use of creosote	blending	analogous data (naphthalene and phthalic anhydride manufacture)		
	bulk impregnation plants	1987	<0.5 to 51	Heikkila et al.
		na	1.3 to 8	EASE
	packing plants (for DIY outlets)	na	1.3 to 8	EASE
	brush application	1995	0.14 to 2.9	HSE
Manufacture of mothballs		1995	3.3 to 10*	industry
		na	4.6 to 21	EASE
Manufacture of grinding wheels		nk	2.9 / 5.4	industry
	With LEV	na	1.4 to 3.1	EASE
	Without LEV	na	6.9 to 20	EASE
Manufacture and use of coal tar paints/ membranes		na	2.5 to 5	EASE
During the professional use of consumer products: Creosote products Damp-proof laying Coal tar soaps and shampoos		1995 na NA	2.91 76 negligible	ECOS SCIES FLUSH

* Excludes estimate of exposure to naphthalene particulate (see Section 4.1.1.1.7).
nk - not known
na - not applicable

Higher exposures result during mothball manufacture as this is carried out generally in the open work place, although extraction ventilation may be in use. In addition, exposure is to both the particulate and vapour. The highest reported result for naphthalene vapour was 10 mg/m^3 and for total particulate was 36 mg/m^3, although the latter is considered to be predominantly other components used in the manufacture. Exposure was predicted using EASE to be 2.6 to 16 mg/m^3 for the vapour and 2 to 5 mg/m^3 for the particulate where extraction ventilation is present. This results in a combined exposure to both particulate and vapour of 4.6 to 21 mg/m^3, which correlates reasonably well with measured data. Occupational exposure to both naphthalene particulate and vapour during manufacture is therefore estimated to be in the range 5 to 20 mg/m^3 considering a reasonable estimate of the combined measured data and the combined modelled data. These figures assume the control measures in place at the Belgium plant are similar to the UK plant.

Occupational exposure to naphthalene during the manufacture of grinding wheels is again likely to be higher than exposures on chemical plants. Handling of blends and pressing into moulds is a more open operation, although LEV is used for some operations. Limited industry data was received for exposure to the vapour, with results of 2.9 and 5.4 mg/m^3 8-hour TWA. Exposures were predicted using EASE for operations with and without LEV.

These were 1.4 to 3.1 mg/m^3 8-hour TWA and 6.9 to 20 mg/m^3 8-hour TWA, respectively for combined particulate and vapour exposure.

Occupational exposure at bulk impregnation plants was found in the Heikkila et al. (1987) study to be up to 26 mg/m^3 for a plant operator involved with opening the chamber. It was reported that this was carried out automatically, however, it is assumed that the work required the worker to be in the vicinity of the plant for certain tasks. The EASE model predicts this exposure to be 1.3 to 8 mg/m^3 which again correlates reasonably well with the measured data and will be used in the risk characterisation as the representative exposure.

The highest exposure to naphthalene, for all industries, reported was 51 mg/m^3 (this included indene and methyl naphthalene) for an operator cleaning out a bulk impregnation chamber. Information received from UK plants suggests that this task is rare and always carried out with the operator wearing respiratory protective equipment, protective gloves and clothing. Although it is difficult to estimate what the actual exposure is with the respiratory protective equipment worn, it is assumed that this will at least half exposure to 26 mg/m^3. It is therefore predicted that a reasonable worse case occupational exposure to naphthalene for plant operators would be in the range 26 mg/m^3 considering both the measured and modelled data.

4.1.1.1.13 Dermal exposure (general discussion)

Dermal exposure to naphthalene is likely to be most prominent during manufacture of mothballs and during the use of creosote. During the manufacture of grinding wheels workers handle uncured wheels and dry blends, as well coming into contact with contaminated surfaces. Most of this contact will be to a mixture of grit, binders and naphthalene with a resulting exposure to the latter of 0.3 to 1.5 mg/cm^2/day. During moth ball manufacture workers may be directly handling naphthalene. Exposure was predicted using EASE to be 0.1 to 1 mg/cm^2/day, however, workers use scoops to transfer materials and wear gloves to reduce exposure. Exposure is therefore likely to be at the bottom of the range. **Table 4.8** provides a summary of the dermal exposure data used in this exposure assessment.

The same range is predicted for some workers handling creosote, although this will be reduced to 0.025 to 0.25 mg/cm^2/day as the maximum concentration of naphthalene is 25%. The extent of contact will also depend on whether this is during blending, bulk impregnation, packaging or brush application, and the above is the highest likely exposure range. The last mentioned is likely to give the greatest dermal contact. Due to the unpleasant nature of creosote on the skin including possible irritation it is assumed that workers will takes steps to avoid contact and for most tasks will wear gloves. It was reported by blenders and users of creosote in the UK that operators generally wear gloves to reduce this dermal exposure. Dermal exposure at the bottom of the range is therefore likely.

Table 4.8   Summary of occupational exposure data (dermal) used in this exposure assessment

Industry	Year of sampling	Range (mg/cm²/day)	Source
Manufacture of naphthalene	na	0 to 0.1	EASE
Manufacture of phthalic anhydride	na	0 to 0.1	EASE
Blending and use of creosote - predicted exposure is the highest likely range and operators wear gloves, therefore exposure is likely to be at the bottom of this range.	na	highest range 0.025 to 0.25	EASE
Manufacture of mothballs - exposure at the bottom of the range is likely at operators wear gloves.	na	0.1 to 1	EASE
Manufacture and use of coal tar paints/membranes	na	0.001 to 0.02	EASE
Manufacture of grinding wheels	na	0.3 to 1.5	EASE
During the professional use of consumer products: Creosote products Damp-proof laying Coal tar soaps and shampoos	na	 22 mg per 2-hour application 9 mg per 1-hour application 4.4 mg per day	 ECOS DERMAL FLUSH

na - not applicable

4.1.1.2   Consumer exposure

Naphthalene is distilled from coal tar and petroleum but coal tar is, by far, the major source (see occupational exposure assessment for details). In 1992 about 2,000,000 tonnes of coal tar were distilled in Western Europe producing about 200,000 tonnes of naphthalene. Most of this was used industrially as precursors for other substances and the consumer use was small.

Consumer use of naphthalene is nowadays restricted to creosote, moth repellents, some minor applications in the building industry and a small number of coal tar products such as soap and shampoo. These will be dealt with separately.

4.1.1.2.1   Creosote

Creosote, which is a used within the EU for wood preservation, is the collective name for blends of distillate of oils obtained from coal tar within the boiling point range of 200-400°C. The British Standard for creosote (BS 144: Part 1: 1990) specifies three different types. Type 3 is for consumer use but no naphthalene content is specified for this type in the British Standard although it often contains varying quantities. If the creosote was derived from low temperature coking then it contains virtually no naphthalene. If it was derived from high temperature coking then it will contain up to 20 % naphthalene initially and may have more added before it is sold for consumer use.

Across Europe there are important differences in the use of creosote. Overall, within the EU, about 40,000 tonnes is used for consumer use. However, consumer use is banned in Germany and in Italy the use is minimal whereas in the UK consumer use of creosote amounts to between 10,000 and 15,000 tonnes annually as estimated by the industry. About 50% of the creosote cans sold for consumer use in the UK contain significant quantities of naphthalene (some brands may contain up to 45% but overall the average concentration is 10%) so this usage represents between 1,000 and 1,500 tonnes of naphthalene annually for the UK alone.

Consumer use of creosote usually involves brushing the liquid onto wooden fencing, gates or sheds and this is often done in the open in a variety of weather conditions. The most common size of container is 4 to 5 litres although smaller and larger (up to 10 litres) sizes are available. Its use in the UK is restricted to external timbers (under the Control of Pesticides Regulations, 1986) and label instructions include the need for good ventilation. Exposure is therefore sporadic and variable. It is likely that a consumer would use creosote for only a few days a year. Sales of creosote to consumers peak in the early spring and autumn when the weather is most suitable for application.

Inhalation and dermal exposure

Measured data

There is very little measured data on exposure to naphthalene during creosote brushing. A recent research project sponsored by the UK Health and Safety Executive (ECOS, 1995) investigated the consumer exposure to creosote constituents during some typical uses during the UK springtime. Five operators wore PVC gloves over cotton sampling gloves and coveralls fitted with absorbent pads to measure potential dermal contamination and wore filters and personal absorbent tubes to measure potential inhalation. They then painted creosote from 4 litre cans onto a variety of surfaces using appropriate 2.5 cm or 10 cm brushes for up to two hours. Naphthalene in air levels ranged from 0.14 mg/m^3 to 2.91 mg/m^3 as average values during this brushing procedure and the operators using creosote containing the higher levels of naphthalene produced the highest average air levels.

Results for the dermal pads were widely variable and there was evidence that some elements of the deposited creosote were volatilised during the course of the experiment. There was also evidence of absorption of naphthalene vapour from the air onto the pad. Both these processes are likely to take place on human skin. Because of the ongoing volatilisation during the course of the experiment naphthalene levels found on the pads are likely to be lower than the real contamination produced.

Levels of naphthalene on the pads varied from less than 5 to 492 $\mu g/100 \text{ cm}^2$. The highest levels being found on the front of the left thigh. As emphasised earlier, it was likely that levels were even higher at some stage of the experiment and were reduced by volatilisation during the course of the experiment.

Calculation for inhalation and dermal exposure

Inhalation

Modelling is very difficult because these procedures are variable and are carried out in the open air. However, based on the data from the ECOS experiment (see preceding section) we can calculate the potential exposures likely to be seen under UK conditions. Assuming a person paints creosote onto a fence for 2 hours and wears no respiratory protective equipment then they may inhale air containing an average naphthalene value of 2.91 mg/m^3 (the maximum average seen in the tests) at a rate of 1.3 m^3/hour (EPA default), a total of 7.6 mg for this single event.

Dermal

Using the WHO Standard protocol for field surveys of exposure to pesticides (1982) adapted for European bodies, because the ECOS experiment used dermal pads arranged as for this protocol, we can calculate a value for the potential dermal contamination. This is done by multiplying the

level found on the individual pads by a factor which takes into account the proportion of the body area contained within. The calculations and the reasoning are shown in Annex A. We have used the maximum value seen on each pad from the five volunteers and multiplied this by the appropriate factor. The dermal contamination is the totals from the pads divided by two with the inner glove levels added on and this works out at a value of 22 mg of naphthalene.

Other data on brush application

Another HSE-sponsored study has looked at the dermal exposures of amateur users of pesticide products (Roff, 1995). Brushing techniques were examined in this study which involved the application of spirit-based woodworm fluid (with an added fluorescent dye) to a lattice fence. Six subjects, wearing minimal clothing, brushed on the test liquid via a 10 cm brush for between half an hour and an hour. The weather was typical for a British springtime with only a light wind. All-body contamination was very variable and between 0.05 mg (0.03 ml) and 17 mg (10.6 ml) of liquid was found on the dermal surfaces afterwards. The light clothing was found to have a major protective effect and the highest contamination was found on the unprotected hands. This study is proceeding. This report also made reference to an EPA study of unknown antiquity (EPA) which has suggested that on an unprotected skin up to 6 ml of fluid would be retained.

The total inhalation exposure and dermal contamination figures of 7.6 mg and 22 mg, respectively, from our calculation from the ECOS study, will be taken forward to the risk assessment section.

4.1.1.2.2 Moth repellents

The pure grade of naphthalene (see occupational exposure section) is used for this application. Worldwide usage of naphthalene for moth repellents amounts to 30,000 tonnes but much of this is in Africa and Asia. Within the EU about 1,000 tonnes of naphthalene is used in the production of mothballs and of this the UK produces or repacks about 50 tonnes annually.

Moth repellents are used to repel insects from clothes although there is a minor use when formulated with other scents where they are used as a vertebrate repellent for gardens. For vertebrate repellent use the naphthalene is present at a concentration of 0.5% w/w and the product is put down at a rate of up to 30 g/m^2. As the product is left in the open air, air levels are not likely to build up.

When used as an insect repellent the moth repellents, which are between 97 and 99.9% naphthalene, are either left in drawers or, for the type produced in the shape of a doughnut, hung in wardrobes. Mothcrystals, which are smaller than mothballs and have a greater surface area, are more easily volatilised and can also be used. There are recommended usage patterns for these products which, if followed, can give an idea of the potential dose to consumers. The US Department of Agriculture recommends 165 g/m^3 (Neal, 1979) although the maximum US EPA registered use rate is 330 g/m^3. We can calculate exposure to naphthalene in the home from the use of moth repellents in wardrobes and drawers by using some experimental data. However, the exposure to naphthalene from the wearing of clothes stored in moth repellents is far more difficult to quantify.

Inhalation and dermal exposure from moth repellent use in the house

Measured data

A recent study (Recochem, 1995), which was performed to GLP, looked at naphthalene exposure during the experimental use of mothballs. The mothballs were distributed, according to the label conditions, in three bedrooms and adjacent closets in three different houses at a use rate of 330g/m^3. The first exposure scenario consisted of a person, wearing an air-sampler working in the house all day and opening the mothball-containing drawers and wardrobe at intervals. The second exposure scenario represented a bed-ridden person who was present continuously. A static sampler was placed at the head of the bed. Dermal exposure was measured by a person handling the mothballs whilst wearing gloves which were then tested for naphthalene residue.

The naphthalene levels found in the air in the closed areas, such as closets and drawers, ranged from 1 to 12 µg/l (mg/m^3), while the levels in the bedrooms ranged from 0.52 to 0.82 µg/l (mg/m^3). The maximum dermal exposure to naphthalene was estimated to be 0.104 mg from one handling of the mothballs.

Calculation for inhalation and dermal exposure

Inhalation

Based on the data shown in the above experiment we can calculate some possible exposures. Using the level of naphthalene in air of 12 mg/m^3 (the maximum seen in the closed drawers) for one hour, and the maximum level seen in the bedrooms (0.82 mg/m^3) for the other 23-hours, with a breathing rate of 1.3 m^3/hr (EPA default for an active person) for 8-hours, which includes the peak, and a breathing rate of 1.1 m^3/hr (EPA default for a consumer in the house) for the other 16-hours we can calculate a daily inhalation exposure of 37.6 mg.

Dermal

Assuming a dermal exposure of 0.104 mg per "handling" there is unlikely to be more than 4 handlings a day while the mothballs are in use (assuming 4 separate periods of opening and using a mothball-containing drawer) which equates to a dermal exposure of 0.416 mg.

These values of 37.6 mg (inhalation) and 0.4 mg (dermal) will be taken forward to the risk assessment and used as a potential continuous exposure.

Exposure from the wearing of clothes stored with moth repellents

There is a potential exposure to naphthalene from the wearing of clothing which has been stored with moth repellents. The use of christening gowns for babies has been highlighted in the toxicology section as a being a source of forseeable mis-use. It is difficult to quantify potential exposures in these situations as there is scope for the use of large quantities of naphthalene being retained in the clothing and hence being a source of considerable exposure.

4.1.1.2.3 Building industry: the use of damp-proofing and paints

Polythene sheeting is now the most common damp-proofing material in buildings but a naphthalene-containing emulsion based on coal tar may be used as well or instead of this. This emulsion of rubber and coal tar pitch can be used as a damp-proofing membrane for both walls

and floors. It contains about 1% naphthalene although this figure may exceptionally rise to 2%. When used on walls, where it is applied in two coats to produce a layer 1-1.5 mm thick, it is covered by a layer of at least 12 mm of plaster. The same thickness is used on floors, particularly cellars, where it is covered by a coarse layer of a concrete screed. If thinner layers of covering are used and where ventilation is poor or where hot water pipes have been put through the concrete there may be a build up of the naphthalene odour and this may lead to complaints.

Although the use of this damp proofing is predominantly by tradesmen it is available on the DIY market. We can model the consumer use of this compound and calculate potential exposures to naphthalene during the laying of these damp proof courses.

Model scenario for inhalation and dermal exposure from the laying of damp proofing

Inhalation

We assume a room with a volume of 18 m^3 which has 0.2 air changes an hour. If 15 kg of emulsion (containing 1% naphthalene) is used and the consumer spends an hour laying the material we can input this data into the US EPA model SCIES. The nearest scenario available is that of FLOOR WAX/POLISH. This scenario envisages the material being used like a floor polish and being spread over a floor and left to evaporate. In practice it is likely to overestimate the evaporation of naphthalene because it does not allow for the formation of a skin on the surface of the emulsion which may reduce evaporation.

This model predicts that the peak naphthalene level in the room during laying is going to be 95 mg/m^3 and that the average level during the hour spent laying the emulsion will be 76 mg/m^3. With a breathing rate of 1.3 m^3/hr (EPA default) this means that there will be an inhalation exposure to naphthalene of 99 mg. This value will be used in the risk assessment.

Dermal

In the circumstances as described above and assuming that no protective gloves are worn then there will be dermal exposure to the emulsion. Given a density of the formulation of 1.07 then the US EPA model DERMAL suggests that there will be exposure to 9 mg of naphthalene. This value will be used in the risk assessment.

4.1.1.2.4   Exposure following damp-proofing

In the UK several complaints, concerning a build up of naphthalene odour in premises some time following the use of this damp-proofing, have been investigated by the manufacturers. The usual reason for problems was that the screed or plaster was not thick enough. Measurements were taken some time after the complaint and may not represent the peak levels. Most measurements were taken at 1 metre above ground and the air levels of naphthalene found varied from 0 to 0.45 mg/m^3 (Maslen, 1995). This highest level was found in an office refurbishment where the specification had not been adhered to. The highest air naphthalene level seen in a domestic dwelling was 0.293 mg/m^3.

Calculation of the inhalation exposure from damp proofing following laying

The value of 0.45 mg/m^3 will be taken as the worst case and assuming 24-hour residence at a breathing rate of 1.1 m^3/hour (EPA default for sedentary occupation) there is a possibility of a

total daily inhalation of 11.9 mg of naphthalene. This is a continuous exposure and will be taken forward to the risk assessment.

Dermal exposure from damp proofing following laying is expected to be negligible.

There are associated products such as coal tar paints which also contain naphthalene. These paints, which contain 2% naphthalene, dry rapidly and are used for marine and agricultural purposes throughout Europe. They are used out of doors in situations where no significant consumer exposure is likely.

Coal tar epoxy paints are also available but these usually contain only very low levels of naphthalene-less than 0.1 % is typical. These are specialist paints which are used on steel or concrete in damp locations. Their use is decreasing and there is no consumer application because of the two-pack procedure and mixing requirements.

Coal tar polyurethane sealers may also contain naphthalene. These are applied by brush for roof repair and although there is some consumer and trade use most of the manufacture is exported outside of the EU. Consumer exposure is not going to be significant because levels of naphthalene in the sealer are less than 1% and it is applied in the open air.

Overall therefore, only the exposure from naphthalene in the damp proof course is considered to be significant. This will be 99 mg (inhalation) and 9 mg (dermal) from the laying of the membrane which will be a single event and a value of 11.9 mg daily from inhalation exposure following the laying.

4.1.1.2.5 Coal tar soaps and shampoos

There is a minor use in medicated soaps and shampoos. Coal tar solution BP which can be used in these products contains a 20 % solution of coal tar which itself contains 13% naphthalene. Hence coal tar solution BP has a 2.6% naphthalene content. The medicated soap or shampoo will contain a 7.5% solution of coal tar BP solution meaning that there will be a 0.2% naphthalene content in the product.

Inhalation and dermal exposure

Model scenario for inhalation and dermal exposure

We can model the daily exposure to naphthalene from the use of shampoo and coal tar soaps.

For a shampoo the assumption is that there is a rinse-off coefficient of 10% (ECETOC, 1994). This means that 10% of the product stays on the hair and this 10% is available for absorption through the scalp. The typical quantity of shampoo used per daily application is 12 g and thus 1.2 g is the level of dermal exposure. This quantity of coal tar shampoo will contain 2.4 mg of naphthalene. Inhalation is assumed to be negligible.

The daily use of coal tar soap per person will be assumed to be 10g. The EPA model FLUSH (EPA, 1992) assumes 18.6 g of daily soap use per household and the Technical Guidance Document (page A VI-6) suggests 0.8 g per application and 3-6 applications per day. Therefore the value of 10 g is a high figure. However the value of 10 g will be used because there are some users of these soaps who regard them as more than just another detergent. Assuming a 10% rinse-off coefficient again this leaves 1 g of soap available on the skin. This will contain 2 mg of naphthalene. Inhalation of naphthalene is again assumed to be negligible.

The combined total for naphthalene dermal exposure from soap and shampoo is therefore, 4.4 mg and this value will be taken forward to the risk assessment.

4.1.1.2.6   Overall consumer exposure to naphthalene

The potential exposures to naphthalene can be split into a continuous exposure from circumstances where naphthalene is present daily and a one-off or acute exposure from circumstances which recur infrequently.

Continuous exposures results from the use of moth repellents, damp proofing following its laying, and from soaps and shampoos which together could produce a daily total of 54.3 mg (0.77 mg/kg/day for a 70 kg person). It would be unlikely that all of these exposures would be together concurrently but a reasonable worst-case scenario should include them all.

