European Union Risk Assessment Report

ANTHRACENE

CAS No: 120-12-7
EINECS No: 204-371-1

RISK ASSESSMENT

GENERAL NOTE

This document contains two different reports:

- **Part I Environment** (Final approved version awaiting for publication) – pages 2-129
- **Volume 78 Part II Human Health** (Publication: EUR 22237 EN) – pages 130-245

European Union Risk Assessment Report

ANTHRACENE

CAS No: 120-12-7

EINECS No: 240-371-1

RISK ASSESSMENT

FINAL APPROVED VERSION

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ANTHRACENE

CAS No: 120-12-7

EINECS No: 204-371-1

RISK ASSESSMENT

April 2008

Greece

FINAL APPROVED VERSION

Rapporteur for the risk assessment of Anthracene is Greece

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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 Health and Environmental Risks (SCHER) 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 and confirmed in the Johannesburg Declaration on Sustainable Development at the World Summit on Sustainable Development, held in Johannesburg, South Africa in 2002.

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.

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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[4]

CAS Number: 120-12-7
EINECS Number: 240-12-7
IUPAC Name: Anthracene

PBT Assessment`

Anthracene meets the vP, vB and T criteria and hence is considered as a vPvB and PBT substance. Therefore, there is a need for limiting the risks; risk reduction measures that are already being applied shall be taken into account. (Conclusion iii).

Environment

Aquatic compartment (incl. sediment)

Conclusion (ii) applies to production of anthracene. No further information is considered nessesary.

Conclusion (ii) applies for production of pyrotechnics.

Sediment

Conclusion (ii) applies to production of anthracene. No further information is considered nessesary.

Conclusion (ii) applies for production of pyrotechnics.

Waste water treatment plants

Conclusion (ii) is reached for the functioning of waste water treatment plants based on a qualitative evaluation. There are insufficient data available to obtain $PNEC_{micro\text{-}organism}$. However, based on the assumption that the PNEC has to be in the µg/l range or higher, it is not expected that anthracene will pose a risk for micro-organisms in a STP.

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

[4] Conclusion (i) There is a need for further information and/or testing.
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 being applied already.
Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

Terrestrial compartment

Conclusion (ii) applies to production of anthracene. No further information is considered nessesary.

Conclusion (ii) applies for production of pyrotechnics.

Atmospheric compartment

Conclusion (ii) is reached for the atmospheric compartment based on a qualitative evaluation. There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.

Secondary poisoning

In the absence of sufficient toxicity data, a PNECoral can not be derived. Therefore, a realistic quantitative risk assessment for secondary poisoning can not be made. It is possible to refine the assessment by generating further information to address the uncertainties in the above mentioned endpoint but, given the findings for the PBT/vPvB assessment, this is considered a low priority at this stage.

Human health

See separate report.

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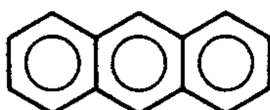
1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS Number: 120-12-7
EINECS Number: 204-371-1
IUPAC Name: Anthracene
Molecular formula: $C_{14}H_{10}$
Structural formula:



Molecular weight: 178.24
Synonyms: Paranaphthalene, p-naphthalene, green oil, tetra olive

1.2 PURITY/IMPURITIES, ADDITIVES

Technical grade anthracene is approximately 97% pure, the main impurities being the following: phenanthrene (1.0%); carbazole (1.0%); naphthothiophene (0.4%); dibenzo[b,c]thiophene (0.3%); acridine (0.2%); acetophenone (0.4%).

Higher-grade anthracene can be obtained by treatment with air to oxidize 9,10-dihydroanthracene to anthracene, and by further recrystallisation, leading to a product with a low nitrogen (carbazole) content. Anthracene can generally be further separated from the higher boiling carbazole (b.p. 354°) by further distillation with a lower-boiling hydrocarbon fraction as reflux medium, or by azeotropic distillation with ethylene glycol. Azeotropic distillation is also used to separate the anthracene-accompanying tetracene (naphthacene) and to obtain very pure anthracene, which is used for **scintillation counting.**

1.3 PHYSICO-CHEMICAL PROPERTIES

Anthracene is a colorless crystalline solid, with violet fluorescence.

Melting point
A value of 218°C is reported in IUCLID (1995) [Lang and Eigen, 1967], in Merck Index (1983), in Ulmann's Encyclopaedia [Collin and Höke, 1985] and in Clar (1964). A closely similar value (217°C) is reported in ECDIN as well as in Sax (1975). IUCLID (1995) also reports lower values of 214°C [Hausigk and Koelling, 1968] and 216.4^{o}C [Karcher et al., 1985]. The value of 216.4^{o}C is adopted in the present.

Boiling point
Two values (340°C and 342°C) are reported in IUCLID (1995) and in several other publications (e.g. 340°C in Lang and Eigen, 1967; 342 ^{o}C, Lax and Synowietz, 1964). The value of 340°C is reported in CRC (1987) as "corrected", while in ECDIN the value of 339.9^{o}C is reported as the value at 760 mm Hg. The value of 342°C is adopted in the present Report.

Relative density
IUCLID (1985) gives values of 1.252 (25/4°C) [Collin and Höke, 1985] and 1.283 (25/4°C) [Lide, 1991]. The same value is given in Lax and Synowietz (1964), while a value of 1.25 (27/4°C) is also reported in ECDIN and in Dean (1979). The value of 1.283 (25/4°C) is adopted in the present Report.

Vapour pressure
Three values are given in IUCLID (1995). Sonnefeld et al. (1983) obtained a value of 8.0×10^{-4} Pa (25°C) using a method closely similar to one of those recommended by Dir. 67/548/EC. Their system was based on dynamic coupled-column liquid chromatography that is direct coupling of a gas saturation system to HPLC, used at the ambient temperature range. Commercially available anthracene was employed (purity > 98%). However, through the provisions of the system (prewashing/ conditioning of the saturation column) the measurements concerned pure anthracene.
Jandris and Forcė (1983) measured the vapour pressure of anthracene by analyzing the vapour concentration using laser-induced molecular fluorescence. The material employed was 99.9% pure. Fluorescence intensity data over the range 25-150°C were obtained and employed to calculate first the vapour pressure at 101°C, which was then extrapolated to 25°C, yielding a value of 9.1×10^{-9} atm (9.2×10^{-4} Pa).
A third value of 0.026 Pa, is reported in IUCLID (1995) citing a secondary reference [Neff, 1979] where no information on the method employed is given.

In the present Report, the value of 9.4×10^{-4} Pa (25°C) is adopted, which is determined by gas saturation/effusion method (Mackay 1992).

Water solubility
Different values of water solubility of anthracene have been reported. The value of 0.041 mg/l (25°C) [Schwartz, 1977] reported in IUCLID (1995), has been determined at 25°C by direct analysis of a saturated solution using UV and fluorescence analytical methodology. Other values reported in IUCLID (1995) are 0.032 mg/l (20°C) [Hashimoto et al., 1984), 0.037 mg/l (22 °C) [May et al., 1983] and 0.044 mg/l (25.3 °C, but using tap water) [Whitehouse, 1984]. A value of 0.073 mg/l (25°C) [Mackay et al., 1977], measured fluorimetrically after extraction of a saturated solution with cyclohexane, was not selected as it was reported in only one study, while the selected value is close to those reported by all other studies.
Values of the solubility of anthracene in saline water are also reported in IUCLID (1995), including 0.0324 mg/l (25.3°C, salinity of 36.5 o/oo; measured by dynamic column liquid chromatography) [Whitehouse, 1984] and 0.021 mg/l (20°C, salinity of 35 o/oo; measured after extraction of a saturated solution with benzene) [Hashimoto, 1984].
Finally, a value of 0.047 mg/l (25^0C) has been determined using generated column methods (Mackay 1992). In the present Report, this value of 0.047mg/l (25°C) is adopted.

Solubility in other solvents.
Anthracene is reported to be slightly soluble in benzene, chloroform and carbon bisulfide and less soluble in ether and alcohol, but no quantitative information in given [Collin and Höke, 1985].
Partition coefficient
IUCLID (1995) gives a compilation of measured and calculated values for $\log P_{ow}$ in the range of 3.45-4.8, as reported by Sangster (1989). Among the values reported, two, 4.50 and 4.54, were based on direct measurement (shake-flask), the latter in particular being measured at 25°C [Karickhoff et al., 1979]. Yoshida et al. (1983) derived by calculation, using equations

related to molar refraction, the values 4.21 and 4.71, and compared them to a measured value of 4.50. In another study, Geyer et al., (1984) a value of 4.54 was reported, derived from measurements carried out in accordance with the Guidelines of Directive 67/548/EC (OECD method 107). The value of 4.68 (log value) is adopted in the present Report, as it is based on slow stirring methods using average values (Mackay 1992).

Henry constant
The selected value of 4.3 (Pa m^3/mol at 25^0C) is based on batch/gas stripping/wetted-wall column, (Mackay 1992).

Flash point
A single value of 121^oC (close cup) is given in IUCLID (1995), derived from Sax (1992).

Autoflammability
A single value of 540^oC at 1,013 hPa (1 Atm.) is given in IUCLID (1995), derived from Nabert and Schoen (1963).

Explosivity
Information given in IUCLID (1995), derived from Nabert and Schoen (1963), indicates a low explosion limit of 45 g/m^3 (20^oC, 1 atm) or 0.6% by volume, while no high explosion limit is given. It is also indicated that dust is possibly explosive.

Oxidising properties
Anthracene is not an oxidizing agent.

Table 1.1: Summary of physico-chemical properties

Property	Value	References
Physical state	Colorless crystalline solid with violet fluorescence	
Molecural weight	178.2	Mackay, 1992
Melting point (^{0}C)	216.4	Mackay, 1992
Boiling point (^{0}C)	342	Mackay, 1992
Relative density	1.283	Mackay, 1992
Vapour pressure (Pa, 25^oC)	$9.4x10^{-4}$	Sonnefeld et al. (1983)
Water solubility (mg/l)	0.047	Mackay, 1992
Partition coefficient n-octanol/water (log value)	4.68	Mackay, 1992
Flash point (^{0}C)	121	IUCLID, 1995
Autoflammability (^{0}C) at 1013 hPa (1 Atm.)	**540**	IUCLID, 1995
Explosive properties (g/m^3)	45	at 20^oC, 1 atm, Nabert and Schoen,1963
Oxidizing properties	not an oxidizing agent	
Henry's constant (Pa. m^3/mol at 25 ^{o}C)	4.3	Mackay, 1992

1.4 CLASSIFICATION

1.4.1 Current classification

-

1.4.2 Proposed classification

Anthracene had not been classified in the context of Directive 67/548/ concerning dangerous subtances.
Based on the toxicity with fish, invertebrates and algae and the lack of biodegradability in standard test systems, the following classification and labeling, according the annex I of Directive 67/548/ was proposed and agreed by the TC C& L for environmental effects **:**
N, R50 /R53 (very toxic to aquatic organisms /may cause long-term adverse effects in the aquatic environment), with the following specific concentration limits.

Classification	**Toxicity**
N;R50-53	L(E)C50 < 1mg/l **0.001 < L(E)50 < 0.01mg/l**
	$C_n \geq 0.25$ % : **N;R50-53** 0.025 % $\leq C_n < 0, 25$ %: **N;R51-53** 0.0025 % $\leq C_n < 0,025$ % : **N;R52-53** $C_n < 0,0025$ % : N.C.

An M-factor of 100 has also been agreed by the TC C & L env.

Safety phrases :
S60 (this material and its contain must be disposed of as hazardous waste),
S61 (avoid release to the environment. Refer to special instructions/Safety data sheet.).

As far as the human health is concerned, the classification and labelling proposed on the basis of the current RAR is :
Xi , R38 (irritating to skin),
S37(wear suitable gloves).

In November 2005 the human health (HH) Classification and labelling Group (TC C&L) in the context of directive 67/548/EEC agreed that the rapporteur Classification proposal Xi; R38 should not be applied to reflect photo irritancy. The TC C & L in March 2006 agreed that this substance should also be assigned the new Note (X) that would be generalized for photo irritation and photo sensitization. The Note X should be discussed at the next meeting (TC C&L H H) on the basis of the NL proposal. The classification of the substance should then be concluded on the basis of this discussion.

2 GENERAL INFORMATION ON EXPOSURE

2.1 GENERAL COMMENTS ON RELEASES AND EXPOSURE

Releases of and exposure to anthracene can occur during the production and use of anthracene and anthracene-containing products. Anthracene is produced from light anthracene oil (a fraction of coal tar distillation) and its use is restricted to the industrial production of an aldehyde (which ceased operating in the EU as of 2003), the manufacture of pyrotechnics and in scientific research laboratories.

Several products containing anthracene as part of complex mixtures but not involving addition of isolated commercial anthracene, such as coal tar itself, coal tar-containing products (paints, waterproof membranes etc.) and creosote, are outside the scope of the present Report (Council Regulation 793/93). Nevertheless, all these products may affect, through anthracene releases during their production and use, the background environment concentrations.

Background environmental concentrations of anthracene can also be affected by releases arising from incomplete combustion of organic matter, as occurring during fossil fuel combustion or in various workplaces (e.g. carbon anode/graphite, silicon carbide, aluminium, iron and steel production plants and others).

A site in the UK recently announced that it produces crude Anthracene. It supplies CAS number 90640-81-6 which refers to Anthracene oil, anthracene paste; Anthracene oil fraction. The anthracene side stream is an anthracene rich oil fraction produced during the distillation of coal tar. The product does not therefore fall under the ESR risk assessment of anthracene which considers only pure anthracene, supplied under CAS Number 120-12-7. The site produces 40% anthracene which is exported to China and 20% anthracene which is blended with other oils to produce Carbon Black feedstock sold in Europe. The remaining balance of oil from the plant is blended into creosote.

There is a Czech anthracene producer with a maximum capacity of 2.450 metric tons per year. Last years, the real production was from 1033 to 1885 metric tons per year. This production occurred outside the EU of 15 Member-States and therefore it does not fall within the scope of the present RAR.It may, however, already be considered in the risk assessment of Coal Tar Pitch (EC, 2008).

2.2 ANTHRACENE PRODUCTION

Anthracene is present in coal tar, from where it can be recovered efficiently. Hence recovery from coal tar and, in particular, from anthracene oil (one of coal tar’s distillate fractions), constitutes the basis for the industrial production of anthracene. In view of the importance of coal tar distillation in anthracene production, and because the production and use of certain coal tar distillation products constitute sources of human exposure to anthracene, coal tar and its distillation products are discussed in some detail below.

The recent RAR studies the effects of the production of anthracene. All other data are for informative purposes.

2.2.1 Coal tars

Coal tars are by-products of the destructive distillation of coal, called carbonization or coking. The composition and properties of a coal tar depend mainly on the temperature of carbonization and, to a lesser extent, on the nature of the coal used as feedstock. Coal tars are complex mixtures of hydrocarbons, phenols and heterocyclic (oxygen-, sulphur- and nitrogen-containing) compounds. Probably as many as 10,000 compounds are actually present in coal-tars, of which over 400 have been identified. Two main classes of coal tars are recognised, depending on the temperature of carbonization, namely high-temperature (>700°C) and low-temperature coal-tars (<700°C), which differ significantly in their chemical composition. The anthracene content of high-temperature coal tars (low-temperature ones contain negligible amounts of anthracene) has been reported by several sources as shown in Table 2.1.

Table 2.1: Anthracene content of different coal tar types

Mc Neil, 1983		Kleffer et al., 1981		Novotny et al., 1981		Marlich & Leukevitch, 1978		Collin & Höke, 1985	
coal tar type	Content	coal tar type	content	coal tar type	content	coal tar type	content	coal tar type	content
coke-oven (UK)	1%	high tempe-rature	1.5%	coke-oven	5.5%	high tempe-rature	1.1%	high tempe-rature	1.5%
coke-oven (FRG)	1.8%								
coke-oven (US)	0.75%								
CVR (UK)	0.26%								
low tempe-rature	0.06%								
Lurgi	0.32%								

Based on the above figures as well as figures from other sources, a value of 1.5% is adopted in the present Report as a representative level for the anthracene content of high temperature coal tars.

2.2.1.1 Coal-tar distillation products (tar oils)

Coal tar distillation is conducted at 10 distillation plants in Europe (1 each in Germany, Belgium, France, the Netherlands, Italy, Denmark, and 2 each in the U.K. and Spain) [Betts, pers. commun., 2000]. The amounts of coal-tar produced and distilled in the EU during 1997-1999 are given in Table 2.2 (the 1998 and 1999 production figures do not include data for Germany) [Betts, pers. commun., 2000].

Table 2.2: Volumes of coal-tar production and distillation in the European Union

Year	produced (tonnes)	distilled (tonnes)
1997	1,266,000	1,799,000
1998	1,109,000	1,810,000
1999	825,000	1,767,000

The important common distillation fractions and residues obtained from coal tar by high-temperature processes, including light anthracene oil (the starting material for anthracene production), are shown in Table 2.3.
Anthracene oil (distillation temperature 300-450°C) is a semisolid, greenish brown crystalline material. It is obtained in two fractions from the primary distillation of coal tars. The lower-boiling fraction (light anthracene oil) has a high content of phenanthrene, anthracene and carbazole. The higher-boiling fraction (heavy anthracene oil) has a high content of fluoranthene and pyrene [IARC, 1983a; Collin & Zander, 1982]. Light anthracene oil, which makes up about 20% of coal tar and usually has an anthracene content of 6-7%, is used as the starting material for the production of pure anthracene [Franck & Stadelhofer, 1987; Collin & Höke, 1985]. The value of 6% as the anthracene concentration in light anthracene oil is adopted in the present Report and taken forward to risk characterization.

Table 2.3: Primary distillation fractions and residues obtained from high temperature coal tar distillation

	coal tar fraction	dist. range	main components
a	light oil/overheads	<180°C	Mainly toluene, xylene, benzene and indene-naphthalene
b	carbolic oil	180-205°C	Mainly higher alkylbenzenes, phenol and alkylphenols, indene, xylene and naphthalene
c	Naphthalene oil	200-230°C	Mainly naphthalene and methylnaphthalene
d	creosote oil/wash oil	230-290°C	Mainly alkynaphthalenes, naphthalene, diphenylacena-phthene, fluorene, plus some higher phenols
e	light anthracene oil	260-310°C	Mainly anthracene, phenan-threne and carbazole, with small amounts of fluorene and pyrene; this fraction is used for anthracene recovery
f	heavy anthracene oil/ base oil	>310°C	Mainly polynuclear aromatic compounds of higher molecular weight

g	medium-soft pitch	residue	40-50% polynuclear aromatic compounds of 4-7 rings

2.2.1.2 Creosote

While according to IARC the term “creosote oil” refers to one of the coal-tar distillation fractions (distillation temperature 230-290°C), this term is commonly used to describe the material used for timber impregnation and is made up of a blend of several coal-tar distillation fractions. It is emphasised here that creosote blending does not involve the addition of pure anthracene.

Creosote, in its best known and most commonly used form, is a mid-heavy distillate of coal tar, boiling range 200-400°C, with varying composition because of the different blending procedures employed in its production. It is generally described as a yellow-dark-green-brown oily liquid consisting of aromatic hydrocarbons (including anthracene, naphthalene and phenanthrene derivatives), some tar acids (phenol, cresols and xylenols) and tar bases (e.g. pyridine and lutidine derivatives) [IARC, 1983a; McNeill, 1983]. About 160-200 compounds are present in creosote [IARC, 1983a; Nestler, 1974]. However, only a limited number (about 30) have been identified, 20 of which are present at levels exceeding 1% and make up the major portion of creosote. Polycyclic aromatic hydrocarbons (mostly unsubstituted) generally account for 75-85% of creosote [Lorenz and Gjovik, 1972]. The maximum benzo[a]pyrene content of WEI Grade A creosote is 500 ppm and that of WEI Grades B and C 50 ppm [WEI, pers. commun., 2000].

The anthracene content of some creosotes is given in Table 2.4. The anthracene content of “impregnation oil” is said in additional reports to vary from as low as 0.16% to as high as 7% [Lehman et al., 1984; Danish EPA, 1995; Ingram, 1982]. In the EU, creosote is generally manufactured to grades specified by the West European Institute for Wood Preservation (WEI). Three creosote types are specified, based on their density, the distillation ranges and other physicochemical characteristics, without any reference to their anthracene content.

Table 2.4: Anthracene content of some creosotes

Component	Creosote type			
	A probably a mixture (classes 3, 4, 5 and 6) [Staesse, 1954]	B classes 3, 4, 5 and 6 [Staesse, 1954]	C average of 9 creosote oil samples (class 3) [Nestler, 1974, Staesse, 1954]	D typical creosote (classes 3, 4, 5 and 6) used for the impregnation of railway sleepers [Andersson et al., 1983]
Anthracene	2.0	*	1.5	7.0

* the anthracene content is included in that of phenanthrene (17.4%)

Recent information gives the following picture regarding the anthracene content of creosote preparations:

a) Analyses carried out by WEI for type A and B creosotes show anthracene contents of 1.1% and 1.5%, respectively [Betts, pers. commun., 2000]. According to the same source, other analyses gave 1.7% (AWPA P1-65) and 1.3% (P1/P13 Industry composite Test Material).

b) Analyses carried out by the Swiss independent institution EMPA indicate the following concentrations: For two creosote companies, anthracene concentrations of 0.9% and 0.8% (WEI type A), 0.55% and 0.7% (WEI type B) and 1.36% and 1.05% (WEI type C). These figures are in broad agreement with the results of analysis carried out by one of the above companies indicating contents of 0.7% (WEI type A), 0.45% (WEI type B) and 0.8% (WEI type C) [Höke, pers. commun., 2000].

c) The UK Health and Safety Executive have measured the anthracene content of 3 creosote products available to amateur users and found them to depend on the colour of the product as follows [HSE ECOS, unpublished]:

Golden Brown 0.07%

Dark Brown 9.9%

Dark Brown 11.6%

Finally, anthracene concentrations in creosote lie at a maximum of 1.5%. A higher content would create problems with crystallization and workability of the oil [WEI, pers. commun., 2000].

In the present Report the typical anthracene content of commercial creosote is assumed to be 1.5%.

2.2.2 Anthracene production from coal tar

According to information provided by the only European manufacturer of anthracene (operating in Germany), the procedure employed for its production involves, as feedstock, light anthracene oil containing about 6% anthracene [Frank & Stadelhofer, 1987]. In the present Report 6% is taken as the anthracene content of light anthracene oil used for the production of anthracene. Table 2.5 below shows a typical composition of this fraction.

Table 2.5: Composition of light anthracene oil (%) (from Frank & Stadelhofer, 1987).

component	%
Dimethynaphthalenes	0.7
Acenaphthenes	3.1
Dibenzofuzan	4.0
Fluorene	7.7
Methylfluorene	10.6
Dibenzothiophene	1.9
Anthracene	5.8
Phenanthrene	18.8
Carbazole	3.8
Methyphenanthrenes	12.3

Fluoranthenes	8.0
Pyrene	4 1
Other aromatics	19.2

Anthracene is recovered from anthracene oil by the combined application of crystallization and distillation (vacuum distillation), while the product is further refined by recrystallisation. Figure 2.1 shows the flow diagram for the recovery of pure anthracene from anthracene oil.
The first stage in the production of anthracene is the recovery of a concentrate (25-30% anthracene content) by crystallization, which can be carried out in two stages to increase the yield. The final crystallisate, known as "anthracene cake" or crude anthracene, is generally concentrated to around 50% by vacuum distillation. Subsequent refining to yield anthracene of purity over 95% is normally achieved by recrystallisation. The quality of the anthracene thus obtained is of technical grade with a typical composition shown in Table 2.6.

Figure 2.1. Flow diagram for the recovery of anthracene from anthracene oil
(from Frank & Stadelhofer, 1987)

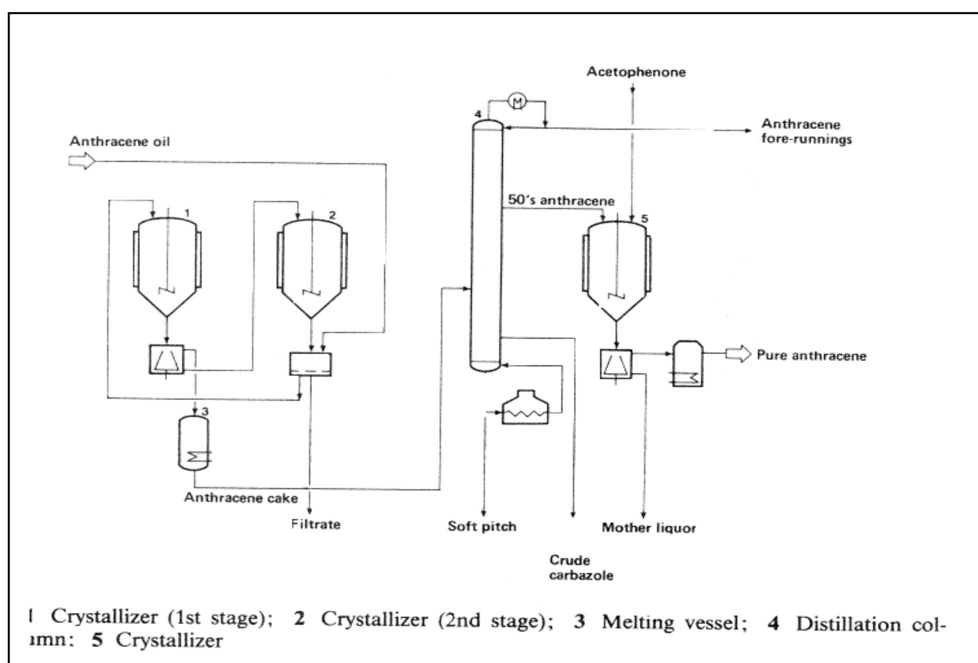


Table 2.7 shows the amounts of anthracene produced in the EU during recent years [Höke, pers. commun., 2000; 2002]. These data indicate that production of pure anthracene dropped to around 1,000 tpa or less during recent years. Approximately 99% of the 1999 production was exported to outside the EU. No importation of anthracene into the EU appears to take place.

Table 2.6: Typical composition of technical grade anthracene (%)

component	%
Anthracene	≥97.0
Phenanthrene	≤1.0
Carbazole	≤1.0
Naphthothiophene	≤0.4
Dibenzo[b,c]thiophene	≤0.3

Acridine	≤0.2
Acetophenone	≤0.4

Table 2.7: Anthracene production volumes in Europe

Year	production (tonnes)*					
	Pure	40% (liquid) (approx.)	50% (liquid) (approx.)	50% (solid) (approx.)	total 50% (approx.)	total crude (approx.)
1987	8,000					
1989	7,500					
1990	1,300					
1991	1,500	-	5,600	-		5,600
1992	3,600	40	420	280	700	740
1993	2,100	600	2,080	1,480	3,550	4,150
1994	2,100	400	2,890	2,490	5,380	5,780
1995	1,900	70	899	480	1,280	1,350
1996	1,800	-	1,030	-	1,030	1,030
1997	1,700	-	780	-	780	780
1998	1,600	-	1,400	-	1,400	1400
1999	550	-	930	660	1590	1590
2000	1,190	-	600	1330	1930	1930
2001	1,150	-	-	400	400	400

* The crude anthracene figures refer to amounts in addition to those of the pure product [Höke, pers. commun., 2000; 2002].