A consumer could also be exposed infrequently to naphthalene from the use of creosote. An exposure from this use would amount to an extra 29.6 mg. The laying of a damp proof course could add an extra 108 mg of naphthalene exposure on that particular day.

4.1.1.3   Humans exposed via the environment

Tables 4.9 and **4.10**, which repeat **Tables 3.32** and **3.33** from the environment section, give the concentrations in human uptake and the daily human doses arising from releases from production (based on site D), processing and the manufacture of creosote and mothballs, and for releases at the regional level.

Table 4.9   Concentration in human intake

	Regional	**Production (site D)**	**Processing**	**Mothballs manufacture**	**Grinding wheels manufacture**
Air	0.137 µg/m3	0.396 µg/m3	1.05 µg/m3	3.94µg/m3	1.92 µg/m3
Drinking water	0.0346 µg/l	0.264 µg/l	2.36 µg/l	0.142 mg/l	0.28 mg/l
Fish	14.8 µg/kg	113 µg/kg	20.4 µg/l	60.7 mg/kg	120 mg/kg
Leaves of plants	0.367µg/kg	1.1 µg/kg	2.82 µg/l	10.7µg/kg	5.33 µg/kg
Root of plants	0.841 µg/kg	1.52 µg/kg	120 µg/l	4.89 mg/kg	8.25 mg/kg
Meat	0.0059 µg/kg	0.0182 µg/kg	0.0617 µg/l	1.26 µg/kg	2.23 µg/kg
Milk	0.00186 µg/kg	0.0058 µg/kg	0.0195 µg/l	0.399µg/kg	0.707 µg/kg

Table 4.10 Daily human dose via indirect exposure (mg/kg (body weight)/day)

	Regional	Production (site D)	Processing	Mothballs manufacture	Grinding wheels manufacture
Air	$2.93 \cdot 10^{-5}$	$8.49 \cdot 10^{-5}$	$2.25 \cdot 10^{-4}$	$8.45 \cdot 10^{-4}$	$1.08 \cdot 10^{-4}$
Drinking water	$9.88 \cdot 10^{-7}$	$7.53 \cdot 10^{-6}$	$6.73 \cdot 10^{-5}$	$4.06 \cdot 10^{-3}$	$8.0 \cdot 10^{-3}$
Fish	$2.42 \cdot 10^{-5}$	$1.85 \cdot 10^{-4}$	$3.35 \cdot 10^{-5}$	0.0997	0.197
Leaves of plants	$6.29 \cdot 10^{-6}$	$1.82 \cdot 10^{-5}$	$4.84 \cdot 10^{-5}$	$1.84 \cdot 10^{-4}$	$9.14 \cdot 10^{-5}$
Root of plants	$4.61 \cdot 10^{-6}$	$8.34 \cdot 10^{-6}$	$6.57 \cdot 10^{-4}$	$2.68 \cdot 10^{-2}$	$4.53 \cdot 10^{-2}$
Meat	$2.53 \cdot 10^{-8}$	$7.84 \cdot 10^{-8}$	$2.65 \cdot 10^{-7}$	$5.43 \cdot 10^{-6}$	$9.61 \cdot 10^{-6}$
Milk	$1.49 \cdot 10^{-8}$	$4.62 \cdot 10^{-8}$	$1.56 \cdot 10^{-7}$	$3.2 \cdot 10^{-6}$	$5.66 \cdot 10^{-6}$
Total	$6.55 \cdot 10^{-5}$	$3.04 \cdot 10^{-4}$	$1.03 \cdot 10^{-3}$	0.132	0.25

It can be seen that the daily human intake for humans via the environment based upon typical human consumption and inhalation rates at the regional level is $6.55 \cdot 10^{-5}$ mg/kg/day and the highest local exposure (manufacture of grinding wheels) is 0.25 mg/kg/day. These two figures will be taken forward to the risk characterisation.

4.1.1.4 Combined exposure

The worst-case combined body burden would be for a person who works in the naphthalene processing industry with the highest inhalation exposure (from mothball manufacture or from the manufacture of grinding wheels) and is exposed via the environment in the locality of a grinding wheel manufacturing plant and who also uses consumer products containing naphthalene. The exposures for these component parts are presented in **Table 4.11**. Daily dermal occupational exposure is not included, but would obviously further increase the combined exposure.

Table 4.11 Data comprising combined exposure scenario

Source of exposure	Exposure
Occupational source (Mothball manufacture)	20 mg/m^3 (equivalent to about 3 mg/kg/day)
Indirect via the environment　　　regional 　　　　　　　　　　　　　　　local	6.55×10^{-5} mg/kg/day 0.25 mg/kg/day
As a consumer	0.77 mg/kg/day
Total　　　　　　　　　　　regional 　　　　　　　　　　　　local	3.8 mg/kg/day 4.0 mg/kg/day

The consumer exposure could be increased by the infrequent use of creosote products (29.6 mg) and the activity of damp proof laying (108 mg). These would be per event which would be expected to be infrequent. The use of creosote may be for a few (2-3) days per year with damp proof laying being a single event.

4.1.2   **Effects assessment (Hazard identification and dose (concentration)-response (effect) relationship)**

4.1.2.1   **Toxicokinetics, metabolism and distribution**

4.1.2.1.1   **Studies in animals**

In vivo studies

Inhalation

No published data are available on the toxicokinetics of naphthalene following inhalation although consideration of the molecular structure would suggest it is likely to be well absorbed following inhalation exposure. In a review document (NTIS, 1987) reference was made to an unpublished study (Buckpitt, 1985) in which it was apparently stated that an 8-hour exposure to 100 ppm (~520 mg/m^3) naphthalene resulted in a body burden of 150-200 mg/kg in rats. This suggests extensive uptake via the lungs. No further details were given.

Oral

Three groups of Sprague-Dawley rats were treated orally with a single administration of 2 mg ^{14}C-naphthalene; the dose/unit body weight was not stated (Bakke et al., 1985). One group of 16 animals were bile duct cannulated, the second group of 4 animals were uncannulated but with an altered intestinal microflora (germ free), and the third group of 13 animals were standard uncannulated rats. Urine, faeces and bile were collected for 72-hours after administration of naphthalene. In the standard uncannulated rats 75.6% of the administered radioactivity was recovered in the urine in 24-hours. At 72 hours approximately 83% of the radioactivity had been recovered in the urine, 6% in the faeces and 4% remained in the carcass with no account made for the remaining radioactivity. In the cannulated rats the 24-hour urine and bile contained 30% and 66.8% of the ^{14}C dose, respectively (faecal data not presented for 24-hour time period). At 72 hours, approximately 30% of the radioactivity had been recovered in the urine, 69% was contained in the bile, less than 1% was excreted in the faeces and 0.2% remained in the carcass.

As part of the above study the metabolites were identified and quantified (see Figure 4.1). In the standard uncannulated rats the urinary metabolites identified at 24-hours were: an N-acetyl cysteinyl naphthalene conjugate (38.1% of the administered radioactivity), 1,2-dihydro 1-2-dihydroxynaphthalene (dihydrodiol) glucuronide (23.9%), dihydroxynaphthalene (4.9%), naphthols and naphthol glucuronides (4.6%) and 1,2-dihydro-1-hydroxy-2-methylthio-naphthalene glucuronide [CH$_3$S-metabolite] (4.6%). At 24-hours in cannulated rats, 14.4% of the administered dose was present in the urine as N-acetyl cysteinyl naphthalene conjugate, and 14.5% as the dihydroxynaphthalene glucuronide conjugate. Naphthols, thionaphthols, CH$_3$S-metabolites or their respective glucuronides and sulphates were not detected in either the 24-hour urine or bile. The major urinary metabolites of germ free rats were N-acetyl cysteinyl naphthalene (89%) and dihydroxynaphthalene glucuronide (4%).

Only traces of naphthols and no CH$_3$S-metabolites could be detected indicating that intestinal microflora are probably involved in the production of both these metabolites. Overall, this study shows that there is rapid and complete absorption of naphthalene from the gastro-intestinal tract.

Extensive metabolism occurs and elimination is rapid, primarily through the urinary route, following enterohepatic circulation.

Groups of five Wistar rats were administered a single dose of 0, 30, 75 or 200 mg/kg naphthalene by gavage (Summer et al., 1979). Urine was collected over a 24-hour period and analysed for mercapturic acid. Naphthalene administration increased the total urinary excretion of mercapturic acids from the control rate of 94 μmol/kg/24-hour to 502 μmol/kg/24-hours, with mercapturic acids of naphthalene increasing in a dose-dependent manner of 92, 186 and 408 μmol/kg/24-hour with 30, 75 and 200 mg/kg, respectively. The increased amount of mercapturic acid excreted in the urine in 24-hours at the three dose levels is equivalent to 39, 32 and 26% of the administered dose of naphthalene in this form. Hepatic glutathione (GSH) levels were decreased to 17% of the pre-treatment levels at 6.5-hours after administration of 200 mg/kg naphthalene (data not provided for the other doses). The GSH levels returned to control values 24 hours after administration.

As the eye has been an organ of interest in relation to naphthalene, GSH levels in the whole optical lens, capsule-epithelium, cortex and nucleus of 6-10 rats/group at 4, 8, 12 and 24 hours after a single oral dose of 0 or 1,000 mg/kg naphthalene have been reported (Xu et al., 1992). GSH levels in the capsule-epithelium were reduced to 60% of control values at 8 hours and recovered 12 hours post-administration. GSH levels in the other lens tissues studied remained equivalent to pre-dose levels over a 24-hour period. The only major metabolite of naphthalene found in the eyes of the rats was naphthalene dihydrodiol which was detected in the lens and aqueous humour.

In a limited study, an unstated number of rats and rabbits were administered a single dose of 0 or 500 mg/kg naphthalene by gavage and urine samples were collected for 48-hours and analysed for metabolites (Corner and Young, 1954). Treated animals from both species were observed to excrete 1-and 2-naphthol, 1-naphthylsulphuric acid, 1-naphthylmercapturic acid and 1,2-dihydronaphthalene-1,2-diol. The latter metabolite showed optical activity in both the free state and when conjugated with glucuronic acid, with rabbits excreting the dextrorotatory form only and rats primarily the laevorotatory form. No quantitative details were given.

Figure 4.1   Proposed Pathways for Naphthalene Metabolism (from ATSDR draft report, update 1993)
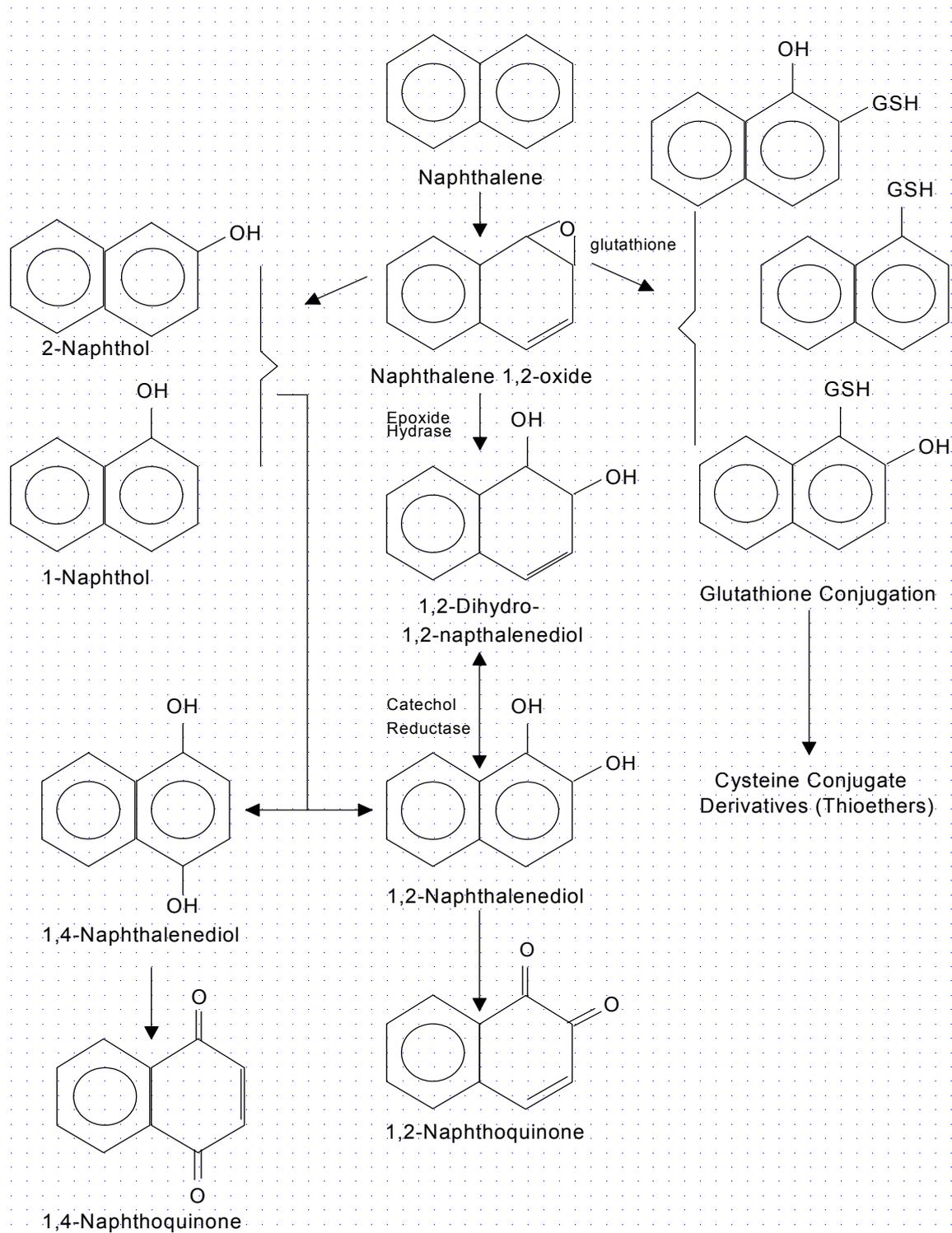
A single sample of blood plasma was extracted from a rabbit following repeated daily administration of 1,000 mg/kg naphthalene by gavage for 6 days and analysed for metabolites (van Heyningen and Pirie, 1976). The presence of 1,2-dihydro-1,2-dihydroxynaphthalene (dihydrodiol), 2-hydroxy-1-naphthyl sulphate, and naphthyl glucosiduronic acid were detected in the plasma although no quantitative information was provided. In the same paper 1,2-dihydroxy-naphthalene and 1,2-naphthoquinone was reported to be present in the aqueous humour of the eye.

A total of 4 chimpanzees were administered a single dose of 0, 30, 75 or 200 mg/kg naphthalene by gavage (Summer et al., 1979). Urine was collected over a 24-hour period and analysed for mercapturic acids of naphthalene. The total excretion rate of mercapturic acids in control animals was noted to be 18 mol/kg/24-hour and was similar in the high dose group at 17 mol/kg/24-hour. A decrease in hepatic GSH levels was not observed with 200 mg/kg naphthalene administration (urinary mercapturic acid excretion rate and GSH levels at other doses not determined). Reference was made in this paper to a preliminary unpublished study in chimpanzees that apparently showed that the majority of the naphthalene metabolites excreted in the urine were glucuronide and sulphate conjugates. GSH conjugates were not detected in the bile. These results are in contrast to the situation in rats where GSH depletion occurs in the liver and glutathione conjugates are detected in the urine, following exposure to naphthalene.

Dermal

No information was available, however the highly lipid soluble nature of naphthalene suggests that dermal absorption is likely.

Intraperitoneal

Rapid excretion of naphthalene in the urine has also been demonstrated following intraperitoneal (i.p.) dosing in rats. Such studies have shown that unconjugated metabolites account for 5-20% and conjugated metabolites for 80-95% of the metabolites excreted in the urine (Chen and Dorough, 1979; Horning et al., 1980).

Swiss Webster mice (4 to 5 per group) were treated with a single i.p. administration of 400 mg/kg ^{14}C-naphthalene 30 minutes after administration of vehicle or piperonyl butoxide (PIP), which inhibits P450 cytochromes (Warren et al., 1982). Glutathione depletion in the lung, liver and kidneys was less with PIP than vehicle pre-treatment. Also there was a 75% reduction in the amount of covalently bound radioactivity observed in PIP pre-treated animals. In comparison, administration of 200 mg/kg ^{14}C-naphthalene following pre-treatment with diethyl maleate (which reduces glutathione levels) substantially increased covalent binding in the liver, kidney and lung compared to vehicle pre-treated controls.

As part of the same study a single dose of 0, 25, 50, 100, 200, 400 and 600 mg/kg ^{14}C-naphthalene was administered i.p. to groups of 3-4 mice. Between 25 and 200 mg/kg, covalent binding of radioactivity in the liver, kidney and lung showed a near linear increase with administered dose. However, above 200 mg/kg the amount of covalently bound radioactivity in the tissues increased supralinearly. It was noted that this larger increase in binding coincided with almost complete depletion of tissue glutathione. At 600 mg/kg, glutathione levels were reduced to 10% of control in all three tissues. These data indicate that naphthalene is metabolised by cytochrome P450s to intermediates which may conjugate with glutathione or become bound to tissue, presumably by interacting with macromolecule sulphydryl groups.

Five mice were treated i.p. with a single administration of 300 mg/kg ^{14}C-naphthalene, 48 hours after i.p. pre-treatment with *p*-xylene (such treatment selectively reduces pulmonary cytochrome P450 levels) (Buckpitt and Warren, 1983). With the *p*-xylene pre-treatment, a significant decrease in metabolism of naphthalene, as measured by cytochrome P450 activity, was observed only in lung tissue. However, *p*-xylene had little effect on naphthalene-induced glutathione depletion and covalent binding in the lung. In the same study administration of 200 mg/kg ^{14}C-naphthalene, 2-hours after pre-treatment with buthionine sulfoximine (which reduces hepatic and renal glutathione levels but not pulmonary levels), gave rise to a substantial decrease in glutathione levels in the lungs as well as the liver and kidney. An increase in covalently bound radioactivity in lung, liver, kidney and muscle of 295, 319, 423 and 490%, respectively was also observed. The increase in covalent binding in muscle tissue (which contains no detectable P450 activity) indicated the presence of reactive metabolites rather than parent naphthalene in this tissue. The results indicate that reactive naphthalene metabolites are generated in the liver and are then able to circulate from the liver to other organs such as the lung and kidney where conjugation with glutathione occurs and, with extensive glutathione depletion, covalent binding of the reactive metabolites to tissue macromolecules occurs.

As part of a study reporting on the effects of a number of chemicals, 0 or 500 mg/kg naphthalene was administered to an unstated number of rats (Gandy *et al*, 1990). Animals were sacrificed at 1, 2, 4, 8 and 16 hours after dosing and the liver, testes and epididymis examined for glutathione content. Hepatic glutathione levels decreased steadily down to 11% of controls by 16 hours after dosing. Testicular glutathione levels were reduced to 77% of controls one hour after dosing, but were approximately equivalent to and higher than controls from two hours onwards. Epididymal levels of glutathione were reduced to 53% of controls by 4 hours post exposure with little increase in glutathione levels at 16 hours.

In vitro studies

Preparations of nasal mucosa, lung, liver and kidney microsomal incubations from the mouse, rat and hamster along with monkey lung and liver tissue preparations were treated with ^{14}C-naphthalene to investigate the rate of naphthalene metabolism *in vitro* (Buckpitt et al., 1987; Buckpitt et al., 1992). The highest rates of naphthalene metabolism were observed in mouse lung and liver microsomal incubations. The rate of metabolism in rat, hamster and monkey lung microsomal preparations was 12, 37 and 1%, respectively of that in mouse lung. In both the rat and hamster the hepatic rate of naphthalene metabolism was greater than the pulmonary rate. Naphthalene metabolism by kidney microsomes was less than 5% of either lung or liver preparations in the mouse. In rat, mouse and hamster microsomal fractions, 3 stereoisomeric glutathione conjugates of two epoxide intermediates were noted. From these stereoisomers the prevalence of the epoxides was determined, and it was found that in the mouse lung and the olfactory regions of the rat and hamster 1R, 2S-naphthalene-1,2-oxide was the dominant enantiomer, whereas 1S, 2R-naphthalene-1,2-oxide was the major enantiomer in the other tissues studied. The rate of naphthalene metabolism was noted to be highest in regions where the 1R, 2S-naphthalene-1,2-oxide was the dominant enantiomer.

4.1.2.1.2   Studies in humans

In vivo studies

1-Naphthol has been detected in urine samples taken from a group of 123 coke plant workers who were exposed to naphthalene, benzene and other aromatic hydrocarbons (Bieniek, 1994). The maximum excretion of 1-naphthol was found 2 to 3-hours after the end of an 8-hour working shift. Naphthalene in the breathing zone air was also monitored and a linear relationship between naphthalene air concentration (0-6 mg/m^3) and the concentration of 1-naphthol in urine was noted. There was no attempt made to identify other naphthalene metabolites and it is impossible to conclude whether or not the 1-naphthol was a metabolite of naphthalene or the other aromatic hydrocarbons.