2.3 USES OF ANTHRACENE AND ANTHRACENE-CONTAINING PRODUCTS

Until recently, the main uses of anthracene, which could give rise to releases, were two specific types of chemical synthesis. As discussed in the next paragraph, these processes have stopped in recent years. The only known remaining uses of anthracene relates to the use of small amounts of anthracene in pyrotechnics and in scientific research laboratories.

Other products containing anthracene are creosote, tar paints, waterproof membranes and related products containing coal tar distillates. These products contain anthracene as part of a complex mixture and do not involve addition of pure anthracene.

Another potential source of exposure to anthracene, which no longer exists and therefore does not need to be considered here, is related to the use of anthracene oil and coal tar in cosmetics products such as soaps, lotions, oils, shampoos and gels. The use of anthracene oil for such purposes was prohibited by Directive 76/768/EC, while more recently Directive 97/45/EC prohibited the use of coal tar in these products.

2.3.1 Uses of anthracene

According the latest information available, practically all consumption of anthracene in the EU, which until recently was carried out by 2 main industrial users, has now stopped and

almost all anthracene produced in Europe is exported. Figures on anthracene consumption during recent years users are shown in Table 2.8.

Most of the material used by one of these users (consumer 1, Table 2.8) went to the manufacture of anthraquinone. However, production of anthraquinone at this plant has stopped since the end of 1998. Consumer 2 used the quantities of anthracene indicated below for chemical synthesis of anthracene-9-aldehyde. According to information recently provided by this consumer, as of 2003 this use of anthracene ceased and anthracene-9-aldehyde will no longer be produced [Höke, pers. commun., 2003]. Consequently neither of these processes need be further discussed in the present Report. However, in view of the use of anthracene in synthesis of anthracene-9-adelhyde until very recently, this process will be considered in following Sections for illustrative purposes.

Table 2.8: Anthracene consumption volumes in Europe

Year	Consumption within EU (tonnes)			Production (pure anthracene; tonnes, from Table 2.7)
	Consumer 1	Consumer 2	Total	
1995	1,937	n.d.	1,937	1,900
1996	1,330	n.d.	1,330	1,800
1997	1,679	19.2	1,699	1,700
1998	1,675	13.5	1,689	1,600
1999	none	6.8	6.8	550
2000	none	none	none	1,190
2001	none	none	none	1,150
2002	none	7.0	7.0	no data available

Small quantities (approximately 0.2 tonnes per year) of anthracene are sold to a company operating in the EU for the manufacture of pyrotechnics.

Small amounts of anthracene are also used in scientific research laboratories. In accordance with Council Directive 79/831, this type of use does not come under the terms of the present Report and for this reason it will not be discussed further.
Small quantities (approximately 0.2 tonnes per year) of anthracene are sold to a company operating in the EU for the manufacture of pyrotechnics.

In conclusion, practically no use of anthracene takes place in Europe. Consequently only the limited use of anthracene for the production of pyrotechnics will be considered in the context of this Report.

2.3.2 Uses of anthracene-containing products (indirect uses)

2.3.2.1 Uses of creosote

Creosote is used almost exclusively in wood impregnation. Recent estimates put the amount of creosote used in the EU at approximately 107,000 tpa [Sorgo, 1996]. There are 9 bulk wood impregnation plants in the EU [WEI, pers. commun., 2000].

The marketing and use of creosote in the EU are strictly regulated by Directive 2001/90 (adapting to technical process Annex I of Directive 76/769 concerning the restriction of the marketing and use of certain dangerous substances and preparations). This Directive does not permit the use of creosote for wood treatment. By derogation, it permits the use of special creosote (containing <0.005% benzo[a]pyrene and <3% water-extractable phenols) only in industrial installations or by professionals for in situ re-treatment. In addition, this kind of creosote may be place on the market only in containers of capacity ≥20 l, and may not be sold to consumers. Apart from other labelling restrictions, the packaging of this creosote should mention “For use in industrial installations or professional treatment only”. It is concluded from this that consumers cannot use creosote.

2.3.2.2 Tar paints, waterproof membranes and related products containing coal tar distillates

Coal tar and its distillates are used in some specialist paints, damp-proofing materials, waterproof membranes, coal tar epoxy paints and coal tar poly-urethane sealers. It is understood that tar paints are no longer used in Germany and that Scandinavian countries are moving away from them. Coal tar paints usually contain 0.5% anthracene, while the anthracene content of other products seems to be below 0.5% [IARC, 1983a].

No information on the number of plants or the production volumes of these products in Europe is available.

2.4 RELEASES OF ANTHRACENE DURING COMBUSTION AND RELATED INDUSTRIAL PROCESSES

Anthracene is produced during incomplete combustion of organic matter. Therefore it is emitted as a component of vehicle exhaust gases, as well as during various industrial processes such as carbon anode/graphite, silicon carbide, aluminium, iron and steel production plants and others.

2.5 SUMMARY OF INFORMATION ON RELEASES DURING PRODUCTION AND USE OF ANTHRACENE AND ANTHRACENE-CONTAINING PRODUCTS

Environmental releases of anthracene can arise mainly through the production and use of anthracene itself, during the production and use of anthracene-containing coal-tar distillates, and during combustion processes. The production of anthracene has been declining significantly during recent years in Europe, ranging around approximately 1,000 tonnes since 1999. Of this production, 99% was exported to outside the EU. On the other hand, more than 1.7 million tonnes of coal-tar, containing over 25,000 tonnes of anthracene, were distilled in Europe during 1999. Thus the production and use of coal-tar derivatives (especially creosote, 107,0000 tpa used for wood treatment in Europe) represents a significantly greater potential source of environmental release of anthracene. In 1998, anthracene production in Europe involved 1,600 tonnes of pure anthracene plus approximately 1,400 tonnes of crude (50%), making a total of 2,300 tonnes of anthracene. In 1999, 550 tonnes of pure anthracene were produced. Taking 1.5% as the typical anthracene content of coal-tar, these quantities would have been derived from the distillation of 153,000 and 37,000 tonnes of crude-coal tar, respectively, corresponding to 8.5% and 2.1% of the total amount of coal-tar distilled in Europe during these two years. Thus, the distillation of coal tar for the ultimate purpose of production of anthracene contributes to under 10% and appears to be following a decreasing trend, and should be seen in this context.

2.6 EXISTING LEGISLATIVE CONTROLS CONCERNING ANTHRACENE AND ANTHRACENE-CONTAINING PRODUCTS

Classification

Anthracene:

Anthracene has not been classified in the context of Directive 67/548 concerning dangerous substances.

Anthracene oils:

All kinds of anthracene oils (different distillation fractions) are classified according to Directive 94/69 (21st adaptation to technical progress of Directive 67/548) as

carcinogen, category 2; R45 - May cause cancer

labelling: T; S53 (Avoid exposure – obtain special instructions before use); S45 (In case of accident or if you feel unwell, seek medical advice immediately - show the label where possible)

Such classification may be considered as not necessary, depending on benzo[a]pyrene content.

CREOSOTE AND CREOSOTE OILS

All types of creosote and creosote oils are classified according to Directive 94/69 as

carcinogen, category 2; R45

labelling T, R42, S53, S45

Such classification may be considered as not necessary, depending on benzo[a]pyrene

content.

Occupation

Directive 91/332 sets a limit value for occupational exposure to "coal tar volatiles" of 0.2 mg/m^3 (8-hour TWA).

Marketing and uses

CREOSOTE

Directive 2001/90 (7th Adaptation to technical progress of Annex I of Directive 76/769) sets strict restrictions on the marketing and use of creosote. Creosote may not be used in the treatment of wood except, by derogation, in the following case:
It may be used for wood treatment in industrial installations, or by professionals covered by Community legislation on the protection of workers for in situ re-treatment, only if it contains <0.005% benzo[a]pyrene and <3% water-extractable phenols. Such creosote may be placed on the market only in packaging of capacity $\leq$20 l, and it may not be sold to consumers. It may also be specially labelled as "For use in industrial installations or professional treatment only".

Further provisions restrict the use of creosote-treated wood: The use of wood treated in industrial installations or by professionals as described above, which is placed on the market for the first time or retreated in-situ, is permitted for professional and industrial use only, e.g. on railways, in electric power transmission and telecommunications, for fencing, for agricultural purposes (e.g. stakes for tree support) and in harbours and waterways.

The restrictions concerning creosote-treated wood do not apply to wood which was treated with creosote before this Directive came into operation and which is placed on the second-hand market for re-use. However, such wood may not be used:
— inside buildings, whatever their purpose,
— in toys,
— in playgrounds,
— in parks, gardens, and outdoor recreational and leisure facilities where there is a risk of frequent skin contact,
— in the manufacture of garden furniture such as picnic tables,
— for the manufacture and use and any re-treatment of:
— containers intended for growing purposes,
— packaging that may come into contact with raw materials, intermediate or finished products destined for human and/or animal consumption,
— other materials, which may contaminate the products, mentioned above.

Cosmetics

According to Directive 76/768 concerning cosmetics, the use of "anthracene oil" is not permitted, while according to Directive 97/45 (adapting to technical progress Directive 74/768) the use of "crude and refined coal tars" is also not permitted.

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 General discussion

3.1.2 Environmental releases

Anthracene occurs in fossil fuels and is released into the environment as a product of incomplete combustion of organic matter (IARC, 1983). It has been identified in the mainstream smoke of cigarettes, cigar and pipe smoke, mainstream smoke of marijuana cigarettes, exhaust emissions from gasoline engines, samples of charcoal-broiled steaks, edible oils, surface water, tap water, waste water, and dried sediment of lakes (IARC, 1983). In addition, anthracene has been identified in emissions from open burning of scrap rubber tires, in high octane gasoline, in coke oven emissions, and in emissions from asphalt processes (Verschueren, 1996). Anthracene is emitted from the open burning of scrap rubber tires at an average concentration of 53 mg/kg of tire (De Marini et. al., 1995)

3.1.2.1 Release from production

Current production of pure anthracene in Europe is estimated at 1.150 tonnes per annum. Table 3.1 presents data from anthracene production Industry Plant Production information has been obtained from only one company in Europe, which is assumed to be the only one operating in the EU.

Table 3.1: Data on annual production of pure anthracene by anthracene production Industry Plant:

1994: 2,100t.	1995: 1,900t.	1998: 1,600t.	1999: 550t.
2000: 1,190t.	2001: 1,150t		

In addition to the production volumes of pure anthracene, the following volumes of crude anthracene have to be included in the balance of direct anthracene products: (anthracene production Industry Plant, 2002)

Table 3.2: Production of crude anthracene.

Year	Anthracene (40%) [tons]	Anthracene (50%), liquid [tons]	Anthracene (50%), solid [tons]	Total anthracene (50%) [tons]	Total
1991		approx. 5600			approx. 5600
1992	approx. 40	approx. 420	approx. 280	approx. 700	approx. 740
1993	approx. 600	approx. 2080	approx. 1480	approx. 3550	approx. 4150
1994	approx. 400	approx. 2890	approx. 2490	approx. 5380	approx. 5780
1995	approx. 70	approx. 800	approx. 480	approx. 1280	approx. 1350

1996		approx. 1030		approx. 1030	approx. 1030
1997		approx. 780		approx. 780	approx. 780
1998		approx. 1400		approx. 1400	approx. 1400
1999		approx. 930	approx. 660	approx. 1590	approx. 1590
2000		approx. 600	approx. 1330	approx. 1930	approx. 1930
2001	-	-	approx. 400	approx. 400	approx. 400

According to information of anthracene production Industry Plant total anthracene production volume would be at best about 1,000 tpa produced by just one company. However, the value estimated from 2001 figures of pure and crude anthracene is 1350 tpa. This includes 400 tonnes of crude anthracene production and 1150 tonnes as pure production. This most precise estimation of 1350 tpa will be used to calculate the PEC at local, regional and continental levels:

Local:	1350 tonnes per annum based on an average manufacturing production
Regional:	1350 tonnes per annum based on an average manufacturing production in a European region
Continental	0 as there is only one site producing anthracene in EU

All the releases occur in the region, as there is one site, so the continental releases should be zero. In addition 99% of anthracene exported abroad and outside EU (to Japan and Czech Republic). So, only 13.5 tonnes anthracene remains in EU. (This RAR was constructed before the enlargement of EU).

A site in the UK recently announced that it produces crude Anthracene. It supplies CAS number 90640-81-6 which refers to Anthracene oil, anthracene paste; Anthracene oil fraction. The anthracene side stream is an anthracene rich oil fraction produced during the distillation of coal tar. The product does not therefore fall under the ESR risk assessment of anthracene which considers only pure anthracene, supplied under CAS Number 120-12-7.

As it is mentioned that the measured data used for tar distillation in the coal tar pitch RAR were also applicable for the production of pure anthracene, industry's suggestion that the same effluent stream was assessed in both reports has been adopted. Therefore the site-specific release estimation made in the coal tar pitch RAR has been used. The default release estimation for anthracene production remains for informative reasons.

Releases to air

Site specific-information

Limited information was available regarding releases of anthracene to the environment during production. Emissions to air were reported by the above company to be 135 kg/year. But due to decline in the production volume and the extended implementation of exhaust gas incineration, the emissions have been appreciably reduced down to about 25 kg/year. This corresponds to 0.02 kg/tonne produced. Based on these data, an emission factor of 0.02 kg/tonne anthracene produced calculated. Local, regional and continental release calculated respectively over 300 days per year.

An estimate for the diffuse emissions to air from tar processing and pitch production was given in the RAR for CTPHT. This is 37 g/d = ~13 kg/year (Tab. 3.13, RAR323_0705_env). As these calculations have been done after more recent site specific informations they will be adopted in the present report.

Default values

For a production of 1350 tpa the number of days of production emission can be estimated as 122 from Table B1.1. This is equivalent to a production of 11.07 tonnes/day.

The default values for emission factor to air given in the Technical Guidance Document for a chemical industry Category 9, with a vapor pressure 9.4 x 10^{-4} Pa is 0 for air (assuming MC 1c-Intermediated stored off-site/dedicated equipment). That means that for EUSES calculations anthracene emissions to air are 0.

As we have a site-specific emission factor to air of 0.02kg/tonne product, these factors will be used instead of default factors. Thus the emissions to air becomes:

Table 3.3: Releases of anthracene to air during production

Environment	Emission factor	Production volume (tpa)	Release
Local	0.02 kg/tonne produced	1350	0.09(kg/day)
Regional	"	1350	27(Kg/year)
Continental	"		

Water

Site specific-information

As there appears to be only 1 plant in the EU, the regional releases will be the total release from the plant and the continental release will be zero. However emissions to water from the anthracene production Industrial Plant were estimated to 268 kg/year. This emission rate is out of date. The situation has considerably improved by establishing more efficient pre-cleaning. An on-site terminal biological wastewater treatment plant has been put in operation and removes all residual anthracene below detection limits. The mean influent concentration is approximately 25µg/l. The average total annual water flow is 750,000 m^3. That means the emission into the biological plant is approximately 19kg/year. After WWTP, total release into the effluent is significantly below 1kg/year, according the anthracene production Industrial Plant site specific information. This corresponds to 0.0008 kg/tonne produced. Based on these data, an emission factor of 0.0008 kg/tonne anthracene produced will be used. Local, regional and continental releases are calculated respectively over 300 days per year.

Table 3.4: Releases of anthracene to water during production

Environment	Emission factor	Production volume (tpa)	Release
Local	0.0008 kg/tonne produced	1350	0.0036 kg/day
Regional	"	1350	1.08 kg/year

The emission data for water which related to tar distillation/processing are also applicable to anthracene production. There is no separate waste-water stream for anthracene production, i.e. all process waste-waters are combined, going into one on-site WWTP. This biological plant is subjected to a regular control for proper function by the environmental agency of the respective German federal state. Again, three campaigns were conducted during the first half of 2006. The samples were taken for 20 min from the effluent of the plant. These are site specific information given by the anthracene production Industrial Plant.

Anthracene levels in the effluent of the anthracene production Industry Plant on-site Waste-Water Treatment plant (WWTP):

Date	Time of sampling from…to	Concentration [µg/L]	Extrapolated total annual emission to water [g/a]
31 Jan. 2006	12:45 – 13:05	0.02	15
16 March 2006	19:15 – 19:35	0.07	52.5
12 June 2006	10:55 – 11:10	0.04	30
Average		0.043	32.5

Release of anthracene in waste water from coal tar pitch production is 0.10g/d for 350 days production. This release is from calculations after industry information and as the model of coal tar pitch production cover anthracene production too, we will use this value for further calculations (Table 3.15, R323_0705_env).

Default values

Default values from the Technical Guidance Document have been used to estimate the emission factors to water based on anthracene solubility of less than 1mg/l and a very low vapor pressure of 9.4×10^{-4} Pa at 25°C. The release of anthracene to waste water is therefore 0.003 during production (TGD, Appendix I, IC=9). Release is calculated based on production over 122 days per year for the local and regional situations according to EUSES. The releases calculated to water are:

Local releases = 33.2 kg/day to waste water
Regional releases = 4,050 kg/year to waste water

For 122 days release is calculated at 0.0088kg/day.

Soil

Site specific-information

As we had site - specific information for water and air and we have used 300 days of production, the same days we would also be used for the soil and these calculations we would use for PECs calculations

In the CTPHT RAR, the estimates of the excess local soil concentrations caused by tar processing and pitch production are 51 ng/kg/dwt in agricultural soil (averaged over 30 days) and 100 ng/kg/dwt in grassland (averaged over 30days) for anthracene, respectively (R323_0705, Tab. 3.50and3.51). As these calculations have been conducted after more recent site-specific information we will adopt this value as PECs for soil. We must clarify that there

are no direct emissions to soil from the site and the concentrations in soil arise mainly from atmospheric deposition (or the application of sewage sludge if this is appropriate).

Default values
Default values from the Technical Guidance Document will be used to estimate the emission factors. According to TGD emission factors to soil are 0.0001. The releases calculated to soil are given in next Table for 122 day of production.

Table 3.5: Releases of anthracene to soil

Environment	Emission factor	Production volume (tpa)	Release
Local (production)	0.0001	1350	1.11 kg/day
Regional (production)	0.0001	1350	135 kg/year

Values found using TGD are not comparable with site-specific information calculations . Moreover these values are overestimated.

3.1.2.2 Release from formulation

Release during production of pyrotechnics (formulation)
The release of Anthracene in water during the manufacturing process (mixing of composition) is not more than 0.25%, because this is a dry mixing process which is operated in a closed system, so that during this process no anthracene is emitted. The above amounts are only a result of cleaning processes.

There are a number of companies in the EU using anthracene in the production of pyrotechnics. Some of these are 4 in the UK, 2 in Germany and one in France and in Italy. It has been assumed that there are about 10 plants using anthracene in EU in the production of pyrotechnics. We have information from only one plant in Germany with a production of 10000 t per year. For this amount they use 400 kg anthracene.
So it has been assumed that an amount of 4,000 kg of anhracene is used for the production of pyrotechnics. Using TGD and IC=0, others, we have release factors: 0.0025 for air, 0.02 for water and 0.0001 for soil and 300 days of production according table B 2.8. Site specific information for water gives an emission factor of 0.0025.

In the present RAR, as well as in the RAR of naphthalene, assessments use the A and B tables from the TGD for both the formulation and processing (use) Both use the tables for Industry Category 0, the catch-all category when the use does not fit into one of the others. Site specific information for water gives also for the processing (use) an emission factor of 0.0025.

The physico-chemical properties of the substances are different, which leads to different emission factors to air, anthracene having a lower vapour pressure than naphthalene which leads to a lower emission factor in both formulation and processing. The basic assumptions underlying the estimates are the same. There are no differences in the approach as far as the emission factors are concerned.

In the RAR on naphthalene the use of 2 tonnes of naphthalene per year on local scale was used based on information provided from a UK maker of pyrotechnics. On a regional and continental scale, the use of naphthalene in formulation was assumed to be 4 and 11 tonnes, respectively.
For anthracene, information for one German site using 400 kg per year is available. This tonnage was considered representative for the use on a local scale. From this a total EU use of 4 tonnes is estimated.

Table 3.6: Releases of anthracene during the production of pyrotechnics (formulation)

Environment	Emission factor	Production volume (kgpa)	Release
Local	Air 0.0025 Water 0.0025	400	0.0033 kg/day 0.003 kg/day**
Regional	Air 0.0025 Water 0.0025 Soil 0.0001	400	1 kg/year 1 kg/year 0.04kg/year
Continental	Air 0.0025 Water 0.0025 Soil 0.0001	3600	9 kg/year 9 kg/year 0.36 kg/year

** = not relevant, because the process is not continuous every day.
Air and soil: No data available.

These default values have been used for the purposes of the assessment to represent a worst-case scenario. The formulation is reported to be water-free. The default value for release to water from the use of anthracene, as well as the use of naphthalene in pyrotechnics, is therefore likely to be an overestimate of the emission factor in practice as indicated in the EC naphthalene risk assessment (EC, 2003).

Processing (use of pyrotechnics)
The naphthalene assessment assumes that the use of pyrotechnics is diffuse, and that there are therefore no real local emissions. Hence only regional and continental emissions are estimated, with 10% in the region and 90% in the continent.

The use of pyrotechnics is widespread throughout the EU and so no local assessment must be carried out. The figure of 400 Kg is the regional release, as it is 10% of the evaluated total amount of anthracene (4000 Kg) used for the production of pyrotechnics inside the EU .
The use of the substance as a component of black smokes suggests that external use is most likely, such as on film locations etc, but it may be used also indoors in theatres and in film studios. It seems unlikely that there will be continuous use (not all productions call for such smoke) and so a disperse use pattern seems reasonable.Therefore no calculations will be made on local estimates for processing.

Using TGD (table A 3.16) and IC=0, others, we have release factors: 0.001 for air and 0.01 for soil.

Table 3.7: Releases of anthracene during the use of pyrotechnics (processing)

Environment	Emission factor	Production volume (kgpa)	Release

Regional	Air 0.001 Water 0.0025* Soil 0.01	400	0.4 kg/year 1 kg/year 4 kg/year
Continental	Air 0.001 Water 0.0025* Soil 0.01	3600	3.6 kg/year 9 kg/year 36 kg/year

* Emmision factors for water are given from site specific information .

Release during production of anthracene-9-aldehyde

This type of production has ceased and these calculations below have only informational value.

Emissions during the production of anthracene-9-aldehyde were estimated on 6.8 tonnes and are formulated at one site.

The releases of anthracene during the processing of anthracene-9-aldehyde 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 3 (Chemical Industry, formulation), MC3 with a vapour pressure of 9.4 x 10^{-4} Pa are 0.00001, 0.02 and 0.0001 respectively (from Table A3.3). As process data received, it confirmed that production of anthracene-9-aldehyde was a water free process, zero emission factor for water will be used in our calculations. Number of emission days calculated can be obtained from Table B3.2 as 14. Local and regional emissions are given below (the continental emissions are zero as there is only one site in the EU).

Table 3.8: Releases of anthracene to air, water and soil during production of anthracene-9-aldehyde

Local	= 0.0049 kg/day to air
	= 0 kg/day to waste water
	= 0.049 kg/day to soil
Regional	= 0.068 kg/year to air
	= 0 kg/year to waste water
	= 0.68 kg/year to soil

As this refers to an outdated application of anthracene, we will not use these elements for the RAR of anthracene.

3.1.2.3 Indirect releases

Release from production of coal tar

1,767,000 tonnes of coal tar containing 1.5% of anthracene (26505 tonnes) were processed in 1999 at 10 sites (Table 3.11) (CTPHT RAR).

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 9 = Mineral oil and fuel industry, processing, vapor pressure 9.4 x 10-4 Pa

and MC1c (Intermediates stored off-site/dedicated equipment)) are 0.0001 to air, 0.0005 to water and 0.001 to soil. Emission days calculated using TGD at 350, according to default values given in Table B 3.7 and a fraction of 0.05 of the main source.

Table 3.9: Emission estimates from coal-tar plants using TGD in 1999

Location	Production (tonnes/year)	Amount of anthracene present (tonnes/year)	Number of days	Estimated anthracene emission (kg/year)		
				Air (0.0001)	Water (0.0005)	Soil (0.001)
Germany	500,000	7,500	350	750	3,750	7,500
Belgium	120,000	1,800	350	180	900	1,800
France	150,000	2,250	350	225	1,125	2,250
Denmark	170,000	2,550	350	255	1,275	2,550
UK1	80,000	1,200	350	120	600	1,200
UK2	120,000	1,800	350	180	900	1,800
Spain1	260,000	3,900	350	390	1,950	3,900
Spain2	70,000	1,050	350	105	525	1,050
Netherlands	140,000	2,100	350	210	1,050	2,100
Italy	157,000a	2,355	350	236	1,180	2,360
Total	1,767,000	26,505		2,651	13,253	26,505

We have information on the annual amounts of coal tar distilled at 9 of the 10 plants. So it's easy to calculate the production of the final plant. The largest coal-tar distillation site in the EU is 500,000tonnes/year. Therefore, these tonnages could be used for the worst case local assessment and for the regional assessment as this site produces more than 10% of the total EU production. We have site specific information for air only from two plants, both from UK.

Table 3.10: Site specific information from two plants in UK.

year	1st plant	2nd plant
2001	98kg	17kg
2002	37kg	11kg
2003	34kg	11kg

We will use the most recent information for further calculations. These give an emission factor of $4.25x10^{-4}$ kg/tones for air for the worst case scenario, if we divide 34 kg with 80,000 tonnes per year. This is equal to 0.60 kg/day.

According to previous RUTGERS site specific data, emission into water were 19kg/a before and 1kg/a after WWTP. Denmark plant had calculated emission to 3kg/a. One company from the UK gave release levels for anthracene in the water from WWTP typically <1kg/year. This site specific information gives gave an emission factor of $3.8x10^{-5}$ kg/tones for Germany and $1.7x10^{-5}$ kg/tones for Denmark. If we take into account the worst case scenario for releases before WWTP. Using the emission factor of $3.8x10^{-5}$ kg/tones it would be equal to 0.054 kg/day.

As it is already mentioned, we will use the most recent site specific information for further calculations, that RUTGERS industry has provided us, in October 2006,.

Releases from use of creosote
Creozote is prohibited in EU for private use but not for professional use. It is estimated that about 100,000 tonnes/annum of creosote are used in the EU and it is thought that about 1,5% of this represent the level of anthracene. 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 9 = Mineral oil and fuel industry) are 0.0001 to air, 0.0005 to water and 0.001 to soil. It has been assumed that 60,000 tonnes of creosote (containing 900 tonnes of anthracene) are used in the EU for the bulk impregnation of timber. Regional and continental releases are given in Table 3.9.

Table 3.11: Regional releases of anthracene during bulk impregnation of timber

	Anthracene in creosote	Emission factors	Releases Kg/year
Regional	90 tonnes/annum	Air 0.0001 Water 0.0005 Soil 0.001	Air 9 Waste Water 45 Soil 90
Continental	810 tonnes/annum	Air 0.0001 Water 0.0005 Soil 0.001	Air 81 Waste Water 405 Soil 810

The releases of anthracene 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 9 (Mineral oil and fuel industry)are 0.0001 to air, 0.0005 to water and 0.001 to soil for a vapor pressure of 9.4 x 10-4 Pa for private use. Regional and continental emissions are given in Table 3.10. In this assessment it has been assumed that 40,000 tonnes/annum of creosote containing 1.5% anthracene is used in domestic applications in the EU.

Table 3.12: Releases of anthracene during use of creosote

	Anthracene in creosote	emission factors	releases kg/year
Regional	60 tonnes/annum	Air 0.0001 Water 0.0005 Soil 0.001	Air 6 Waste Water 30 Soil 60
Continental	540 tonnes/annum	Air 0.0001 Water 0.0005 Soil 0.001	Air 54 Waste Water 270 Soil 540

EU prohibited the use of creosote as it's classified as carcinogen Category 2. However we supposed that amounts of creosote continue to exist in EU. This tonnage was mainly covered by UK private use practice in the past. Releases of anthracene during private use of creosote will be not included in calculations of PECs, following the script of TGD even if, in this way, it does not appear what happens with the remainder sums of anthracene from creosote.

The use of creosote is not a downstream use of anthracene. **So this scenario will not be covered by this RAR**. The creosote is considered under the Biocides directive.

Release during fuel combustion

Diesel fuelled engines emit anthracene to the atmosphere (Choudhury, 1982, Lopez et al., 1987, Dorie et al., 1987). Anthracene/phenanthrene was emitted from a gasoline power turbine to the air at concentrations ranging from 0.53 to 106.00 ng/m^3, with an average from 8 samples of 17.31 ng/m^3 (Robertson et al., 1980). Anthracene was found in 4 gasoline fuels at levels ranging from 0.33, 2.6, and 2.7 mg/l and was released to the air by automobiles in the gaseous phase at an average concentration of 5.3, 12, 21, and 28 µg/km. It was also released as particulates at an average concentration of 0.11, 0.08, 0.24 µg/km and undetectable levels, respectively (Westerholm et al., 1988).

Exhaust emissions from gasoline engines contained anthracene at concentrations ranging from 534 to 642 µg/l fuel burned (IARC, 1983).

Fine aerosol emission rates of anthracene from heavy-duty diesel trucks, noncatalyst-equipped and catalyst-equipped automobiles were 1.6, 5.1, and 0.11 µg/km, respectively (Rogge et al., 1993). Total anthracene concentrations in air samples from the Queensway road tunnel, UK, and the Baltimore Harbour Tunnel, US were 51.1 and 37.6 ng/m^3, respectively (Smith & Harrison, 1996).

According to Panorama of Transport from European Commission, in 1998 passenger cars cover a distance of 3,776 million km and buses and trucks a distance of 415 million km. Using the worst case scenario for anthracene emissions of 28µg/km for passenger cars and 0.11µg/km for heavy-duty trucks and buses, we could calculate releases of anthracene to air (Table 3.11). For regional emissions we used France for passenger cars as it performed 740.3 million km (over 10% of total) and Italy for trucks as it performed 89.2 million km (over 10% of total)

Table 3.13: Releases of anthracene to air during production fuel combustion

	Emission rates of Anthracene in passenger cars	releases in air kg/year	Emission rates of Anthracene in trucks	releases in air kg/year	Total Kg/year
Regional	28µg/km	20.73	0.11µg/km	0.01	20.74
Continental	28µg/km	85	0.11µg/km	0.45	85.45

Release during burning of wood

Anthracene was detected in emission samples collected during the burning of six different fuel types in wood stoves, concentration in mg/m^3:

wood chips/briquettes, 0.061 to 2.79
virgin beech wood, 1.06 to 15.6;
scrap wood, 2.72 to 4.04
sorted domestic waste, 0.087 to 1.77
pentachlorophenol preserved pine wood, 1.81 to 2.72

rolled up newspapers, 0.351 to 1.76
(Nielsen et al., 1992).

No specific information is available for these releases, so they would not consider for PECs calculations.

Release from offshore activities / production of petroleum
Petroleum is a heterogeneous mixture of organic substances and content and fraction of the substances in the petroleum will change during the production period or life period of the oilfield. In general the fraction of water in the petroleum is increased when the oilfields get older. When the petroleum reaches the platform, different mechanical and chemical methods are used for separating the water from the petroleum. In this process naturally occurring anthracene will partly follow the discharged water to the sea. This discharge of industrial water is called "produced water". The oil companies at the Norwegian Continental Shelf report annually the total releases of substances, including anthracene, to the Authorities. Based on monitoring program over years the concentration of anthracene in the "produced water" is measured to be in the range 0,1-0,3 mg/m^3. The total annual release of "produced water" in 2000 to the Norwegian Continental Shelf was 116,000,000 m^3, and the calculated total release of anthracene to the sea will be 10-35 kg/year. The Norwegian discharge of "produced water" is about 25% of the total release to the North Sea. Total volume released of anthracene from offshore activities / production of petroleum, is minor compared to other releases.

Other releases
Anthracene has been detected in individual environments at the following airborne industrial concentrations (geometric mean):
coke plant, 18.61 $\mu g/m^3$
carbon anode plant, 0.894 $\mu g/m^3$
graphite plant, 0.042 $\mu g/m^3$
silicon carbide plant, 0.006 $\mu g/m^3$
metal recycling plant, 0.040 $\mu g/m^3$
a bitumen paving plant, 0.073 $\mu g/m^3$
(Petry et al., 1996).

Runoff from coal piles contained anthracene at an average concentration of 0.6 µg/l (Stahl et al., 1984).
Coal-fired plants emitted anthracene to the air at concentrations ranging from 0.4 to 100 ng/m^3 (Junk et al., 1984).
Anthracene was identified as a stack emission and in grate and fly ash from coal combustion (Junk & Ford, 1980).

Anthracene was also detected as a stack emission and in grate ash of waste incinerators (Junk & Ford, 1980).
A sludge incinerator emitted anthracene to the air at levels from 0.2 to 7.7 µg/sample (Lao et al., 1985).
Incinerator fly ash contained anthracene at conc. from 10 to 500 ng/g with an average from 5 samples of 146 ng/g (Eiceman et al., 1979).
Anthracene was identified as a fine particle released from a natural gas-fired space heater and water heater; emission rates were 1.9 pg/kJ and 0.26 pg/kJ for the first series of filters and backup filters within the samplers, respectively (Rogge et al., 1993).

Releases of anthracene observed from various sources and some of these are listed below.

Table 3.14: Releases of anthracene to the environment from various sites: (Verschueren, 1996).

Emissions from space heating installation burning:	coal (underfeed) stoker): 0.85 x 10^{-3} µg/10 x 10+6 Btu input
gasoil:	3.9 mg/10 x 10+6 Btu input
In gasoline:	1.55 x 10^{-3} µg/l
In exhaust condensate of gasoline engine:	0.53-0.64 x 10^{-3} µg/l gasoline consumed
Emissions from typical European gasoline engine:	18.2-392.5 µg/l fuel burnt
In gasoline (high octane number):	2.6 x 10^{-3} µg/l
In an outlet waterspray tower of asphalt hot-road-mix process:	1,6 µg/m^3
In an outlet of asphalt air blowing process:	220 µg/m^3
Emissions from open burning of scrap rubber tires:	50000-56000 µg/kg tire
Emissions from open burning of scrap rubber tires:	0-1000 µg/kg of tire

The most difficult in these releases is to harmonize the units, as the measurements are all on a different basis. Anyhow, no specific information is available for these releases, and they are not considered for PECs calculations.
The following Table 3.15 gives information for emission factors from USEPA from various sources

Table 3.15: Emission factors from USEPA from various sources

Source	**Anthracene emission factor**
Pulverized coal: dry bottom (bituminous coal)	2.1x10^{-7} lb/ton bituminous coal burned.
Cyclone furnace (bituminous coal)	2.1x10^{-7} lb/ton bituminous coal burned.
Pulverized coal: dry bottom (tangential) bituminous coal)	2.1x10^{-7} lb/ton bituminous coal burned
Pulverized coal: dry bottom (subbituminous coal)	2.1x10^{-7} lb/ton subbituminous coal burned
Cyclone furnace (subbituminous coal)	2.1x10^{-7} lb/ton subbituminous coal burned
Pulverized coal: dry bottom (tangential) (subbituminous coal)	2.1x10^{-7} lb/ton subbituminous coal burned
Pulverized coal: dry bottom, wall fired	2.1x10^{-7} lb/ton lignite burned
Pulverized coal: dry bottom, tangential fired	2.1x10^{-7} lb/ton lignite burned
Cyclone furnace	2.1x10^{-7} lb/ton lignite burned
Grade 6 oil: normal firing	1.22x10^{-6} lb/1000 gallons residual oil (No. 6) burned

Source	**Anthracene emission factor**
Grade 6 oil: tangential firing	$1.22x10^{-6}$ lb/1000 gallons residual oil (No. 6) burned
Natural gas: boilers >100 million Btu/hr except tangential	<$2.4x10^{-6}$ lb/million cubic feet natural gas
Natural gas: boilers <100 million Btu/hr except tangential	<$2.4x10^{-6}$ lb/million cubic feet natural gas
Natural gas: tangentially fired units	<$2.4x10^{-6}$ lb/million cubic feet natural gas
Bark fired boiler	$3.3x10^{-6}$ lb/ton bark burned
Wood/bark fired boiler	$3.3x10^{-6}$ lb/ton wood/bark burned
Wood fired boiler	$3.3x10^{-6}$ lb/ton wood burned
Internal combustion engines; industrial; reciprocating; diesel	$1.87x10^{-6}$ lb/million Btus heat input
Internal combustion engines; industrial; large bore; diesel	$1.23x10^{-6}$ lb/million Btus heat input
Asphalt heaters: Distillate oil	$1.8x10^{-7}$ lb/gallon oil burned
Asphalt concrete, conventional batch mix plant: rotary dryer, natural gas fired	$3.1x10^{-7}$ lb/ton hot mix asphalt produced
Asphalt concrete, drum mix plant: rotary drum dryer/mixer, natural gas fired	$2.1x10^{-7}$ lb/ton hot mix asphalt produced
Asphalt concrete, drum mix plant: rotary drum dryer/mixer, oil fired	$3.6x10^{-6}$ lb/ton hot mix asphalt produced
Crematoria	$3.24x10^{-7}$ lb/each body burned
Open burning of refuse	$1.3x10^{-3}$ lb/ton waste burned
Open burning of refuse	$1.43x10^{-2}$ lb/ton waste burned
Open burning of refuse	$2.6x10^{-3}$ lb/ton waste burned
Open burning of refuse	$8x10^{-4}$ lb/ton waste burned
Open burning of car tires	$5.31x10^{2}$ lb/1000 tons tires burned
Open burning of car tires	9.92 lb/1000 tons tires burned
Anthracite coal combustion (residential)	$3.3x10^{-2}$ lb/ton anthracite burned
Natural gas combustion (residential)	<$2.4x10^{-6}$ lb/million cubic feet natural gas burned
Catalytic woodstoves (residential)	$8.0x10^{-3}$ lb/ton wood burned
Non-catalytic woodstoves (residential)	$9.0x10^{-3}$ lb/ton wood burned
Non-catalytic woodstoves: conventional (residential)	$1.4x10^{-2}$ lb/ton wood burned

Total solid fuel consumption in the EU for energy generation in 2000 was estimated to be 211.7 million tonnes (Integrated Pollution Prevention and Control (IPPC). Using the emission factor of 2.1×10^{-7} lb/ton given in Table 3.15 (equivalent to approximately 9.4×10^{-8} kg/tonne) gives a very approximate total EU emission of 20 kg/year.

Another paper presents the first attempt to quantify the production, cycling, storage and loss of PAHs in the UK environment. Over 53 000 tonnes of PAHs (sum of 12 individual compounds) are estimated to reside in the contemporary UK environment, with soil being the major repository. If soils at contaminated sites are included, this estimate increases dramatically. Emission of PAHs to the UK atmosphere from primary combustion sources are estimated to be greater than 1000 tonnes PAHs per annum, with over 95% coming from domestic coal combustion, unregulated fires and vehicle emissions. It is estimated that

approximately 210 tonnes of PAH are delivered to terrestrial surfaces each year via atmospheric deposition. Therefore, inputs of PAHs to the UK atmosphere outweigh the outputs by a factor of over 4. This may be explained by enhanced particulate deposition near point sources, PAH degradation in the atmosphere and transport away from the UK with prevailing winds. Disposal of waste residues is estimated to contribute a further 1000 tonnes of PAH per year to the terrestrial environment. It is illustrated that the use of creosote has the potential to release considerable quantities of PAHs to the UK environment. Temporal trends in PAH cycling are then considered. There is good evidence to suggest that air concentrations and fluxes to the UK surface are now lower than at any time throughout this century. Nonetheless, the UK PAH burden is still increasing at the present time, principally through retention by soils. However, there are marked differences in the behaviour of individual compounds: there is evidence, for example, that phenanthrene concentrations in soils have declined since the 1960s, although soil concentrations of benzo[a]pyrene and other heavier PAHs have continued to increase through this century. Volatilisation of low molecular weight PAHs accumulated in soils over previous decades may be making an important contribution to the current atmospheric burden. The major uncertainties identified by data on this budget are: (1) the lack of PAH concentrations in some environmental matrices; (2) the possible importance of contaminated soils as a major repository and source of PAHs; (3) the lack of emission data (especially vapour phase releases) for some PAH sources; (4) the importance of biodegradation and volatilisation as loss mechanisms for low molecular weight PAHs in soils; and (5) the importance of creosote use in the PAH cycle. (Wild and Jones, 1995).

3.1.2.4 Summary of releases

Releases of anthracene from major sources under worst case conditions, are summarised for the local, regional and continental environments in Tables 3.16, 3.17, 3.18. These values have been used in the assessment to calculate PECs.

Table 3.16: Summary of emission estimates in air

Process	tonnes	Emissions from site specific informations	Emission form default values
Production of anthracene	1350	Local 37 g/day*	90 g/day
		Regional 13 kg/year	27 kg/year
		Continental -	-
Production of pyrotechnics (formulation)	4	-	Local 0.0033 kg/day
		-	Regional 1 kg/year
		-	Continental 9 Kg/year
Production of pyrotechnics (processing)	4	-	
		-	Regional 0.4 kg/year
		-	Continental 3.6 Kg/year

*: 37 g/day represent not only anthracene production, but is the related to tar-distillation and pitch production, as the process emissions cannot be easily allocated to single operations. Therefore, this value is supposed to represent a high worst-case when solely linked to anthracene production

Table 3.17: Summary of emission estimates in water

Process	Tonnes	Emissions from site specific	Emission form default values

		informations	
Production of anthracene	1350	Local 3.6g/day	Local 33,200 g/day
		Regional 1.08 kg/year	Regional 4,050 kg/year
Production of pyrotechnics (formulation)	4	-	Local 3 g/day
		-	Regional 1 kg/year
		-	Continental 9 Kg/year
Production of pyrotechnics (processing)	4	-	
		-	Regional 1 kg/year
		-	Continental 9 Kg/year

Table 3.18: Summary of emission estimates in soil

Process	tonnes	Emissions from site specific informations	Emission form default values
Production of anthracene	1350	Local (agricultural soil) $51x10^{-9}$ g/day/dwt Local (grassland) 10^{-7} g/day/dwt	Local 1,110 g/day
		Regional -	Regional 135kg/year
		Continental -	Continental ?
Production of pyrotechnics (formulation)	4	-	
		-	Regional 0.04 kg/year
		-	Continental 0.36 Kg/year
Production of pyrotechnics (processing)	4	-	
		-	Regional 4 kg/year
		-	Continental 36 Kg/year

Site specific emissions for the production of anthracene and default emissions from the production of pyrotechnics are used for PEC calculations.

3.1.3 Environmental fate

Chemical processes, including ozone and hydroxide, radical and photochemical reactions, degrade atmospheric anthracene. The degradation of vapour-phase atmospheric anthracene is expected to be faster than particle-sorbed anthracene. The atmospheric life-time of anthracene may vary from hours to days. The long-range transport of anthracene indicates the particle-sorbed anthracene may have a half-life of the order of days (USEPA 1987).

Generally, the biodegradation of non-chlorinated aliphatic and aromatic hydrocarbons is affected by their bioavailability. Hydrocarbons are very poorly soluble in water. They are easily adsorbed to clay or humus fractions in the soil, and pass very slowly to the aqueous phase, where they are metabolized by microorganisms. Surfactants that increase their solubility and improve their bioavailability can accelerate degradation.

3.1.3.1 Degradation in the environment

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3.1.3.1.1 Degradation in the aquatic environment (incl. sediment)

Hydrolysis

Hydrolysis of anthracene is not expected. Studies showed hydrolysis <0.001 $hour^{-1}$ at $25^{0}C$ in Southworth, 1997, and no hydrolysable in Mabey et al, 1982 and Howard et al, 1991.

Photolysis in water

The degradation of anthracene in water by O_3, by O_3 in combination with H_2O_2 and UV-irradiation, and by UV-irradiation alone was studied to compare the efficiency of various oxidation processes at different pHs. The chemical reaction rate constants were calculated on the basis of kinetic curves of anthracene destruction. The results confirm that anthracene oxidation proceeds mostly with mol. O_3 and the best method for its reduction is simple ozonization in acidic or neutral media. The velocity of anthracene autoxidation is rather low and depends neither on pH nor H_2O_2 addition. Anthracene concentration may be reduced remarkably also by UV-irradiation. (Trapido and Veressinina, 1994).

Anthracene is photoreactive. The effect of added SiO_2 on the photochemistry of anthracene is examined in cyclohexane. Adsorption of anthracene onto the SiO_2 surface from cyclohexane follows a Freundlich adsorption isotherm indicating interactions between anthracene and the surface over a distribution of adsorption sites. In the absence of O_2, addition of SiO_2 to a cyclohexane solution of anthracene leads to a significant increase in the rate of photolysis without a large concomitant change in product identity. The 9,10-photodimer of anthracene is the major product under O_2-deficient conditions. With O_2 mediation, added SiO_2 has dramatic effects on both the photoproduct distribution and the kinetics of photodecomposition. The complicated mixture of oxygenated products formed from photolysis in O_2-contg. Slurries arise from thermal decomposition of a single primary photoproduct, anthracene 9,10-endoperoxide. The polarity/polarizability of the SiO_2/cyclohexane interface is a major factor influencing the rate of photolysis in the oxygenated SiO_2/cyclohexane slurries. The photolysis rate constant for a 3 x 10^{-7} M aqueous solution of anthracene was measured to be 5.16 x 10^{-4}/sec (Zingg, S. P., et al, 1993). This corresponds in an estimated half life of 20min for photolysis.

The fate of anthracene, a representative polycyclic aromatic hydrocarbon, was followed in a large outdoor stream microcosm. The major nonadvective route for the removal of anthracene was photolytic degradation to anthraquinone (half-life 43 min). The anthraquinone also photolyzed rapidly in this shallow stream system. Excluding the plastic channel liner, the sediment acts as the major sink for anthracene, absorbing 0.2% of the 14-day input dose. The periphyton community was the second most important sink, absorbing 0.04% of the input dose. All other compartments were of significantly less importance on a mass basis. Anthracene (11 mgxliter-1) caused photo-induced 100% mortality of the bluegill sunfish in 9 hr in the upstream reach. Fish at the downstream station survived for ~26 hr and all died within 1 hr of each other. Other organisms, clams and dragonfly larvae, started to die off toward the end of the 14-day input period. (Landrum P F, et al, 1984).

Anthracene in distilled water was rapidly degraded under exposure to natural sunlight, with a photolysis half-life of about 35 minutes under midday sunlight in midsummer at 35 deg north latitude. Under average winter solar conditions at the same latitude coordinates, anthracene's photolytic half-life was 4.8 hr and 1.6 hr for summer conditions (Callahan et al, 1979).

Photolysis of anthracene (350 nm) in aerated water yields endoperoxide and 9,10-anthraquinone as the major primary products. Photolysis of anthracene in oxygen-deficient aqueous solutions yields the three isomers of 10,10'-dihydroxy-9, 9'-10, 10'-tetrahydro-9, 9'-bianthryl as the primary product (Sigman, M. E., et al, 1991).

Photoinduced degradation of anthracene in water was monitored spectroscopically. Three types of gas stream (ozone/nitrogen, oxygen/nitrogen, and ozone-oxygen/nitrogen) were used to supply oxidants to the aquatic solutions. The use of an O_2-free ozone/nitrogen gas stream allows the elucidation of the role of ozone in the degradation process without possible complications from oxygen. A light source of > 300 nm was employed to excite the aromatic compounds. The measured degradation rate constants under these conditions support that the interaction between the excited states of PAHs or ferricyanide and ozone plays a role in their photoinduced decomposition. (Schutt et al, 1997).

In Bertilsson and Widenfalk (1979) study, as solar ultraviolet radiation degrades and alters the quality of natural organic matter as well as organic pollutants in surface waters, .photochemical degradation of three PAHs; anthracene, phenanthrene and naphthalene, in water was studied. Anthracene and phenanthrene were rapidly photodegraded (half-lives of 1 and 20.4 hours, respectively), while the photochemical half-life of naphthalene exceeded 100 hours. Hence photodegradation is most likely a less important removal mechanism for the latter compound. The influence of humic substance additions (0–25 mg C l^{-1}) on degradation rates was also assessed, and while photodegradation of anthracene was not affected by these additions, phenanthrene photodegradation slowed down considerably at the higher humic substance concentrations. These differential responses of anthracene and phenanthrene can at least partially be explained by differences in the spectral absorbance of the two compounds. In contrast, ionic strength did not have any appreciable effect on the estimated photodegradation rates of either compound. The influence of PAHs on growth of aquatic bacteria was assessed in dilution cultures with and without exposure to PAHs and simulated solar UV radiation. Separately, neither PAHs nor simulated solar UV radiation had any effect on bacterial growth. However, when combined, a marked inhibition of bacterial growth could be observed in water obtained from a clearwater lake. This could be due to the formation of toxic photodegradation products such as quinones (detected in our incubations) or other reactive species that affect bacteria negatively. Hence, in addition to influencing the fate and persistence of PAHs in aquatic systems, solar radiation and natural organic matter and regulate the toxicity of these compounds to indigenous micro-organisms.

Table 3.19, may show different values of half lives of anthracene via photolysis in those studies a result has arise.

Table 3.19 : Photolysis of anthracene

Season	Environment	Experim. time	Temp. ^0C	Removal rate constant	Half live	References

		(hours)		$(hours^{-1})$	(hours)	
	Adsorption of anthracene onto the SiO_2 surface from cyclohexane			$5.16x10^{-4}/sec$	20min	Zingg, S. P., et al, 1993
	Degradation to anthraquinone				43 min	Landrum P F, et al, 1984
Summer	Direct sunlight in distilled water	24			1.6	Callaghan et al, 1979
Winter	Direct sunlight in distilled water	24		0.15	4.8	Callaghan et al, 1979
	Surface waters				1	Bertilsson & Widenfalk,1979
Midsummer sunlight	Slow clear water		25	0.004-0.238		Southworth, 1977
Midsummer	Near surface water				0.75	Harris, 1982
	Inland water				108	Zepp & Schlotzhauer, 1979
	Inland water with sediment				124.8	Zepp & Schlotzhauer, 1979
	Distilled water			0.66	1	Fukuda et al, 1988
	High pressure mercury lamp or sunlight			$0.0023\ min^{-1}$	0.5	Wang et al. 1991

Summary

The half-life for photolysis in water lies in the range 20 minutes and 124.8 hours depending on the experimental conditions used. The highest value in this range corresponds to photolysis in winter solar conditions. The lower photolysis rate constant for a 3 x 10^{-7} M aqueous solution of anthracene was measured to be 5.16 x 10^{-4}/sec (Zingg, S.p., et al. 1993)

Biodegradation

Simulation study has been performed resulting in a dt50 of 141 d in the water and 57-210 d in the sediment (Lee et al., 1983).

Anthracene was confirmed to be poorly or non-biodegradable in MITI test. According to MITI I modified test system (Guideline OECD 301C) 100 mg/l substance, 30 mg/l sludge showing that 1.9% degradation of anthracene by BOD Therefore anthracene seems to be not readily biodegradable(MITI, 1992). Although mass balance problems exist in ready-tests, they don't remove the fact that anthracene is not degraded in STP and thus finds its way to the environment, where the biotic degradation is even slower, (PBT working group factsheet, rev 5, 2003).

The rate constant of micrological degradation of anthracene in Third Greek water incubated 18 h at 25 ^oC was $0.061 \pm 0.007h^{-1}$. No degradation was observed in autoclaved Third Greek water, distilled water, or water from Walker Branch over the same time period. The anthracene concentration in the Third Greek water, prior to the addition of 1 ppb anthracene, was below the detection limit by fluorescence (<0.05) . Analysis of the ^{14}C fractions in this experiment indicated that about 5 percent of the ^{14}C was converted to CO_2, with the remainder present in 1 N NaOH metabolites. Fluorescence and ^{14}C techniques agreed well in estimating anthracene degradation rates. Further study of the role of microbial degradation in bedded sediments in the removal of PAHs from the water column is also necessary. (Southworth 1979).

Mackay et al. (1992), suggested half lifes of anthracene above 300-1,000 hours for water and 420-1,250 days days for sediments. These values are based on modeling data.

Significant degradation with gradual adaption was reported for anthracene (5 and 10 mg/l) incubated sewage seed (43% and 26% degradation after 7 days, 92% and 51% degradation after 28 days and 3 weekly subcultures) (Tabak et al, 1981). Extremely high removal rates are reported there for several substances that proved non degradable in othertests and there fore the results were judged not plausible.

For degradation by bacteria from estuary half lifes for anthracene and B{a} of more than 145 and 1750 days, respectively, were found (Gerlach, 1981 in CTPHT RAR).

In static experiments complete decomposition for naphtthalene and phenanthrene, partial decomposition for anthracene was found(Richards, & Shieh, 1986 in CTPHT RAR).

A pilot-scale treatability study, 100% of the anthracene present in contaminated groundwater was biodegraded over a period of 36 days in publicly owned treatment works employing activated sludge treatment (Smith et al, 1981).