1-Naphthol was detected in urine samples from two psoriatic patients who were being treated daily for 4 weeks with dermal applications of coal tar which contained naphthalene (Hansen et al., 1993). During the treatment period the patients were in contact with a total of 118 mg naphthalene. It was not stated whether or not treatment sites were covered with dressings. One patient showed very high levels of 1-naphthol (approximately 2,750 mmoles/mol creatinine) in a urine sample taken after one week of treatment. 1-Naphthol was not detected in urine samples taken on days 18 and 22, despite continued treatment. The 1-naphthol may not have been detected in the later urine samples for several reasons, such as analytical problems, enzyme induction of another metabolic pathway or reduced skin absorption of naphthalene due to recovery of the psoriasis. There was no attempt made to identify other metabolites of naphthalene.

Urine samples were collected from an infant hospitalised because of haemolytic anaemia following ingestion of unknown quantities of naphthalene mothballs (Mackell et al., 1951). 1- and 2-naphthol and 1,2- and 1,4-naphthoquinone were identified in the samples for up to 8 days of hospitalisation. 1-Naphthol was present in the largest, unspecified quantities.

No other toxicokinetic data are available. However, clear signs of systemic effects have been observed in humans following ingestion of naphthalene mothballs, inhalation of naphthalene vapour from clothes which had been stored with mothballs and dermal contact with clothes which had been impregnated with naphthalene (see Sections 4.1.2.2 and 4.1.2.6). Therefore the evidence indicates that naphthalene is absorbed by all routes. Neonatal haemolytic anaemia also occurred following ingestion of naphthalene mothballs by pregnant woman, indicating that naphthalene and/or a metabolite crosses the placental barrier (see Section 4.1.2.9).

In vitro studies

Naphthalene 1,2-dihydrodiol and three glutathione conjugates were identified when ^{14}C-naphthalene was added to microsomal fractions of fresh samples of human lung tissue in the presence of glutathione and glutathione transferases (Buckpitt and Bahnson, 1986).

Naphthalene 1,2-dihydrodiol was identified as the major metabolite when ^{14}C-naphthalene (100 µM) was incubated with human hepatic microsomes (Tingle et al., 1993). The addition of trichloropropene oxide (an epoxide hydrolase inhibitor) to the human microsomes decreased the ratio of 1,2-dihydrodiol to 1-naphthol from approximately 9:1 to 0.1:1. Co-incubation of the human microsomes with glutathione also decreased the covalent binding to protein by 64% indicating competitive binding to glutathione.

4.1.2.1.3   Summary of toxicokinetics

The limited information available in humans indicates that naphthalene is readily absorbed by all routes of exposure and animal data shows that almost complete and rapid absorption occurs following ingestion.

In humans, naphthalene is metabolised to 1-naphthol, 2-naphthol and 1,2- and 1,4-naphthoquinone. *In vitro* studies in human liver microsomes and human lung preparations indicate that epoxide hydrolase is involved in the metabolic pathway by which naphthalene is metabolised to naphthalene 1,2-dihydrodiol.

Metabolism in rodents is chiefly by P450 oxidation, with subsequent glutathione conjugation, as well as epoxide hydroxylation to naphthalene 1,2-dihydrodiol. Glutathione conjugation has not been shown to occur in non-human primates. There is some evidence that significant enterohepatic recirculation of naphthalene metabolites occurs in rodents. *In vitro* studies show that the rate of naphthalene metabolism in mouse lung tissue is approximately 3, 8 and 100 times greater than that observed in lung tissue from hamsters, rats and monkeys, respectively. There are no data available to indicate whether or not similar differences in metabolic activity between species also occur in the nasal tissues.

The urine is the main route of rapid excretion in humans and animals.

4.1.2.2   Acute toxicity

4.1.2.2.1   Studies in animals

Inhalation

In a report published in abstract form only, it was stated that when an unspecified number of mice were exposed nose-only to 0.38 mg/m^3 naphthalene for four hours, bronchiolar damage was observed (Buckpitt et al., 1982). No further information was provided on the type or severity of the damage. However no bronchiolar damage was observed following a 1-hour exposure to 0.38 mg/m^3. A 4-hour exposure of between 0.09 and 0.17 mg/m^3 was stated to result in "slight" pulmonary damage. These findings are questionable given that only 'minimal to mild' broncho-alveolar changes were observed at 50 mg/m^3 in a 2-year mouse carcinogenicity study (Section 4.1.2.8).

Oral

A series of well-conducted, unpublished acute toxicity limit tests were available in which groups of 5 male and 5 female Sprague-Dawley rats were treated with 2,000 mg/kg naphthalene (Hazleton, Report Nos. 008304, 008305 and 008306, 1990). Over a 14-day period of observation 2 deaths occurred in a total of 30 animals. Diarrhoea was reported in 17 of the 30 animals on days two to nine. No macroscopic changes were observed. An oral LD$_{50}$ of greater than 2,000 mg/kg for rats is therefore indicated.

An LD$_{50}$ of 2,300 mg/kg was determined in a group of 40 male and 40 female rats (Gaines, 1969). The lowest oral lethal dose was 1,500 and 2,000 mg/kg for males and females, respectively.

LD_{50} values of 533 mg/kg and 710 mg/kg were determined in 40 male and 47 female CD-1 mice, respectively (Shopp et al., 1984). Deaths occurred between 5-hours and 5 days and were preceded by depressed breathing and ataxia. Haemolytic anaemia was not observed in the CD-1 mice.

In an early poorly conducted study a single oral dose of 400 mg/kg or 1,500 mg/kg naphthalene was administered in the diet to two dogs (Zuelzer and Apt, 1949). On the eighth day there was a reduction of the haemoglobin to 6.6 gm/100 ml and 10.2 gm/100 ml (from 9.3 and 14.4 gm/100 ml) for the low and high dose, respectively. Both animals showed an increase in the number of Heinz bodies in erythrocytes, and reticulocytosis began on the 7th day reaching a maximum on the 10th day. Lethargy, vomiting and diarrhoea were also noted in the dog treated with the higher dose. Complete recovery was achieved 1-2 weeks after administration.

Dermal

No deaths were observed in 40 Sherman rats of either sex following dermal administration of 2,500 mg/kg naphthalene (Gaines, 1969), indicating that naphthalene is of low dermal toxicity in this species. No further information was available.

In an unpublished study, only available as an unpublished abstract, an LD_{50} value of greater than 2,000 mg/kg was reported in rabbits (Landis International, 1995).

Parenteral studies

A number of intraperitoneal studies have been carried out using rats, mice and hamsters (O'Brien et al., 1985; Rassmussen et al., 1986; O'Brien et al., 1989; Wells et al., 1989 and Plopper et al., 1992a,b). In rats necrosis and exfoliation of nasal olfactory epithelium was observed at 200 mg/kg and above. No histopathological changes were noted in the terminal bronchioles, lobar bronchus, trachea or kidney with up to 1,600 mg/kg. In mice a different pattern of results was obtained; changes, ranging from slight cell swelling, through vacuolation, exfoliation and necrosis, occurred at 50 mg/kg in the Clara cells and at 100 mg/kg and above in the non-ciliated cells of the terminal bronchioles, at 300 mg/kg and above in the trachea and lobar bronchus and at 400 mg/kg in the nasal epithelium. Kidney damage was also noted in mice with 400 mg/kg. There was also an increase in cataract formation at 500 mg/kg and above in C57BL/6 mice, but not DBA/2 mice at up to 2,000 mg/kg. In hamsters there was vacuolation in cells of the lobar bronchus and necrosis of olfactory epithelium at 400 mg/kg but no tracheal damage at the highest dose of 800 mg/kg.

In a briefly reported study, 1 ml volumes of 4 or 5 mg/ml of either naphthalene, 1-naphthol or 2-naphthol was injected intravenously (i.v.) into the marginal vein of an unstated number of albino rabbits (Mackell et al., 1951). From blood samples taken from the opposite ear, no haemolysis was observed with the naphthalene or 2-naphthol treated animals. However, 6% haemolysis was noted with the animals administered 4 mg of 1-naphthol and 9% with 5 mg of 1-naphthol. This study indicates that 1-naphthol, a metabolite in humans, has haemolytic properties in rabbits.

4.1.2.2.2   Studies in humans

There are a great many case reports in the literature of acute haemolytic anaemia produced by naphthalene. The signs and symptoms of haemolytic anaemia associated with naphthalene exposure are well described (e.g. Gosselin et al., 1984, Mack, 1989).

The first signs and symptoms of toxicity are usually dark urine, pallor, abdominal pain, fever, nausea, vomiting and diarrhoea. On clinical examination the liver and spleen were enlarged. Haematological effects are fragmentation of red blood cells with anisocytosis and poikilocytosis, jaundice, anaemia with a reduction in haemoglobin levels and haematocrit values and resulting reticulocytosis and leucocytosis. More severe reactions also include Heinz body formation, haemoglobinuria and mild methaemoglobinaemia. In young children deaths have occurred due to kernicterus (a severe neural condition associated with high levels of bilirubin in the blood). In older children and adults renal failure may occur. Liver damage has also been described, but as a rare occurrence.

Individuals who are deficient in G-6-PD are particularly sensitive to haemolytic anaemia produced by naphthalene (Gosselin et al., 1984). This deficiency is genetically determined and occurs more often in males. The defect results in an inability by the red blood cell to maintain a balance between reduced and oxidised glutathione which in turn results in an increased susceptibility to oxidative attack by exogenous chemicals. It seems probable that the oxidative attack, following exposure to naphthalene, can occur following redox cycling of the naphthalene metabolites 1-naphthol and the quinone. The deficiency is known to be more common in Blacks, Greeks, Italians, Sephardic Jews, Orientals and Filipinos.

Inhalation

No reports were found of acute naphthalene toxicity following a single inhalation exposure.

Oral

Naphthalene was used in the past as an antihelminithic agent. It has not been possible to obtain any details of this use, although some sources (e.g. ACGIH, 1991) indicate that the dose levels used were in the range 0.1-0.5 g three times daily, approximately equivalent to 4-20 mg/kg/day. However no other details are given, particularly with respect to whether or not there were any side effects at these dose levels.

Twelve cases of oral ingestion by young children of naphthalene-containing mothballs have been reported (Melzer-Lange and Walsh-Kelly, 1989; Todisco, 1991; Zuelzer and Apt, 1949; Shannon and Buchannon, 1982; Zinkham and Childs, 1958; Mackell et al., 1951). The majority of the children were aged between 1-3 years. Seven were male and 2 female (the sex of the remaining three cases was not specified). The first signs of toxicity were usually seen within hours to up to 2 days after exposure. Haemolytic anaemia was diagnosed in all cases and signs and symptoms were similar to those described above, with haemoglobin levels falling to 2-6 g/100 ml in 10 cases (average haemoglobin concentration in children aged one year is 12.5 g/100 ml; Wright, 1971). No deaths occurred. In one case haemolysis was reported to have begun 24 to 72 hours after exposure (Shannon and Buchannon, 1982). G-6-PD deficiency was reported in all of the cases (8) in which it was investigated. The amount of naphthalene ingested was not known for any of these cases although consumption apparently ranged from between having sucked one mothball to approximately half its size to swallowing whole two to three mothballs. According to one review naphthalene mothballs usually weigh between 500 and 3,600 mg and contain 100% naphthalene (Mack, 1989). It should, however, be noted that further consumption, and perhaps repeated exposure may have occurred without the knowledge of the parents. Thus no firm conclusions regarding any dose-response relationship can be drawn.

Quantitative details of intake levels of naphthalene producing effects in children are available in the secondary literature. However such data on doses received are old and are difficult to

substantiate and therefore should be used with caution. For example, Sollmann (1957) mentions a very early report which apparently stated that 2 g naphthalene taken over a 2-day period killed a 2 year old child (Prochownik, 1911), but it has been impossible to obtain a copy of the original report.

A few cases of haemolytic anaemia following ingestion of naphthalene have also been reported in teenagers or adults. An early report described a case study of a 16 year old female who had deliberately consumed approximately 6 g of naphthalene, although it is not stated how this estimation was made (Gidron and Leurer, 1956). Within 12-hours she was suffering from abdominal pain and vertigo. On day 2 after the ingestion her erythrocyte count had approximately halved, her urine had darkened in colour and she complained of pain in the kidneys. Despite a blood transfusion on day 2 she had become jaundiced on day 3. Treatment, including another blood transfusion continued. By day 7 the jaundice had subsided. On day 8 her erythrocyte count began to rise and the urine returned to a normal colour. Pain in the kidneys was reported to have continued for "some days". G-6-PD status was not assessed. Based on the requirement for two blood transfusions it seems possible that the estimated 6 g of naphthalene ingested represents a lethal dose to humans.

Haemolytic anaemia (with no red blood cells being seen on blood microscopy) was reported in a female who had drunk approximately 50 ml of an oil which was reported to contain a "high concentration" of naphthalene (Ostlere et al., 1988). The female was apparently not G-6-PD deficient. Her sister also drank the oil and did not show any signs of toxicity.

A secondary literature source cited an incident occurring in 1902 in which severe pain in the bladder and a severe impairment in vision were reported within nine hours of a man taking 5 g unpurified naphthalene over a 13-hour period (Grant, 1974). Vision apparently remained severely impaired 1 year after the incident. Due to the age of the report, the unpurified nature of the naphthalene and the lack of other similar reports, despite its past use as a medicine, no conclusions should be drawn from this report.

Dermal

There is no information available.

In vitro studies

Blood samples were taken from healthy volunteers and mixed *in vitro* with between 0.001-1 ml of either naphthalene, 1-naphthol or 2-naphthol (Mackell et al., 1951). Naphthalene was shown to have no haemolytic activity on the human erythrocytes at any of the concentrations tested. 1-Naphthol gave 74% haemolysis at 1 mg/ml, and there were still some signs of haemolysis at 0.001 mg/ml. 2-Naphthol gave a haemolytic response at all dose levels although this response was less than that observed with 1-naphthol. This study indicated that 1-naphthol is responsible either partially or wholly for the haemolytic anaemia observed in humans.

4.1.2.2.3   Summary of single exposure studies

There is no information on the effects of naphthalene following acute inhalation or dermal exposure in humans. Acute oral exposure to naphthalene causes haemolytic anaemia, which may be fatal. Individuals deficient in G-6-PD are more susceptible to the effects of naphthalene. The first signs of toxicity are usually seen within 2 days. There is little quantitative information available, although severe haemolytic anaemia, which may have proved lethal in the absence of clinical

intervention, was reported in a female who had ingested approximately 6 g naphthalene. An *in vitro* study backed up by an *in vivo* study in rabbits has demonstrated that the anaemia is caused by the naphthalene metabolite, 1-naphthol.

Naphthalene is of low toxicity in rats, with mice being more sensitive. However, studies in animal models (mainly rats, mice and rabbits) have indicated that the toxic effects of naphthalene seen in these species are different from those in humans. Of the species studied, only dogs (in a poorly conducted study) demonstrated naphthalene-induced haemolytic anaemia. Rabbits showed signs of haemolytic anaemia with 1-naphthol but not with naphthalene.

It appears that rodents are not suitable animal models for the acutely toxic human health effects of naphthalene in relation to haemolytic anaemia. Thus, while the LD_{50} results from the rat suggest relatively low acute toxicity in this species, the available information in humans indicates significant toxicity. Very severe haemolytic anaemia occurred in one case report (of a 16 year old female) at an estimated single oral dose of approximately 6 g. It is possible that this represents a lethal dose given that a number of blood transfusions were required.

4.1.2.3   Irritation

4.1.2.3.1   Studies in animals

Skin

In a well conducted unpublished study, 500 mg naphthalene was administered dermally to 6 rabbits for 4 hours and the animals observed for 6 days (Pharmakon, 1985a). In 3 animals, very slight to well defined erythema was visible from 30 minutes to 5 days after administration, with slight fissuring of the skin noted in three animals from 72 hours to the end of the study. There were no signs of oedema in any of the animals at any timepoint. By day 6 after administration all scores had returned to zero.

A brief unpublished report describes a study conducted using 6 rabbits (Reprotox, 1980a). Although the amount of naphthalene administered, the type of dressing and the length of time over which the application took place were not stated, the study appears to be a standard test for skin irritation. Twenty four hours after application of naphthalene, 4 rabbits showed grade 1, and 2 animals grade 2 erythema. Grade 1 and 2 oedema was noted in 4 and 1 animal, respectively. At 72 hours 4 animals still showed grade 1 or 2 erythema and 3 animals showed grade 1 oedema. Naphthalene was described in the study report as being "slightly irritating".

Eye

In a brief unpublished report of what appears to be a standard study, an unstated amount of naphthalene was applied to the eyes of 6 rabbits (Reprotox, 1980b). On the second day temporary iritis was seen in one animal, five animals showed a very slight conjunctival reddening and two slight conjunctival swelling. No ocular abnormalities were noted on day 7. Overall an irritation score of 1.6 was calculated and naphthalene was described as being "non-irritant".

4.1.2.3.2   Studies in humans

Skin

Secondary literature sources such as Gerarde (1960) state that "naphthalene is a primary skin irritant" and cite early references which apparently report generalised erythema in workers handling clothes sprinkled with naphthalene moth repellent (White, 1934) and reversible moist dermatitis in workers handling a mineral oil which apparently contained up to 1.5% naphthalene (Eisner, 1924). Despite its continued use there are no more recent reports of skin irritation in humans. It is unclear whether this is due to an awareness of the hazard and thus care in handling or because other substances with skin irritant properties were present in early activities using naphthalene. Overall it is difficult to draw any conclusions on the irritancy of naphthalene from the information available.

Eye

Secondary literature sources such as Gerarde (1960), Grant (1974) and Gosselin (1984) state that naphthalene vapours may cause "eye irritation". However the majority of the supporting references cited in these sources focus on eye lens opacity formation and not irritation reactions (see Section 4.1.2.6). The only other relevant information from the secondary literature is a report which stated that eye irritation occurs in humans following exposure to 15 ppm naphthalene vapour and above (Grant, 1974). It was also stated that "probably no corneal damage has been produced by naphthalene vapours" in humans. However a reference was not cited and it has not been possible to substantiate this statement.

As above, despite the continued use of naphthalene, there are no more recent reports of eye irritation in humans.

4.1.2.3.3   Summary of irritation

Although no conclusions can be drawn regarding the irritant properties of naphthalene from studies in humans, data from animal studies indicate that it is only a slight skin and eye irritant insufficient to warrant classification.

4.1.2.4   Corrosivity

The studies in animals in Section 4.1.2.3 indicate that naphthalene is not corrosive to the skin or eyes.

4.1.2.5   Sensitisation

4.1.2.5.1   Studies in animals

In a flawed unpublished study, naphthalene was tested for skin sensitising potential by the Buehler method (Pharmakon, 1985b). At the induction stage, 400 mg naphthalene as solid flakes was applied dermally to 20 guinea pigs for 3 six-hour periods. Fourteen days after the last induction period the animals were challenged at a naive site with 400 mg naphthalene. No

positive sensitisation reactions were observed in any of the naphthalene induced animals. It is unclear whether the skin of the test animals was moistened prior to the application of the naphthalene at the induction or challenge stage, and therefore the stringency of the test is unclear. Skin sensitisation reactions were observed in a positive control group induced and challenged with (0.3%) 1-chloro-2,4-dinitrobenzene (DNCB). Overall, although flawed this study is considered to have shown a negative result.

In a poorly reported skin sensitisation study based on the guinea pig maximisation method, a 24-hour open application dermal challenge, with 0.1% or 1% naphthalene in acetone, did not produce any sign of a sensitisation reaction in any of the 24 animals (Okada et al., 1985). However no information was provided on the use of positive controls and at the induction stage no signs of naphthalene-induced irritation were reported at the concentrations examined. However, 2-naphthol was also tested and was positive in all 16 animals tested. Overall considering the quality of the reporting and the low challenge concentrations of naphthalene used, no firm conclusions can be drawn from this study.

There is no information available on the potential of naphthalene to produce respiratory sensitisation in animals.

4.1.2.5.2   Studies in humans

There is no information available on skin or respiratory sensitisation. However, since naphthalene has been in the public and commercial domain for many years and its use has involved dermal contact, the absence of case reports in humans indicates that naphthalene is not a skin or respiratory sensitiser.

4.1.2.5.3   Summary of sensitisation

There is no information available on skin sensitisation in humans or respiratory sensitisation in humans or animals. In animal skin sensitisation studies, negative results were obtained in both a maximisation and a Buehler study, although both had limitations in either conduct or reporting. However, in view of the use of naphthalene for many years in occupational and consumer settings, the absence of reports that naphthalene produces skin or respiratory sensitisation suggests that these endpoints are not of concern for human health and no further information is required.

4.1.2.6   Repeated dose toxicity

4.1.2.6.1   Studies in animals

Inhalation

Studies in rats

In a well conducted unpublished study, groups of 10 male and 10 female rats were exposed nose only for 6 hours/day, 5 days a week for 13 weeks to 0, 2, 10 or 58 ppm (approximately 0, 10, 50 or 300 mg/m^3) vapourised naphthalene (Huntingdon Research Centre, 1993a). A gross pathological

examination was carried out on a wide range of tissues and a microscopic examination was carried out on a range of tissues including the lungs, liver, kidneys, adrenals, testes, eyes and optic nerve. Prior to terminal sacrifice, samples of blood were taken from all rats for haematological and clinical chemistry evaluation. In high dose animals body weight gain was reduced by 43% and 34% in males and females, respectively and was associated with reduced food consumption. There were no toxicologically significant haematological or clinical chemistry findings observed. Similarly, no significant changes were noted in organ weight or gross pathology.

Microscopic analysis of the nasal epithelium revealed treatment-related effects at all dose levels. The severity of the effects was dose-related. At the highest exposure level (300 mg/m^3) changes included erosion of the olfactory epithelium, hyperplasia of basal cells in the olfactory epithelium and loss of Bowmans' glands. At the lowest exposure level (10 mg/m^3) changes in olfactory epithelium were less marked but included slight disorganisation, mild erosion (in one rat), minimal atrophy, rosette formation (an attempt at proliferative repair by the olfactory neuro-epithelium), occasional degenerate cells, loss of Bowmans' glands and minimal hyperplasia. There were no treatment related effects observed in the lungs or nasal respiratory epithelium at this dose. There were no observed changes in the nasal passages of control animals. In one low dose rat there was evidence of squamous metaplasia of the respiratory epithelium, however as this lesion was not seen in the other rats at higher doses this lesion was not considered toxicologically significant. The effects at 10 mg/m^3 were generally minimal in severity and seen in only small numbers of animals, and therefore appear to represent the low end of the dose-response curve for nasal effects. Overall, signs of damage to the olfactory epithelium were seen at all doses down to 10 mg/m^3 (2 ppm), and a NOAEL cannot be identified for local effects.

In a well conducted unpublished study, groups of 5 male and 5 female rats were exposed nose only for 6 hours/day, 5 days a week for 4 weeks to 0, 1, 3, 10, 29 or 71 ppm (approximately 0, 5, 15, 50, 150 or 370 mg/m^3) vapourised naphthalene (Huntingdon Research Centre, 1993b). Investigations were similar to the 13-week study performed in the same laboratory. Results were similar to those observed in the 13-week study. High dose animals showed approximately a 50% reduction in body weight gain associated with reduced food consumption. There was no evidence of systemic toxicity. Local effects were observed with signs of proliferative repair in the nasal olfactory epithelium changes observed at all doses down to 5 mg/m^3 (1 ppm), and therefore a NOAEL for local effects cannot be identified.

For both the 4 and 13-week studies the mechanism by which the observed effects in the olfactory nasal epithelium arise is unclear, although the effects may be mediated by locally produced metabolite(s) of naphthalene. The relevance of these effects to human health is uncertain, as there may be significant species differences in local metabolism. However, there is no evidence to indicate that these effects are not relevant to human health.

Studies in mice

Groups of between 4 and 10 male and female B6C3F$_1$ mice were exposed to 0, 10 or 30 ppm naphthalene by inhalation for 6 hours daily, 5 days a week for 14 days (NTP, 1992). It was stated that no biologically significant changes in haemolytic parameters were observed at any dose level. Other signs of toxicity were not assessed and a general NOAEL cannot be identified from this limited study.

In a carcinogenicity study by the same group of workers, groups of 140 B6C3F$_1$ mice were exposed to 0 or 10 ppm/day (0, 50 mg/m^3/day) and groups of 270 to 30 ppm/day (150 mg/m^3/day) naphthalene vapour for 6-hours/day, 5 days/week for up to 104 weeks (NTP,

1992). Full details of this study are given in section 4.1.2.8. An increase in the incidence of benign alveolar/bronchiolar adenomas was observed in the high dose females and both treated groups showed minimal to mild inflammatory changes in the lungs and nasal passages.

Oral

Studies in rats

No standard repeated exposure toxicity studies have been reported in rats. The studies available have focused on one or a few specific organs and/or manifestations of toxicity.

A dose of 0 or 1,000 mg/kg/day naphthalene was administered by gavage to a total of 12 male albino rats for 10 days (Rao and Pandya, 1981). A statistically significant increase (38%) in relative liver weight was noted in treated animals. There was no change in kidney weight. There was evidence of lipid peroxidation in both the liver and the eye although a statistically significant increase in lipid peroxidation products was only obtained for the liver. No other systemic effects were assessed.

Wistar rats (4 or 5 per group) were administered 0 or 1,000 mg/kg/day naphthalene for 18 days by gavage (Yamauchi et al., 1986). Evidence for increased lipid peroxidation in the liver and serum appeared on the 4th day, reaching a maximum on the 7th and 18th day in the blood and liver, respectively. The increases in serum and hepatic lipid peroxide levels were accompanied by a decrease in the content of glutathione (GSH) in the optical lenses to 64% and 67% of control values on the 4th and 12th day, respectively. Lens opacities in treated animals were observed on the 14th day of treatment. However no information was provided as to the number of rats developing cataracts during the study. No other systemic effects were reported.

Over a nine-week period male 24 Blue Spruce pigmented rats were treated by gavage with up to 750 mg/kg/day naphthalene (100 mg/kg/day for the first 2 weeks, increasing to 750 mg/kg/day over 4 weeks and continued at this level for the next 3 weeks) (Germansky and Jamall, 1988). At the end of treatment a 200% increase in lipid peroxidation (measured as thiobarbituric acid-reactive substances) was observed in liver tissue relative to controls. There was no increase in lipid peroxidation observed in the lung, eye or heart.

In an early chronic exposure study described in more detail in Section 4.1.2.8, no signs of toxicity, including damage to the eyes, was noted in BD rats treated with 0 or 10-20 mg/day naphthalene in the diet 6 days/week for 100 weeks (Schmahl, 1955). This dose is equivalent to 50-100 mg/kg/day, assuming that BD rats weigh 200 g.

Several studies have been conducted to specifically investigate optical lens opacity formation in rats treated with naphthalene (Koch et al., 1976; Tao et al., 1991a, b; Xu et al., 1992; Murano et al., 1993). In three of these studies, five different strains of rat (Brown-Norway, Sprague-Dawley, Long-Evans, Wistar and Lewis) were treated with 0 or 1,000 mg/kg/day by gavage for 6 to 11 weeks. In the fourth study 0 or 700 mg/kg were administered by gavage for 14 weeks. In general the first optical lens lesions (water clefts) were observed after 7 days of treatment and opacities were first noted during the 3rd or 4th week of treatment. Both Xu and Tao reported decreased glutathione and water soluble protein levels in the lens. One study reported the occurrence of 2/6 deaths and another recorded systemic effects which included diarrhoea, decreased body weight gain, hair loss and death although the number of animals involved was not given. No other signs of toxicity or pathological findings were reported in the studies. The metabolite naphthalene 1,2-dihydrodiol was identified in the lens and aqueous humour by Xu. This paper also noted that there was no GSH depletion and no cataract formation when

1,000 mg/kg/day 1-naphthol was administered to rats over 4 weeks. This evidence suggests that the cataract formation may be due to the metabolite 1,2-naphthoquinone.

Studies in mice

In a briefly reported study 10 female CD-1 mice per dosage group were administered 0, 125, 250, 500, 1,200 or 2,000 mg/kg/day naphthalene by gavage for 8 days (Plasterer et al., 1985). There was 100% mortality of the animals with 500 mg/kg/day and above. No histopathological information was provided on the deceased animals and no other systemic effects were assessed.

A total of 388 male and female CD-1 mice were treated with 0, 27, 53 or 267 mg/kg/day naphthalene by gavage for 14 days (Shopp et al., 1984). Mortality in the highest dose group occurred in 10/96 males and 3/60 females. A statistically significant decrease in terminal body weight of 10% was noted in high dose group males. Although gross pathological examination of the organs was stated to have been carried out, no information other than in relation to organ weight was provided. High dose males showed a statistically significant decrease (30%) in thymus weight. In high dose females there was a 29% decrease in spleen weight and a 16% increase in lung weight with both changes being statistically significant. Immunologically, naphthalene treatment had no effect on humoral response or the delayed hypersensitivity response. Dose-related changes were observed in the serum chemistry with statistically significant decreases in blood urea nitrogen (BUN) (30%-36%) and albumin (15%) and increases in cholesterol (51%), bilirubin (23%) and globulin (21%) being noted in high dose males and/or females. However, these changes are considered to be of little toxicological significance. Although no further information was given, it was stated that there was no naphthalene induced cataract formation or haemolytic anaemia observed at any dose level. A NOAEL of 53 mg/kg/day for 14 days was indicated.

As part of the same study 388 CD-1 mice were administered 0, 5.3, 53 or 133 mg/kg/day naphthalene by gavage for 90 days (Shopp et al., 1984). Gross pathological examination of organs was stated to have taken place as with the 14-day study. Although there were no treatment related effects on body weight or mortality, a statistically significant decrease was observed in absolute brain (9.5%), liver (15%) and spleen (32%) weights for females in the highest dose group compared to controls. A statistically significant decrease of 35% in BUN was noted in the high dose group. None of these changes were seen in males. Overall total serum protein levels increased in the 53 and 133 mg/kg/day dose groups by 27% and 25%, respectively, and these increases were reflected by increases in albumin and globulin. However, these changes are considered to be of little toxicological significance. Naphthalene administration had no observed effect on hepatic glutathione levels or mixed function oxidase activity. Although no further information was given, it was stated that there was no naphthalene-induced cataract formation or haemolytic anaemia observed at any dose level. A NOAEL of 133 mg/kg/day for 90 days was indicated.

Studies in rabbits

A total of 35 pigmented rabbits were administered 0 or 1,000 mg/kg naphthalene every other day by gavage for 15 to 180 days (Orzalesci et al., 1994). Slit-lamp and indirect ophthalmoscopy was used to examine the eyes specifically for retinal changes, although gross lens changes were also noted. Ten percent of the animals were sacrificed after developing a complete cataract within 15 days of the start of treatment. In 70% of the remaining treated animals, focal lesions were observed in the eye on the periphery of the retinal fundus beginning three weeks after the start of administration. No other signs of toxicity were assessed.

In a brief report a group of 28 rabbits (16 Dutch pigmented and 12 albino) were administered 1,000 mg/kg/day naphthalene by gavage for up 28 days (van Heyningen and Pirie, 1976). The onset of eye effects ranged from 1 to 8 days and the severity of the effects in the lens and retina among the animals was reported to be very variable. No differences in initiation or severity of the effects were determined between pigmented and albino rabbits.

Thirty nine rabbits (Dutch and two albino strains) were administered 1,000 mg/kg/day naphthalene by gavage for an unspecified length of time (van Heyningen and Pirie, 1967). Within 10 days of treatment some animals showed brown cortical areas in the lens of the eye and yellowing of the eye fluids. Overall, lens opacities were stated to have been observed in over half the animals administered naphthalene. Treatment-related signs of ill-health were reported in the animals including loss of appetite, haemorrhaging of the ears and intestine and in some cases death, although the numbers of animals showing these effects were not stated.

Studies in dogs

In a poorly conducted study with no controls, an average daily dose of 220 mg/kg/day was administered in the diet to a single dog over 7 days (Zuelzer and Apt, 1949). During an observation period of 36 days, lethargy, ataxia and diarrhoea were observed beginning on the fifth day of treatment. Also on the fifth day of treatment the white blood cell count rose from 14,400 to 25,500 and Heinz bodies appeared in the majority of erythrocytes. On the ninth day there was a reduction of the haemoglobin to 2.4 gm/100 ml, red blood cell count to $1.3 \cdot 10^6$ and haematocrit to 7.5 volumes % (from 13.1 gm/100 ml, $6.78 \cdot 10^6$ and 41.5, respectively). The clinical signs and reductions in haematological parameters resolved over 36 days. An optical examination was not conducted.

Dermal

In a well conducted unpublished study, groups of 10 male and 10 female rats were dermally exposed 6 hours/day, 5 days/week for 13 weeks to 0, 100, 300 or 1,000 mg/kg/day naphthalene applied to unmoistened skin under an occlusive dressing (Bushy Run, 1986). Samples of blood were taken for haematological and clinical chemistry evaluation at 4 weeks from 5 male and 5 female animals per dose group and from all rats prior to terminal sacrifice. No treatment related deaths or changes in bodyweight gain occurred. In high dose animals, very mild local skin irritation was indicated by an increased incidence of excoriated skin and papules from day 7 until the end of the study. A dose-related increase in blood urea nitrogen (BUN) was noted at 4 weeks but was not observed at 13 weeks and was therefore of doubtful toxicological significance. No other toxicologically significant findings were observed in either the clinical chemistry, haematology or urinalysis. A limited (7%) but statistically significant decrease in testes weight was observed in high dose animals. However, given the limited magnitude of the observed result and the absence of this finding in other repeat dose studies, this finding is considered to be due to chance. A NOAEL of 1,000 mg/kg/day was identified for systemic effects in this study although mild skin irritation was observed at this dose.

4.1.2.6.2   Studies in humans

Inhalation

Several cases of adverse health effects have been reported following repeated exposure to naphthalene. The principal route of exposure appears to be inhalation although dermal exposure

to the vapour may also have occurred and the possibility of additional oral exposure cannot be discounted.

Eighteen cases of haemolytic anaemia, following exposure to naphthalene vapours, have been reported (Shannon and Buchannon, 1982; Valaes et al., 1963; Dawson et al., 1958; Cock, 1957; Grigor et al., 1966). The majority of the cases were neonates. Fourteen were male and 4 female. Exposure to naphthalene vapour was via clothing and blanketing which had been stored with naphthalene mothballs. The signs and symptoms of anaemia were as described above in Section 4.1.2.2. Two cases of neonatal kernicterus were reported and death occurred in one of the neonates. G-6-PD deficiency was reported in 11/17 cases where it was investigated. One study, which included 6 neonates who were G-6-PD deficient and another 7 who were not, stated that the haemolysis was more severe in those who were deficient (Valaes et al., 1963). The level and duration of exposure was not known in any of these cases, although hospital admissions were commonly made within two weeks of birth. Dermal exposure to solid naphthalene may have occurred in one case for which it was stated that the clothing was "impregnated" with naphthalene mothballs (Dawson et al., 1958).

No conclusions can be drawn, with respect to the role of naphthalene exposure, from a single case report of aplastic anaemia in a 68-year old woman, who had been employed in a clothing resale shop for 39 years, where she was exposed to paradichlorobenzene and naphthalene (Harden and Baetjer, 1978).

A poorly reported paper described eye effects in a group of 21 workers who were involved in manual processes where they came into contact with solid, molten and presumably vaporised naphthalene (Ghetti and Mariani, 1956). The exposure duration is unclear from the report but appears to vary from 1-5 years. Optical lens opacities were noted in 8 workers. However "almost all" of the lesions were pin-point peripheral opacities of the nucleus of the lens, which "largely unaffected" the vision of the individuals. These opacities were described as "slight" (could only be detected by slit lamp). Also, the individuals themselves were reported to be unaware of any damage. However details of two of the cases were presented, and in these two cases cataracts and more marked diffuse opacities were reported. Overall it is not clear from the information provided, whether the effects reported were in excess of that expected in the general population.

A secondary literature source (Grant, 1974) reported three cases of decreased visual acuity, chorioretinitis or lens cataract formation in men occupationally exposed to naphthalene during the early 1900s (Van der Hoeve, 1906; Gottstein et al., 1926). Other signs of naphthalene toxicity did not occur and naphthalene exposure levels (to the solid and/or vapour) were not known. Similarly Gosselin (1984) cited another early reference which apparently claimed that corneal ulceration and cataracts were noted in a worker who had been exposed to naphthalene vapour and dust (Adams and Henderson, 1930). No conclusions as to the potential of naphthalene to cause eye damage can be drawn from these early case reports in view of the lack of information on exposure to other chemical or physical agents which may act as confounders.

Oral

Haemolytic anaemia was observed in a 15 year old male who was reported to have developed a liking for sucking naphthalene mothballs and a 19 year old female who "intermittently sucked and chewed" naphthalene mothballs during her pregnancy (Zinkham and Childs, 1958). Signs and symptoms were the same as those described for acute ingestion of naphthalene. Both individuals were G-6-PD deficient. There was no indication of level or duration of exposure in either case.

Dermal

No information specifically on dermal exposure is available, although dermal exposure to naphthalene solid and vapour may have occurred in the studies summarised in the inhalation section.

4.1.2.6.3   Summary of repeated exposure studies

There are no epidemiological studies on the human health effects of naphthalene, and the only human information available derives from a limited number of early case-reports which provide no quantitative data on the levels or duration of exposure. A principal human health effect is haemolytic anaemia which in some cases has been of marked severity in humans exposed by inhalation to naphthalene vapour and by ingestion to solid naphthalene. Dermal exposure to the solid and vapour was also likely in these cases.

Animal studies reveal species differences in response to naphthalene. Haemolytic anaemia was noted in a dog following oral dosing of 220 mg/kg/day for 7 days but not in rodents even with high/prolonged exposures. Cataract formation was the principal effect seen in rats and rabbits following oral exposure to 700 and 1,000 mg/kg/day, respectively in studies ranging from 10-180 days, but this effect was not seen in mice with similar exposures. The lack of reliable reports of cataracts in humans despite the widespread use of naphthalene and high dose accidental exposure, suggests that cataract formation is unlikely to be a significant health effect in humans.

Limited signs of nasal olfactory inflammation were reported in 28-day rat inhalation study at 5 mg/m3. More marked changes in the olfactory epithelium were noted in a 90-day rat inhalation study at the lowest exposure level of 10 mg/m3. These nasal effects became more marked with increasing levels of naphthalene exposure. In a 104-week carcinogenicity study in mice, signs of nasal, olfactory and pulmonary inflammation were noted at 50 mg/m3, the lowest exposure concentration used. A NOAEL could not be identified for local respiratory effects from these studies.

General signs of toxicity, including death, were reported in rats and rabbits orally dosed with 700 and 1,000 mg/kg/day, respectively. It is apparent that mice are more susceptible to naphthalene than are rats or rabbits following oral treatment, with 100% mortality reported at 500 mg/kg/day in an 8-day study. For systemic effects, a NOAEL of 133 mg/kg/day was identified in a 90-day oral mouse study.

In the 90-day rat inhalation study there was no evidence of systemic toxicity with exposures up to 300 mg/m3, although a reduction in body weight gain of up to 43% associated with reduced food consumption was observed at this top does. By the dermal route a NOAEL for systemic toxicity of 1,000 mg/kg/day (the highest dose tested) was identified in a 90-day rat study.

4.1.2.7   **Mutagenicity**

4.1.2.7.1   *In vitro* **studies**

Bacterial studies

In a well-conducted preincubation assay naphthalene did not produce an increase in revertants in *Salmonella typhimurium* strains TA 98, TA 100, TA 1535 and TA 1538 in the presence or absence of hamster and Aroclor-induced rat liver S9 (NTP, 1992; Mortlemans et al., 1986). Naphthalene was tested up to cytotoxic concentrations.

Negative results were obtained in a plate-incorporation assay in Salmonella typhimurium strains TA 97, TA 98 and TA 100, in the presence or absence of phenobarbitone-induced rat liver S9 (Sakai et al., 1985). As above, naphthalene was tested up to cytotoxic concentrations.

A negative result was also obtained in a more limited study using Salmonella typhimurium TA 98 and TA 1535 in the presence of Aroclor-induced rat liver S9 (Narbonne et al., 1987). This study was performed with S9 only. A replicate experiment was not performed although naphthalene was tested up to cytotoxic concentrations.

Negative results were claimed in another four briefly reported studies using 2 or 4 of the above strains of Salmonella typhimurium (Bos et al.; 1988, Epler et al., 1979; McCann et al., 1975; Purchase et al., 1978). These studies were conducted in the presence of rat liver S9 only. It is not clear if naphthalene was tested up to cytotoxic concentrations and the data were not presented in these study reports. Despite the inadequacies in reporting, these studies support other negative findings. One of these studies also employed the "taped plate" assay (Bos et al., 1988). Negative results were again obtained.

Salmonella typhimurium strains UTH8414 and UTH8413, as well as TA 98 and TA 100 were tested, in the presence and absence of Aroclor-induced rat liver S9, apparently with up to cytotoxic concentrations of naphthalene (Connor et al., 1985). As above experimental results were not presented, but a negative result was claimed.

Naphthalene was also tested for mutagenic activity, as measured by resistance to 8-azaguanine, in Salmonella typhimurium strain TM677 (Kaden et al., 1979). The study was conducted in the presence, but not the absence of phenobarbitone- and Aroclor-induced rat liver S9. Negative results were again obtained.