Biodegradation of five chemicals (aniline, anthracene, chlornitrophen (CNP), fenitrothion (FNT) and linear alkylbenzene sulphonate (LAS)) by aquatic bacteria in three different types of ponds was determined according to the cultivation method developed by this group. The degradability toward these chemicals was varied among the ponds, except for LAS which was decomposed well in all samples. Higher degradability towards the two agrochemicals, CNT and FNT, was found in the pond surrounded by paddy fields, whereas aniline and anthracene were decomposed more rapidly in the pond located in the industrial area. Water from the pond in the botanical garden, with the least exposure to any chemicals, exhibited the lowest degradation toward all chemicals tested. There was no significant seasonal variation in the biodegradation of chemicals in these ponds. It was deduced that biodegradability toward certain chemicals could be a result of acclimatization of the microbial community by chemical contamination present and past, suggesting the possible use of biodegradation profiles as an indicator for chemical pollution in the aquatic environment. Nishihara et al (1997).

The marine cyanobacteria, *Oscillatoria salina Biswas*, *Plectonnema terebrans* Bornet *et Flahault*, and *Aphanocapsa species*, degraded Bombay High crude oil when grown in artificial seawater nutrients and in natural seawater. Oil removal was measured by gravimetric and gas chromatographic methods. About 20 to 90% of aromatic compounds (incl. anthracene) disappeared within 10 days. The cultures were grown photoautotrophically

in filter-sterilized artificial seawater nutrients (ASN as recommended by NFMC) with salinity 25‰ and pH of 5.7-8.2. The cultures were maintained under 12:12 light and dark cycle at 28° C. Light was provided by two fluorescent lamps of 40W placed at distance of approximately 40cm. After 10 days the percentage degraded of anthracene was 90.6 for *Oscillatoria salina*, 62.7 for *Plectonnema terebrans* and 41.9 for *Aphanocapsa species* (Raghukumar et al, 2001).

Methanotrophic bacteria were enriched from marine sediments and screened for their ability to biotransform polycyclic aromatic hydrocarbons (PAHs). The methanotrophic enrichment degraded anthracene to below detectable levels in 15 days. The addition of the methanotrophic enrichment to a marine culture grown on PAHs as the sole C source increased the transformation rate of anthracene. A possible mechanism for the increased transformation rate was the rapid oxidation of PAHs by methane monooxygenase, forming an intermediate that is more bioavailable for utilization by the PAH-degraders (Rockne et al, 1998).

Rhodococcus species isolated from polluted river sediment was studied to detect if this isolate could degrade high molecular mass polycyclic aromatic hydrocarbons. The *Rhodococcus species* could utilize anthracene (53%), as sole C and energy sources. In a study of anthracene degradation by a *Rhodococcus species*, the identification of ring-fission products indicated at least 2 ring-cleavage pathways. One results in the production of 6,7-benzocoumarin, previously shown to be chemical produced from the product of meta-cleavage of 1,2-dihydroxyanthracene, a pathway well established in Gram-negative bacteria. The second is an ortho-cleavage of 1,2-dihydroxyanthracene, which produces 3-(2-carboxyvinyl) naphthalene-2-carboxylic acid, a dicarboxylic acid ring-fission product. This represents a novel metabolic pathway only identified in Gram-positive bacteria. (Dean-Ross et al, 2001).

The ratio of the concentration of the oxidation product anthraquinone to that of its parent polycyclic aromatic hydrocarbon anthracene is reported for several coastal marine sediments. The ratio ranges from 0.317 in a highly contaminated industrialized harbor to 2.81 in a remote, less contaminated site. McKinney et al (1999) hypothesize that differences in this ratio result from the input source of PAHs, with input from atmospheric deposition at remote sites resulting in a predominance of anthraquinone (ratio >1), and direct discharge to highly contaminated industrialized harbors resulting in a predominance of anthracene (ratio <1). To support this hypothesis, the fate of anthracene in the marine environment was studied with respect to conversion to its oxidation product, anthraquinone. Once associated with sediments, anthracene is believed to be relatively persistent; however, it can potentially be subjected to oxidation via biological (microbial degradation) and chemical (chemical oxidation and photooxidation) processes. An assessment of the extent of oxidation of anthracene associated with sediments was conducted both under conditions simulating those found in the marine environment and under rigorous conditions by exposure to UV radiation. Results show that while anthracene associated with marine sediments does not readily undergo oxidation to anthraquinone under conditions normally encountered in the marine environment, under extreme conditions anthracene is photooxidized by exposure to UV radiation. The extent of oxidation is influenced by sediment characteristics such as percent organic C, humic acid content and sediment surface area.

Due to the large variations in anthracene half-lives it is decided to use the study of Lee et al. (1983), as a valid simulation study, as well as the suggested mean half-lives by Mackay et al. (1992), for the risk assessment.

3.1.3.1.2 Degradation in soil

Abiotic degradation

As a means to remediate soil contaminated by polycyclic aromatic hydrocarbons, a combined process involving EtOH washing followed by a Fenton oxidation reaction was investigated. Artificial loamy soil was contaminated with various representative polycyclic arom. hydrocarbons (i.e. anthracene) at concentrations 10 times higher than regulatory soil standards of The Netherlands or Canada, and then washed 4 times in EtOH, which reduced the concentration of polycyclic aromatic hydrocarbon contamination to below the regulatory standard. Fenton oxidation of EtOH solutions showed a removal efficiency of 73.3-99.0% for anthracene. Since each of the nonremediated polycyclic aromatic hydrocarbons are easily biodegradable, these results indicate that the proposed treatment can be successfully applied to polycyclic aromatic hydrocarbon-contaminated soil. The main reaction products resulting from Fenton oxidation of EtOH solutions for anthracene were anthraquinone. (Lee and Hosomi, 2000).

A method is described for effectively pretreating soil highly contaminated with anthracene (500 mg anthracene/kg soil), where Fenton oxidation with ethanol was applied to increase anthracene removal. In addition to detecting the optimum amounts of ethanol, Fe^{2+}, and H_2O_2 needed to achieve maximum removal efficiency of anthracene from simulated soil (andisol particles), i.e., $\cong$97%, we also employ GC-MS and HPLC to detect the main oxidation product generated by the optimized Fenton reaction: 9,10-anthraquinone. The biodegradability of 9,10-anthraquinone is much more rapid than that of anthracene, i.e., biodegradability of 90 vs. 30% over 30 days. Thus, this treatment can be effectively applied to remove anthracene prior to disposal at industrial waste sites. (Lee et al, 1998)

Degradation of anthracene, by Fenton's reagent was studied using batch, pseudo-continuous, and continuous dosing modes. The effect of organic solvent, i.e., methanol and ethanol, on the PAH degradation was examined. Results indicated that Fenton's reagent could effectively degrade all selected PAH. In the continuous dosing mode, the reaction follows a time-squared kinetic expression, i.e., C = C0 exp (-kobst2). Results also showed that methanol and ethanol inhibit degradation of target PAH due to competition for OH- radicals. (Oiang et al, 2001)

The feasibility of using a combination of soil washing and Fenton oxidation to remove polycyclic aromatic hydrocarbons (PAH) from soil and subsequently destroy them in the resulting wastewater solution was investigated by Saxe et al (2000). Three well-characterized New Jersey soils, representing a wide range of organic C content, were artificially contaminated with a mixture of anthracene, phenanthrene, and fluoranthene to simulate PAH-contaminated field soil. Batch laboratory-scale surfactant-enhanced soil washing was used to remove PAH from these soils. Two nonionic octylphenyl ethoxylate-type surfactants, Triton X-100 and Igepal CA-720, were employed. PAH concentration in wash solution containing 1% surfactant was increased by over an order of magnitude above the level possible with water alone. The resulting solutions were treated with Fenton's reagent (H_2O_2 plus Fe^{2+}) to destroy PAH in solution. Greater than 99% of PAH parent material was destroyed in the Triton X-100 wash solution from the low organic sandy soil. Mass spectrometric analysis of the treated material indicated that partially degraded surfactant mols. constituted the major reaction residuals. The extent of disappearance of parent PAH in soil-washing wastewater for

a given dose of Fenton's reagent was inversely proportional to the water solubility of the species (anthracene > fluoranthene > phenanthrene).

Biodegradation

The biodegradation rate of PAHs in soil found under laboratory conditions shown half lives from soil microcosms to 48 and field conditions to 120 days and half lives from long term field experiment at 7.9 years (Wild et al., 1991) Twenty nine anaerobically digested, lagoon dried sewage sludges were analysed for polynuclear aromatic hydrocarbons (PAHs). These sludges had been applied to the plots of a long term agricultural experiment from 1942 to 1961. The exact dates of sludge production and treatment are unknown, although they are likely to be 1–5 years prior to sludge application dates. The sludges had a mean PAH (defined as the sum of the compounds measured) concentration of 50 mg/kg, with a range of 18–125 mg/kg. The most abundant compound was benzo[ghi]perylene with a mean concentration of 10 mg/kg. A trend is apparent in the PAH content through time, increasing until 1948 to over 125 mg/kg, then decreasing to 29 mg/kg by 1961. These changes are tentatively attributed to changes in air quality and smoke emissions. The PAH content of contemporary sludges is also reviewed)

The relation between the properties of PAHs and their persistence in soils was evaluated in a study of Maliszewska-Kordybach (1998). The degradation of anthracene in 10 different soils was studied in laboratory experiments. The soil samples artificially contaminated with the mixture of PAHs on the level of 20 mg of each hydrocarbon per kg of soil were incubated during 180 days at dark at 20^0C and analyzed periodically for PAHs content. The range and the rate of PAHs disappearance from soils was correlated with their volatilization ability (characterized by Henry's constant H,), bioavailability (characterized by water solubility. Ws), and sorption affinity (characterized by 3 parameters: octanol-water partition coeff. log Kows, first-order connectivity index 1 , and mol. surface area MSA). The strength of the relationship between PAHs properties and the range of their degradation, depended on the contact time. The highest correlation coefficients corresponded to the parameters related to surface area MSA and 1.

Contaminated soil from a former manufactured-gas plant (MGP) site was treated in bench-scale, slurry-phase biol. reactors, in which the biodegradation of 7 polycyclic aromatic hydrocarbons (PAH), including anthracene, was followed. In the 1st phase of the project, no difference in PAH removal was observed between reactors operated at solids residence times (SRT) of 18 and 35 days. A modified reactor with an 18 d SRT was operated during the 2nd phase of the project. From 42 to >87% of the PAH of interest were removed over a 3-mo operating period. Sorption of hydrophobic surfactant was apprx.25 g/kg soil solids for the untreated soil and 47 g/kg soil solids for the treated soil. Above these doses, the surfactant was able to solubilize 40% of most of the PAH. Rates of PAH solubilization were rapid, with approximately 80% of the concentration reached within 3-4 h for both the untreated and treated soils. Additional of the surfactant to treated soil from the reactor did not enhance PAH degradation. (Aitken et al, 1998)

The effect of a nonionic surfactant, Tween 80, on the biodegradation of PAHs in acidic loamy soil spiked with 400 mg/kg anthracene was assessed in a bench-scale composting system. PAH-spiked soil was mixed with pig manure at a ratio of 25% (wt./wt. dry wt). Tween 80

was applied at a rate of 20, 40, or 100 mg/kg (wt./wt. dry wt) to the composting mass at day 0 or 14 of the composting period. Tween 80 enhanced the removal of anthracene. The effect of Tween 80 on biodegradation of PAHs was also time-dependent with significantly higher degradation for treatment added at Day 14 than Day 0, especially at a Tween 80 concentration of 100 mg/kg. The highest removal efficiency of PAHs in the spiked soil was obtained in the treatments with Tween 80 application rate of 20 to 40 mg/kg applied at Day 14 of composting process. (Fang et al, 2001)

Greenhouse studies using pots and microcosms were conducted to investigate the persistence and fate of nonlabeled and 14C-labeled anthracene in sludge treated soil. Results indicated that anthracene degraded rapidly (t1/2. apprx.3 wk) from the experimental systems. On completion of the experiments (21 wk), 10% of the anthracene added to soil was recovered intact. Recoveries of $14CO_2$ indicated significant mineralization of anthracene. Trivial amounts (0.2%) of 14C were recovered as volatile organisms and in plant materials, but large amounts were recovered from soil. A considerable proportion of the anthracene derived 14C in soil had been converted to bound residue. No evidence was obtained for uptake of intact anthracene by ryegrass (*Lolium multiflorum*), soybean (*Glycine Max (L.) Merr*.), and cabbage (*Brassica oleracea var. capitata L.*) (Goodin, J. D., et al., 1995).

In addition, Kästner et al examined the metabolism and formation of bound residues of anthracene with different labels in the soil-compost mixture. Under similar culture conditions, the metabolism of [9-^{14}C]anthracene was significantly slower than the metabolism of [1,2,3,4,4a,5a-^{14}C]anthracene.The formation of CO_2 from [1,2,3,4,4a,5a-^{14}C]anthracene began after a lag phase of 15 days, compared to a lag phase of 35 days in the case of [9-^{14}C]anthracene. The mineralization phase occurred at the same time in both experiments (90 to 100 days). However, the levels of extractable activity in the experiment performed with [1,2,3,4,4a,5a-[^{14}C]anthracene decreased rapidly within 40 days, whereas more than 100 days were needed to reach similar concentrations in the extracts in the [9-^{14}C]anthracene experiment. The total level of mineralization of [9-^{14}C]anthracene (67.2%) was slightly higher than the total level of mineralization of [1,2,3,4,4a,5a-^{14}C]anthracene (62.4%), whereas the nonextractable bound residues accounted for 20.7% of the [9-^{14}C]anthracene and 28.5% of the [1,2,3,4,4a,5a-^{14}C]anthracene. This indicates that the transformation and carbon partitioning of the A-ring label were significantly different from the transformation and carbon partitioning at the C-9 position. A higher level of residues was observed with elevated metabolism of [1,2,3,4,4a,5a-^{14}C]anthracene in the soil-compost mixture.

Soil samples from wood treatment site were contaminated with 25% (wt./wt.) creosote in a study of Atagana et al (2000). Aerobic assessment of indigenous soil microorganisms capable

of catabolizing creosote oil components was carried out using phenol, pyrrole, anthracene, phenanthrene, o-cresol, p-cresol, and m-cresol, which were found to be in relatively high concentrations in the samples as indicator compds. In all enrichment reactors, which were poised with different concentrations. i.e. 50, 100, 500, 1000, 5000, 10000, 15000, 20000, 25000 and 30000 ppm of the different compounds, increase in microbial activity was seen to be inversely related to pH which reached the min. at 5.32. The rate of decrease in concentration of the indicator compounds was seen to vary from one compound to another, and from one concentration to the other. The decrease tended to reach a peak at 5000 ppm. However, relatively high microbial activity and loss in hydrocarbon concentration continued to be noticed up to 25000 ppm. Fifteen microbial associations were isolated from the enrichment cultures. Optimization of hydrocarbon degradation by soil microorganisms was carried out by soil amendments based on the C:N:P ratio of the samples used. The lower amendments which corresponds to C:N:P ratios of 25:1:1, 20:1:1, 15:1:1, 10:1:1, had the highest microbial activity and corresponding loss in hydrocarbon concentrations; 68.7%, 61.1%, 56.3%, 63.8%, respectively.
The use of vegetation to increase the degradation of anthracene was investigated in a greenhouse experiment (Reilley et al, 1996). Anthracene was added to a contaminated, land-farmed soil and a similar uncontaminated soil at a rate of 100 mg/kg. Four plant species were grown in each soil; after 4, 8, 16, and 24 wk of plant growth, soil and plant material were sampled and analyzed for anthracene. Vegetated soils had significantly lower concentrations of anthracene than the unvegetated soils, ranging from 30-44% more degradation in the vegetated soils. Enhanced biological degradation in the rhizosphere appears to be a mechanism of dissipation. Leaching, plant uptake, abiotic degradation, mineralization to CO_2, and irreversible sorption were shown to be insignificant in the overall mass balance of anthracene. The presence of plants may enhance the clean-up of anthracene-contaminated soils during in situ remediation.

The availability of anthracene in soil for biological degradation was detected by Willumsen et al (1997) as a function of aging of the soil in the presence of liquid solvents, in order to simulate the conditions following contamination by non-aquatic phase liquids (NAPL). Two soils and clay were aged in the presence of a solution of anthracene in a non-aquatic solvent. The availability of the remaining anthracene to bacteria was detected by contacting the sample with an active mixed culture in a slurry bioreactor. The parameters investigated during the aging step were moisture content solvent polarity and time of aging the soil in the presence of the anthracene in a carrier solvent. When the non-aquatic solution of anthracene was in aged with soil for 90 days, the subsequent bioreactor treatment reduced the residual anthracene concentration to 4 mg/kg dry soil. The presence of soil organic material had no effect. The bioavailability of anthracene in the slurry bioreactor was only reduced when the soil had been added for 180 days in the presence of the solvent solution. This reduction in availability was consistent with the formation of large anthracene crystals in the soil matrix, and slow diffusion within soil aggregates.

A mixed bacterial culture was used to degrade anthracene that had been impregnated into a representative high-clay soil. The activity of the culture was sustained over a period of months in repeated batch operation, in which fresh soil was inoculated with 20% spent slurry from the previous run. Maximum degradation rates of 100-150 mg anthracene/kg soil/day were achieved throughout the experiments. Evolution of carbon dioxide from the bioreactor showed that degradation and mineralization of anthracene occurred simultaneously, and that 55% of the anthracene was mineralized. When the culture was switched from anthracene as sole carbon source to a mixture of three polynuclear aromatic hydrocarbons, the culture was

able to degrade each of these in the sequence: anthracene, phenanthrene and finally pyrene. (Banerjee et al, 1995)

The white-rot fungi *Trametes versicolor* PRL 572, *Trametes versicolor* MUCL 28407, *Pleurotus ostreatus* MUCL 29527, *Pleurotus sajor-caju* MUCL 29757, and *Phanerochaete chrysosporium* DSM 1556 were investigated for their ability to degrade anthracene in soil. The fungi were grown on wheat straw and mixed with artificially contaminated soil. In a heterogeneous soil environment, the fungi have different abilities to degrade PAH, with *Trametes* showing little or no accumulation of dead-end metabolites and *Phanerochaete* and *Pleurotus* showing almost complete conversion of anthracene to 9,10-anthracenedione. In contrast to earlier studies, *Phanerochaete* showed the ability to degrade the accumulated 9,10-anthracenedione while *Pleurotus* did not. In a heterogeneous soil system, the PAH degradation pattern for white-rot fungi can be quite different from that in a controlled liquid system. (Anderson, 1996).

Seven common 3- to 7-ring (R) polycyclic aromatic hydrocarbons (PAH) as well as PAH derived from lignite tar were spiked into 3 soils (0.8 to 9.7% of org. carbon). The disappearance of the original PAH was detected for the freshly spiked soils, for soils incubated for up to 287 d with their indigenous microflora, and for autoclaved, unsterile and pasteurized soils inoculated with *basidiomycetous* and *ascomycetous* fungi. (Gramss et al, 1999). Three to 12 d after spiking, 22 to 38% of the PAH could no longer be recovered from the soils. At 287 d, 88.5 to 92.7%, 83.4 to 87.4%, and 22.0 to 42.1% of the 3-, 4-, and 5- to 7-R PAH, respectively, had disappeared from the unsterile, uninoculated soils. In organic-rich unsterile soils inoculated with wood- and straw-degrading fungi, the degradation of 3- to 4-R PAH was not accelerated by the presence of fungi. In organic-poor unsterile soil, these same fungi delayed the net degradation of PAH possibly for 2 reasons. Mycelia of *Pleurotus* killed most of the indigenous soil bacteria expected to take part in the degradation of PAH, whereas those of *Hypholoma* and *Stropharia* promoted the development of opportunistic bacteria in the soil, which must not necessarily be PAH degraders. Contemporarily, the contribution of the fungi themselves to PAH degradation may be negligible in the absence of soil orgacic matter due to the lower production of ligninolytic enzymes. Thus, fungi degrade PAH irrespectively of their molecular size in organic-rich and wood chip-amended soils which promote fungal oxidative enzyme production.

The biodegradation of anthracene at 15 and 30^{0}C was observed by Lisowska et al (1994). Two *Pseudomonas* strains: *Pseudomonas sp*. 61 (NCIMB 13262) and *Pseudomonas mendocina* (NCIMB 13264) isolated from soil polluted by crude oil, were able to degrade anthracene at both temperatures to the same extent. Additional nitrogen and phosphorus had no influence on the degradation process. Also, results indicate that anthracene is degraded under cometabolic conditions with ethanol in *Pseudomonas sp*. 61 at both 15 and 30°C.

Both microcosm and respirometric experiments have been used in a study of Lors et al (2001) to evaluate the intrinsic degradation of PAHs in soil from a former coke site. The study has been carried out in solid phase over a period of 8 mo, at 20°C, under aerobic conditions, using a soil sample which contained about 1 g/kg dry soil of the 16 PAHs from the USEPA list and about 1 g/kg dry soil of metallic pollutants, such as Zn, Pb, Cu, and Cd. The microbial degradation activity was assessed by monitoring the CO_2-production and O_2-consumption as well as the decrease of the PAH content in the soil samples. Experiments on sterilized samples were also carried out, in order to evaluate the importance of abiotic processes in the degradation. Part of the most volatile and sol. PAHs (2 and 3 ring PAHs) are rapidly

eliminated (within 7 days) by abiotic processes. Afterwards, biological phenomena become dominant in the PAHs degradation contributing to a 25% decrease in total PAH content. This concerned mainly 3- and 4-cycle PAHs which were degraded by 30 and 25%, respectively. Of the 3-cycle PAHs, (e.g. anthracene) were degraded to the highest degree. O_2-consumption and CO_2-production revealed a high microbial activity which is associated with the decrease in the PAH concentrations.

The ligninolytic enzyme system of white-rot fungi is able to partially oxidize polycyclic aromatic hydrocarbons (PAH), yielding better soluble metabolites. This increase in solubility and, hence, bioavailability, may be important for the further oxidation of these PAH-metabolites by bacteria. The biodegradability of anthracene and its oxidation products was tested by Meulenberg et al (1997) with soil as the inoculum. Anthracene was hardly degraded during an incubation period of 60 days. Degradation of the soluble substrate phthalic acid was very fast: 60% conversion was reached within 2 days. O_2 uptake rates with anthraquinone were low and approximately linear in time. Intact cultures and extracellular culture supernatant of the white-rot fungus *Bjerkandera* BOS55 produced anthraquinone as the end-product from anthracene oxidation. Two intermediary formed products are most probably hydroxyanthracene and dihydroxyanthracene. From soil and activated sludge, several bacteria were isolated on anthraquinone as the sole source of C and energy. Incubation of anthracene in culture supernatant of *Bjerkandera* BOS55, to which suspensions of these isolates were added, showed intermediary accumulation of anthraquinone, which was subsequently further degraded. These results clearly show that anthracene can be mineralized by mixed cultures of *Bjerkandera* BOS55 and isolated bacteria.

Two studies were conducted to detect the combined effect of sequestration and laboratory-scale bioremediation on the bioavailability of polycyclic aromatic hydrocarbons in soil. After the compounds had aged for 140-203 days in soil, bacteria capable of degrading the compounds were added, and the availability of the hydrocarbons after bioremediation was detected. Aging decreased the amount of anthracene available to bacteria as shown by increases in the amount of the compound remaining after bioremediation and to earthworms (*Eisenia foetida*) as shown by lower tissue concentrations, percentages assimilated, and bioconcentration factors (Tang et al, 1998, 1999).

Degradation of anthracene at concentrations of 5.10^{-6} mol/dm^3 by 2 single strains of *Bacillus alvei* and *Arthrobacter* species and a defined mixed culture of 7 microbe strains immobilized by sorption on activated C, clinoptilolite, and zeolite 5A was tested. *B. alvei* immobilized on all 3 sorbents removed 66-85% of anthracene whereas the Arthrobacter species and mixed bacterial culture removed anthracene at 66-94% and 54-94%. Highest efficiency was achieved on activated C bed; lowest on zeolite 5A. (Bilyk et al, 1999)

The white-rot fungi *Phanerochaete chrysosporium* ATCC 24725, *Pleurotus ostreatus* ATCC 32783, *Lentinus edodes* ATCC 24462, and *Trametes versicolor* ATCC 42530 were studied for their ability to degrade anthracene. One hundred percent of 15 ppm anthracene was degraded in 10 days by both *P. chrysosporium* and *T. versicolor*. At 40 ppm anthracene inhibited the mycelial growth of *P. chrysosporium*. (Kim et al, 1998)

A study by McNally et al (1998) presents evidence for the biodegradation of three- and four-ringed PAHs (incl. anthracene) under strict anaerobic, denitrifying conditions. Three pseudomonad strains, isolated from contrasting environments, were used in this study. All three strains were known PAH degraders and denitrifiers. Degradation proceeded to

nondetectable levels (<0.001 mg/L) in 12-80 h for anthracene. The rates of anaerobic degradation were typically slower than under aerobic conditions, except for strain SAG-R which had similar removal rates for all three and four-ring PAHs. Denitrification activity was verified by monitoring nitrate utilization and nitrous oxide production. Although none of the pseudomonads were adapted to the denitrifying conditions, only the pseudomonad isolated from a noncontaminated site (strain KBM-1) consistently exhibited an adaptation period, which approximated 12 h. This study supports growing evidence that the degradation of aromatic hydrocarbons coupled to denitrification may be an important factor affecting the fate of these compounds in natural and engineered systems.

The use of white-rot fungi has been proposed for bioremediating polycyclic aromatic hydrocarbon (PAH)-polluted sites. In order to enhance their bioavailability, PAH solubilization can be increased in water/solvent mixtures. The oxidation of anthracene in the presence of cosolvents by the white-rot fungus, *Bjerkandera* strain BOS55 was studied. Acetonne and ethanol at 5% were toxic to this fungus when added at the time of inoculation. However, when solvents 20% (vol./vol.) were added to 9-day-old cultures, ligninolytic activity as indicated by Poly R-478 dye decolorization and anthracene oxidation was evident for several days. Since 20% solvent was toxic to cells, the oxidation of anthracene can be attributed to extracellular peroxidases, which were shown to tolerate the solvent. Solvent additions of 11-21% (vol./vol.) acetone or ethanol increased the rate of anthracene bioconversion to anthraquinone in liquid medium by a factor of 2-3 compared to fungal cultures receiving 1-3% solvent (Field et al, 1995).

A ligninolytic fungal strain, F 898, was grown in media containing and anthracene as C source. Various levels of lignin peroxidase (LiP), manganese peroxidase (MnP) and laccase activities were detected following growth for 3, 6, and 10 days. Regardless of the C source, however, MnP activity levels were higher than LiP and laccase activities. Degradation of the aromatic compounds, measured by reversed-phase high-performance liquid chromatography (HPLC) on a C18 column, varied with each C source. About 27% of the anthracene was degraded. (Clemente et al, 1999).
The study of Binet et al, (2000) investigates the dissipation of a mixture of eight PAHs, ranging from 3 to 6 rings, in the rhizosphere of ryegrass (*Lolium perenne L.*). Two pot experiments were conducted with or without plants using soil spiked with 1 g/kg of PAHs in a growth chamber. The first experiment was carried out shortly after spiking and the second after 6 months of ageing. At the end of both experiments, the extractable concentrations of all PAHs were lower in rhizospheric than before, but was still significantly higher in the rhizospheric soil. Total culturable microflora were higher in the rhizospheric than non-rhizospheric soil, but was at the same level in spiked and non-spiked soil. The number of PAHs degraders, established by a modified MPN procedure, was not significantly different in the freshly spiked rhizospheric and non-rhizospheric soils, but was significantly higher in the rhizosphere of the aged spiked soil than non-rhizospheric soil. PAH dissipation was lower after soil ageing.