Negative results were also reported in a non-standard assay using Escherichia coli K12 when the potential of naphthalene to increase the induction of prophages was assessed (Mamber et al., 1984). The study was conducted in the presence of Aroclor-induced rat liver S9.

Overall, naphthalene is clearly not genotoxic in bacterial test systems.

Mammalian cell studies

Positive results were obtained in a well-conducted cytogenetics assay, performed to modern protocols (NTP, 1992; Galloway et al., 1987). Chinese hamster ovary cells (CHO) were exposed for 2-hours to naphthalene in a suitable solvent in the presence of Aroclor-induced rat liver S9. A harvest time of 20 hours was employed. A statistically significant, dose-related increase in the percentage of metaphases with aberrations (excluding gaps) was observed. In the absence of S9, an exposure time of 8-10 hours and harvest times of 10 and 20 hours were employed. There was

no increase in the percentage of metaphases with aberrations in the absence of S9. Cytotoxicity was not reported in this study.

Negative results were obtained in a well conducted unpublished *in vitro* unscheduled DNA synthesis (UDS) assay, in which rat hepatocyte cultures were incubated for 18-20-hours with 0.16 to 5,000 μg/ml naphthalene (Pharmakon, 1985c). The highest dose scored was 16 μg/ml with cytotoxicity stated to have been observed at concentrations of 50 μg/ml and above although no further information was provided. A negative and positive control group were included and produced acceptable results.

No mammalian cell gene mutation studies have been conducted.

A sister chromatid exchange (SCE) assay was conducted in the presence and absence of Aroclor-induced rat liver S9, (NTP, 1992). CHO cells were incubated with naphthalene in the absence of S9 for 26-hours. With S9 the exposure time was 2-hours. Although dose-related increases in the number of SCEs/chromosome were noted with and without S9, the number of SCEs were at most increased by only 50% compared with the solvent controls. In addition, with S9 the effect was noted in only one of two trials. Overall, the results of this study are negative.

As part of a study on *in vitro* metabolism of naphthalene, SCE induction was investigated in human peripheral lymphocytes in the presence of uninduced human liver microsomes (Tingle et al., 1993). The lymphocytes were exposed to naphthalene for 2 hours and harvested at 72 hours. Although the data were provided only in graphical form, there was apparently no increase in SCE frequency in the naphthalene treated lymphocytes.

In an alkaline elution assay rat hepatocytes were exposed to naphthalene for 3-hours (Sina et al., 1983). No increase in the incidence of alkali-labile single-strand breaks was observed.

It was claimed in an abstract, that naphthalene produced chromosomal damage in the cells of preimplantation mouse embryos which had been collected 72 hours after conception and treated with naphthalene with and without rodent liver S9 (Gollahon et al., 1990). The significance of the results from this non-standard study is unclear.

4.1.2.7.2   *In vivo* studies

In a well-conducted bone marrow micronucleus study groups of 5-10 mice were administered a single oral dose of 0, 50, 250 or 500 mg/kg naphthalene (Harper et al., 1984). With the top dose 2/10 deaths occurred. There was no increase in the frequency of micronuclei in polychromatic erythrocytes 24 hours after treatment. A second sample time was not employed. The P/N ratio was not recorded but as naphthalene is known to be absorbed following ingestion and is lipophilic, it is most likely that the cells of the target tissue were exposed. Animals were also treated with 1,500 mg/kg, however 100% lethality occurred and micronuclei were not assessed in these animals.

Negative results were also obtained in a well conducted unpublished bone marrow micronucleus study in which three groups of 10 mice were administered a single intraperitoneal dose of 250 mg/kg naphthalene and sacrificed at 30, 48 or 72 hours after administration (Pharmakon, 1985d). This dose was selected following 100% mortality in mice treated ip with 500 mg/kg and above in a preliminary study. In the main study there were no statistically significant increases in the number of micronuclei per 1,000 polychromatic erythrocytes at any of the timepoints. A

decrease of 35% in the P/N ratio was noted at 72 hours after administration. A negative and positive control group were included and produced acceptable results.

Negative results were obtained in a well conducted unpublished liver unscheduled DNA synthesis (UDS) study, in which groups of 4 rats were administered a single oral dose of 0, 600, 1,000 or 1,600 mg/kg naphthalene and sacrificed 2 or 14-hours after administration (RTC, 1999). The dose levels of naphthalene used were based on results from the previously evaluated rat acute oral toxicity studies, in which a single administration of 2,000 mg/kg naphthalene produced diarrhoea in 17/30 animals and mortality in 2/30 rats. Consequently, for this study 2,000 mg/kg was perceived as the maximum tolerated dose (MTD), and the highest dose level administered (1,600 mg/kg) represented 80% of this MTD, whilst a ratio of 1.6 to 1.7 was applied for the derivation of the lower test groups. The induction of UDS was evaluated using the autoradiographic method. At 1,600 mg/kg, clinical signs reported were piloerection and reduced activity in an unstated number of animals. Reduced activity was also observed in all animals treated with 1,000 mg/kg and in an unstated number of animals from the low dose group. No cytotoxic effects were observed in hepatocyte preparations at any dose level, and therefore the dose levels of 1,000 and 1,600 mg/kg were selected for scoring. In the naphthalene dose groups there were no statistically significant increases in mean net grains per nucleus at either the 2 or 14-hour sacrifice times and no more than 1% of cells were seen in repair. The negative and positive control groups produced acceptable results.

It is reported that when 359 mg/kg naphthalene was administered orally to 7 rats 21 and 4 hours before sacrifice there was apparently no increase in hepatic DNA damage as determined by alkaline elution (Kitchin et al., 1992). However, as this result was part of a paper investigating 110 other chemicals, and presented in tabular form only, no further information is available.

No other information is available.

4.1.2.7.3   Studies in humans

There is no information available.

4.1.2.7.4   Summary of mutagenicity

Naphthalene has given reproducible negative results in bacterial mutation assays, and was negative in an *in vitro* UDS assay. It was however found to be clastogenic in CHO cells in the presence but not the absence of S9. Two *in vitro* studies using CHO cells and human peripheral lymphocytes were negative for induction of SCE. Naphthalene was found to be negative in two *in vivo* bone-marrow micronucleus tests and an *in vivo* rat liver UDS study. Overall, the balance of evidence indicates that naphthalene is not genotoxic.

4.1.2.8   **Carcinogenicity**

4.1.2.8.1   **Studies in animals**

Inhalation

Groups of 49 male and 49 female F344/N rats were exposed to 0, 10, 30 or 60 ppm naphthalene vapour (>99% pure) (approximately equivalent to 0, 50, 150 or 300 mg/m^3) in inhalation chambers for 6 hours/day, 5 days/week for 105 weeks (NTP, draft report 2000). Additional groups of 9 male and female rats were exposed to 10, 30 or 60 ppm naphthalene for 18 months for evaluation of toxicokinetic parameters. All animals were observed twice daily with clinical findings and body weights recorded every 4 weeks beginning at week 5 and every 2 weeks beginning at week 92. Complete necropsy and microscopic examinations were performed on all core study animals.

Survival rates of all exposed groups were similar to those of chamber controls. Survival rates at the end of the study in control, low, medium and high dose males were 24/49, 22/49, 23/49 and 21/49, respectively. The corresponding rates in the females were 28/49, 21/49, 28/49 and 24/49, respectively. At termination, mean body weights of all exposed groups of male rats were 5-6% lower than those of controls. No significant differences were noted in mean body weights of the treated females compared to control animals. There were no treatment-related clinical signs of toxicity in any of the treatment groups.

Neuroblastoma of the nasal olfactory epithelium was observed in males from the 30 and 60 ppm groups (4/48 and 3/48, respectively) and in all exposed groups of female rats (2/49, 3/49 and 12/49 at 10, 30 and 60 ppm, respectively). This neoplasm did not occur in chamber control rats or male rats exposed to 10 ppm. In addition, this tumour has not been observed in the historical chamber control rats in NTP 2-year inhalation studies. Increases were also observed in adenomas of the respiratory epithelium in males from all exposure groups (control: 0/49, 10 ppm: 6/49, 30 ppm: 8/48 and 60 ppm: 15/48) and females from the 30 and 60 ppm exposure groups (control: 0/49, 30 ppm: 4/49 and 60 ppm: 2/49). Compared to concurrent chamber controls the increases in respiratory epithelium adenomas were statistically significant in males but not females. The draft report states that nasal adenomas have not been observed in NTP historical chamber control rats. No lung tumours were observed.

In addition to the nasal neoplasms, the incidences of a variety of non-neoplastic lesions of the nasal tract in both sexes were statistically significantly greater in naphthalene exposed animals than controls. These lesions included, in the olfactory epithelium: atypical (basal cell) hyperplasia, atrophy, chronic inflammation, and hyaline degeneration; in the respiratory epithelium: hyperplasia, squamous metaplasia, hyaline degeneration, and goblet cell hyperplasia; and glandular hyperplasia and squamous metaplasia. In general, the severity of the olfactory and glandular lesions increased with increasing exposure concentrations.

Overall this study demonstrated an increase in the incidence of respiratory epithelial adenomas in naphthalene exposed males from 10 ppm and females from 30 ppm and olfactory epithelial neuroblastomas (a very rare tumour type) in males from 30 ppm and females from 10 ppm. These tumours occurred at sites where non-neoplastic inflammatory changes also occurred and are considered to be treatment-related.

Groups of 70 male and 70 female B6C3F1 mice were exposed to 0 or 10 ppm naphthalene vapour and groups of 135 males and 135 females to 30 ppm naphthalene vapour (>99% pure)

(equivalent to 0, 50 and 150 mg/m^3/day) in inhalation chambers for 6 hours/day, 5 days/week for 104 weeks (NTP, 1992). All animals were observed daily and body weights recorded at least monthly. Necropsy was performed on all animals. Complete histopathological examinations were performed on control and high exposure concentration animals and on all animals found dead or killed moribund prior to the end of the study. Histopathology of the lungs and nasal cavities was also performed on low exposure concentration mice. Serial slit-lamp biomicroscopy and indirect ophthalmoscopic examinations were performed on 5 animals of each sex from all groups at 6-month intervals.

Survival rates were generally good, particularly in the exposed groups. Survival of control males was significantly lower than exposed males. Survival rates at the end of the study in control, low and high dose males were 26/70, 52/69 and 118/133, respectively. The corresponding rates in the females were 59/69, 57/65 and 102/135, respectively. (The low survival in the control males was reported to be due to "wound trauma" and secondary infection resulting from increased fighting in the group). No significant differences were noted in mean body weights of the treated animals compared to control animals. There were no treatment-related clinical signs of toxicity in any of the treatment groups and there were no treatment-related ocular changes in any of the selected animals throughout the study.

A statistically significant increase occurred in the incidence of alveolar/bronchiolar adenomas in high-exposure females (controls: 5/69, 7%; 10 ppm: 2/65, 3%; 30 ppm: 28/135, 21%; historical incidence and range in NTP inhalation studies in female mice: 5.8%, 0-10%). One alveolar/bronchiolar carcinoma was also noted in a high-dose female (1%) but as the historical control incidence is 2.8% (range 0-6%) no significance can be placed on this finding. Exposed males also showed an increased incidence in alveolar/bronchiolar adenomas and carcinomas. However these increases were not statistically significant and/or were within historical control values (adenomas: 7/70 (10%); 15/69 (22%); 27/135 (20%); 69/478 (14.4%), carcinomas: 0/70; 3/69 (4%); 7/135 (5%); 30/478 (6.3%), in control, low and high exposure and NTP historical controls, respectively).

Non-neoplastic changes were only seen in the lungs and nose. A dose-related increase in alveolar and bronchial inflammation (3/139 (2%); 34/134 (25%); 108/170 (63%) in 0, 10 and 30 ppm groups) with macrophage accumulation, lymphocyte infiltration and alveolar epithelial hyperplasia was noted in all groups. The severity of the lung effects was described as minimal to mild but was reported to be more pronounced in exposed animals than controls. Virtually all of the exposed animals, and none of the controls, showed nasal epithelium inflammation with olfactory epithelium metaplasia and respiratory epithelium hyperplasia in the nose. These effects mainly occurred in the posterior nasal cavity and were described as minimal to mild.

Overall this study demonstrated an increase in the incidence of benign adenomas in female mice at a site where non-neoplastic inflammatory changes also occurred. There was no increase in malignant tumours. Other than the non-neoplastic changes in the lungs and nose no other signs of general toxicity were noted and it is possible that the study could have included a higher concentration of naphthalene. That is, the study could have been more rigorous but is none the less, adequate.

In a limited and briefly reported inhalation study groups of 30 male and female Strain A/J mice were exposed to 0, 10 or 30 ppm naphthalene (equivalent to 0, 50 and 150 mg/m^3/day) for 6 hours/day, 5 days/week for 6 months (Adkins et al., 1986). Survival was unaffected by treatment. Body weight and signs of toxicity were not reported. Macro- and microscopic examinations were only conducted on the lungs. There was an increase in the incidence of lung

adenomas, although it is not clear if this increase was statistically significant (Controls: 21%, 10 ppm: 29%, 30 ppm: 30%). No other details were given. Overall due to the high incidence of lung adenomas in controls, the small numbers of animals used and the limited study length, no meaningful conclusions can be drawn from these findings.

Oral

In an early study groups of 28 BDI and BDIII rats (sex not specified) were treated with 0 or 10-20 mg/day naphthalene in the diet, 6 days/week for 100 weeks (Schmahl, 1955). Animals were kept under observation until they died. Survival was unaffected by treatment and there were no signs of toxicity throughout the study. It was also reported that there was no damage to the eyes. Autopsy was reported to be "thorough" and any organs showing abnormalities were examined histologically. There was no increase in the incidence of any tumours in treated animals. Overall, considering the small number of animals used, no firm conclusions as to the carcinogenic potential of naphthalene can be drawn from this early study.

Dermal

There is no information available.

Parenteral

In an early study groups of 38 rats (strain and sex unspecified) were treated with 7 subcutaneous injections of 0 or 500 mg/kg naphthalene, administered once every two weeks for 14 weeks, and were observed for up to 18 months after the end of the treatment period (Knake, 1956). There were no signs of toxicity, including no evidence of haemolysis and corneal damage. Survival in treatment and control animals was poor with a survival rate of approximately 50% being noted in both groups 6 to 7 months after the end of treatment, and no treated and only 4 control animals surviving to the end of the observation period. The poor survival was apparently due to pathogenic infections and the generally poor condition of the animals. Sarcomas were noted at different sites in 15% of test animals and 3% controls. Overall, considering the small number of animals and the poor survival, no firm conclusions can be drawn from this early study.

In another early study groups of 10 BDI and BDIII rats (sex not specified) were treated weekly with 0 or 20 mg naphthalene by subcutaneous or intraperitoneal injection for 40 weeks (Schmahl, 1955). Animals were kept under observation until they died. Survival was unaffected by treatment and there were no signs of toxicity throughout the study. It was also reported that there was no damage to the eyes. Autopsy was reported to be "thorough" and any organs showing abnormalities were examined histologically. There was no increase in the incidence of any tumours in treated animals. Overall, considering the small number of animals, administration route and frequency and the low doses of naphthalene used, no firm conclusions can be draw from this early study.

Other studies

Cultures of Syrian baby hamster kidney cells and human diploid lung fibroblasts were treated with naphthalene in the presence of rat liver S9 (Purchase et al., 1978). There was no increase in transformation frequency. The study was conducted with S9 only. Similarly, negative results were obtained in a cell transformation assay testing naphthalene in mouse mammary gland whole tissue cultures (Tonelli et al., 1979). This second test was conducted with and without the addition of hormones. No conclusions can be drawn from this non-standard study.

4.1.2.8.2   **Studies in humans**

Two brief reports are available of four cases of laryngeal cancer which occurred in workers engaged in the purification of naphthalene (Wolf, 1976; 1978). It is difficult to define from the reports whether the author identified these four cases independently or whether they were brought to his attention by an external source. However, it is clear from the reports that all the cases were smokers and were exposed to other substances including coal tar volatiles. Overall, no conclusion can be drawn from these reports regarding the role, if any, of naphthalene in the production of these cancers.

4.1.2.8.3   **Summary of carcinogenicity**

No conclusions can be drawn from the limited information available in humans. However the carcinogenic potential of naphthalene has been well investigated in animals. In a recently conducted carcinogenicity study in rats, currently only available as a draft document, an increase in the incidence of respiratory epithelial adenomas and olfactory epithelial neuroblastomas (a very rare tumour type) was observed even with the lowest exposure concentration of 10 ppm. In another carcinogenicity study, female mice showed an increase in the incidence of a benign tumour (alveolar/bronchiolar adenomas), to which this species is prone, following inhalation exposure to naphthalene. In view of the negative results obtained in the *in vivo* genotoxicity studies, naphthalene is considered to be non-genotoxic. Given this, the tumours in the animal studies are considered to arise via a non-genotoxic mechanism and consideration must therefore be given to other potential mechanisms underlying the carcinogenic response.

In relation to the rat nasal tumours, the tumours develop only at the sites where non-neoplastic inflammatory changes also occur (changes such as atrophy, hyperplasia and metaplasia). Thus, it is considered that the development of the nasal tumours in the rat is a consequence of chronic tissue injury, for which an identifiable threshold of effect will exist, although currently not identified. However, the available data do not allow the identification of a threshold for chronic tissue damage, nor is there any clear information on whether or not local tissue metabolism is involved in the toxicity of naphthalene to the nasal epithelium. Mechanistic studies have indicated that a particular stereoisomer is formed in significant amounts in rat and hamster nasal olfactory epithelium. This stereoisomer may be implicated in the toxicity of naphthalene and may explain the observed effects in this tissue in repeat inhalation studies in rats. However, there is currently insufficient evidence to support or refute this hypothesis, nor is there any information on the formation of this metabolite in humans. Given this, it is not possible to determine whether or not the effects seen in the rat are relevant for humans. In addition, there are anatomical differences in the nasal passages between rats and humans, and differences in breathing pattern (rats are obligate nasal breathers) which may affect airflow and deposition patterns of naphthalene. Thus, there is some uncertainty concerning the relevance of the rat nasal effects to human health. However, overall, it is not possible to dismiss the rat nasal olfactory data as being of no relevance for humans and consideration will be given to this endpoint in the risk characterisation.

The development of naphthalene-induced mouse lung adenomas is unlikely to be of relevance to human health due to species differences in pulmonary metabolism. *In vitro* studies with lung microsomal preparations (Section 4.1.2.1) clearly showed that mouse lung preparations metabolised naphthalene at substantially greater rates (up to 100-fold) than those from hamster, rat or monkey. Furthermore, intra-peritoneal dosing of 50 mg/kg naphthalene led to specific toxicity to Clara cells in the lungs of mice, but no such toxicity was observed in rats even at

1,600 mg/kg (Section 4.1.2.2.1). In addition, no lung tumours were seen in rats. Hence, the pattern of toxicological evidence indicates that the mouse is more susceptible to the pulmonary toxicity of naphthalene than other species, and therefore the observed pulmonary adenomas seen in mice at 30 ppm (150 mg/m^3) are not considered to be of relevance to human health.

4.1.2.9   Toxicity for reproduction

4.1.2.9.1   Studies in animals

<u>Effects on fertility</u>

No studies investigating effects on fertility are available. In a carcinogenicity study mice showed no histopathological changes in the epididymis, prostate, seminal vesicle, testis or ovary following inhalation of 30 ppm (150 mg/m^3/day, estimated to be approximately 45 mg/kg/day, assuming a 30 g mouse breathes in 1.8 l/hour, and that 100% naphthalene is absorbed) naphthalene vapour for 6-hours/day, 5 days/week for 104 weeks (NTP, 1992).

<u>Developmental studies</u>

Rabbits

In a well-reported study groups of 25 rabbits were treated by gavage with 0, 20, 80 or 120 mg/kg/day on days 6-19 of gestation (Navarro et al., 1992). Caesarean sections were conducted on day 30. There were no signs of maternal toxicity in any of the treatment groups and there were no differences in the number of resorptions, live and dead fetuses, litter size, fetal body weight and no increase in the incidence of external, skeletal or visceral malformations. The potential to produce developmental effects at maternally toxic doses was not assessed.

The dose levels used in the above study were based on a preliminary study which was cited in the above report. Rabbits were treated with 0, 75, 150, 300 or 500 mg/kg/day, presumably according to the above protocol. Maternal deaths (at least 40%) occurred with 150 mg/kg/day and above. There were no signs of fetotoxicity. Pups were apparently not assessed for malformations. It is difficult to draw firm conclusions from the brief report of this preliminary study which did not assess occurrence of malformations.

In a well conducted unpublished study, groups of 18 rabbits were administered 0, 40, 200 or 400 mg/kg/day naphthalene by gavage on days 6-18 of gestation (Pharmakon, 1985e). Caesarean sections were conducted on day 29. Two high dose animals aborted on days 18 and 23 of gestation which was considered to be due to maternal toxicity. There were no naphthalene induced maternal deaths or statistically significant changes in maternal body weight. However, at 200 and 400 mg/kg/day increased dyspnoea, cyanosis and salivation were reported. Examination of the dams and offspring indicated no differences in the number of implantations, post-implantation loss, number of live and dead foetuses, litter size, fetal body weight, or fetal sex distribution in any of the treatment groups. Overall, no developmental effects were observed at a naphthalene dose of up to 400 mg/kg/day, at which maternal toxicity was evident.

Rats

In a well conducted but poorly reported study groups of 25 female Sprague-Dawley rats were treated by gavage with 0, 50, 150 or 450 mg/kg/day naphthalene on days 6-15 of gestation (Navarro et al., 1991). Caesarean sections were performed on day 20. Maternal body weight gains (corrected for gravid uterine weight) were decreased by 22 and 29% with 150 and 450 mg/kg/day, respectively. These animals were also lethargic and showed slow respiration rates during the treatment period. There were no differences in the number of corpora lutea per dam or the number of dead or live fetuses per litter in any treatment group. However there was a 2-fold increase in the number of resorptions per litter with the top dose compared to the controls. It was not stated whether these were considered to be early or late resorptions. Pups from top dose animals showed a slight increase in the number of litters with visceral malformations and slight dose-related increases in percentage of fetuses per litter with visceral malformations was also noted. However these increases, which were principally due to increased incidence of enlarged lateral ventricles in the brain, were not statistically significant. Overall this study provides some evidence of fetotoxicity occurring at maternally toxic doses with no fetotoxicity occurring at doses which were not maternally toxic.

The dose levels used in the above study were based on a preliminary study which was cited in the above report. Rats were treated with 0, 100, 400, 500, 600 or 800 mg/kg/day, presumably according to the protocol above. Severe maternal toxicity was noted with the top two doses. With the top dose 67% of dams died and total resorptions occurred in 33% of the survivors. No further details were presented of the toxicity seen with 600 mg/kg/day. "Slight" maternal and fetal toxicity was noted with 400 and 500 mg/kg/day although no details were given. Pups were not assessed for malformations. Overall fetal toxicity was apparently observed at maternally toxic doses. However it is difficult to draw firm conclusions from the brief report of this preliminary study which did not assess the occurrence of malformations.

No signs of maternal or fetotoxicity or fetal developmental effects were noted in a study in which "small" groups of pregnant rats were treated intraperitoneally with naphthalene (Hardin et al., 1981). In contrast evidence of delayed development was claimed in a brief abstract, in a study conducted by the same group of workers using the same intraperitoneal dose of naphthalene (Harris et al., 1979). Maternal toxicity was not reported. No conclusions, with respect to effects on human health, can be drawn from these two studies due to the route of exposure employed.

Mice

In a poorly conducted study in CD-1 mice, groups of 50 females were treated by gavage with 0 or 300 mg/kg/day naphthalene on days 7-14 of gestation (Plasterer et al., 1985). Dams showed a 15% decrease in survival and a 26% reduction in body weight. There was a statistically significant decrease (18%) in the number of live pups/litter, although a corresponding increase in the number of dead pups/litter was not noted. There was no change in pup weight. Gross examinations were apparently performed on the pups but it is not clear if visceral and skeletal examinations were conducted. The number of resorptions was not assessed. Overall, evidence of fetal toxicity was observed in this limited study at a dose producing severe maternal toxicity.

In vitro

No conclusions, with respect to effects on human health, can be drawn from three non validated studies in which mouse embryos were treated *in vitro* with naphthalene (Iyer et al., 1990; 1991).

4.1.2.9.2   Studies in humans

<u>Effects on fertility</u>

There is no information available on the effects of naphthalene on fertility in humans.

<u>Developmental effects</u>

A new-born baby was noted to be suffering from "profound" haemolytic anaemia (Zinkham and Childs, 1958). Haemolytic anaemia was also observed in the mother who was reported to have "intermittently sucked and chewed" naphthalene mothballs during her pregnancy. The mother was G-6-PD deficient. There was no indication of level or duration of exposure.

A similar case was reported in which a 26 year old female was suffering from haemolytic anaemia, following ingestion of an unspecified number of naphthalene mothballs during the third trimester of her pregnancy (Anziulewicz et al., 1959). She gave birth to a male who was also suffering from haemolytic anaemia on the first day post partum. Ethnic origin and G-6-PD status were not reported and there was no quantitative information on exposure.

4.1.2.9.3   Summary of toxicity for reproduction

In relation to fertility, there is no information available in humans and there are no animal studies specifically investigating such effects. However in a two-year carcinogenicity study mice showed no histopathological changes in the gonads or accessory sex organs following inhalation of 150 mg/m^3 naphthalene. No testes changes were observed in a 90-day inhalation study in rats at 300 mg/m^3.

With respect to developmental toxicity, the only information available in humans comes from cases of haemolytic anaemia in infants born to mothers also suffering haemolytic anaemia, following ingestion of unquantified doses of naphthalene during their pregnancy. In rats fetotoxicity, but not malformations, was observed at doses causing significant maternal toxicity (450 mg/kg/day). Maternal toxicity was also noted at lower doses without fetotoxicity (150 mg/kg/day). Fetotoxicity was also observed in mice at doses which produced severe maternal toxicity (300 mg/kg/day). In rabbits, no developmental effects were seen in one study at a dose causing mild maternal toxicity, or in another study at a dose close to those producing pronounced maternal toxicity. Overall naphthalene only produces fetotoxicity at maternally toxic doses in animals, and does not produce developmental toxicity at maternally subtoxic doses.

4.1.3   Risk characterisation

4.1.3.1   General aspects

There is a reasonable animal toxicity database available for naphthalene. However, it is apparent that the main experimental species (rats, mice and rabbits) do not provide a suitable model for naphthalene-induced haemolytic anaemia, a principal toxicological effect of naphthalene in humans. There is some evidence from a single dog study to indicate that this species may develop naphthalene-induced haemolytic anaemia, but due to the limited nature of the study few conclusions can be drawn.

Therefore, the only option is to use information from the available human case reports for assessing the risks of haemolytic anaemia. In humans, the occurrence of haemolytic anaemia has been reported in at least 30 individuals, typically following single or repeated oral intake of naphthalene mothballs but also following inhalation and dermal exposure to naphthalene from clothing. In some cases (particularly neonates) the naphthalene-induced haemolytic anaemia proved fatal, although it is not possible to determine the doses involved from the reports available. Overall, due to a lack of quantitative information on the exposures producing haemolytic anaemia in humans, the nature of the dose-response relationship cannot be identified. Furthermore, it is apparent that individuals who are deficient in the enzyme glucose-6-phosphate dehydrogenase are more susceptible to the haemolytic effects of naphthalene than are the general population, and as the enzyme status of the individuals was not always given in the case reports this adds further uncertainty to the assessment of risk for this endpoint.

Given these uncertainties, whilst it is clear that humans are susceptible to haemolytic anaemia following exposure to naphthalene via the oral, dermal or inhalation routes there is insufficient information available for an adequate risk characterisation. In the occupational setting, it is unclear whether contemporary levels of exposure might be associated with the overt or sub-clinical development of haemolytic anaemia. In view of the clear species differences in relation to this endpoint, and the lack of a suitable animal model, it was proposed that a workplace health survey should be conducted. However, investigation into the feasibility of such a study revealed that the only population exposed to a high level of naphthalene (but no other confounding exposures) was the workforce of a mothball manufacturing plant. There were only 17 workers who would qualify for a study and it was concluded that this population was too small for any meaningful conclusions to be drawn from such a study. Thus, the NOAEL for this endpoint cannot be clarified any further.

With regard to other information and endpoints, the limited toxicological information available in humans indicates that naphthalene is readily absorbed by all routes of exposure and animal data shows that almost complete and rapid absorption occurs following ingestion. In humans, naphthalene is metabolised to 1-naphthol, 2-naphthol and 1,2- and 1,4- naphthoquinone. *In vitro* studies in human liver microsomes and human lung preparations indicate that naphthalene is also metabolised to naphthalene 1,2-dihydrodiol via a pathway which involves epoxide hydrolase. Metabolism in rodents is chiefly by P450 oxidation, with subsequent glutathione conjugation, as well as epoxide hydroxylation to naphthalene 1,2-dihydrodiol. Glutathione conjugation has not been shown to occur in non-human primates. There is some evidence that significant enterohepatic recirculation of naphthalene metabolites occurs in rodents. *In vitro* studies show that the rate of naphthalene metabolism in mouse lung tissue is approximately 3, 8 and 100 times greater than that observed in lung tissue from hamsters, rats and monkeys, respectively. There are no data available to indicate whether or not similar differences in metabolic activity between

species also occur in the nasal tissues. The urine is the main route of rapid excretion in humans and animals.

In relation to acute toxicity, naphthalene is of low single dose toxicity in rats, but there appears to be some species differences in that the mouse shows a greater sensitivity to the acutely lethal effects of naphthalene than the rat, presumably reflecting differences in metabolism. However, given that neither species shows haemolytic anaemia at the doses administered this information is not considered to be relevant to humans.

There is no information on the irritant properties of naphthalene in humans. However, data from animal studies indicate that it is only a slight skin and eye irritant. There is no information available on skin sensitisation in humans or respiratory sensitisation in humans or animals. In animal skin sensitisation studies, negative results were obtained in both a maximisation study and a Buehler study although both had limitations in either conduct or reporting. There is no evidence to suggest that humans would react differently to the animals and overall, in view of the long-standing and widespread use of naphthalene (including domestic), the absence of reports of skin or respiratory sensitisation suggests that these endpoints are not of concern for human health and no further information is required.

With regard to systemic effects other than haemolytic anaemia following repeat exposure, cataract formation appears to be the principal effect reported in rats and rabbits given high doses of naphthalene (700 and 1,000 mg/kg/day, respectively) by the oral route. Although there are a limited number of early case-reports of cataract formation in humans these are of uncertain reliability and provide no information on exposure to other chemical or physical agents which may act as confounders. The lack of reliable reports of cataracts in humans despite the widespread use of naphthalene or any reports of cataracts following high dose exposure from poisoning incidents in the past 60 years, suggests that cataract formation is unlikely to be a significant health effect in humans and therefore this endpoint is not evaluated further.

In a 90-day inhalation study in the rat there was no evidence of systemic toxicity with exposures up to 300 mg/m^3, although a reduction in body weight gain of up to 43% associated with reduced food consumption was observed at this top dose. This is likely to have been a consequence of the olfactory effects and is not clear evidence of systemic toxicity. By the dermal route, no systemic toxicity was seen at 1,000 mg/kg/day (the highest dose tested) in a 90-day rat study.

With regard to local effects following repeated exposure, in a 90-day rat inhalation study, minimal degenerative changes were observed in the nasal olfactory epithelium (but not lungs) even at the lowest exposure level of 2 ppm (10 mg/m^3), with similar but less marked changes also seen in a 28-day study at 1 ppm (5 mg/m^3). At higher exposures, damage to the olfactory epithelium was more pronounced. In rats exposed by inhalation for 2 years, chronic inflammatory changes in the olfactory and respiratory epithelium were seen at all exposure levels, from 10 ppm (50 mg/m^3) and above. These lesions included atrophy, hyperplasia and metaplasia. In general, the severity of the lesions increased with increasing exposure concentrations. In mice, minimal signs of inflammation were noted in the lungs and nasal passages at 10 ppm (50 mg/m^3) at the end of a 2-year study. An intra-peritoneal injection of 50 mg/kg naphthalene in mice led specifically to Clara cell damage, but no such damage was observed in rats even at 1,00 mg/kg, although it was noted that there was necrosis of rat nasal olfactory epithelial cells at 200 mg/kg.

Mechanistic studies have indicated that compared to monkey, mouse lung (primarily metabolically active Clara cells) may be particularly susceptible to naphthalene toxicity due its high rate of metabolism and the formation of a particular stereoisomer of a metabolite. Other

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studies have indicated that this particular stereoisomer is also formed in significant amounts in rat and hamster nasal olfactory epithelium, which may explain the observed effects in this tissue in repeat inhalation studies in rats.

Overall, there are clear species differences in naphthalene-induced lung damage, with the mouse showing greater susceptibility than rat/monkey, and it is considered that the monkey is likely to be a more reliable model than the mouse for the assessment of the effects of naphthalene on the human lung. In this regard, *in vitro* data indicated metabolism of naphthalene in mouse lung tissue to be 100-fold faster than in monkey lung tissue. In relation to nasal olfactory effects, there are anatomical and possibly metabolic differences between rats and humans, and thus there is some uncertainty concerning the relevance of the rat nasal effects to human health. However, it is not possible to dismiss the relevance to human health of these rat nasal olfactory data.

Naphthalene has given reproducible negative results in bacterial mutation assays, and was negative in an *in vitro* UDS assay. Two *in vitro* studies using CHO cells and human peripheral lymphocytes were negative for induction of SCE. It was however, found to be clastogenic in CHO cells in the presence but not the absence of S9. Naphthalene was also found to be negative in two *in vivo* bone-marrow micronucleus tests in mice and an *in vivo* liver UDS study. Overall, the balance of evidence indicates that naphthalene is not genotoxic.

No conclusions on carcinogenicity can be drawn from the limited information available in humans. However, the carcinogenic potential of naphthalene has been well investigated in animals. In the most useful animal carcinogenicity study available, a 2-year inhalation study in the rat, an increase in the incidence of respiratory epithelial adenomas and olfactory epithelial neuroblastomas (a very rare tumour type) was observed even with the lowest exposure concentration of 10 ppm (50 mg/m^3). In another study, female mice showed an increase in the incidence of benign lung tumours (alveolar/bronchiolar adenomas), to which this species is prone, following inhalation exposure to naphthalene. In view of the negative results obtained in the *in vivo* genotoxicity studies, naphthalene is considered to be non-genotoxic. Given this, the tumours in the animal studies are considered to arise via a non-genotoxic mechanism and consideration must therefore be given to other potential mechanisms underlying the carcinogenic response.

In relation to the rat nasal tumours, the tumours develop only at the sites where non-neoplastic inflammatory changes also occur (changes such as atrophy, hyperplasia and metaplasia). Thus, it is considered that the development of the nasal tumours in the rat is a consequence of chronic tissue injury, for which an identifiable threshold of effect will exist, although currently not identified. It is possible that the damage to the rat olfactory epithelium, and subsequent development of tumours at this site, may be mediated by a locally produced metabolite(s) of naphthalene at the target tissue. However, the available information does not allow the clear identification of which metabolite(s) may be involved, nor is there any information on the comparative metabolic capacity of rat and human nasal tissue in relation to the formation of any such metabolite(s). In addition, there are significant anatomical differences in the nasal passages of rats and humans and differences in breathing patterns (rats are obligate nasal breathers), which are associated with differences in the local airflow and deposition in the upper respiratory tract of the rat and human. Therefore, there is some uncertainty surrounding the relevance of the naphthalene-induced rat olfactory epithelium damage for human health. However, overall, the effect cannot be dismissed as being of no relevance to humans.

Given that the underlying mechanism for the development of nasal tumours in the rat is considered to be the chronic inflammatory damage seen at this site, it is follows that prevention

of local tissue damage would prevent subsequent development of tumours. However, a NOAEL for local tissue damage cannot be identified from the available studies. It is clear from inhalation studies conducted in rats that even with 28-day exposure to naphthalene, damage to nasal olfactory tissue occurred at 5 mg/m^3, the lowest concentration level used. In view of the similar findings in the 90-day and 2-year studies, it is anticipated that the mild nasal effects observed at 5 mg/m^3 in the 28-day study would have been likely at least to have been present and possibly to have progressed to more severe effects, had the duration of the study been extended to two years. Therefore, the LOAEL of 5 mg/m^3 from the 28-day study will be used in the risk characterisation for repeated inhalation toxicity, including carcinogenicity.

In relation to fertility, there is no information concerning effects on humans exposed to naphthalene and there are no animal fertility studies. In two-year carcinogenicity studies, neither mice nor rats showed any histopathological changes in the gonads or accessory sex organs following inhalation of up to 300 mg/m^3 naphthalene. No testes changes were observed in a 90-day inhalation study in rats at 300 mg/m^3. Overall, there are no grounds to indicate that naphthalene would adversely affect fertility.

With respect to developmental toxicity, the only information available in humans comes from cases of haemolytic anaemia in infants born to mothers also suffering haemolytic anaemia, following ingestion of unquantified doses of naphthalene during their pregnancy. In rats, a two-fold increase in the number of resorptions but no malformations, were observed at doses causing significant maternal toxicity (450 mg/kg/day). Maternal toxicity was also noted at lower doses without evidence of resorptions (150 mg/kg/day). Fetotoxicity (18% decrease in the number of live pups/litter) was also observed in mice at doses which produced severe maternal toxicity (300 mg/kg/day). In rabbits, no developmental effects were seen in one study at a dose causing mild maternal toxicity, or in another study at a dose close to those producing pronounced maternal toxicity. Overall naphthalene does not demonstrate developmental toxicity at doses which are not maternally toxic. Therefore there is considered to be no concern for human health from this endpoint.

Overall, the hazardous properties of naphthalene have been evaluated to the extent that the minimum data requirements according to Article 9(2) of Regulation 793/93 have been met. The key health effects of haemolytic anaemia, repeated inhalation toxicity and carcinogenicity have been identified. For haemolytic anaemia, it is not possible to identify a NOAEL from the available data. For repeated inhalation toxicity, the key effect of concern is local damage to the upper respiratory tract. The available data do not allow the identification of a NOAEL; in a 28-day study in rats, damage to nasal olfactory tissue occurred at 5 mg/m^3, the lowest concentration level used. In relation to carcinogenicity, naphthalene is not genotoxic *in vivo* and thus the tumours are considered to arise via a non-genotoxic mechanism. The tumours develop only at the sites where non-neoplastic inflammatory changes also occur. Thus, it is considered that the development of the nasal tumours in the rat is a consequence of chronic tissue injury, for which an identifiable threshold of effect will exist, although currently not identified. Given that the underlying mechanism for the development of nasal tumours in the rat is considered to be the chronic inflammatory damage seen at this site, it follows that prevention of local tissue damage would prevent subsequent development of tumours. However, as indicated, a NOAEL for local tissue damage cannot be identified from the available studies. It is clear from inhalation studies conducted in rats that even with 28-day exposure to naphthalene, damage to nasal olfactory tissue occurred at 5 mg/m^3, the lowest concentration level used. In view of the similar findings in the 90-day and 2-year studies, it is anticipated that the mild nasal effects observed at 5 mg/m^3 in the 28-day study would have been likely at least to have been present and possibly to have progressed to more severe effects, had the duration of the study been extended to two years.

Therefore, the LOAEL of 5 mg/m^3 from the 28-day study will be used in the risk characterisation for repeated inhalation toxicity, including carcinogenicity.

There are no concerns for irritation, sensitisation, mutagenicity or for effects on reproduction. Thus, **conclusion (ii)** is reached for these endpoints, for all exposure scenarios.

4.1.3.2   Workers

The relevant routes of occupational exposure are inhalation and dermal. In calculating body burdens arising from these routes of exposure, it has been assumed that a worker breathes 10 m^3 per day, that absorption is 100% by both routes and that a worker weighs 70 kg. For dermal exposure, an exposed area of 2,000 cm^2 has been assumed.

4.1.3.2.1   Manufacture

Haemolytic anaemia

The manufacture of naphthalene gives rise to a maximum 8-hour TWA of up to 6.3 mg/m^3 and a dermal exposure of up to 0.1 mg/cm^2/day. The resulting total body burden is about 4 mg/kg/day. A NOAEL for haemolytic anaemia cannot be identified and therefore a margin of safety cannot be calculated. Given the absence of a NOAEL and of information on the dose-response characteristics for this endpoint, any significant body burden (values in the mg/kg range) is considered to give rise to concern. Thus, **conclusion (iii)** is reached for this endpoint.

Repeated inhalation toxicity and carcinogenicity

The 8-hour TWA of up to 6.3 mg/m^3 is above the LOAEL of 5 mg/m^3 for local damage to the respiratory tract. Thus the MOS is less than 1, which raises concerns for human health and **conclusion (iii)** is reached.

4.1.3.2.2   Uses

Phthalic anhydride manufacture

The use of naphthalene in the chemical synthesis of phthalic anhydride gives rise to a maximum 8-hour TWAof up to 2 mg/m^3. Cleaning of the process plant vessels which takes place every 2-3 years would be expected to give rise to higher levels of exposure. However the use of appropriate personal protective equipment (compressed air-line breathing apparatus) is expected to be routinely used in such cleaning processes and would be expected to significantly reduce exposure. Dermal exposures for this scenario are in the range 0-0.1 mg/cm^2/day.

Haemolytic anaemia

The body burden arising from combined inhalation and dermal exposure is about 3 mg/kg/day. As for manufacturing, exposures of this magnitude are considered to be of concern, and thus **conclusion (iii)** is reached.