Mineralization

The comparative mineralization of eight polycyclic aromatic compounds in five soils collected from an abandoned coal tar refinery in eastern Ohio was detected. The soils showed differences only in total extractable hydrocarbon content of the soil chem. characteristics measured. The compounds studied included five polycyclic aromatic hydrocarbons (icluding anthracene). Mineralization was measured by serum bottle radiorespirometry. . Two of the soils with eight to 15 times the hexane-extractable hydrocarbon content consistently showed

more rapid initial rates and higher overall extents of mineralization compared to the other three soils. Overall extents of mineralization ranged from 10 to 60% for anthracene, after 64 days. (Grosser et al, 1995).

Experiments were carried out to evaluate the impact of the addition of ripe compost on the degradation of 2 14C-labeled hydrocarbon model compounds (incl. anthracene) in soil. The addition of mature compost (20% dry wt./dry wt.) stimulated significantly the disappearance of the extractable fraction of anthracene. With compost, 23% of the labeled anthracene was transformed into $14CO_2$ and 42% was fixed to the soil matrix irreversibly. In the unsupplemented control reactor more than 88% of the original anthracene could be recovered. Compost could stimulate the depletion of hydrocarbons by either mineralization or the formation of nonextractable bound residues (humification). The latter process might be a significant route of depletion in soil especially, for those hydrocarbons that are mineralized only slowly. (Kaestner et al, 1995)

Anthracene was mineralized in the soils after 64 days as previous detected from Grosser et al (1995). Extents of mineralization by indigenous soil microbiota appear to be more dependent on the chemical characteristics of the soil and not total biomass and activity. Cultures capable of degrading anthracene were obtained following enrichment techniques. A *Mycobacterium sp*. capable of degrading these compounds was isolated and reintroduced into two of the soils, resulting in mineralization enhanced above that of the indigenous soil microbial population. These data indicate that the future success for bioremediation methods relies on the characterization of environmental parameters affecting microbial degradation as well as the isolation of microbial populations that can reduce toxicity in the environment.

3.1.3.1.3 Atmospheric degradation

A relative-rate method with gas chromatography and long-path differential optical-absorption spectroscopy was used to detect rate constant (1.3 x 10^{10} cm^3/mol. s) at 298 K. At an assumed OH bulk concentration of 1 x 10^6molecule/cm^3, these rate constant lead to atmosphere lifetimes of approximately 2 h (Biermann, Heinz W., et al., 1985).

The rate constant for the vapour-phase reaction of anthracene with photochemically produced hydroxyl radicals has been measured to be 1.12 x 10^{-10} cm^3/molecule.sec at 52 deg C. This corresponds to an atmospheric half-life of about 3.4 hours at an atmospheric concentration of 5 x 10^5 hydroxyl radicals per cm^3 (Atkinson et al, 1989).

K_{OH} for anthracene is 1.30.10^{-10} as the reaction rate is recalculated to match the reaction rate constant for reaction with NO_3 radicals, (EC, 2001b), Slooff *et al*. (1989).

Gas-particle phase partitioning (EC, 2001) for anthracene gives vapour pressurePa (a) x $10^{-4.}$ The lifetime of anthracene with respect to gas-phase reaction with hydroxyl (OH) radicals, nitrate (NO_3) radicals and ozone (O_3) (EC, 2001b) was 2.1 hours for summer and 10 hours for winter.

Summary of gas-particle phase partitioning given by EC, (2001) for anthracene which has 3 rings is 3% (measurements made in Oslo, January/February 1979, Thrane and Mikalsen, 1981) and 0.5% (measurements made in Torrance, California, February 1986, Arey *et al*.,

1987, with a vapour pressure of 8.7 x 10^{-4} Pa and Sonnefeld et al. 1983 with a vapour pressure of 9.4x 10^{-4} Pa (in 25°C), that it has been adopted in the present RAR.

Representative lifetimes of anthracene with respect to gas-phase reaction with hydroxyl (OH) radicals, are 2.1 hours for summer and 10 hours winter calculated using rate coefficients summarised by Atkinson and Arey (1994) and Brubaker and Hites(1998). 24 hour-average summer and winter OH concentrations of 1 x 10^6 molecule cm^{-3} (0.04 pptv) and 2 x 10^5 molecule cm^{-3} (0.008 pptv) assumed for boundary layer UK (Collins *et al*., 1995).

3.1.3.1.4 Summary of environmental degradation

The rate constant for the vapour-phase reaction of anthracene has been measured to be 1.12 x 10^{-10} cm^3/molecule.sec at 52 deg C. This corresponds to an atmospheric half-life of about 3.4 hours at an atmospheric concentration of 5 x 10^5 hydroxyl radicals per cm^3. These values will be used in EUSES analysis.

Using TGD, half lives for anthracene are calculated:

In surface water: 150 days
In bulk soil: 3,000 days
In sediment: 30,000 days

Suggested half-life classes of PAHs in various environmental compartments according to Mackay et al., 1992 are:

Class	Half-life (h)	
	Mean	Range
1	17	10-30
2	55	30-100
3	170	100-300
4	550	300-1000
5	1700	1000-3000 (42 -125 days)
6	5500	3000-10000 (125 – 420 days)
7	17000	10000-30000 (420 – 1250 days)
8	55000	> 30000

For anthracene is:

Water	Soil	Sediment
4	6	7

According to Mackay et al (1992), half lives of anthracene are 300-1000 hours for water, 125-420 days for soil and 420-1,250 days for sediment.
According to Lee et al, (1998), anthracene released to the ground would sorb to the soil. It would be minimal problem for groundwater contamination. Anthracene would be subject to biodegradation in the ground. It would not hydrolyze due to the lack of functional groups. The half-life for biodegradation of terrestrial released is about 100 to150 days. The strong adsorption to particulate matter makes it less bioavailable. Most of the anthracene released to the atmosphere would volatilize or sorb to particulate matter. It would be subject to photolysis immediately. It has very short half-life associated with atmosphere fate. It is on the order of

hours to days depending on the degree of adsorption. Non-adsorbed compounds will have a shorter half-life than adsorbed compounds. It is broken down via ozonation and hydroxide radical photolysis. Half-life in surface water – borne export experiment was 27.44 day and for volatilization 13.18 days. An estimation of 95% clean up in the environment gives a period of 53 months for anthacene.

However, experimentally determined half-lives in sediments exhibit a wide range in reported values from days to months. Results indicate that anthracene can be regarded as inherently biodegradable (in the general sense, rather than in relation to specific inherent biodegradability tests). There are no standard ready biodegradability tests with maybe the exception of Tabak et al study from 1981. Although the overall study involved the adaptation of the culture through sequential subcultures, the initial exposure over seven days involved the use of an unadapted inoculum derived from settled domestic waste water. The concentrations of anthracene used were a little lower than would be recommended in the standard tests. There is no information on the level of inoculums used. The substance was added dispersed in a solvent and no solvent control appears to have been used. For these reasons it is not possible to determine whether the test shows significant degradation of anthracene. At the PBT-WG-meeting in June 2003 the following was agreed. “New data provided by industry confirmed the P-criteria”. On the basis of the above, even anthracene would appear to meet the screening vP criterion.

3.1.3.2 Distribution

Sonochemical degradation of 3 polycyclic aromatic hydrocarbons (incl. Anthracene) in aquatic solution was examined. Additional chemicals used were benzoic acid, fulvic acid, humic acid, O_2, and N. All PAH exhibited first order kinetics for degradation in pure water. When benzoic acid, humic acid, or fulvic acid were added to the solution, the decay rate constant decreased with increasing concentration of added aid. Decay rate constant for anthracene with O_2 saturation was not statistically distinguishable from that of air-equilibrated solutions. (Laughrey et al, 2000)

Based on a classification scheme (Swann et al., 1983), measured soil Koc values ranging from 2600 (Hu et al., 1995) to 8600 (Brusseau, 1993) indicate that anthracene is expected to have slight to no mobility in soil.

Estimation of removal of anthracene in STP according to EUSES 2.0 is 1.5% to air, 25.2% to water, 73.1% to sludge, 0.2% degraded and 74.8% removal

3.1.3.2.1 Adsorption

Anthracene is expected to absorb to suspended solids and sediment in water (USEPA, 1987). In order to determine the Koc for PAHs we propose to use the equation of Karickhoff *et al.* (1979): $\log K_{\text{oc}} = \log K_{\text{ow}} - 0.21$

The data for monoaromatic compounds and PAHs for sediments (Karickhoff et al., 1979) but also for soils (Karickhoff, 1981) fit well to this equation. Similar results are presented for PAHs by other authors by means of the most appropriate techniques (De Maagd et al., 1998a). For anthracene this gives a log Koc value of 4.47

3.1.3.2.2 Volatilisation

Henry constant of anthracene is 4.3 Pa m^3/mol at 25^0C according to Mackay, 1992. This value is used in RAR for CTPHT and we agree with them.
Volatilisation of anthracene from moist soil surfaces may be important given a measured Henry's Law constant of 4.88X10-5 atm-m^3/mole (Alaee et al., 1996). However, adsorption to organic matter may attenuate this process. Anthracene is not expected to volatilize from dry soil surfaces based on an extrapolated vapor pressure of 2.67X10-6 mm Hg (Scala, 1982).

Anthracene is expected to volatilize slowly from water. Estimated volatilization half-lives for a model lake is 1.2 and 13 days, respectively. However, volatilization is expected to be attenuated by absorption to suspended solids and sediment in the water (Lyman et al, 1990)

The volatilization half-life of an environmental pond 2m deep is estimated to be about 12.76 days, ignoring absorption. When considering maximum absorption the volatilization half-life increases to16.7 months (USEPA, 1987)

3.1.3.3 Accumulation and metabolism

The quality of data in studies has been evaluated according to Klimisch et al (1997) study. The approach proposes to indicate a measure of the study/data reliability with the following categories:
Validity 1: Reliable without restrictions
Validity 2: Reliable with restrictions
Validity 3: Not reliable
Validity 4: Not assignable

Bluegills (*Lepomis macrochirus*) were exposed to 14C-labeled anthracene in water. Rates of uptake and biotransformation within the fish were followed by 14C counting and thin-layer and liquid chromatography. The initial uptake-rate coefficient for anthracene (KU = 36/h) was independent of exposure concentration. The presence of dissolved humics did not affect anthracene. Biotransformation of the anthracene was constant at 0.22 nmol/g/h, with approximately 92% of the residue unmetabolized at 4 h. 6% of the anthracene was found in liver and gall bladder. Depuration rates were 1st-order and yielded half-lives of 17 h for anthracene. The estimated bioconcentration factors (BCF) for anthracene in whole fish (K_U/K_D) were 900, (Validity 2) for total 14C activity, but only 675 for parent material (Validity 3). The predicted BCF was 1,209. The bioconcentration factors for various tissius were: 1,890 for gall bladder, 561 for liver, 641 for viscera, 555 for brain and 42 for carcass. These BCFs were considerably lower than those predicted from the octanol-water partition coefficient because of biotransformation. (Spacie, et al, 1983). (Validity 2)

The bioaccumulation of 5 PAHs in fathead minnows (*Pimephales promeals*) was studied in a static experimental set-up according to the so-called ‘adjusted Banerjee method’ (De Maagd, 1996; chapter 4). This study was designed to quantify the role of biotransformation in the bioaccumulation process. PAHs were added to tap water by a generator column. Fish (7-11) of on average 0.52 g were added to an aquarium with 1.5 L of water. The concentrations of the parent compounds in both water and fish in the static systems were analyzed using HPLC

during 48 hours on 7 to 11 fish in time. Fish were fed daily until two days prior to the experiment. In the modelling of the concentrations, the amount of fish and the volume of the water were adjusted every time a sample was taken. The bioaccumulation with and without biotransformation was determined by running parallel tests with and without the addition of piperonyl butoxide (PBO), a known biotransformation inhibitor for substrates binding to the site of cytochrome P450-isoenzymes. To distinguish between loss due to abiotic processes and biotransformation controls without fish were used as well. The uptake rate determined from the concentration in fish was not in accordance with the uptake rate determined from the decrease of concentration in the water phase. Therefore, the recovery of anthracene from fish exposed via water had to be slightly adjusted downwards in comparison with homogenized fish spiked with the PAHs dissolved in hexane (18 %).The calculated BCF values in the absence of PBO were 6800 for anthracene. The amount of fish in the aquarium (more than 3 grams/L) is three times more than the upper limit of what is recommended in OECD guideline 305. However, in the modified Banerjee method this amount of fish is necessary to reduce the water concentration in such a way that the uptake can be modelled from the concentration in water (Validity : 2) (RAR CTPHT 3230705env).

In case of a test performed by De Voogt et al. (1991. the Banerjee method was used, but not the adjusted Banerjee method, which implies that the concentrations in fish were not determined. Solutions of the PAHs in 1:1 tap water:deionised water were prepared by the generator column technique. The tested concentrations were about one tenth of the experimental LC50. Guppies (Poecilia reticulata; 0.135 g) were added to test vessels with 3 liters of water at a load of 1 to 4 gram fish per liter. Water samples were taken at 8 time points during 48 hours. Controls without fish were run to check the loss of compound due to other factors than accumulation in fish. It is not clear if the fish were fed or not. Concentrations in water were analyzed using HPLC.

BCFs were calculated from the uptake and depuration rate constant which were both determined from the decrease in aqueous concentrations during the uptake phase. The BCFs were 2230 ± 490 L/kg for fluorene, 7260 ± 2110 L/kg for anthracene and 4810 ± 2860 L/kg for pyrene. Without determination of the concentration of the parent compound in fish metabolism can not be taken into account. (Validity : 3) .

Moreover, for comparison a regular semi static (renewal) bioaccumulation test was carried out. In this test, 2 liter of solution in the test vessels was renewed every 12 hours for pyrene and anthracene and every 24 hours for fluorene. Eight to ten guppies were placed in each test vessel leading to a load of about 0.5 to 0.7 gram fish per liter. For pyrene two guppies were sampled on days 1, 3, and 7. For fluorene and anthracene two guppies were samples on days 1, 2, and 4. PAHs were analyzed using HPLC. The BCF was determined by dividing the final concentration in fish by the average concentration in water during the last renewal period. These BCF values are 1050 L/kg for fluorene, 4550 L/kg for anthracene and 11300 L/kg for pyrene. (Validity : 2 ; preferred values) .

With the concentration at the end of the static experiment according to the Banerjee method together with the corresponding initial and final aqueous concentrations, a mass balance could be made. The mass balance was 125% for fluorene, 101% for anthracene and 62% for pyrene. BCF values determined as the concentration in fish divided by the concentration in water at the end of the static experiment are not given in the study but can be derived from the presented data. These BCF values after 48 hours of exposure would be 3500 L/kg for fluorene, 6000 L/kg for anthracene and 2700 L/kg for pyrene. (Validity : 2), (RAR CTPHT R3230705env.

In the study by Djomo et al. (1996), the uptake and elimination of four radiolabelled PAHs by zebrafish (*Brachydanio rerio*) was studied. The used experimental design differs at some points from the OECD guideline 305 (OECD, 1996). The study was performed in the

presence of sediment in a static system. The four tested PAHs were for more than 90% sorbed to this sediment containing 0.95% organic carbon. Only one concentration was tested. This concentration was much higher than the natural background concentration of the sediment. The sediment/water system was allowed to equilibrate for 48 hours, which is claimed to be enough to reach equilibrium between the two phases.
“The loading of the fish is at the upper limit of the recommended range from the OECD 305 guideline, i.e. 1.0 gram of fish per liter water. However, water concentrations were nearly constant during during the exposure period, which may be attributed to the buffering capacity of the sediment present in the test system. The uptake period lasted for 30 days. Fish that were exposed for 24 hours to the PAHs were used in a depuration experiment in clean water. Uptake an elimination rate constants were determined. The BCF could be determined as the quotient of these two constants. The resulting BCFs are 10400 L/kg for anthracene, 13400 L/kg for phenanthrene, 4300 L/kg for pyrene, and 3600 L/kg for benzo[*a*]pyrene. However, there are two reasons why the BCFs determined in this way can not be considered as reliable.
First, after a strong initial uptake, the uptake curves showed a decrease in the concentration in fish after 1 day to a more or less constant level after 20 days. Therefore, the uptake rate constants that are determined from the initial part of the curve (not specified, but probably from the 7 time points in the first 24 hours), could be erroneous and are probably too high. If the constant levels from 20 to 30 days are used and the BCF is calculated as the quotient of the concentrations in fish and water, the resulting BCFs are 33 L/kg for anthracene, 8 L/kg for phenanthrene, 11 L/kg for pyrene, and 60 L/kg for benzo[*a*]pyrene.
Second, all concentrations in water and fish were from total radioactivity determined by liquid scintillation counting (LSC). This may lead to an overestimation of the BCF, because metabolites of the PAHs formed in the fish contribute to the total radioacitivity as well as the parent compound.
For these reasons the BCF values that are deduced from this study by dividing the uptake rate and the elimination rate constant are considered to be not valid.” (Validity : 3) (RAR CTPHT 3230601env).
Goldfish, reared in aquarium containing 1 mg/l of 17 different aromatic compounds e.g. anthracene accumulated the compounds at 0.017-0.8 ppm. Uptake of the compounds is being correlated (0.98) with their water-oil partition coefficients in a linear relation. BCF was measured at 162. (Ogata, et al, 1984). (Validity 3)

The concept of “Environmental Hazard Profile” developed at the study of Freitag et al (1985) has been tested with 100 ^{14}C-labelled organic compounds. Concentration factors in activated sludge, in algae and fish were determined. The microbial degradation of the chemicals to CO_2 in activated sludge and the decomposition to CO_2 under artificial light were determined. Ranking of compounds is given in the order of falling concentration factors and accumulation in rats respectively, and decreasing rates of decomposition. Relationship between chemical structure and accumulative and degradative behaviours is demonstrated using some selected groups of chemicals, such as benzenes, phenols, biphenyls and polyaromatic hydrocarbons. Correlations between the octanol/water partition coefficient, concentration factors and rates of decomposition could be established. Evaluation of test compounds was possible using hazard profiles obtained by the sum of single test results. BCFs for various organisms were: 7,770 for algae, 910 for fish and 6,700 for activated sludge. The % retention for rat was 0.7). (Validity 4)

Rainbow trout (*Salmo gairdneri/Oncorhynchus mykiss*) were exposed for 72 h to 14C-labeled anthracene alone and in an oil shale retort water. Tissue levels of anthracene were analyzed at 24, 48, and 72 h to detect nonsteady-state bioconcentration factors (BCFs), and uptake and

depuration rates were calculated from anthracene disappearance in exposure waters and metabolite appearance in depuration waters. Uptake rates (14.6-16.6 h) were similar to previously reported values; however, depuration rates (0.00158-0.00188 h) were very low. Consequently, measured nonsteady-state BCFs after 72 h (9000-9200) (k_u/k_d 14.6/0.00158) for anthracene were higher than expected, probably because fish were not fed and had low excretion rates. Measured and estimated anthracene BCFs were lower in retort water exposures than in single-compound exposures because of slower uptake and faster depuration. Slower uptake during retort water exposures was attributed to either decreased bioavailability of anthracene or rate-limiting transport of contaminants from uptake sites to storage and processing sites. Induction of mixed-function oxidases may have increased depuration from fish exposed to retort water. Thus, bioconcentration of individual compounds from complex chemical mixtures may be difficult to predict based on single-compound kinetics. (Linder et al, 1985). (Validity : 3)

Adult rainbow trout were exposed to a single oral dose of 8 PAHs, and fish were sampled at intervals between 5 and 48 days after exposure. Regression analyses on whole fish indicate levels declined significantly for 4 of the compounds monitored. The biological half-live was established as 7 days for anthracene (Niimi, A.J. and Palazzo, V., 1986). (Validity : 4)

The log of octanol/water partition coeff. (Kow) values for numerous organic chemicals were presented in Geyer et al (1984) publication. These values covered a wide range of values to enhance the potential utility of the correlation to predict bioaccumulation potential. There was an obvious linear relation between lipophilicity expressed as log Kow and log bioaccumulation potential of organic chemical in *Chlorella fusca*. BCF was measured at 7,770. (Validity : 3)

The effect of binding of polycyclic aromatic hydrocarbons (PAHs) to dissolved humic material (DHM) on the uptake and bioaccumulation of PAHs is examined in the cladoceran *Daphnia magna*. To the extent that a PAH binds to DHM, which is related to its hydrophobicity, it becomes unavailable for uptake by the organisms. The structure-activity relationship developed here suggests that the high affinity for binding to DHM and the resultant decrease in bioavailability might mitigate the biological impact of those very hydrophobic contaminants having the greatest potential for bioaccumulation and transfer to man via food chains.(Mc Carthy et al, 1985) (Validity : 3)

The bioaccumulation potentials by aquatic biota from aquatic solution were detected for 7 polycyclic aromatic hydrocarbons (PAH). The PAH tested using *Daphnia. Pulex*. Bioaccumulation kinetics were described as a 1st order approach to equilibrium in a 2-compartment model (water and *Daphnia*), using a 2-stage technique to est. uptake and elimination rates, while accounting for decreasing aquatic concentrations. The calculated n-octanol-water partition coefficient was a good predictor of bioaccumulation potential of PAH in *Daphnia*. Estimations of equilibrium concentration factors obtained by the two methods were in good agreement, and increased dramatically with increasing molecular weight within the series of compounds. The calculated *n*-octanol-water partition coefficient was shown to be a good predictor of bioaccumulation potential of PAH in *Daphnia*. PAH were concentrated from a high of about 10,000-fold for benz(a) anthracene to a low of about 100-fold for naphthalene. (Southworth, G. R. et al, 1978). (Validity : 4)

Daphnia pulex accumulated anthracene rapidly from aromatic hydrocarbon-contaminated water. At a time of approximately 4 h, Daphnia had accumulated 760 times the concentration of anthracene in the water. The total aromatic hydrocarbon excretion data indicated a 3 compartmental system of excretion in *Daphnia*, a rapidly eliminated compartment (apprx.30%), a more slowly eliminated compartment (apprx.60%), and a tightly bound residue of 8%. (Herbes and Risi, 1978). (Validity : 4)

Uptake, depuration, and biotransformation rates of 14C-labeled anthracene were detected for *Pontoporeia hoyi*, the dominant benthic invertebrate in the Great Lakes, at 4, 7, 10, and 15°C. The uptake rate constants for anthracene increased from 136/h to 215/h over the temperature range studied and were seasonally dependent. The depuration rate constant at the apparent optimum temperature of 7°C was 0.015/h. The biotransformation ability of *Pontoporeia hoyi* is low, and degradation of anthracene was undetectable even after exposures of 48 h. The bioconcentration factor can be predicted from the uptake and depuration kinetics to be approx. 16,800 at 4°C. (Landrum, P. F. 1982). (Validity : 3)

The acute toxicity of anthracene to the glochidial larvae of the paper pondshell (*Utterbackia impercillis*) was characterized in the lamboratory using two sets of experiments. Mean BCF factors across all treatment were 346±166. The highest BCF factor observed was 420 at 0.0026mg/l. (Weinstein and Polk, 2001) (Validity : 2)

Uptake, depuration and biotransformation rates of selected unsubstituted polycyclic aromatic hydrocarbons in *Stylodrilus heringianus* were determined in Frank et al studie (1986). There were compared to rates for other bethnic organisms, particularly *Mysis relicta* and *Pontoporeira hoyi*. Uptake from lake and sediment from lake Michigan, where Stylodrilus heringianus is an abundant ologochaete, was determined. Indipented sediment uptake experiment involved two sediment particle size matrices. Depuration rate constants ranged from 0.013 to 0.020 h^{-1}. Biotransformation over 48 h was negligible. Bioconcentration and PAH uptake relative to oxygen uptake were peridicted. (Validity : 3)

All 6 hydrocarbons tested were excreted from the gills of Dolly Varden char (*Salvelinus malma*), although less of the largest and least polar compounds were excreted. Approximately equal amounts of the administered 14C-labeled phenol, cresol, and toluene were excreted from the gills, but the amount decreased for naphthalene and even more so for anthracene (1.0%). The size of the hydrocarbon appeared to be a more important factor in gill excretion than partition coefficient (log of octanol/water partition). Only small amounts of administered C-14 (1.9%) of anthracene were recovered. The tissue distribution of the C-14 at 24 h was the result or summation of several processes occurring simultaneously but at different rate for each compound and process (Thomas and Rice, 1982). (Validity : 3)

The fingerprint of 14 polycyclic aromatic hydrocarbons (PAHs) was investigated in biopsy, fur, blood, liver and feces of live and dead specimens of two Argentinean populations of southern sea lion (*Otaria flavescens*). One colony lives in Mar del Plata harbor, which is particularly polluted with petroleum, the second (control) colony lives at Punta Bermeja (Patagonia). The highest concentrations of the five carcinogenic PAHs were found in the Mar del Plata sea ions. Anthracene was detected in the fur of seal lions at 59.81 and 14.29ng/ml fresh weight, from Mar de Plata and Punta Bermeja respectively. In blood anthracene was detected in average concentrations of 81.32 and 110.3 ng/g dry weight and in liver at average concentrations of 0.30 and 10.57 ng/g flesh weigh, from Mar de Plata and Punta Bermeja respectively. (Marsili, L. et al, 1997). (Validity : 3)

According to the Technical Guidance Document, the bioconcentration in earthworm can be described as a hydrophobic partioning between the pore water and the phases inside the organism. This equilibrium partioning approach (EP) can be modelled according to the following equation as described by Jager (1998):

$$BCF_{earthworm} = (0.84 + 0.012K_{ow}) / RHO_{earthworm}$$

where for $RHO_{earthworm}$ by default a value of 1 ($kg_{wwt}.L^{-1}$) can be assumed.

The BCF values in earthworm for anthracene calculated based on the equation described in the EU TGD (2003) was 580 for a Log Kow of 4.68.

BCFs for various species are shown in table 3.20. Only studies with an observed or calculated result are presented in the table.