Repeated inhalation toxicity and carcinogenicity

The ratio between exposure to airborne naphthalene and the level (LOAEL of 5 mg/m^3) at which minimal degenerative effects were observed in the rat nasal olfactory epithelium is 2.5. Given the severity of this endpoint, this MOS is not considered to be sufficient to allow for differences in toxicokinetics and toxicodynamics between and within species and thus **conclusion (iii)** is reached.

Other chemical syntheses

Other chemical syntheses include the use of naphthalene in the synthesis of compounds such as dye intermediates and naphthalene sulphonic acids. No data were available from these industries but occupational exposure via inhalation is predicted to be similar to that reported during the manufacture of phthalic anhydride. Thus, the same conclusions are reached, namely **conclusion (iii)** applies for both health endpoints of concern.

Blending and use of creosote

1. Blending: No data were available for the blending of creosote but occupational exposure via inhalation is predicted to be similar to that reported during the manufacture of phthalic anhydride. Dermal exposure is predicted to be in the range 0.025-0.25 mg/cm^2/day. However, operators wear gloves and therefore exposure is likely to be at the bottom of this range.

2. Bulk impregnation plants: Exposure via the lungs to naphthalene vapour is highest due to exposure to creosote vapour by workers in timber bulk impregnation plants. An 8-hour TWAexposure value of 51 mg/m^3 has been recorded. However, it is assumed that use of RPE will have reduced exposure to at least 26 mg/m^3 and although this value is within an exposure range of 5-30 mg/m^3 predicted by the EASE model, it is at the top end of the range and may considerably overestimate typical exposure. It should also be noted that this value is not for pure naphthalene, but includes indene and methyl naphthalene, but since the percentage of naphthalene is not known, 100% is assumed for calculations. Dermal exposure is predicted to be in the range 0.025-0.25 mg/cm^2/day. However, operators wear gloves and therefore exposure is likely to be at the bottom of this range.

3. Packaging plants (for DIY outlets): Delivery of creosote to packaging plants by road tanker and transferral to storage may lead to occupational exposure, but these are likely to be short-term intermittent tasks. Inhalation exposure has been modelled using EASE and the 8-hour TWA was estimated to be 8 mg/cm^3. Factors to be taken into account when considering the MOS include that the filling of containers (upon which the EASE model is based) is likely to be an intermittent task. On the other hand this task may not always be carried out using an automatic system. Exposure during manual filling is expected to be higher. Dermal exposure is predicted to be in the range 0.025-0.25 mg/cm^2/day. However, operators wear gloves and therefore exposure is likely to be at the bottom of this range.

4. Brush application: Occupational exposure to naphthalene occurs from the brush application of creosote. Exposure levels in the occupational environment have not been measured. However, Section 4.1.1.2, consumer exposure). For a professional painter therefore a full shift may involve exposure with up to 2.9 mg/m^3 8-hour TWA. Dermal exposure is predicted to be in the range 0.025-0.25 mg/cm^2/day. However, operators wear gloves and therefore exposure is likely to be at the bottom of this range.

Haemolytic anaemia

All scenarios in the blending and use of creosote result in exposures and resultant body burdens which are comparable with or higher than those estimated for manufacture of phthalic anhydride. Thus, the same conclusions are reached, namely these exposures are of concern and **conclusion (iii)** applies.

Repeated inhalation toxicity and carcinogenicity

The lowest inhalation exposure is for blending, estimated to be similar to that for manufacture of phthalic anhydride (up to 2 mg/m^3 8-hour TWA). Thus, the MOS is 2.5. Given the severity of this endpoint, this MOS is not considered to be sufficient to allow for differences in toxicokinetics and toxicodynamics between and within species and thus **conclusion (iii)** is reached. All other scenarios result in higher exposures and resultant MOSs of less than 1 and therefore are also considered to be of concern. Overall, **conclusion (iii)** is reached for blending and use of creosote.

Mothball manufacture

The EASE model predicts an exposure range of 2.6-16 mg/m^3 due to the vapour and 2-5 mg/m^3 due to particulate matter giving a combined inhalation exposure prediction of 4.6-21 mg/m^3. This range appears to correlate reasonably well with measured data. No information was available on the particle size and therefore 100% is assumed to be inhalable. Dermal exposure is predicted to be in the range 0.1-1 mg/cm^2/day. However, exposure is likely to be at the bottom of this range, as operators wear gloves.

Haemolytic anaemia

The resultant body burden arising from these exposure levels is at least 6 mg/kg/day, assuming minimal dermal exposure. A body burden in this range is considered to give rise to concern and therefore **conclusion (iii)** is reached.

Repeated inhalation toxicity and carcinogenicity

The worst-case exposure to vapour and airborne particulates is approximately 4-fold greater than the level at which effects have been observed in rats and thus the MOS is less than 1. Therefore there are concerns for human health and **conclusion (iii)** is reached.

Manufacture of grinding wheels

Occupational exposure to naphthalene during the handling of blends and pressing into moulds is an open operation, although LEV is used for some operations. Limited industry data was received for exposure to the vapour, with results of 2.9 and 5.4 mg/m^3 8-hour TWA. Exposures were predicted using EASE for operations with and without LEV. These were 1.4 to 3.1 mg/m^3 8-hour TWA and 6.9 to 20 mg/m^3 8-hour TWA, respectively, for combined particulate and vapour exposure. Dermal exposures are predicted to be in the range 0.3-1.5 mg/cm^2/day.

Haemolytic anaemia

These exposures are higher than those for other scenarios for which concerns have been identified. Thus, the body burdens arising from exposure during the manufacture of grinding wheels are also considered to give rise to concern, and **conclusion (iii)** is reached.

Repeated inhalation toxicity and carcinogenicity

Exposure to vapour and airborne particulates with LEV is up to 3.1 mg/m^3. Comparing this with the LOAEL of 5 mg/m^3 gives an MOS of 1.6. This is considered to be insufficient to allow for differences in toxicokinetics and toxicodynamics between and within species and thus **conclusion (iii)** is reached. For scenarios without the use of LEV, combined exposure to vapour and particulates is above the LOAEL for nasal damage in rats. Therefore the MOS is less than 1 and there are concerns for human health, leading to **conclusion (iii)**.

Manufacture and use of coal tar paints and waterproof membranes

The EASE model predicts an exposure range of 2.5-5 mg/m^3, although it is appreciated that as the application is generally outdoors that exposure is likely to be lower. Dermal exposure is predicted to be in the range 0.001-0.02 mg/cm^2/day.

Haemolytic anaemia

The body burden arising from inhalation and dermal exposure is about 1.3 mg/kg/day. A body burden of this magnitude is considered to give rise to concerns and thus **conclusion (iii)** is reached.

Repeated inhalation toxicity and carcinogenicity

The ratio between exposure to airborne naphthalene and the level at which minimal degenerative effects were observed in the rat nasal olfactory epithelium is 1. Given the severity of this endpoint, this MOS is not considered to be sufficient to allow for differences in toxicokinetics and toxicodynamics between and within species and thus **conclusion (iii)** is reached.

Professional use of naphthalene containing consumer products

Professional use of naphthalene is predicted to give rise to exposures similar to those for consumers where use is event-based, that is for the use of creosote products and for damp-proof laying. Exposure during a single event of the same duration will be the same as for consumers (unless PPE is used). However, the duration of each event and the frequency of events may be greater (for example, for a professional painter). Given that use will probably be more frequent comparison of exposure to a repeated dose inhalation LOAEL is appropriate and can be made in the risk characterisation, even though each event may be an acute exposure (this situation will be different for consumers, Section 4.1.3.3).

Daily exposure to coal tar soaps and shampoos is also predicted to be similar to that for consumers, given the worst-case assumptions made in the consumer assessment.

Haemolytic anaemia

Combined inhalation and dermal exposures for creosote products and damp-proof laying give rise to body burdens in the mg/kg range and thus raise concerns for human health, leading to **conclusion (iii)**. The professional use of coal tar soaps and shampoos is estimated to result in dermal exposure only, of up to 4.4 mg/day, leading to a body burden of 0.06 mg/kg/day. The exposure estimate is derived from worst-case assumptions and therefore is likely to overestimate exposure. Therefore, although a NOAEL cannot be derived for this endpoint, the body burden arising from this exposure scenario is considered to be sufficiently low to minimise concerns for this endpoint and **conclusion (ii)** is reached.

Repeated inhalation toxicity and carcinogenicity

The inhalation exposure arising from the professional use of creosote products is 2.91 mg/m^3, resulting in a MOS of 1.7. This MOS is not considered to be sufficient to allow for differences in toxicokinetics and toxicodynamics within and between species and therefore conclusion (iii) is reached. For inhalation exposure during damp-proof laying, exposures are clearly in excess of the rat LOAEL. Thus there are concerns for human health, and again conclusion (iii) is reached. For the professional use of coal tar soaps and shampoos, inhalation exposure is considered to be negligible and thus there is no concern for inhalation toxicity. Conclusion (ii) is therefore reached for this scenario.

Tables 4.12 and **4 .13** present the information as stated above for each occupational activity.

Table 4.12 Risk characterisation for inhalation exposure to workers

Activity leading to naphthalene exposure	Inhalation exposure (mg/m^3)	LOAEL (rat) (mg/m^3	Ratio LOAEL/ inhalation exposure (MOS)*	Conclusion for local respiratory effects	Conclusion for systemic effects
Manufacture of naphthalene	6.3	5	<1	iii	iii
Synthesis of phthalic anhydride	2	5	2.5	iii	iii
Other chemical syntheses	2	5	2.5	iii	iii
Blending of creosote	2	5	2.5	iii	iii
Bulk impregnation with creosote	8	5	<1	iii	iii
Packaging plants	8	5	<1	iii	iii
Brush application of creosote	2.9	5	2	iii	iii
Mothball manufacture	20 incl. particulates	5	<1	iii	iii
Manufacture of grinding wheels: with LEV	3.1	5	1	iii	iii
Without LEV	20	5	<1	iii	
Manufacture of coal tar paint /membranes	5	5	1	iii	iii
Professional use of consumer products					
Creosote products	2.91	5	<2	iii	iii
Damp-proof laying	76	5	<1	iii	iii
Coal tar soaps & shampoos	negligible	5	N/A	ii	ii

* It should be emphasised that considerable uncertainty surrounds the significance of these values in view of the possible substantial species differences between rats and humans for local nasal effects. Where conclusion (iii) is reached the uncertainty is considered to be outweighed by the small magnitude of the MoS.

Table 4.13 Risk characterisation for dermal exposures

Industry	Range (mg/cm²/day*)	Conclusion for systemic effects
Manufacture of naphthalene	0 to 0.1	iii
Manufacture of phthalic anhydride	0 to 0.1	iii
Blending and use of creosote - predicted exposure is the highest likely range and operators wear gloves, therefore exposure is likely to be at the bottom of this range.	highest range 0.025 to 0.25	iii
Manufacture of mothballs - exposure at the bottom of the range is likely at operators wear gloves.	0.1 to 1	iii
Manufacture of grinding wheels	0.3 to 1.5	iii
Manufacture and use of coal tar paints/membranes	0.001 to 0.02	iii
During the professional use of consumer products: Creosote products Damp-proof laying: Coal tar soaps and shampoos	 22 mg per 2 hour application 9 mg per 1 hour application 4.4 mg per day	 iii iii ii

*   Except where other units indicated

Overall occupational risk assessment conclusions:

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

There is no concern for inhalation effects or for haemolytic anaemia for workers in during the professional use of coal tar soaps and shampoo.

For all other occupational scenarios, conclusion (iii) applies:

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied should be taken into account.

4.1.3.3   Consumers

Consumer exposure may be acute, related to a specific event, use or application of a particular product, for example application of creosote or damp proofing; or repeated, for example the application of coal tar soaps and shampoos; or continuous, related to emissions from products in use over a period of time after their application. In addition, exposures should be viewed together, with the acute event-based exposure being peaks against a (usually) lower background of repeated or continuous exposure.

Haemolytic anaemia

In relation to haemolytic anaemia, the available data do not allow the identification of a NOAEL nor the dose-response characteristics for this endpoint. In the absence of such information, any significant body burden (values in the mg/kg range) is considered to give rise to concerns for

human health. The risks related to exposure of infants to textiles (clothing/bedding) which have been stored for long periods with naphthalene moth repellent raises significant concern. There is documented evidence for the development of severe haemolytic anaemia resulting from such use, although there is no quantitative information available on the level or duration of exposure to naphthalene in these cases. There are no consumer exposure scenarios for which both inhalation and dermal exposures are considered to be negligible, particularly when considering body burdens for infants, and therefore **conclusion (iii)** applies for all consumer exposure scenarios for this endpoint.

<u>Repeated inhalation toxicity and carcinogenicity</u>

Creosote application

The use of creosote products by consumers is assumed to be a single, rare event. The concerns for local respiratory effects are associated with long-term repeated exposure and therefore this health effect is not of concern for this exposure scenario. Therefore **conclusion (ii)** is reached.

Moth Repellent

The highest airborne concentration likely to be encountered by consumers on a regular daily basis from a single activity is from exposure to naphthalene moth repellent in a closed area (for example, when opening and using a drawer containing mothballs several times every day). This is higher than the LOAEL for nasal olfactory effects in rats. Similarly, moth repellent in the bedroom may give rise to exposure levels of the same order of magnitude of the rat LOAEL. The LOAEL is based upon local nasal effects from a 4-week study where exposure was only for a few hours, 5 days per week. The comparison of periodic exposure (short periods per day) is therefore justified. These MOSs give rise to concerns and **conclusion (iii)** is therefore reached.

Damp-proofing laying

Damp-proof laying by consumers is assumed to be a single, very rare event. The concerns for local respiratory effects are associated with long-term repeated exposure and therefore this health effect is not of concern for this exposure scenario. Therefore **conclusion (ii)** is reached.

Following damp-proofing

Comparison to a repeated exposure study is appropriate here since the scenario would involve someone living or working in treated premises. The MOS for this scenario is 11. This is considered to be insufficient to allow for differences in toxicokinetics and toxicodynamics within and between species. **Conclusion (iii)** is therefore reached.

Coal tar soaps and shampoos

Use of these products is considered to result in negligible inhalation exposure (Section 4.1.1.2.5). It is therefore concluded that there is no risk of local nasal effects. Consequently, **conclusion (ii)** is reached.

Table 4.14 and **Table 4.15** indicate the estimated airborne concentration of naphthalene from the various sources of exposure to naphthalene from consumer products or activities.

Table 4.14 Risk characterisation for consumer inhalation exposures of naphthalene – local respiratory effects

Consumer activity leading to naphthalene exposure	Inhalation Exposure (mg/m^3)	Ratio LOAEL[1] / inhalation exposure (MOS [#])	Conclusion for local nasal effects
Creosote application	2.91	not appropriate **	ii
Moth repellent (Bedroom)	0.82	6	**iii**
Moth repellent (closed areas)	12.00	0.4	**iii**
Damp-proofing (laying)	76 *	not appropriate **	ii
Damp-proofing (post laying)	0.45	11	**iii**
Coal tar soaps and shampoos	Negligible	negligible	ii

1) rat LOAEL 5 mg/m^3
[#] It should be emphasised that considerable uncertainty surrounds the significance of these values in view of the possible substantial species differences between rats and humans for local nasal effects.
* A peak inhalation exposure of 95 mg/m^3 was estimated by the SCIES model.
** Based on likely risk from acute exposure.

Table 4.15 Risk characterisation for consumer inhalation exposures of naphthalene – systemic effects (haemolytic anaemia)

Consumer activity leading to naphthalene exposure	Body burden (Inhalation exposure)	Body burden (Dermal exposure)	Total body burden	Conclusion on systemic effects (haemolytic anaemia)
Creosote application	7.6 mg/event	22 mg/event	29.6 mg/event	iii
Moth repellent (Bedroom)	22 mg/day	Negligible	22 mg/day	iii
Moth repellent (closed areas)	15.6 mg/day	0.4 mg/day	16 mg/day	iii
Damp-proofing (laying)	99 mg/day *	9 mg/event	108 mg/day	iii
Damp-proofing (post laying)	11.9 mg/day	Negligible	11.9 mg/day	iii
Coal tar soaps and shampoos	Negligible	4.4 mg/day	4.4 mg/day	iii

* A peak inhalation exposure of 95 mg/m^3 was estimated by the SCIES model.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied should be taken into account.

4.1.3.4   Humans exposed via the environment

4.1.3.4.1   Regional exposure

Haemolytic anaemia

In Section 4.1.1.3, the total daily human intake of naphthalene via the environment is estimated to be $6.55 \cdot 10^{-5}$ mg/kg/day for regional sources. It is not possible to quantitatively assess the risks for haemolytic anaemia, because of the absence of information on the NOAEL and dose-response characteristics for this endpoint in humans. However, given the extremely low level of

exposure for the regional scenario, the risk to human health is not considered to be of concern. Therefore **conclusion (ii)** is reached.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

Repeated inhalation toxicity and carcinogenicity

The environmental airborne levels of naphthalene (Section 4.1.1.3) are stated to be 0.137 μg/m^3 which is approximately 5 orders of magnitude lower than the 5 mg/m^3 naphthalene LOAEL for local respiratory effects in rats. Therefore there is no cause for concern for human health from this exposure and **conclusion (ii)** is reached.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

4.1.3.4.2   Local exposure

Haemolytic anaemia

Exposure in the locality of grinding wheel plants is estimated to result in a much higher daily intake (0.25 mg/kg/day) than that for regional exposure and it is not possible to conclude there is no concern for human health. Since a **conclusion (iii)** has been reached for environmental exposure for this use, it is anticipated that exposures will be reduced as a result of environmental risk reduction measures. It is therefore proposed that measured exposure data for this scenario be obtained following environmental risk reduction activity and this data used to reconsider the risk to humans via local environmental exposure. Therefore **conclusion (i)** is reached.

Conclusion (i)   There is a need for further information.

Further information is required on exposures in the locality of grinding wheel plants.

Repeated inhalation toxicity and carcinogenicity

The highest airborne levels are produced in the locality of a mothball manufacturing site, with estimated levels of 4 μg/m^3. This is 3 orders of magnitude lower than the 5 mg/m^3 LOAEL for local respiratory effects in rats. Therefore there is no cause for concern for human health from this exposure.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are already being applied.

4.1.3.5   Combined exposure

It can be seen from Section 4.1.1.4 (and **Table 4.16**) that it is possible to consider a combined exposure to naphthalene from all sources.

Table 4.16 Data comprising combined exposure scenario

Source of exposure		Exposure
Occupational source (mothball manufacture)		20 mg/m^3 (equivalent to about 3 mg/kg/day)
Indirect via the environment	regional local	$6.55 \cdot 10^{-5}$ mg/kg/day 0.25 mg/kg/day
As a consumer		0.77 mg/kg/day
Total	regional local	3.8 mg/kg/day 4.0 mg/kg/day

The total combined exposure incorporating local environmental exposure is estimated to be 4 mg/kg/day. Further occasional exposure could arise from using creosote products (29.6 mg per event) and damp proof laying (108 mg/event). It can be seen that if these events occupied 1 day per year for the use of creosote products and 1 day in 10 years for damp proof laying, the total consumer exposure would be increased slightly when calculated on a daily basis per kg of body weight. However, this additional exposure is considered to be negligible for risk assessment purposes.

Haemolytic anaemia

It is not possible to calculate MOSs for haemolytic anaemia for combined exposure because of the lack of dose response information and a NOAEL in humans. However, the combined exposure is too high to be considered negligible and thus there are concerns. Although further information on the exposures in the locality of grinding wheel plants has been requested, which may result in refinement of the risk characterisation for this scenario, nevertheless, the combined exposures arising from occupational and consumer exposure give rise to concern. Thus, **conclusion (iii)** is reached.

Repeated inhalation toxicity and carcinogenicity

There are concerns for most occupational and some consumer exposures. Therefore, there are concerns for these endpoints for combined exposure and hence the same conclusions as for workers and consumers apply.

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied should be taken into account.

4.2   HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

The physicochemical properties of naphthalene are well known and there is a general consensus as to the values of particular parameters.

Naphthalene has a low vapour pressure (at room temperature), is of low flammability and is not acidic or alkaline. In common with other organic materials it can be made to explode if finely divided and subjected to a source of ignition. However, given the level of control in manufacture and use the risks from such physicochemical properties are small.

Overall risk assessment conclusion for physicochemical properties:

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

5 RESULTS

There are thought to be 8 companies manufacturing naphthalene in the EU. All but one (using petroleum feedstock) of these uses coal tar as the source. Total production is estimated to be 200,000 tonnes/annum of which up to 25% is exported.

The major uses of naphthalene in the EU are in the production of phthalic anhydride (approximately 40,000 tonnes/annum), dyestuffs (46,000 tonnes/annum), alkylated naphthalene solvents (15,000 tonnes/annum), naphthalene sulphonic acids (approximately 24,000 tonnes per annum), mothballs (1,000 tonnes/annum), 2-naphthol (12,000 tonnes per annum), pyrotechnics (about 15 tonnes/annum) and the manufacture of grinding wheels (EU tonnage unknown). Other uses include the manufacture of some pesticides and miscellaneous use in the chemical and pharmaceutical industry (tonnages not known). Tonnage figures given are approximate and do not include naphthalene present in distillates used for the production of creosote (about 10,000 tonnes/annum) or in petroleum streams.

5.1 ENVIRONMENT

Releases of naphthalene to the environment from production and use are likely to occur mainly to the atmosphere, although releases to water and soil have been estimated using the Technical Guidance Document and site-specific information. The major release of naphthalene is to the atmosphere from vehicle emissions. Naphthalene releases can also occur through other fuel sources and during the production of aluminium. Naphthalene is readily volatilised from water and is removed from the atmosphere by reaction with hydroxyl radicals and other reactive species. Naphthalene will adsorb to soil and sediment to a moderate extent. The results of the only standardised screening test for inherent biodegradability for naphthalene suggest that naphthalene is not inherently biodegradable. However, numerous other "non-standard" biodegradation tests suggest that it is easily degraded under aerobic and denitrifying conditions. Naphthalene has therefore been considered to be inherently biodegradable in the risk assessment. Naphthalene is expected to show a moderate potential bioaccumulation but can be metabolised by a number of organisms. Measured levels of naphthalene are available for the major environmental compartments although a lot of the data are for contaminated sites.

Naphthalene is toxic to aquatic organisms and on the basis of evidence available is of low toxicity to terrestrial organisms.

For the aquatic compartment, naphthalene is not expected to cause any adverse effects in water, in a STP or in sediment at background levels. However, very high levels of naphthalene have been measured in water and sediment at contaminated sites and these levels may be expected to cause adverse effects on aquatic organisms. These sites are not contaminated through the production or use of naphthalene itself, but through other activities. PECs calculated from site-specific data for the manufacture of grinding wheels indicate that naphthalene may cause adverse effects in all compartments at the local level. The use of naphthalene in the manufacture of grinding wheels at another plant has indicated that there should be no adverse effects arising from its use at this location. Based on PECs calculated from site-specific data for the local environment at other sites where naphthalene is produced and used naphthalene is unlikely to cause adverse effects in water or sediment.

Based on the available toxicity data for terrestrial organisms, naphthalene is not expected to cause adverse effects in the terrestrial compartment as a result of its production or processing. It

may, however, cause adverse effects as a result of its use in the manufacture of grinding wheels. Naphthalene has been found at very high levels in soil as a result of indirect naphthalene contamination, for example on gasworks sites or hazardous waste dumps. At these levels it may cause adverse effects.

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion is based on site-specific data from one plant using naphthalene in the manufacture of grinding wheels. The calculations indicate that the use of naphthalene in this process is likely to cause adverse effects in water and sediment, to microorganisms in the wastewater treatment plant and in the soil compartment. Information from another plant using naphthalene in the manufacture of grinding wheels has indicated that there should be no adverse effects arising from its use at this location.

It is recognised that for both sediment and soil the PNEC used is derived from the surface water PNEC using the equilibrium partitioning method, and so in both of these cases the PNEC could be revised through toxicity testing. However, risks have only been identified for one site using naphthalene for this purpose. This site is developing plans to reduce releases to water, air and sludge so the rapporteur does not think it necessary to require such testing to be carried out. The planned risk management strategy will be based on the above conclusions to ensure that emissions to water (and hence sediment) and sludge (and hence the terrestrial compartment) are limited.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those that are being applied already.

This conclusion applies to release of naphthalene to the aquatic (including sediment) and terrestrial compartments from naphthalene production and its use as an intermediate, in pyrotechnics and in mothballs. There is also no risk to microorganisms from production or any of these uses.

5.2   HUMAN HEALTH

5.2.1   Human health (toxicity)

5.2.1.1   General

There are considerable difficulties in attempting to construct a human risk assessment for naphthalene due principally to significant species differences in response to this substance. The endpoints for which there are concerns are haemolytic anaemia, nasal effectsgenotoxicity and carcinogenicity.

It is clear that humans are susceptible to haemolytic anaemia following exposure to naphthalene via the oral, dermal or inhalation routes. In the occupational and consumer settings, it is unclear whether contemporary levels of exposure might be associated with the overt or sub-clinical development of haemolytic anaemia. No suitable investigation could be designed to clarify this situation or identify an NOAEL and so the possibility remains that these exposures to naphthalene may be of concern. Consequently conclusion (iii) is reached.

In relation to nasal effects, minimal damage to the rat nasal olfactory epithelium was observed even at the lowest exposure level of 5 mg/m^3 in a 28-day study. At higher and longer exposures, damage to the olfactory epithelium was more pronounced. However, there are significant anatomical and possibly also metabolic differences between the upper respiratory tract of the rat and humans. Therefore the relevance of the naphthalene-induced olfactory epithelium damage in rats for human health is uncertain, although there are no grounds to dismiss these findings in rats as not being of relevance.

For genotoxicity, naphthalene has given reproducible negative results in bacterial mutation assays, and was negative in an *in vitro* UDS assay. Two *in vitro* studies using CHO cells and human peripheral lymphocytes were negative for induction of SCE. It was however, found to be clastogenic in CHO cells in the presence but not the absence of S9. Naphthalene was also found to be negative in two *in vivo* bone-marrow micronucleus tests in mice and an *in vivo* liver UDS study. Overall, the balance of evidence indicates that naphthalene is non genotoxic.

For carcinogenicity, no conclusions can be drawn from the limited information available in humans. However, the carcinogenic potential of naphthalene has been well investigated in animals. In the most useful animal carcinogenicity study available, a 2-year inhalation study in the rat, an increase in the incidence of respiratory epithelial adenomas and olfactory epithelial neuroblastomas (a very rare tumour type) was observed in rats even with the lowest exposure concentration of 10 ppm (50 mg/m^3). In another study, female mice showed an increase in the incidence of benign lung tumours (alveolar/bronchiolar adenomas), to which this species is prone, following inhalation exposure to naphthalene. In view of the negative results obtained in the *in vivo* genotoxicity studies, naphthalene is considered to be non-genotoxic, and given that the neoplasia seen in mice and rats lie on a background of inflammatory changes in the tissues affected, they are considered to be a result of chronic tissue injury.

5.2.1.2   Workers

The industries where occupational exposure to naphthalene was found to be highest were mothball manufacture, the manufacture of grinding wheels and the use of creosote, in particular bulk impregnation. The blending of creosote and packaging into containers for do-it-yourself use are not considered to result in high exposures as it is contained in semi-enclosed plant with short-duration exposure during specific tasks. During manufacture and use in chemical synthesis it also used in semi-enclosed plant with exposure during tasks such as sampling and maintenance. The results of exposure from tar distillation plants and during the synthesis of phthalic anhydride, although limited, reflect the levels likely in these industries.

In relation to haemolytic anaemia, the available data do not allow the identification of a NOAEL nor the dose-response characteristics for this endpoint. In the absence of such information, it is considered that any significant body burden (values in the mg/kg range) arising from inhalation and/or dermal exposure gives rise to concern. Thus, conclusion (iii) is reached for this endpoint for all occupational exposure scenarios, except the professional use of coal tar soaps and shampoos.

In relation to local effects on the respiratory tract following repeated inhalation exposure, and carcinogenicity, there are concerns for human health from all occupational scenarios, except the professional use of coal tar soaps and shampoos.

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied should be taken into account.

There is no concern for inhalation effects or for haemolytic anaemia for workers during the professional use of coal tar soaps and shampoo.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

5.2.1.3   Consumers

Consumer exposure may be acute, related to a specific event, use or application of a particular product, for example application of creosote or damp proofing; repeated, for example the application of coal tar soaps and shampoos; or continuous, related to emissions from products in use over a period of time after their application.

In relation to haemolytic anaemia, the available data do not allow the identification of a NOAEL nor the dose-response characteristics for this endpoint. In the absence of such information, any significant body burden (values in the mg/kg range) is considered to give rise to concerns for human health. The risks related to exposure of infants to textiles (clothing/bedding) which have been stored for long periods with naphthalene moth repellent raises significant concern. There is documented evidence for the development of severe haemolytic anaemia resulting from such use, although there is no quantitative information available on the level or duration of exposure to naphthalene in these cases. There are no consumer exposure scenarios for which both inhalation and dermal exposures are considered to be negligible, particularly when considering body burdens for infants, and therefore conclusion (iii) applies for all consumer exposure scenarios for this endpoint.

In relation to local effects on the respiratory tract following repeated inhalation exposure, and carcinogenicity, all scenarios for which there is the potential for repeated inhalation exposure to naphthalene are considered to give rise to concern. Thus, conclusion (iii) applies to the consumer use of mothballs and to exposures arising after damp-proof laying.

Conclusion (iii)   There is a need for limiting the risks; risk reduction measure which are already being applied shall be taken into account

There are no concerns for local effects on the respiratory tract, or carcinogenicity, for those exposure scenarios that are single, rare events, nor for exposure scenarios that result in negligible inhalation exposure. Thus, conclusion (ii) is reached for creosote application, for damp-proof laying and for the use of coal tar soaps and shampoos.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

5.2.1.4   Humans exposed via the environment

5.2.1.4.1   Regional exposure

The environmental airborne levels and estimated total daily human intake of naphthalene arising from regional sources are very low. Although it is not possible to quantitatively assess the risks for haemolytic anaemia, given the extremely low level of exposure for the regional scenario, this exposure does not give rise to concern. Similarly, in relation to local effects on the respiratory

tract and carcinogenicity, these very low exposures do not give rise to concern. Therefore conclusion (ii) applies.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

5.2.1.4.2   Local exposure

Exposure in the locality of grinding wheel plants is estimated to result in a much higher daily intake (0.25 mg/kg/day) than that for regional exposure and it is not possible to conclude there is no concern for human health. Since a conclusion (iii) has been reached for environmental exposure for this use, it is anticipated that exposures will be reduced as a result of environmental risk reduction measures. It is therefore proposed that measured exposure data for this scenario be obtained following environmental risk reduction activity and this data used to reconsider the risk to humans via local environmental exposure. Therefore conclusion (i) is reached.

Conclusion (i)   There is a need for further information.

Further information is required on exposures in the locality of grinding wheel plants.

In relation to local effects on the respiratory tract, and carcinogenicity, the highest airborne levels are produced in the locality of a mothball manufacturing site, with estimated levels of $4 \mu g/m^3$. This is 3 orders of magnitude lower than the $5 mg/m^3$ LOAEL for local respiratory effects in rats. Therefore there is no cause for concern for human health and conclusion (ii) is reached.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

5.2.1.5   Combined exposure

The total combined exposure incorporating local environmental exposure is estimated to be 4 mg/kg/day. Further occasional exposure could arise from using creosote products (29.6 mg per event) and damp proof laying (108 mg/event). It can be seen that if these events occupied 1 day per year for the use of creosote products and 1 day in 10 years for damp proof laying, the total consumer exposure would be increased slightly when calculated on a daily basis per kg of body weight. However, this additional exposure is considered to be negligible for risk assessment purposes.

It is not possible to calculate MOSs for haemolytic anaemia for combined exposure because of the lack of dose response information and a NOAEL in humans. However, the combined exposure is too high to be considered negligible and thus there are concerns. Although further information on the exposures in the locality of grinding wheel plants has been requested, which may result in refinement of the risk characterisation for this scenario, nevertheless, the combined exposures arising from occupational and consumer exposure give rise to concern. Thus, conclusion (iii) is reached. There are also concerns for most occupational and some consumer exposures for repeated inhalation toxicity and carcinogenicity. Therefore, there are concerns for these endpoints for combined exposure and hence the same conclusions as for workers and consumers apply.

Conclusion (iii)   There is a need for limiting the risks; risk reduction measures which are already being applied should be taken into account.

5.2.2   Human health (risks from physico-chemical properties)

There are no significant risks from physicochemical properties.

Conclusion (ii)   There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.

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ABBREVIATIONS

ADI	Acceptable Daily Intake
AF	Assessment Factor
ASTM	American Society for Testing and Materials
ATP	Adaptation to Technical Progress
AUC	Area Under The Curve
B	Bioaccumulation
BBA	Biologische Bundesanstalt für Land- und Forstwirtschaft
BCF	Bioconcentration Factor
BMC	Benchmark Concentration
BMD	Benchmark Dose
BMF	Biomagnification Factor
BOD	Biochemical Oxygen Demand
bw	body weight / *Bw*, *bw*
C	Corrosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
CA	Chromosome Aberration
CA	Competent Authority
CAS	Chemical Abstract Services
CEC	Commission of the European Communities
CEN	European Standards Organisation / European Committee for Normalisation
CEPE	European Committee for Paints and Inks
CMR	Carcinogenic, Mutagenic and toxic to Reproduction
CNS	Central Nervous System
COD	Chemical Oxygen Demand
CSTEE	Scientific Committee for Toxicity, Ecotoxicity and the Environment (DG SANCO)
CT_{50}	Clearance Time, elimination or depuration expressed as half-life
d.wt	dry weight / dw
dfi	daily food intake
DG	Directorate General
DIN	Deutsche Industrie Norm (German norm)
DNA	DeoxyriboNucleic Acid
DOC	Dissolved Organic Carbon
DT50	Degradation half-life or period required for 50 percent dissipation / degradation
DT90	Period required for 90 percent dissipation / degradation
E	Explosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)

EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]
EbC50	Effect Concentration measured as 50% reduction in biomass growth in algae tests
EC	European Communities
EC10	Effect Concentration measured as 10% effect
EC50	median Effect Concentration
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
ECVAM	European Centre for the Validation of Alternative Methods
EDC	Endocrine Disrupting Chemical
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
ErC50	Effect Concentration measured as 50% reduction in growth rate in algae tests
ESD	Emission Scenario Document
EU	European Union
EUSES	European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]
F(+)	(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
FAO	Food and Agriculture Organisation of the United Nations
FELS	Fish Early Life Stage
foc	Organic carbon factor (compartment depending)
GLP	Good Laboratory Practice
HEDSET	EC/OECD Harmonised Electronic Data Set (for data collection of existing substances)
HELCOM	Helsinki Commission -Baltic Marine Environment Protection Commission
HPLC	High Pressure Liquid Chromatography
HPVC	High Production Volume Chemical (> 1000 t/a)
IARC	International Agency for Research on Cancer
IC	Industrial Category
IC50	median Immobilisation Concentration or median Inhibitory Concentration
ILO	International Labour Organisation
IPCS	International Programme on Chemical Safety
ISO	International Organisation for Standardisation
IUCLID	International Uniform Chemical Information Database (existing substances)
IUPAC	International Union for Pure and Applied Chemistry
JEFCA	Joint FAO/WHO Expert Committee on Food Additives

JMPR	Joint FAO/WHO Meeting on Pesticide Residues
Koc	organic carbon normalised distribution coefficient
Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient
L(E)C50	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC50	median Lethal Concentration
LD50	median Lethal Dose
LEV	Local Exhaust Ventilation
LLNA	Local Lymph Node Assay
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LOED	Lowest Observed Effect Dose
LOEL	Lowest Observed Effect Level
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
MITI	Ministry of International Trade and Industry, Japan
MOE	Margin of Exposure
MOS	Margin of Safety
MW	Molecular Weight
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC
NAEL	No Adverse Effect Level
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NOEC	No Observed Effect Concentration
NTP	National Toxicology Program (USA)
O	Oxidizing (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
OC	Organic Carbon content
OECD	Organisation for Economic Cooperation and Development
OEL	Occupational Exposure Limit
OJ	Official Journal
OSPAR	Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic
P	Persistent
PBT	Persistent, Bioaccumulative and Toxic

PBPK	Physiologically Based PharmacoKinetic modelling
PBTK	Physiologically Based ToxicoKinetic modelling
PEC	Predicted Environmental Concentration
pH	logarithm (to the base 10) (of the hydrogen ion concentration {H^+}
pKa	logarithm (to the base 10) of the acid dissociation constant
pKb	logarithm (to the base 10) of the base dissociation constant
PNEC	Predicted No Effect Concentration
POP	Persistent Organic Pollutant
PPE	Personal Protective Equipment
QSAR	(Quantitative) Structure-Activity Relationship
R phrases	Risk phrases according to Annex III of Directive 67/548/EEC
RAR	Risk Assessment Report
RC	Risk Characterisation
RfC	Reference Concentration
RfD	Reference Dose
RNA	RiboNucleic Acid
RPE	Respiratory Protective Equipment
RWC	Reasonable Worst Case
S phrases	Safety phrases according to Annex III of Directive 67/548/EEC
SAR	Structure-Activity Relationships
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange
SDS	Safety Data Sheet
SETAC	Society of Environmental Toxicology And Chemistry
SNIF	Summary Notification Interchange Format (new substances)
SSD	Species Sensitivity Distribution
STP	Sewage Treatment Plant
T(+)	(Very) Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
TDI	Tolerable Daily Intake
TG	Test Guideline
TGD	Technical Guidance Document
TNsG	Technical Notes for Guidance (for Biocides)
TNO	The Netherlands Organisation for Applied Scientific Research
ThOD	Theoritical Oxygen Demand
UC	Use Category
UDS	Unscheduled DNA Synthesis

UN	United Nations
UNEP	United Nations Environment Programme
US EPA	Environmental Protection Agency, USA
UV	Ultraviolet Region of Spectrum
UVCB	Unknown or Variable composition, Complex reaction products of Biological material
vB	very Bioaccumulative
VOC	Volatile Organic Compound
vP	very Persistent
vPvB	very Persistent and very Bioaccumulative
v/v	volume per volume ratio
w/w	weight per weight ratio
WHO	World Health Organization
WWTP	Waste Water Treatment Plant
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)

Appendix 1   EUSES Output

1   Production
2   Processing - use as an intermediate (phthalic anhydride production)
3   Processing - use as an intermediate (generic)
4   Formulation - pyrotechnics
5   Processing - grinding wheel production
6   Formulation - mothballs

Notes

For production (Use Pattern 1), the local emissions included are the largest release to water and to air from the site-specific data; these come from two different sites.

For processing (Use Pattern 2), the local emissions used are the largest releases from the site-specific data for phthalic anhydride production.

For processing (Use Pattern 3), the local emissions are estimated using an emission factor derived from the site-specific data for intermediate use.

Continental and regional emissions: the values for the continental and regional emissions for the individual Use Pattern steps have not been adjusted to match those in the Risk Assessment Report. The total emissions to the region and continent in EUSES have been over-written with the appropriate values from the Risk Assessment Report. These values include the indirect emissions of naphthalene as described in the report.

Euses Calculations can be viewed as part of the report at the website of the European Chemicals Bureau: http://ecb.jrc.it

Appendix 2   WHO Standard Protocol for field surveys of exposure to pesticides and its use in calculating naphthalene exposure

Pads are incorporated in a coverall and the amount of a chemical found on the pad is multiplied by a multiplication factor which takes into account the ratio of the pad area to the area of the body part it represents. Five volunteers were used in a variety of creosote brushing activities. The maximum level of naphthalene found on any type of pad was used in the following calculation.

Pad (1) Head - maximum level seen = 152 µg x multiplication factor (14.5) = 2,204 µg

Pad (2) Chest - maximum level seen = 244 µg x multiplication factor (46.2) = 11,273 µg

Pad (3) was used as a control.

Pad (4) Arm - maximum level seen = 222 µg x multiplication factor (20.2) = 4,484 µg

Pad (5) Thigh - maximum level seen = 492 µg x multiplication factor (36.4) = 17,909 µg

Pad (6) Lower leg - maximum level seen = 126 µg x multiplication factor (36.4) = 4,586 µg

Pad (7) Back - maximum level seen = 83 µg x multiplication factor (46.2) = 3,835 µg

Maximum amount seen on left hand gloves = nil

Maximum amount seen on right hand gloves = 48 µg

The amount of exposure as calculated is divided by 2 to take account of the unevenness of deposition in areas where direct exposure is less likely. The amount on the gloves is then added.

Total amount of exposure calculated over the whole body = 22.169 mg.

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                    naphthalene, Volume 33

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Environment and quality of life series

The report provides the comprehensive risk assessment of the substance naphthalene. It has been prepared by The United Kingdom in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to humans and the environment, laid down in Commission Regulation (EC) No. 1488/94.

The evaluation considers the emissions and the resulting exposure to the environment and the human populations in all life cycle steps. Following the exposure assessment, the environmental risk characterisation for each protection goal in the aquatic, terrestrial and atmospheric compartment has been determined. For human health the scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.

The environmental risk assessment for naphthalene concludes that there is concern for the aquatic and terrestrial compartments arising from the use of the substance in the manufacture of grinding wheels.

The human health risk assessment for naphthalene concludes that there is concern for workers and consumers. For humans exposed via the environment there is a need for further information to adequately characterise the risk in the locality of grinding wheel manufacturing plants.

The conclusions of this report will lead to risk reduction measures to be proposed by the Commissions committee on risk reduction strategies set up in support of Council Regulation (EEC) N. 793/93.

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