Table 3.20: BCFs for various species (fish, Mollusca, Crustacea, Algae, Insecta, Oligochaeta)

Species	**BCF**	**Test system**	**Type**	**Val.**	**References**
Fish					
L. macrochirus	900	S	k_1/k_2 (total)	2	Spacie *et al*. (1983)
P. promelas	6760	S	k_1/k_2 (parent)	2	De Maagd *et al*., 1996
P. reticulata	4550 (pref)	R	Equi (parent)	2	De Voogt *et al*. (1991)
P. reticulata	6000	S	Equi (parent)	2	De Voogt *et al*. (1991)
B. rerio	10400	S	k_1/k_2 (total)	3	Djomo *et al*. (1996)
L. macrochirus	675	S	k_1/k_2 (corrected)	3	Spacie *et al*. (1983)
O. mykiss	9000-9200	R	k_1/k_2 (parent)	3	Linder *et al*. (1985)
O. mykiss	779	R	Equi (parent)	3	Linder *et al*. (1985)
P. reticulata	7260	S	k_1/k_2 (parent)	3	De Voogt *et al*. (1991)
Salmo gairdneri	9,000-9,200				Linder et al. 1985
Salmo gairdneri	143µg/fish	OECD			Nimi et al. 1986
Cyprinus carpio	1,660-2,820	OECD			Japan Chemical industry 1992
Cyprinus carpio	903-2,710	OECD			Japan Chemical industry 1992
Carassius auratus	162	S		4	Ogata et al. 1984
L. melanotus	910	S	k_1/k_2 (unclear)	4	Freitag et al, (1982)
Mollusca					
U. imbecilis (larv.)	345 (highest 420)	R	Equi (parent)	2	Weinstein & Polk, 2001
Macona balthica (marine bivalve)	260	F		3	Clement et al., 1980
Anodonta imbecilla (clam)	Little to no biotrasformation	S		2	Giesy et al., 1982
Crustacea					
Daphnia magna	970	B		3	Newsted and Giesy, 1987
Daphnia magna	511	S		3	Mc Carthy et al., 1985
Daphnia pulex	1,192			4	Southworth, 1978
Daphnia pulex	1,085	S		4	Southworth, 1978
Daphnia pulex	988	S		4	Southworth, 1978
Daphnia pulex	917	S		4	Southworth, 1979
Daphnia magna	319-607	F		2	Leversee et al., 1982
H. azteca	1800-10985	F	k_1/k_2	3	Landrum & Scavia (1983)
P. hoyi	16857-39727	F	k_1/k_2	3	Landrum (1982, 1988)
Algae					
Selanastrum capricornutum	5100-10500	S		3	Mailhot et al. 1987
Chlorellafusca var.vacuolata	7,770	S		3	Freitag et al 1985

Species	BCF	Test system	Type	Val. *	References
Insecta					
Chironomus riparius	804-1,915	F		2	Geisy et al. 1982
Oligochaeta					
L.Variegates	1,369	F	Equi	3	Ankley et al. 1997
Stylodrillus heringianus	5,051	F	k_1/k_2	3	Frank et al. 1986
Activated sludge	6,700	S		3	Freitag et. al 1984,1985

F: flow-through system, R: static renewal system, S: static exposure system, B: Batch test, , k_1/k_2 kinetic : uptake rate/depuration rate , Equi : equilibrium, Val. : Validity
OECD: OECD Guide-line 305 C
* by Klimisch et al, 1997

Summary

Several studies in fish gave BCF values > 5,000. Highest observed or calculated values for bioconcentration factor in fish were 4,550, 6,000 and 6,760 that are valid (validity marking of 2) (Table 3.20). These values indicate that anthracene, has a high or very high bioaccumulation potential. Consequently, there is sufficient evidence that anthracene does meet the B and vB criterion, as it has also been conluded by PBT working group..

3.1.4 Aquatic compartment (incl. sediment)

3.1.4.1 Calculation of predicted environmental concentrations (PEC_{local})

The PECs for anthracene in the aquatic compartment have been calculated according to the methods in the Technical Guidance Document. Site specific information has been used where available to calcualate the PECs for the risk assessment. Default values are shown in the text only for informative purpose. They are not always in agreement with the site specific data.

3.1.4.1.1 Calculation of PEC_{local} for production, for formulation and for processing.

Water

The PEC_{local} for water has been calculated using the equation in the Technical Guidance Document where full details of the calculation can be found. In the absence of other

information, it is assumed that the release occurs to a Waste Water Treatment Plant (WWTP). The WWTP is assumed to have the characteristics of a plant serving a population of 10,000 inhabitants. The default values used in the calculation are appropriated to this WWTP and are obtained from the Technical Guidance Document. The $PEC_{local\ (water)}$ and C_{eff} are calculated as follows:

$PEC_{local\ (water)}$ = $C_{eff} / [(1 + K_{p(susp)} . c_{susp}) . D]$

where:

$PEC_{local\ (water)}$	=	Predicted environmental concentration (g/l)
C_{eff}	=	Concentration of the chemical in the WWTP effluent (g/l)
$K_{p(susp)}$	=	Solid – water partition coefficient of suspended matter (2.3 x 10^3 l/kg)
c_{susp}	=	Concentration of suspended matter in the river (kg/l)
	=	(default = 15 mg/l)
D		Dilution factor (default = 10)

Anthracene is estimated as not readily biodegradable under aerobic conditions and under denitrifying conditions while it is not easily biodegradable under anaerobic conditions. For this risk assessment, it has been assumed that anthracene is not readily biodegradable. Using the Tables in Appendix II (Elimination rates for substances in the wastewater treatment plant based on the SIMPLETREAT model), log K_{ow} of 4.68 and Henry Law 4.3 x 10^{-2} Pa.m^3. mol^{-1} it has been assumed that anthracene is inherent biodegradable and its fate in the waste water treatment plant is: 1.5% to air, 25.2% released to water, 73.1% absorbed to sludge, 0.2% degraded. 74.8% is thus removed in the waste water treatment plant before release into surface water. The above equations have been used to calculate PEC_{local} for the water compartment based on the release of anthracene during production. The $PEC_{effluent}$ is equivalent to the $PEC_{local(surface\ water)}$ before dilution. A dilution rate of 10 is used so values for $PEC_{effluent}$ have been calculated and are in Table 3.21. In some cases the dilution rate will be much greater, but in the absence of any specific information on dilution factor the default value of 10 has been used. No specific data about emission rates of anthracene to waste water in different uses of anthracene have been found.

Table 3.21 : Local PECs calculated for water

Process	$PEC_{surface\ water\ (dissolved)(dissolved)}$ µg/l
Production of anthracene	1.23 x 10^{-5}
Producrion Production of pyrotechnics (form.)	3.62 x 10^{-2}

A re-calculation with new revised data from industry is as follows:
Influent: 19 kg/a
Remaining in water: 25.2 % = 4.9 kg/a ➔ ~14 g/d
According to Simple Treat: Water flow = 2000 m^3/d
Ceff = 14 g/2000 m^3 = 0.007 g/m^3 = 7 µg/L
Dilution factor: 10
Clocal = PEC = 0.7 µg/L

Moreover, based on more site-specific data, an effluent concentration of 1.3 µg/L results, which by using the dilution factor of 10, would give a local PEC of 0.13 µg/L. This is directly after the on-site WWTP. But as in this case a second WWTP is down-stream before discharged into the surface-water (river), anthracene is eliminated below detection limit.
The previous emission data of <1kg/a represented the upper limit from early operation of the new on-site biological WWTP (2001 – 2003); it was taken as a “worst-case” value that could always be met throughout the year with absolute certainty. All later measurements confirmed that the effluent levels of anthracene resulted in distinctly lower annual emissions: The scenario of tables is based on more recent measurements that became available to the Rapporteur of Pitch (NL). The latest measurements of 2006 (mean 32.5 g/a) displayed in Tables indicate that the previous amount of 37 g/a is fairly reproducible and therefore a reasonable annual estimate.
In essence, the default dilution factor of 10 is much higher, and the recipient water is no natural river but a huge sewage canal which still passes a public WWTP before arriving at the final surface water (river). In reality, the overall dilution factor of the effluent after the anthracene production Industrial Plant until the surface water (final river) is about 100,000.

Overview of recent site-specific data as inputs for PEC estimation:

		after WWTP 1 (on-site Industry)		after WWTP 2 (off-site Community)	
		Effluent Conc. (WWTP) [µg/L]	PEClocal [µg/L] DF = 10	PEC [µg/L]	Comment
Process time	Whole year	--	--	--	related to tar distillation and processing
Waste-water flow (WWTP 1)	750,000 m^3/a	--	--	--	
TOTAL ANTHRACENE EMISSION	~37 g/a	~0.049	0.049/10 = 0.005	0.00011 ng/L	

The value of 0.00011 ng/l will be used in further calculation of risk assessment.

Sediment
The values for the $PEC_{local(water)}$ can be used to calculate the $PEC_{local(sediment)}$ using the formula in the Technical Guidance:

$$PEC_{local\ (sed)} = K_{susp\text{-}water} \times PEC_{local\ (water)} \times 1000 / RHO_{susp}$$

Using this formula and the $PEC_{local(water)}$ calculated above, the $PECs_{local(sed)}$ have been calculated. The same equations are be used to calculate regional and continental PECs from the calculated regional and continental PECs for water.

Table 3.22 : Predicted Environmental concentrations for sediment

Process	$PEC_{local(sediment)}$ (µg/kg)	
Production of anthracene	3.65×10^{-2}	
Producrion of pyrotechnics (form.)	107	

New site specific information reffered to anthracene production Industry Plant provides a value of **0.00033 μg/kg$_{dwt}$** . We will adopt this value (**0.00033 μg/kg$_{dwt}$) as PEC for sediment in our calculations.**

3.1.4.2 Measured levels

The fate and transport of anthracene in surface waters will depend on the nature of the water. In most waters, the loss of anthracene is mainly due to photolysis and biodegradation, however, in every shallow fast flowing clear water, volatilisation and photolysis will play dominant roles in determining the fate of anthracene (USEPA, 1987).

Anthracene is ubiquitous in the aquatic environment. It has been detected in industrial effluents, in run off waters, in surface water and sediments, in groundwater, and in drinking water. The industrial effluents that are most likely to contain polynuclear aromatic compounds including anthracene are wastewaters from the fuel industry, shale oil plants, petroleum processing plants, other industries using coal derived products, wastewater treatment plants and aluminium reduction plants. However, anthracene has been reported in effluents from only a few industries

Surface water

Detection of anthracene in UK, in Tamar estuary showed concentrations of 4,9 ppt (Readman et al, 1982).

In Germany in Rhine river in samples collected between 1981 and 1987, anthracene detected in concentrations of 15 and 13 ppt respectively (Malle, 1984).

Measurements in USA, Japan or Chine showed no differences in concentrations between these countries and Europe.

Additional monitoring data on anthracene concentrations in surface water in The Netherlands are given below (Scheepers N, 1993):

Table 3.23 Monitoring data on anthracene concentrations in surface water in The Netherlands

Location	Concentration (μg/l)
De Linde (Kuinre), recently placed wood	<0.005
Beuseberger Waterleiding (Markelo), wood placed approximately one year before measurements	1390
Espelerdwarstocht (Noordoostpolder).	0.88
Kalenbergerbocht (Luttelgeest), recently placed wood	0.006
Repelweg (Kraggenburg), recently placed wood	<6.0
Dedemsvaart (Dedemsvaart), recently placed wood, not filtered sample taken 1 m from the bank	0.056
Dedemsvaart (Dedemsvaart), recently placed wood, filtered sample taken 1 m from the bank	32.73
Dedemsvaart (Dedemsvaart), recently placed wood, filtered sample taken 30 m from the bank with facing	51.0
Dedemsvaart (Dedemsvaart), recently placed wood, filtered sample taken 500 m from the bank with facing	0.405
Gemaalweg (Noordoostpolder), recently placed wood, location 1	0.570

Gemaalweg (Noordoostpolder), recently placed wood, location 1	<1.000

1390 and 51 µg/l values are above the water solubility and should therefore be considered incorrect.

Most recently, in the Netherlands the concentrations of several PAHs are measured yearly in different rivers (RIZA). The range of 90-percentile and mean PAH water concentrations in different Dutch rivers was:

	2000		2001		2002	
Compound	90-perctle. [µg/l]	Mean [µg/l]	90-perctle. [µg/l]	Mean [µg/l]	90-perctle. [µg/l]	Mean [µg/l]
anthracene	< 0.01	< 0.01	< 0.01 – 0.01	< 0.01 - <0.03	< 0.01 – 0.03	< 0.01 - < 0.04

Denzer et al. (1999; COMMPS database) provide further information on concentrations of anthracene in the surface water throughout the EU. Results are based on 10 sampling stations, 54 samples: Results of the aggregation of monitoring data of PAH in surface water (Denzer *et al*., 1999)..

Compound	90-perctle. [µg/l]	Median [µg/l]	ar. Mean [µg/l]	sdev [µg/l]	Sampl. St.	entries used	entries >DL
Anthracene	0.0834	0.0042	0.0141	0.0257	10	54	45

90-perctle. - EU-level 90-percentile of substance concentration (used for exposure scoring); Median - EU-level median; ar. Mean - EU-level arithmetic mean; sdev - standard deviation of arith. mean; Sampl. St. - number of sampling stations from which data were used to calculate the exposure concentrations; entries used - number of measurements used to calculate the exposure concentrations; entries >DL - number of used measurements whith concentrations higher than the corresponding determination limit

Table 3.24 : Concentrations of anthracene in the surface water throughout the EU.

EU level 90-percentile of substance concentration	0.0834 µg/l
EU-level median	0.0042 µg/l
EU-level arithmetic median	0.0141 µg/l

Table 3.25 : Anthracene detected in various rivers in Germany

Emscher: surface water. Highly polluted river	1985: 0.27 µg/l 1987: 0.26 µg/l 1988: 0.025 µg/l 1989: 0.059 µg/l
Lippe, Ruhr, Wupper: Highly polluted rivers	0.010 µg/l
Northern part of the river Rhine: Polluted river, surface water	0.010 µg/l

LWA Gewaessergue-teberícht (1985, 1987, 1988, 1989)

Seawater

Seawater samples from the Baltic Sea collected in November 1993 at depths of 10 to 5 m and concentrations of 164.59 and 464.80 pg/l were measured, with average concentration 333.65

pg/l. Concentrations in samples at depths below 70 m ranged from 37.27 to 558.86 pg/l (Witt, 1995). Another measurement in Baltic Sea near Poland was positive to anthracene.

Groundwater
The possibility of leaching of anthracene from soil to groundwater will depend on soil type. Anthracene is strongly adsorbed to soil and the compound may degrade before it reaches groundwater. Filtration of polluted surface water containing anthracene through sandy soil at a residence time of 100 days did not completely eliminate anthracene in the filtered water. The passage of anthracene through the soil was explained as a breakthrough of the chemical because of the saturation of active sorption sites (USEPA, 1987).

Most measurements have been done in USA and detect concentrations between 68 ppb near a closed coal tar distillation and wood treating plant in St. Louis (Ehlirich et al, 1982) and 4.7 mg/l in water samples collected from the abandoned American Creosote Works facility in FL (Middaugh et al., 1991)
The detection of up to 168.6 μg/l of anthracene was reported in groundwater from a creosote waste site in Conroe, TX.

Drinking water
Concentrations of anthracene in drinking water are below detection limits in Europe in most cases. However, in Netherlands in drinking water derived from bankfiltered Rhine water the concentration was 30 ppt (Piet & Morra, 1983).

In Sweden from May to July (1980), the concentration was 0.04 to 9.7 ppt (Kvesheth & Sortland, 1982) and in tap water from Norway 0.35 ppt (National Research Council, 1983)

High measurements observed in New York State in 1979, when 39 public drinking water wells surveyed, 7 found positive with a maximum concentration of 21 μg/l (Council et al., 1980).

In India, from 10 different locations in Ahmedabad City, anthracene was detected in concentration of 14.91 ng/l (Suffet et al., 1991)

Waste waters
Anthracene was detected in the following wastewaters:

(USEPA, 1981) (number of detections, mean concentration μg/l)

- auto and other laundries, 10 out of 29 samples contained <64 coal mining, 6 out of 46 samples contained 13;
- iron/steel manufacturing, 3 out of 9 samples contained <5,400 leather tanning and finishing, 9 out of 18 samples contained 47 aluminum forming, 11 out of 26 samples contained <14,000
- battery manufacturing, 7 out of 13 samples contained 10 coil coating, 24 out of 78 samples contained 21
- electrical/electronic components, 7 out of 8 contained <10 foundries, 16 out of 16 samples contained 86 (12 out of 12 contained 290); metal finishing 63 out of 125 samples contained 180;
- photographic equipment/supplies, 4 out of 13 contained 3.2 nonferrous metals, 15 out of 75 contained 43 organic chemicals manufacturing/plastics, 23 samples contained 340
- pulp and paperboard mills, 2 out of 16 contained 1

- textile mills, 1 out of 50 samples contained 4.4;
- timber products processing, 11 out of 12 samples contained 3,700

(USEPA, 1987)

- sewage treatment plant in Norway at concentration ranging from <13 to 105 ng/l.
- effluent from coke oven plants in concentration ranging from <0.03 to 0.84 µg/l
- coal gasification plant at a concentration of 0.2 mg/l (Giabbai et al., 1979).
- dissolved air flotation (DAF) wastewater effluent of a class B refinery at a concentration of 168 ng/g (Snider & Manning, 1982).
- sewage sludges from four wastewater treatment works in Switzerland at a mean concentration of 187.8 µg/kg dry weight (Berset & Holzer, 1995).
- The mean concentration of anthracene in ditch water in the Lower Mainland of British Columbia was 0.16 µg/l from farmlands; 81 µg/l from railway ditches with utility poles and 0.13 µg/l in railway ditches without utility poles (Wan, 1994).

Anthracene at a concentration range of <13-105 ng/l was reported in the effluent from a sewage treatment plant in Norway (USEPA, 1987).

Rain – Snow

Between Nov 1974 to March 1975, in rain from Southern Norway, anthracene was detected but not quantified (Lunde et al., 1977).

From 22 samples collected during nine fog events from Sept to Dec 1986 in Dubendorf, a suburb of Zurich, Switzerland, anthracene was detected in concentrations of 0.92 ng/l with range from 0.01 to 3.0 ng/l (Capel et al., 1991). Measurements in USA and Canada showed no differences in quantities.

Sediment

Anthracene was detected in:

- The Baltic Sea, Gulf of Finland at concentrations ranging from 4 to 13 ppb (Poutanen et al., 1981).
- Lake sediments from Greifensee, Switzerland at a concentration of 30 ppb (Carlson et al., 1979).
- Suadafjord, Norway at the following concentrations, ppb dry wt (depth, cm): 12.5 to 727.9 (0 to 2), 7.2 to 1075.2 (2 to 4), 11.6 to 1119.6 (4 to 6), 13.0 to 524 (6 to 8) (Bjorseth et al., 1979).
- Suspended solids, 497 ppb, and sediments, 120 ppb from the Tamar Estuary, UK (Readman et al., 1982).
- A harbour in SW Netherlands at an average concentration of 0.144 ppb (DeLeeuw, 1986).
- 10 stations in Norwegian and Swedish fjords at concentrations ranging from 1.15 to 10,700 ppb (dry wt) (Sportsol et al., 1983).
- Muddy sediments and sandy sediments collected from 7 stations in the Baltic Sea during Oct and Nov 1993 at mean concentrations of 42.05 and 10.39 ng/g dry weight, respectively (Witt, 1995).
- Tilbury Basin on the River Thames at concentrations ranging from 0.30 to 1.92 µg/g dry sediment (Taylor & Lester, 1995).
- 28 out of 30 superlayer and underlayer deep sea sediments from 15 sampling sites in the Gulf of Lion, (Mediterranean Sea, France) at concentrations ranging from 0 to 23.8 pmol/g dry weight sediment (Domine et al., 1994).

- U.K.: Estuary of river Mersey (polluted estuary) at concentrations ranging from n.d.-180 ng/g (related to dry sediment) (Readman et al., 1986)
- Italy: Ligurian sea, at concentrations ranging from 6-42 ng/g (Desideri et al., 1987)
- France: Rhone delta, at concentrations ranging from 8-200 ng/g (Milano & Vernet 1988)
- Germany: Polluted rivers, Rhine: n.d.-190 ng/g, Main: n.d.-4200 ng/g, Different smaller Bavarian and Hessian rivers: n.d.-4340 ng/g, (LWA Gewaessergue-teberícht 1988)

Additional information on measured anthracene concentrations in sediment in The Netherlands (Anonymus, 1999) is given below:

Table 3.26 : Measured anthracene concentrations in sediment in The Netherlands

Location	Concentration (µg/kg dw)
Breevaart (Reeuwijk), half year after placing the facing close to facing	0.37
Breevaart (Reeuwijk), half year after placing the facing a few hundred m far from facing	0.06

Denzer *et al*. (1999; COMMPS database) collected information on concentrations of a number of PAHs in the sediment throughout the EU. The 90-percentile of sample station arithmetic mean concentrations ranged from 468 to 3410 µg/kg measured for anthracene and phenanthrene, respectively. No median or mean values in sediment were available in the COMMPS database. The range of mean PAH sediment concentrations in different Dutch rivers (RIZA) for 2000 was <0.05-6.87 mg/kg for anthracene.

Results of the aggregation of monitoring data of PAH in sediment (Denzer et al., 1999).

Compound	90-percentile conc. [µg/kg]	Sampl. St.	entries used	entries >DL
Anthracene	468.40	55	580	520

3.1.4.3 Comparison between predicted and measured levels

In general, anthracene occurs in ambient waters at a median concentration of <10 µg/l. Anthracene has been reported to be present in groundwater from a few contaminated sites. Detection of anthracene in drinking waters in USA has been reported at maximum concentration of 21µg/ml. However, the measured levels of anthracene in Europe was reported between 0.35 and 30 ppt. Finished waters from 13 different locations throughout the United States, failed to show the presence of any anthracene (USEPA, 1987). Detection limits of anthracene in surface waters, showed concentrations between 0.005 and 1,390 µg/l, but in waste waters, in same cases, levels were above 3,500 µg/l. COMMPS database values ranged around 468 µg/kg.

There are large numbers of reported level data for water. Although these do not come from detailed monitoring studies, when taken together they do give good picture of levels in the aquatic compartment. However, in some cases, compared to PEC levels, the measured levels in waste waters, are much higher.

A large number of reported levels for anthracene in sediment give a good overall picture of anthracene levels. These ranged from not detected concentrations to few mg/l (4-5) in high-

polluted sources, like Main and Hessian River and they seem to be much lower than calculated PECs.
Some high measured values are far above the water solubility. We suppose that these values are either wrong or based on samples which also include particle bound anthracene.

3.1.5 Terrestrial compartment

3.1.5.1 Calculation of PEC_{local}

3.1.5.1.1 Calculation of PEC_{local} for production, for formulation and for processing.

Accumulation of a substance may occur when sludge is applied over consecutive years. As a reasonable worst-case scenario, sludge is assumed to be applied for 10 consecutive years. To provide an indication of the potential persistence of the substance, the percentage of the steady-state situation is calculated. Different averaging times are used for the different end-points: for the ecosystem a period of 30 days after sludge application is used. In order to determine biomagnification effects and indirect exposure to man, an extended period of 180 days is used.
The concentration in groundwater is calculated for indirect exposure of humans through drinking water. The concentration in the pore water of agricultural soil is taken as an indication of potential groundwater levels. It should be noted that this is a worst-case assumption, neglecting transformation and dilution in deeper soil layers.

The TGD equations have been used to calculate a PEC for soil porewater. The local concentrations estimated for soil are given in Table 3.27.

Table 3.27 : PECs calculated for soil

Process	Agric. soil (total) average over 30 days µg/kg	Agric. soil (total) average over 180 days µg/kg	Grassland (total) average over 180 days µg/kg	Groundwater under agricultural soil µg/l
Production of anthracene	51.1×10^{-3}	51.1×10^{-3}	100×10^{-3}	8.66×10^{-5}
Producrion of pyrotechnics (form.)	32.1	31.6	12.5	5.35×10^{-2}

The concentration in pore water can never be higher than the water solubility. Otherwise, there is mistake due to default values using EUSES.

Default calculations for the production of anthracene are inaccordance with those that come up from site-specific information. In the pitch RAR, the estimates of the excess local soil concentrations caused by tar processing and pitch production are **51 ng/kg soil dw for**

agricultural soil and 100 ng/kg soil dw for grassland for anthracene, respectively and **these values will be adopted as PECs for risk assessment calculations.**

3.1.5.2 Measured levels

Concentrations of anthracene in different sites across Europe have as follows:

Soil was found to contain anthracene after the spreading of sewage sludge (Peircysens & Tarradellus, 1987). Smoke from wood burning stoves contained anthracene (Hawthorn et al., 1988).

Anthracene concentrations in the top 10 cm of mineral soil at ten forest sites with increasing distance from the blast furnace plant at IJmuiden, The Netherlands (measured by Van Brummelen et al., 1996)

Distance (km)	0.3	0.5	1.2	1.7	2.0	2.5	2.8	3.4	4.5	6.6
Concentration	49	75	8.9	6.9	1.1	6.1	1.9	2.5	0.4	1.7

Soil anthracene concentration in soil at cattle, vegetable and bulb farms in Netherlands measured 1997 (Groot,2003).

Cattle farms		Vegetable farms		Bulb farms	
Min µg/kg	Max µg/kg	Min µg/kg	Max µg/kg	Min µg/kg	Max µg/kg
<0.5	12	0.71	12.5	0.6	6.7

The concentration of anthracene in the soil surrounding waste pits for natural gas production and processing ranged between 260 and 670 µg/kg (Eiceman et al., 1986) and 36 to 105 mg/kg (Davani et al., 1986). The concentration of anthracene in the soil water of the same pits ranged from 3.5 to 530 µg/l (Davani et al., 1985).

Anthracene was detected in soil samples collected 150, 350 and 900 m from a municipal solid waste incinerator in Italy at 7, 13, and 92 µg/kg, respectively (Morselli et al., 1989).

Anthracene concentrations in the organic surface layer (litter, fragmentation, and humus) of soil, and mineral soil 0.3 to 6.55 km from the blast furnace plant were 56.5, 0 in the litter; 64.4, 0 in the fragmentation; 94.8, 7.8 in the humus; and 49.1, 1.7 µg/kg dry weight in the mineral soil, respectively (VanBrummelen et al., 1993).

Soil samples from sampling areas known to be contaminated with coal tar contained anthracene at concentrations ranging from 10 to 1000 mg/kg (Johnston et al., 1993).

30 soil samples collected from Nova Scotia in Oct 1976 contained anthracene at concentrations ranging from <0.1 to 110 ppb dry wt, median concentration 12 ppb (Windsor & Hits, 1979).

Anthracene's concentration was monitored in an agricultural soil for a period of 100 years; concentration in 1846, 1881, 1893, 1944, 1956, 1966, 1980 and 1986 were 4.5, 13, 9, 4, 10, 9, 13 and 11 µg/kg, respectively (Jones et al., 1989).

Soil samples from Birmingham, UK (urban), Brisbane (urban), Wales (rural), and Hertfordshire (semi-rural) contained anthracene at 2160, 717, 720, and 660 mg/kg, respectively (Smith et al., 1995).
Anthracene was detected in 49 surface soil samples from 49 study sites in Wales at concentrations ranging from 6 to 15,000 mg/kg dry weight (mean concentration 500 mg/kg dry weight). The highest concentration occurred in a soil sample from an industrialized area of the Upper Taff valley (coal-mining valley), the lowest concentration occurred in a soil sample 1 km from the coast (Jones et al., 1989).

Soil samples collected from Rothamsted Experimental Station in Southeast England at various times since the mid-1800s were found to contain anthracene at the following concentrations, μg/kg dry weight (year): 4.5 (1846); 13 (1881); 9 (1893); 4 (1944); 10 (1956); 9 (1966); 13 (1980); and 11 (1986) (Jones et al., 1989).

Soil samples collected 0.3 to 6.55 km from a blast furnace plant in the Netherlands contained anthracene at concentrations of 9.18 and 0.31 mg/m^3, respectively (Van Brummelen et al., 1996).
Additional information on measured concentrations of anthracene in soil in The Netherlands [Esser PM, Suitela W.L.D. 1993]:

Table 3.28 : Measured concentrations of anthracene in soil in The Netherlands

Location	Concentration [μg/kg dw]
Sample from soil taken next to a 45-years old stake, 0-20 cm deep	250
Sample from soil taken next to a 45-years old stake, 20-80 cm deep	40
Sample from soil taken next to a 45-years old stake, 100-120 cm deep	150

3.1.5.3 Comparison between predicted and measured levels

Anthracene has a slight to no mobility in soil. Thus its levels are quite high. In very high-contaminated soil anthracene has concentrations ranging from 1.7 ng to 15,000 mg per kg soil. These values in most cases are higher than default PEC.

3.1.6 Atmosphere

3.1.6.1 Calculation of PEC_{local}

3.1.6.1.1 Calculation of PEC_{local} for production, for formulation and for processing.

A PEC_{local} for air for release of anthracene from production can be calculated. The following equation from the Technical Guidance Document can be used to calculate the concentration in air at 100 m from the site.

$$PEC_{air(local)} = Emission. C_{std}$$

Where

PEC_{air}	=	Concentration in air at the 100 m from the point source (kg/m^3)
Emission	=	Emission rate to air (kg/s)
C_{std}	=	Standard concentration in air at the source strength of 1 kg /s = 24 x 10^{-6} kg/m^3

Table 3.29 : PECs calculated for the atmospheric environment

Process	Annual average local PEC to air ($\mu g/m^3$)
Production of anthracene	$8.45x10^{-3}$
Production of pyrotechnics (form.)	7.54 $x10^{-4}$

The calculated PECs are not taken forward to the risk characterization because only few data are available and because the default calculations are not considered representative .

Local concentrations in air at 100 m from the point source (ng/m^3) at the production site 1 of coal tar reported in CTPHT RAR was:

Site	1								
Anthracene	10								

Site 1, reffered to the Industry Plant of production of anthracene and calculated after new site specific information and we will adopt this value ($10ng/m^3$) as PEC for the air.

3.1.6.2 Measured levels

Fly ash samples from a municipal incinerator contained anthracene at concentrations ranging from 4 to 380 ppb (avg. 148 ppb) (Eiceman et al., 1981). Anthracene was detected in 5% of 1268 samples in the USEPA STORET database at a median concentration of <10.0 ppb (Staples et al., 1985). 27% of 86 U.S. urban runoff samples from 15 cities through July 1982 contained anthracene at concentrations ranging from 1 to 10 ppb (Cole et al., 1984). Anthracene was detected in coke plant wastewater at concentrations ranging from 70.2 to 101 ppb (Walters & Luthy, 1984).
Anthracene concentrations reported from different European cities are as follows:

In Austria, in the centre of Vienna and Linz anthracene was detected in annual average of 34 ng/m^3 (winter: 47 ng/m^3 summer: 23 ng/m^3) (Jaklin et al., 1985,1988).

Concentrations in summer are always lower as in the centre of Paris (Summer: 0.5 ng/m^3 Winter: 10.0 ng/m^3) (Masclet et al., 1984) or in Silesia, Poland where concentrations were 9.4 to 286.3 ng/m^3 and 2.3 to 4.7 ng/m^3 for winter of 1988-89 and summer of 1989 respectively (Broman et al., 1991).
Anthracene has been detected in different sites in Paris. All concentrations were 0.5 to 6.2 ng/m^3 except a scrap smelting plant where the concentration was too high 254 ng/m^3 (Masclet et αl, 1984).

In Scandinavian cities as Copenhagen, Stockholm, Rorvik (Sweden) and Birkenes (Norway), anthracene was detected in concentrations below 1 ng/m^3 in most cases.

In big cities in the UK (London, Birmingham, Manchester), anthracene was detected in concentrations between 0.28 to 5.3 ng/m^3 (Smith & Harrison, 1996).
High concentration of anthracene was observed also in autotunnel near Berlin (> 330 ng/m^3) (Moriske et al., 1987).

In Eastern Europe cities as Budapest concentrations are quite higher (21.5 to 62.8 ng/m^3 (Verschueren, 1983)

Anthracene was detected in ambient air of the western Mediterranean Sea at concentration ranging from 0.3 to 2.5 pg/m^3 (Sirce et al., 1987a,b) and in 14 of 14 samples at concentration ranging from 0.01 to 0.08 ng/m^3 (Masclet et al., 1998).

Anthracene was detected in atmospheric particulate matter collected in Antarctica during the 1990-91 (Italian expedition) at concentrations ranging from 0.9 to 7.1 pg/m^3 (Chiavarini et al., 1994).

3.1.6.3 Comparison between predicted and measured levels

Anthracene has been observed in almost all countries, from very low to very high levels. In Europe anthracene has been detected in range from traces to 330 ng/m^3. Higher concentrations have been found in Eastern Europe and lower in northern cities. In most cases concentrations are equal with the default PEC. Detected levels of anthracene in Europe are not so different from calculated values.

3.1.7 Calculation of $PEC_{regional}$ and $PEC_{continental}$

The TGD equations have been used to predict regional and continental environmental concentrations of anthracene in water, based on a tonnage of 1,350 per year in Europe.
As there is only one site of production of pure anthracene in EU, we think regional and continental risk assessment covered by local risk assessment as it shows not adverse effects to organisms (see risk assessment chapter). So these data have only informative purposes.
The $PEC_{regional}$ has been calculated on the basis of a standardized regional environment as described in the Technical Guidance. This regional environment is a density-populated area of 200 x 200 km with 20 million inhabitants. It has been assumed that a worst case production plant producing 1,350 tonnes per annum is within this area. The $PEC_{continental}$ has been calculated on the basis of a continental box, which has the size of all EU countries together.
Calculations in tables 3.30, 3.31 are based on emission values from tables 3.16, 3.17, 3.18.
Table 3.30 Prediction of regional and continental environmental concentrations of anthracene in surface water

	$PEC_{surface\ water,\ (dissolved)\ (dissolved)}$ µg/l
Regional	1.22x10^{-5}
Continental	1.05x10^{-6}

Table 3.31 : Predicted Environmental concentrations for sediment

Process	$PEC_{regional(sediment)}$ (µg/kg)
Regional (total)	7.06 x 10^{-2}
Continental (total)	6.05 x 10^{-3}

The TGD equations have been used to calculate the PEC for the terrestrial compartment. Based on these estimates, the following local, regional and continental concentrations were calculated:

Table 3.32 : Predicted Environmental concentrations for soil

$PEC_{regional\ (Natural\ soil)}$	9.71×10^{-5} µg/kg
$PEC_{regional\ (Agricultural\ soil)}$	2.31×10^{-3} µg/kg
$PEC_{regional\ (Pore\ water\ of\ Agricultural\ soils)}$	3.92×10^{-6} µg/l
$PEC_{regional\ (Industrial\ soil)}$	7.19×10^{-2} µg/kg
$PEC_{continental\ (Natural\ soil)}$	1.73×10^{-6} µg/kg
$PEC_{continental\ (Agricultural\ soil)}$	1.52×10^{-4} µg/kg
$PEC_{continental\ (Pore\ water\ of\ Agricultural\ soils)}$	2.58×10^{-7} µg/kg
$PEC_{continental\ (Industrial\ soil)}$	7.39×10^{-3} µg/kg

Table 3.33 : PECs calculated for the atmospheric environment

Regional (PEC_{total})	1.91×10^{-7} µg/m^3
Continental (PEC_{total})	3.41×10^{-9} µg/m^3

Regional and condinental PECs have been calculated considering only the production of anthracene.

3.1.8 Calculation of PEC in STP

The STP model (Technical Guidance Document EC, 2003) calculates the emission from the sewage treatment plant to air, the concentration in sewage sludge and the concentration in the effluent. In the production site, the effluent concentration that has been calculated for anthracene was 0.018 ng/l (table 3.72, CTPHT RAR, (EC 2008) and it is directed to off-site (public) sewage treatment plant. Sludge from the STP goes to agricultural soil.

3.1.9 Secondary poisoning

PECoral is not calculated as there is lack of information.

The risk assessment of secondary poisoning is put on hold as the substance is already regarded as a PBT and vPvB substance. Therefore the emissions of anthracene into the environment will be reduced anyhow.

Concentrations used for the exposure assessment of man via the environment
The predicted levels of anthracene in biota and foodstuffs calculated using EUSES are shown in Table 3.26. In EUSES the concentration arising in biota are for biota exposed to anthracene in contaminated areas. The fish is assumed to be exposed to the dissolved surface water concentration at 1,000 m from the waste water treatment plant effluent. The uptake of anthracene by plants is modeled on plants growing on agricultural soil on which sludge from a waste water 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 anthracene by cattle results in concentrations in meat and milk. These are modeled 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 anthracene has been estimated using EUSES at the regional and local levels.

Table 3.34: Secondary poisoning using EUSES

	Concentration in fish for secondary poisoning(fresh water) (mg/kg)	Concentration in earthworms from agricultural soil (mg/kg)
Production of anthracene	1.12×10^{-4}	2.58×10^{-5}
Production of pyrotechnics (form.)	0.135	0.0152

These calculations are based on the preferred BCF value of 4,550 l/kg for fish.

3.1.9.1 Measured /monitoring data

Biota

Measured levels in biota

Mytilus edulis (mussels)	Dutch North sea, coast range	149-243 µg/kg wet weight (dependent on water depth)	Boom (1987)
Limanda limanda (dabs)	Near oil platform	0.1-0.2 µg/kg wet weight (no dependence on distance of the possible source)	McGill et al., (1987)
Mediterranean holothurians	Near sewage outlet of the city of Toulon	1 mg/kg dry weight (in polluted zone)	Milano et al., (1986)

Food

Anthracene has been reported to be present in the following foods:

- Charcoal broiled steaks and barbecued ribs were reported to be 4.5 and 7.1 µg/kg, respectively (USEPA, 1987).
- Charcoal-broiled steaks, 4.5 ppb (Fazio & Howard, 1983)
- Barbecued ribs, 7.1 ppb (Fazio & Howard, 1983)
- Beef patties containing 10, 20 and 30% fat and cooked over mesquite wood at an average concentration of 20, 28 and 31 µg/kg, respectively (Maga, 1986).
- Beef patties with 10 and 20% fat that were cooked over hardwood charcoal (Maga, 1986).
- Beef patties with 30% fat and cooked over hardwood charcoal at an average concentration of 2 µg/kg (Maga, 1986).
- Laboratory prepared charcoal-broiled steaks and commercial barbecued ribs at respective concentration of 4.5 and 7.1 ppb (Lo & Sandi, 1978).
- Cold and hot smoked sausages with the casings contained 25.4 and 10.6 ppb, respectively; 2.4 and 5.6 ppb, respectively with casings removed (Lo & Sandi, 1978).
- 1 out of 6 Italian olive oil samples, 4 µg/kg, and 7 out of 7 Italian virgin olive oil samples at a median concentration of 7 µg/kg (Menichini et al., 1991).
- Edible oils, 0.2 to 402 ppb (IARC, 1983)
- Coconut oil, 36 ppb (Santodonato et al., 1981)

- Vegetable oils at the following median concentrations in Germany, µg/kg: olive oils, 2.2; sunflower oils, 0.3; maize germ oils, 0.1; sesame, 0.2; linseed oil, 4.0; and wheat germ oil, 4.4 (Speer et al., 1990).
- 13 of 25 samples from Finnish margarine, butter, and vegetable oils at concentration ranging from 0.04 to 460 µg/kg (Hopia et al., 1986).
- of 23 samples of Finnish leaf lettuce (Lactuga sativa var. crispa) at concentration of 0.09, 0.10, and 0.19 µg/kg fresh weight (Wickstorm et al., 1986).
- Bakers yeast, 2.6 to 557 ppb (Santodonato et al., 1981
- 25 out of 34 shell-free fresh-frozen and precooked-frozen mussels bought between Sept 1992 and May 1993 on the island of Tenerife, Spain, at concentrations ranging from 1 to 28 ng/g dry weight (Hernadez et al., 1995).
- Alcoholic drinks in France showed anthracene and related compounds in the range of 1 to 10 ppb (Toussaint & Walker, 1979).

In most cases, measured levels of anthracene in biota and foodstuff are lower than calculated values of anthracene, especially from coal tar distillation.

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

The protection goals for the environment are the aquatic, sediment and terrestrial ecosystem, top predators and microbial activity in an STP. This means that a PNEC has to be derived for each of the protection goals. Certain assumptions are made concerning the extrapolation from single-species short-term toxicity data to ecosystem effects:
1. ecosystem sensitivity depends on the most sensitive species; and
2. protecting ecosystem structure protects community function.
These two assumptions have important consequences. By establishing which species is the most sensitive to the toxic effects of a chemical in the laboratory, extrapolation can subsequently be based on the data for that species. Furthermore, the functioning of any ecosystem in which that species exists is protected, provided the structure is not sufficiently distorted as to cause an imbalance. In establishing the size of the assessment factors, a number of uncertainties must be addressed, summarised under the following headings:
1. Intra- and inter-laboratory variation of toxicity data.
2. Intra- and inter-species variations (biological variance).
3. Short-term to long-term toxicity extrapolation.
4. Laboratory data to field impact extrapolation.
The toxicity of most PAHs can be greatly enhanced on exposure of a living organism and/or the chemicals to ultraviolet radiation. There are two major mechanisms involved in photoinduced toxicity of PAHs: photosensitization and photomodification. In the former, production of singled oxygen leads to cellular damage. In the later, photooxidation of PAHs results in new compounds that are often more toxic then their parent PAHs.
From reports below, anthracene clearly meets the T- criterion.

In principle, the PNEC is calculated by dividing the lowest short-term L(E)C50 or long-term NOEC value by an appropriate assessment factor. The size of the assessment factor depends on the confidence with which a PNEC can be derived from the available data. This confidence increases if data are available on the toxicity to organisms at a number of trophic levels, belonging to taxonomic groups and with lifestyles representing various feeding strategies.

Thus, lower assessment factors can be used with larger and more relevant data sets than the base-set data.
The assessment factors also reflect the degree of uncertainty in extrapolation from laboratory toxicity-test data for a limited number of species to the 'real' environment. Assessment factors applied for long-term tests are lower, as there is less uncertainty involved in the extrapolation from laboratory data to the natural environment. Nevertheles, the phototoxic effects, caused by photosensitization, and photomodification, can be observed after a short period of exposure, which explains why for PAHs like anthracene, where photoxicity is most evident, the acute toxicity values are even lower than the chronic toxicity values. (RAR CTPHT, R323 0705env).

3.2.1 Aquatic compartment (incl. sediment)

The quality of data in studies has been evaluated according to Klimisch et al (1997) study. The approach proposes to indicate a measure of the study/data reliability with the following categories:
Validity 1: Reliable without restrictions
Validity 2: Reliable with restrictions
Validity 3: Not reliable
Validity 4: Not assignable

3.2.1.1 Toxicity test results

3.2.1.1.1 Fishes

Acute toxicity

Anthracene is acutely toxic to aquatic organisms in the presence of solar ultraviolet radiation (SUVR).

Roberts et al., (1989) were studied the concentration of contaminated sediment causing an acute lethal effect on *Leiostomus xanthurus* exposed either to sediment or to water which had been in contact with sediment. Initial concentration of anthracene in river was 18ng/kg of sediment. All fish exposed to 100% contaminated sediment were dead within 2h. The lethal time for 50% of the fish (LT50) was estimated graphically to be 57 min. (Validity: 4)

Acute mortality of bluegill sunfish (*Lepomis macrochirus*) dosed with anthracene at 12.7 µg/l and exposed to natural sunlight conditions was observed during a study of anthracene fate in outdoor channel microcosms. No mortality was observed under control conditions (natural sunlight and no anthracene). Fish survived when held in the shade downstream of sunlit contaminated water, arguing against mortality due to toxic anthracene photoproducts in the water. Fish held 48 h in anthracene contaminated water (≈ 12 µg/L), in a shaded channel, died when placed in clean water and exposeeed to sunlight. After 144 h in darkness, fish in anthracene concentrations had decreased to pre-exposure concentrations and no mortality was observed when fish were subsequently exposed to sunlight. This observed photoinduced toxic response in anthracene contaminated fish may represent a significant environmental hazard of polycyclic aromatic hydrocarbons in aquatic environments. (Bowling, J. W. Et al; 1983) (Validity: 4)

The acute toxicity of anthracene to juvenile sunfish in the presence of simulated sunlight was assessed. In this study, 96-h median lethal concentration values were 190-1800-times less (LC50 2.78-26.47µg/l), with concurrent exposure of anthracene and simulated sunlight, than a reported 24-h no-effect anthracene concentration for *L. macrochirus* derived under standard laboratory conditions. A mathematic relation to estimate the potential toxicity of anthracene to fish is presented which closely predicts the results of toxicity studies conducted under natural sunlight. Thus, solar radiation is an important accessory parameter that deserves consideration in the toxicity assessment of polycyclic arom. hydrocarbons in the aquatic environment. (Oris, T. & Giesy, J.P.Jr, 1985) (Validity: 1)

The toxicity of anthracene was assessed in a variety of aquatic organisms under environmentally realistic conditions. A 96-h LC50 value of 11.9 µg anthracene/l - 26.47µg/l was detected for a natural population of juvenile bluegill sunfish at a solar UV intensity equivalent to a depth of 0.6 m in a typical eutrophic north-temperate lake. (Oris, J.T. et al., 1984) (Validity: 1)

The effects of daily light-cycle duration (photoperiod) on the solar UV radiation (SUVR)-induced toxicity of anthracene to juvenile bluegill sunfish (*Lepomis macrochirus*) in a laboratory system under simulated sunlight were examined. Rates of acute mortality were dependent on anthracene concentration and photoperiod. Median lethal time values calculated on the basis of accumulated SUVR exposure time (UV-LT50) were compared with LT50 calculated from real time of exposure (R-LT50) to detect relative rates of phototoxic damage vs. physiological repair during periods of darkness. The comparison of these LT50 indicated that the photoinduced toxicity of anthracene to fish was slowly repaired during periods of darkness but that enough damage accumulated over several SUVR cycles to cause acute mortality. The results from these experiments were incorporated into a relation to predict no-effect in concentrations from the daily light-cycle duration at 1 SUVR intensity. Information from acute toxicity tests was used to extrapolate to chronic no-effect values. No-effect concentrations (NOEC) in water were predicted to range from 1.2 µg/l for 24 h light:0 h dark photoperiod to 13.5 µg/l for a 6 h light:18 h dark photoperiod. (Validity: 1)
A no-effect body burden of 131 µg/kg has been calculated for juvenile bluegill sunfish for a 16 h light: 8 h dark photoperiod at an equivalent depth of 3.0 m in a typical eutrophic system. Thus, considering current natural polycyclic arom. hydrocarbon concnentrations in water and in fish tissue, there exists natural waters in which photoinduced polycyclic aromatic hydrocarbon toxicity may occur. (Validity: 4) (Oris, J.T. and Giesy, J.P.,1986).

The effect of water temperature and oxygen concentration on the photo-induced toxicity of anthracene to juvenile bluegill sunfish (*L. macrochirus*) was detected. Bluegill sunfish were exposed to anthracene (<0.01, 6.20, 11.62, and 20.88 µg/l) and solar UV radiation (SUVR) at different water temps. (20 and 30°) and oxygen concentrations (5.0, 6.9 and 8.1 mg O_2/l) for 120 h in a lab. flow-through system. A trend toward an inverse relationship was found between water temperature and LC50 values, although no significant temperature effect was observed. A nonlinear relationship was found between oxygen concentration and LC50 values, with significantly increased toxicity at the middle oxygen concetration A statistical relationship was developed on the basis of oxygen concentration to predict LC50 values. It is concluded that dissolved oxygen concentration is an important environmental factor in the assessment of photo-induced toxicity of anthracene to fish. (McCloskey, J. T. & Oris, J.T.,1991) (Validity: 3)

Anthracene is acutely toxic to fish when they are exposed simultaneously to solar UV radiation (SUVR). However, the physiological mechanism of acute anthracene photoinduced toxicity is not known. The purpose a study was to investigate possible modes of action associated with simultaneous anthracene and SUVR exposure in bluegill sunfish (*L. macrochirus*). Fish were exposed to anthracene (<0.01 and 7.04 µg/l) and SUVR in a flow-through system. Following 96 h of anthracene and SUVR exposure, fish exhibited IUCLID and significant decreases in whole blood Hb content when compared to no-anthracene controls. Evidence of hemolysis was also observed in anthracene- and SUVR-exposed fish. Using in vitro enzyme analysis, both Na,K-ATPase and Mg-ATPase were significantly inhibited in gill tissue homogenates exposed to anthracene and SUVR when compared to homogenates exposed to SUVR alone. These blood and gill measurements provide evidence of osmotic stress in exposed fish. Apparently, there are numerous sites of acute toxic action with anthracene and SUVR exposure in fish, and this toxicity appears to be associated with a general disruption of cell membrane function. (McCloskey, J. T. & Oris, J.T.,1993). (Validity: 3)

Table 3.35:There are LC50 values (*Lepomis sp.*) e.g. from 2.78 to 26.47 µg/l. Obviously, there is an acute toxicity of anthracene within its solubility limits (IUCLID).

temperature [°C]	tab water [µg/l]	Sea water[1] [µg/l]
4.6	9.6	6.8
12.9	17.7	12.2
21.1	32.2	23.3
25.3	44.2	32.4

[1] salinity 36.5 o/oo

(Validity: 2)

The toxicity of 12 polycyclic arom. hydrocarbons (PAH) to larvae of the fathead minnow (*Pimephales promelas*) in the presence of simulates sunlight was examined. A measure of relative toxicities of the toxic compounds is described in which wave band, radiation intensity, molar extinction coefficients, and molar body concentration of PAH are considered. A structure-lethality relation was developed, based on molecular structure and photochemical properties, that classifies compounds as being phototoxic or nonphototoxic. LC50 for 24 h was calculated at 360µg/l for *P. promelas* under sunlight conditions (Oris, T. & Giesy, J.P.Jr, 1987) (Validity: 2)

The purpose the study of Hall A.T. et al., (1990) was to examine the reproductive effects of anthracene in fathead minnows (*Pimephales promelas*) in the absence of SUVR, and to examine the effects of anthracene both with and without SUVR on the learning of these fish. In the reproductive study, spawning fathead minnows were exposed to either 0, 6 or 12 µg anthracene/l for six weeks, followed by concentration increases to 12 and 25 µg/l for the later two treatment groups for 3 weeks. Eggs were collected daily, put into clean water (i.e., no anthracene) and divided into SUVR and no-SUVR exposure groups. The results to date indicate a significant bioaccumulation of anthracene in the tissues of spawning fish, including their gonads and laid eggs. Reproductive output (number of eggs laid) was lower in all anthracene, no-SUVR exposure groups. Maternal exposure to anthracene also affected the survivorship of eggs (survival) and fry (survival to 96 hr posthatch) reared under SUVR. The effects on learning capabilities of fish were examined to assess the potential neurological effects of anthracene both with and without SUVR exposure. Minnows were exposed to 20

µg/l for one week both with and without SUVR. Each fish was trained to avoid darkness in a shuttlebox conditioning apparatus. The training paradigm included 20 training trials, followed by 10 test trials. Anthracene and SUVR-exposed fish demonstrated increased time to correct response during the test trials. The apparent increase in learning of these fish may have been due to hyperactivity. Their studies have shown that anthracene exposure with SUVR can produce teratological and neurological damage in fish. In addition, anthracene exposure in the absence of SUVR reduces reproductive output. (Validity: 3)

Fresh water fish *Pimephales promelas* exposed for 24 h in anthracene. The fishes were incubated for 0.5 h prior to 0.5 h in sun light exposure. Testing temperature was 14 degree C and each fish has 0.8g weight. LC50 was detected at 360 µg/l (Kagan et al, 1987). (Validity: 2)

Fresh water fish *Orizias latipes* exposed for 24 h in anthracene and LC50 was detected at 210 µg/l (Reutgerswerke, 1991) (Validity: 2)

Table 3.36 : Summary on acute toxixity to fishes

Species	Exposure duration	Endpoint	Effect	Conc (mg/l)	References	Validity *
Leiostomus xanthurus	2 h	LT 50 (57 min)			Roberts et al., 1989	4
Lepomis macrochirus	48 h		Acute mortality	0.0127	Bowling, J. W. Et al; 1983	4
Lepomis macrochirus	96 h	LC50	Survival	0.003-0.026	Oris, T. & Giesy, J.P.Jr, 1985	1
Lepomis macrochirus	96 h	LC50	Survival	0.0119-0.02647	Oris, J.T. et al., 1984	1
Lepomis macrochirus	24 h light:0 h dark 6 h light:18 h dark 16 h light: 8 h dark	NOEC NOEC no-effect body burden		0,0012 0.013.5 0.131	Oris, J.T. and Giesy, J.P.,1986	1 1 4
Lepomis macrochirus	120 h	LC 50	Photo induced toxicity	?	McCloskey, J. T. & Oris, J.T.,1991	3
Lepomis macrochirus	96 h		Significant decreases in whole blood Hb content	0.00001 and 0.007.	McCloskey, J. T. & Oris, J.T.,1993	3
Lepomis macrochirus		LC 50	Survival	0.003 to 0.026	IUCLID	2

Species	Exposure duration	Endpoint	Effect	Conc (mg/l)	References	Validity *
Pimephales promelas	24 h	LC 50	Survival	0.360	Oris, T. & Giesy, J.P.Jr, 1987	2
Pimephales promelas	96 h		Survival, Reproductive effects	0.0006 or 0.012	Hall A.T. et al., 1990	3
Pimephales promelas	24 h	LC 50		0.36	Kagan et al, 1987	2
Orizias latipes	24 hours	LC 50	Survival	0.210	Reutgerswerke, 1991	2

* by Klimisch et al, 1997

Long-term toxicity

Acute mortality of fish, directly attributable to high PAH concentrations in sediment and water, was observed in Elizabeth River, Virginia. *Leiostomus xanthurus* exposed to naturally contaminated sediments from river station 217 and acute mortality was observed within 8 days as well as fin erosion, ulceration of the lateral body surface and several types of lesions of internal organs (Hagris et al., 1984). (Validity: 3)

The long-term effects of anthracene on the reproductive potential of fathead minnows (*Pimephales promelas*) were examined. Sexually mature adult fish were exposed in the absence of solar UV radiation (SUVR) to 0, 6, or 12 μg anthracene/l for 6 wk, followed by concentration increases to 12 and 20 μg anthracene/l for the latter 2 treatment groups for 3 wk (NOEC<6μg/l). Eggs were collected daily, placed into clean water and divided into SUVR and no-SUVR exposure groups. Survivorship and development of eggs was followed until 96 h posthatching. Significant bioconcentration of anthracene was observed in the eggs laid, in the gonads and in the carcasses of the spawning fish in all treatments except controls. Decreased reproductive output (no. of eggs laid) was observed in all anthracene exposed fish. Decreased survivorship of fry which were maternally exposed to anthracene but had no subsequent exposure to SUVR was also observed. These findings provide evidence that anthracene, previously thought to be nontoxic in the absence of SUVR, may cause reproductive impairment in fish. (Hall A.T. et al., 1990, Hall A.T. & Oris, J.T. 1991) (Validity: 2)

Table 3.37 : Summary on long - term toxicity to fishes

Species	Exposure duration	Endpoint	Effect	Conc (mg/l)	References	Validity*
Leiostomus xanthurus				High PAH concentrations in sediment and water	Hagris et al., 1984	3
Lepomis macrochirus	200 h	NOEC (exposed to UV)	Survival	0.0012 - 0.0135	Oris, J.T. and Giesy, J.P.,1986	2

Species	Exposure duration	Endpoint	Effect	Conc (mg/l)	References	Validity*
Pimephales promelas	63 d	NOEC (exposed to sunlight)	Deformities	<0.006	Hall A.T. et al., 1990, Hall A.T. & Oris, J.T. 1991	2
Pimephales promelas	63 days	LOEC (exposed to sunlight)	Survival and hatching	0.012	Hall A.T. et al., 1990, Hall A.T. & Oris, J.	2

* by Klimisch et al, 1997

3.2.1.1.2 Aquatic invertebrates

Acute toxicity

The acute toxicities of 38 hydrocarbons and chlorinated hydrocarbons were detected for 2 planktonic crustaceans, freshwater *Daphnia magna* and saltwater *Artemia*. The vials were not opened during the course of the exposure, and no aeration was carried out, so that the levels of dissolved oxygen decreased. Monitoring of the oxygen levels showed that the lowest levels reached after 48 hours was ~60% of the starting level (this is the minimum level recommended in the OECD test guideline for *Daphnia*). Exposures were conducted in the dark. The results of the study were: *Daphnia* 48 hour LC_{50} 36 µg/l; *Artemia* 24 hour LC_{50} >50 µg/l. The result at 48 hours with *Artemia* was the same as at 24 hours. Effect values were estimated using a graphical method. The study used non-standard conditions in that the exposures took place in the dark, whereas the guidelines recommend a light-dark cycle; however, this is not considered to invalidate the study over the short exposure times. The study can be used in the assessment, but recognising that the exposure concentrations are not certain. In both cases, median lethal concentrations (LC50) at 48 h and 24 h, respectively, were strongly correlated with aquatic solubility, with little direct dependence on chem. structure. In the case of solid chemichals, it is suggested that the appropriate correlation solubility is that of the subcooled liquid chemichal, not that of the solid. Many solids constrained by low water solubility are thus unable to achieve dissolved concentrations sufficient to cause acute toxicity to the test organisms. It is hypothesised that acute toxicity is nonselective and is controlled by organism-water partitioning so that each hydrocarbon group may contribute equally to toxicity and no single group of hydrocarbons is the dominant toxicant. The partitioning characteristics are expressed in 2 math. models which related the LC_{50} to the chemichal properties of solubility and molar volume. An LC_{50} for anthracene of 36µg/l was detected for *Daphnia magna*. (Abernethy et al., 1986) (Validity : 3)

Kagan et al (1985) exposed a number of different organisms to a series of polycyclic aromatic hydrocarbons. Three invertebrates were included: the mosquito *Aedes aegypti*, the brine shrimp *Artemia salina*, and the water flea *Daphnia magna*. Few details of the conditions under which the organisms were cultured are reported. Four to seven repeat exposures were conducted at each concentration. The lamps used to irradiate the solutions are described with a maximum intensity at 350 nm, but a detailed comparison with natural sunlight is not included. Different exposure conditions were used for each organism. For *A. aegypti*, around 20 first instar larvae were incubated in the dark overnight in a solution of the test substance, and were then irradiated for one hour. Survival after this one hour period was the basis for the determination of the LC_{50} as 0.15 mg/l. (The units for the LC_{50} values are not included in the table in the report, but throughout the rest of the paper concentrations are reported as ppm or mg/l. Effect concentrations were determined from plots of survival as a function of

concentration.) With *A salina*, incubation with the substance took place for two hours before irradiation for one hour. Survival was determined immediately after irradiation, and the LC_{50} value was 0.02 mg/l. For *D magna*, adult organisms were incubated for one hour before a one-hour irradiation period; the LC_{50} for survival after this was 0.02 mg/l. The report notes that anthracene was not toxic to any of the organisms in the dark below 1 mg/l, and that solutions of anthracene previously irradiated in the absence of organisms also showed no enhanced toxicity in the dark (there is no indication of the duration of exposures in the dark). The exposures in this study do not follow any standard guidelines. The pattern of exposure, with incubation in the dark followed by irradiation, is not considered to be relevant to most environmental conditions. There is no indication of concentrations in the exposures being measured. The study is therefore considered not valid for the determination of a PNEC. (Validity: 2 for *Aedes aegypti*, 3 for *Daphnia magna* and 3 for *Artemia salina*)

Photoinduced anthracene toxicity to *D. pulex* was investigated by Allred, P. M. and Giesy, J. P. (1985). Adult *Daphnia pulex* were exposed to anthracene in the dark for 24 hours. Then they were exposed to full sunlight for half an hour. Organisms were exposed to 3 nominal anthracene concentrations (3.0, 9.6, and 30.0 μg/l) in static bioassays on clear, partly cloudy, and cloudy days. Photoinduced anthracene toxicity was not observed under laboratory conditions; it occurred only in the presence of solar radiation. Anthracene was only slightly less toxic to organisms transferred into water. This indicates that toxicity resulted from activation by solar radiation of material present on or within the animals and not in the water. Activation appeared to be of anthracene and not anthracene degradation products, since similar concentrations of anthraquinone [84-65-1], the primary and most stable degradation product of anthracene, were not toxic at similar solar radiation intensities. In addition to that, a series of filters was used to selectively remove UV wavelengths from solar radiation to detect the photoactive wavelengths. Mylar film absorbs in the UV-B region (285-315 nm) of solar radiation and Corning 0-52 glass absorbs essentially the entire spectrum of UV wavelengths (285-380 nm). Placement of Mylar film over bioassay beakers diminished photoinduced anthracene toxicity only slightly, whereas Corning 0-52 glass reduced toxicity proportionate to the redn. in UV intensity. Thus, wavelengths in the UV-A region (315-380) are primarily responsible for photoinduced anthracene toxicity. For exposure to full sunlight, the LC50 is estimated to be 1.0 μg/L. (Validity: 2)

The toxicity of anthracene was assessed in a variety of aquatic organisms under environmentally realistic conditions. In the presence of natural or simulated sunlight, anthracene was acutely toxic, at concnentrations within aquatic solubility limits, to freshwater zooplankton, insect larvae, and fish. Less than 15 min was required for 50% immobilization of *Daphnia pulex* at 1.2 μg anthracene/l under natural sunlight (UV-B, 310 nm, = 484 μW/cm2). (Oris, J. T. et al., 1984) (Validity: 2)

degradationdegradationIn saltwater species, acute toxicity occurs at concentrations al low 300μg/l and for freshwater system it can be expected in a wide range in specific PAHs. The median effective concentration after 48h and 24h for immobilization was calculated be computerizing log-probit analysis for *Daphnia magna*. EC50 was calculated at 211μg/l at 24h and 95μg/l at 48h of exposure. (Munoz and Tarazona, 1993). (Validity: 2)

Daphnia magna was examined for toxic effects of anthracene from Smith et al (1988). The method based on batch test with daphnid neonates according to ASTM and EPA standards. Exposed period was 48 h at 20 degree C, age of daphnia was <24 h and endpoint was immobilization. EC50 was detected at 754μg/l. (Validity: 2)

EC50 value of anthracene for *Daphnia magna* was determined at 22µM after preaccumulation period (23 ± 1h) in the dark, exposed to biological effective UV-B dose rate 565mW/m^2 for 15 min followed by 24h in the dark. (Huovinen, P.S. et al., 2001). (Validity: 2)

Newsted and Giesy (1987) used semistatic system 1 day old organisms. *Daphnia magna* was exposed I day to simulated sun (16 h light/ 8 h dark) prior to irradiation with UV light for 24 h. Testing temperature was 23 degree C and pH 7.5. LT50 was detected at 298.5 min in concentration 15µg/l. (in Mekenyan O.G, et al., 1994). (Validity: 2)

Blue mussels (*Mytilus edulis*) were forts acclimated for 3-4 weeks in 10 C water, and then anthracene was injected with 10, 25, 50 or 100µg/mussel. Sampling was after 24 hours (48, 96 and 168 hours for the 100µg/mussel group). There was found to be a dose dependent lysosome destabilization and release of hexosaminidase in the digestive cells after 24 hours. This was accompanied by cytological evidence of cytolysis of the digestive cells. With a dose of 100µg, the destabilization effect persisted for 96 hours, returning to normal by 168 hours. (Moore, M.N. et al., 1978) (Validity: 3)

The acute toxicity of photoactivated anthracene to the glochidial larvae of the paper pondshell (*Utterbackia imbecillis*) was characterized in the laboratory using two sets of experiments. Acute toxicity tests designed to detect the overall sensitivity of glochidia to anthracene were conducted under simulated sunlight (UV [UV]-A = 70 ±0.5 µW/cm2 [mean ± std. deviation]). The median lethal concentration (LC50) and median LD (LD50) of anthracene at 24 h were 1.93 (95% CI, 0.87-3.02) µg/l and 1.94 (1.87-2.00) µg/g. Acute toxicity tests designed to delineate the relationship between the rate of mortality and UV intensity were conducted under one of four different UV intensities (UV-A = 15, 31, 50, and 68 µW/cm2). These findings suggest that glochidia of freshwater mussels are sensitive to photoactivated anthracene at environmentally relevant concnentrations, that the time-dependent mortality of glochidia can be accurately predicted through evaluating the product of tissue residue and light intensity, and that species-dependent factors may alter the predicted relative potencies of anthracene. (Weinstein, J. E. et al. 2001). (Validity: 2)

Culicid mosquito larvae were sensitive to anthracene phototoxicity with a 24-h median lethal concentration (LC50) value of 26.8 µg/L at a solar UV intensity 5 times less than summer max. in Michigan. (Oris, J. T. et al., 1984) (Validity : 2)

To quantify the effect of environmentally realistic variation in light spectra on toxicity, brine shrimp (*Artemia salina*) assays were conducted under various light spectra and with 3 PAHs (pyrene, fluoranthene, and anthracene) of known phototoxicity potential. Based on the absorbance spectra of these PAHs, it was predicted that toxicity, quantified using immobilization as the endpoint, would not vary significantly among light spectra in anthracene assays. The results supported these assumptions. In the anthracene exposures, the glass filter IT50 (62.2 min) was not different from the KCr filter IT50 (63.8 min). These results indicate that quantifying the spectral characteristics of PAH-contaminated aquatic environments may be not an important component of risk assessment at these sites for anthracene. (Diamond, S.A. et al. 2000) (Validity : 2)

In the study of Pelletier et al (1997), lavrae and juvenils of the bivalve, *Mulinia lateralis* and juveniles of the mysid shrimp, *Mysidopsis bahia*, were exposed to individual known

phototoxic PAHs, as well as the water-accomodated fractions of several petrolium products containing PAHs. LC_{50s} and EC_{50s} exceeded the water solubility of anthracene. (Validity: 2)

The fresh water coelenterate *Hydra attenuata* was used for a developmental toxicity screening assay and, in a modified form, aquatic toxicity assessment of effluents. Because Hydra is markedly insensitive to UV irradiation it occurred that it might also be manipulated into a photosensitization assay. There are a few in vitro phototoxicity assays available and if one could be devised with Hydra, marked efficiency would result from use of the same animal for multiple assays. Adult Hydra was exposed to a series of concentrations for 24 hrs followed by 24 hrs in standard hydra medium. One-half of the test populations received exposure of UVA radiation during the first 16 hrs of chemical exposure. Except during actual UV exposure periods, all animals were maintained in total darkness. A Blakray lamp (20mw/cm2 with peak emission at 365nm) was placed 29 cm from the test dishes. A phototoxicity index was determined through the comparison of light and dark NOAEL concentrations. Adult Hydra in its usual media is relatively insensitive to UVA radiation. Exposure to the known phototoxicant anthracene resulted in greater than 100X increase in toxicity in the presence of UVA. (Newman & Johnson, 1991).(Validity: 3)

Table 3.38 Acute toxicity in aquatic invertebrates

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
Waterflea (*Daphnia magna*)	48 h	LC_{50} in dark	OECD	0.036	Abernethy et al., 1986	3
Waterflea (*Daphnia magna*)	0 h	EC_{50} (exposed to UV)		0.020	Kagan et al, 1985	3
Waterflea (*Daphnia pulex*)		LC_{50}		0.001	Allred & Giesy, 1985	2
Waterflea (*Daphnia magna*)	14 min	EC_{50} (exp. to UV)	Immobilisation	0.0012	Oris, J. T. Et al., 1984	2
Waterflea (*Daphnia magna*)	24- 25 h	EC_{50} (exposed to sunlight and in dark)	Immobilisation	0.0012	Oris, J. T. Et al., 1984	2
Daphnia magna	24h 48h	EC_{50}		0.211 0.0095	Munoz and Tarazona, 1993	2
Daphnia magna	48 h	EC_{50}	ASTM and EPA standards	0.754	Smith et al, 1988	2
Daphnia magna	23 ± 1h	EC_{50}		22μM	Huovinen, P.S. et al., 2001	2
Daphnia magna	24 h	LT50 298.5 min	Survival	0.015	Newsted	2

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
		in concentration			and Giesy, 1987	
Daphnia pulex	Static bioassay	Photoinduced toxicity	Not observed under laboratory conditions; it occurred only in the presence of solar radiation	0,0030, 0,0096, and 0,0300	Allred, P. M. and Giesy, J. P. (1985)	3
Mytilus edulis	24 h		Lysosome destabilization and release of hexosaminidase in the digestive cells	0.010, 0.025, 0.050 or 0.100 mg/mussel	Moore, M.N. et al., 1978	3
Paper pondshell (*Utterbackia imbecillis*)	24 h	LC_{50} (exposed to UV)	Survival	0.00193	Weinstein, J. E. et al. 2001	2
Culicid mosquito larvae	24 h	LC_{50}	Survival	0.0268	Oris, J. T. Et al., 1984	2
Mosquito (*Aedes aegypti*)	24 h	LC_{50} (exposed to sunlight and in dark)		<0.001	Kagan et al, 1985	2
Brine shrimp (*Artemia salina*)	1 h	EC_{50} (exposed to UV)		0.020	Diamond, S.A. et al. 2000	2
Brine shrimp (*Artemia salina*)	48 hour	LC_{50}		>0.05	Abernethy et al., 1986	+2
Brine shrimp (*Artemia salina*)	2 hour	LC_{50}		0.020	Kagan et al (1985	3
Brine shrimp (*Artemia salina*)	10 h	EC_{10}		0.023	Peachy & Crosby, 1996	
Mysid (*Mysidopsis bahia*)	48 hours	LC_{50} (exposed to UV)		0.0036	Pelletier et al, 1997	2
Mysid (*Mysidopsis bahia*)	48 hours	LC_{50} (exposed to fluorescent light)		0.535	Pelletier et al (1997	2

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
Bivalve (*Mulinia lateralis*) (embryo-larvae)	48 hours	LC_{50} (exposed to UV)		0.00647	Pelletier et al. 1997	2
Bivalve (*Mulinia lateralis*) (embryo-larvae)	48 hours	LC_{50} (exposed to fluorescent light)		4.260	Pelletier et al. 1997	2
Bivalve (*Mulinia lateralis*) (juvenile)	96 hours	LC_{50} (exposed to UV)		0.0689	Pelletier et al. 1997	2
Bivalve (*Mulinia lateralis*) (juvenile)	96 hours	LC_{50} (exposed to fluorescent light)		13.3	Pelletier et al. 1997	2
Polychaete (*Nereis areaceodentata)*	96 hours	LC_{50}		0.051	DEFRA	
Hydra attenuate	24 hrs	NOAEL	Phototoxici ty		Newman & Johnson, 1991	3

* by Klimisch et al, 1997

Long-term toxicity

Daphnia magna were exposed to anthracene in the presence or absence of ecologically relevant intensities of UVR for 21 days. Exposure to 8.2µg/l anthracene in the absence of UVR significantly reduced the number of neonates produced by 13.8%. However, exposure to UVR in the absence of anthracene had no significant effect on the fecundity of *D. magna*. Concurrent exposure of *D. magna* to UVR and anthracene resulted in further reduced survival and fecundity. Exposure of *D. magna* to 7.2µg/l anthracene and 117µW/cm^2 UV-A radiation resulted in 70% mortality or 69% decrease in production of neonates by *D. magna* that survived (Holst and Giesy, 1989). (Validation: 3)

Photoenhanced effects of anthracene exposure on reproduction of *Daphnia magna* in terms of total clutch size and survival over a 21-d period reported by Foran J.A. et al (1991). NOEC in UV exposure was detected at 1.9-2.2µg/l NOEC without UV exposure detected at 2.2µg/l. (Validation: 2)

Table 3.39 : Long-term toxicity in aquatic invertabrates

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
Midge (*Chironomus tentans*)	10 d	LC_{50} (exposed to UV)	Survival	0.006	EPA, 2000	2
Amphipod (*Hyalella azteca*)	10 d	LC_{50} (exposed to UV)	Survival	0.0056	Lee et al, 2002	
Waterflea (*Daphnia*	21 d	NOEC		0.0019 -	Foran J.A.	2

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
magna)		(exposed to UV)		0.0022	et al, 1991	
Waterflea (*Daphnia magna*)	21 d	NOEC		0.00063 ppm	Holst and Giesy, 1989	3
Waterflea (*Daphnia magna*)	21 d	70 % mortality and 70% decrease in production of neonates in survivors (exposed to UV)		0.0072	Holst and Giesy, 1989	3

* by Klimisch et al, 1997

3.2.1.1.3 Algae

Acute toxicity

Algae appeared to be slightly more resistant to photo-induced toxicity of anthracene than fishes and invertebrates. An environmental hazard assessment suggests that some aquatic systems are sufficiently contaminated by PAH that a hazard to natural algal communities due to photo-induced toxicity of PAH may be present (Gala and Giesy, 1992). (Validation: 2)

The ability of carotenoid pigments to protect the green alga, *Selenastrum capricornutum*, from the photo-induced toxicity of the linear three-ring PAH, anthracene, was investigated. Fluridone, a carotenoid biosynthesis inhibiting herbicide, was utilized to generate algal cells with different levels of colored carotenoids so that the protection provided by colored carotenoid pigments in algal cells to the photo-induced toxicity of anthracene could be measured. The percent inhibition of [14C]bicarbonate incorporation due to the photo-induced toxicity of anthracene was inversely correlated to the concentrations of colored carotenoid pigments in *S. capricornutum* cells. (Gala and Giesy, 1993). A dose-response relationships among anthracene concentration, ultraviolet (UV) radiation intensity, and algae growth rate and ^{14}C-bicarbonate incorporation were determined. The 22h EC50 for specific growth rate was inversely related to UV-A radiation intensity and ranged from 37.4 to 3.9µg/l anthracene. The threshold for photo-induced toxicity of anthracene was 1.5–3µg/l anthracene, however, no UV-A radiation threshold was evident for many of the measurement of toxicity studies. No effects on growth rate was observed at concentration of anthracene less than 3µg/l (NOEC) an any UV-A intensity tested. However, algal cells of *Selenastrum capricornutum* exposed to anthracene concentrations greater than 12µg/l exhibited a lesser growth rate al all UV-A intensities than oh the controls. Growth rate was also inhibited at concentrations of 5-6µg/l anthracene in combination with UV-A intensities greater than 400µW/cm^2. EC50 and EC10 were estimated at a range of 37.4 to 3.9 µg/l and 7.8 to 1.5 µg/l. Therefore, authors suggest that a toxicity threshold (NOEC) of 1.5µg/l anthracene may be more ecologically relevant (Gala and Giesy, 1992). (Validation: 2)

The photoinduced toxicity of anthracene to the green alga *Sellenastrum capricornutum* was assessed by the use of flow cytometry to measure cell size, cellular chlorophyll concentration,

and cell viability. Anthracene was slightly toxic in the absence of UV-A radiation. The detection of the direct toxicity of anthracene in this study at a concentration of 19 µg/l anthracene resulted from the use of sensitive flow cytometry measures. There was a significant interaction between anthracene and UV-A radiation, which, in combination, caused significant toxic effects on *S. capricornutum*. The most sensitive flow cytometric measure of toxicity was the stress index (SI), which was predictive of longer term effects on cell growth. The 28-h EC50 and EC10 for SI for *S. capricornutum* were 16.1 and 8.3 µg/L anthracene, respectivelly, at 125 µW/cm2 UV-A. All combinations of anthracene and UV-A that inhibited alga growth also caused a significantly greater number of nonviable cells. The flow cytometric methods used in this study proved to be sensitive, predictive measures of the direct and photoinduced toxicity of anthracene and UV-A radiation to *S. capricornutum.* (Gala & Giesy, 1994). (Validation: 2)

The effects of selected polycyclic arom. hydrocarbons (PAHs) on the growth of *S. capricornutum* in 3 light regimens were examined In gold fluorescent light anthracene at 40 µg/l had no effect on algal growth. In cool-white fluorescent light, 30% inhibition of algal growth occurred with 40 µmol/l anthracene totally inhibited growth. (Cody, T. E. et al; 1984). (Validation: 2)

When the alga *Selenastrum capricornutum* was exposed to anthracene at 1, 10 and 100% saturation for 2 hours duration, no inhibition of photosynthesis was observed. The LC50s for the polycyclic aromatic hydrocarbon photoproducts generated in sunlight were an order of magnitude lower than the LC50s for the polycyclic aromatic hydrocarbons applied in intact form (Huang et al., 1995) *Nitzschia closterium*, *Isochrysis galbana* and *Platymonas subcordiformis* were stressed by anthracene at different concentrations for 72 h. Then the relative increasing rates and the relative contents of chlorophyll a were detected. For anthracene, their sensitivities from high to low were *N. closterium, I. galbana* and *P. subcordiformis*. Their 72-h EC50s were 0.060 mg/L, 0.065 mg/L and 0.094 mg/L respectively. (Huang, J. et al. 2000) (Validation: 2)

Toxic effects of anthracene on *Chlorella pyrenoidosa, Chlorella vulgaris* and *Chlorella protothecoides* in relation with their heterotrophic character were compared. It was demonstrated that the consequence of their resistant ability was *C. protothecoides* (heterotrophic) > *C. pyrenoidosa* (autotrophic) > *C. vulgaris* (autotrophic) > *C. protothecoides* (autotrophic). The EC50 values were 2.53 mg/L (24 h), 1.47 mg/L (96 h), 1.27 mg/L (96 h), 0.85 mg/L (96 h), respectively. (Yan, X. et al. 1999) (Validation : 2 and 3 for *Chlorella pyrenoidosa* NOEC)

EC50 for *Chlorella fusca* was detected at 536µg/l from Hutchinson et al (1980). Remark of this study is that nominal concentration was 10 times as high as water solubility. (Validity: 3)

In study of Oris, J.T. et al., (1984), a freshwater green alga was not adversely affected by the light-anthracene combination. Evidence exists which suggests that anthracene is only 1 of many PAHs that can caused photoenhanced toxicity, and concentrations of these compounds are expected to increase in surface waters as a result of increased use of fossil fuel for heat and energy. No adverse effect in concentrations range between 3 and 30µg/l was detected for *Chlorella pyrenoidosa.* That was a photoinduced toxicity method in laboratory and field. Prior to irradiation *Chlorella* was exposed for 24 h in the dark. Irradiation time was 3 h with Uvb, 310nm. Intensity in laboratory test was 130µW/cm^2 and in field test 180-1126µW/cm^2.(Validity: 2)

The toxic effect of aromatic hydrocarbons on the productivity of various marine planktonic algae, *Dunaliela biocula, Phaeodactylum tricornutum* and *Isochysis galbana*, increasing with number of aromatic rings. Taxonomic differences in the sensitivity to aromatic hydrocarbons were not demonstrated (Jensen et al., 1984). (Validity: 4)

Table 3.40 : Acute toxicity in alga

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity *
Freshwater alga (*Selenastrum capricornutum)*	22 h	NOEC	Inhibition of growth	<0.003	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*		NOEC	Inhibition of growth	0.04	Cody, T. E. et al; 1984	2
Freshwater alga (*Selenastrum capricornutum)*	22 h	EC_{50} (exposed to UV)	Inhibition of growth	0.0066	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*	22 h	EC_{50} (exposed to UV)	Inhibition of growth	0.0121	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*	22 h	EC_{50} (exposed to UV)	Inhibition of growth	0.0374	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*	22 h	EC_{50} (exposed to UV)	Inhibition of growth	0.0039	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*	22 h	EC_{10} (exposed to UV)	Inhibition of growth	0.0078-0.0015	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*	22 h	NOEC	Inhibition of growth	0.0015	Gala and Giesy, 1992	2
Freshwater alga (*Selenastrum capricornutum)*	28 h	EC50 EC10	Inhibition of growth	0.0161 0.0083	Gala and Giesy, 1994	2 2
Freshwater alga (*Selenastrum capricornutum)*		NOEC	Inhibition of growth	0.040	Cody, T. E. et al; 1984	2
Nitzschia closterium	72 h	EC 50		0.06	Huang, J. et al. 2000	2
Isochrysis galbana	72 h	EC 50		0.065	Huang, J. et al. 2000	2
Platymonas subcordiformis	72 h	EC 50		0.094	Huang, J. et al. 2000	2
Chlorella pyrenoidosa		NOEC	Inhibition of growth	0.003 - 0.03	Yan, X. et al. 1999	3
Chlorella	96 h	EC 50		1.47	Yan, X. et al.	2

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity *
pyrenoidosa					1999	
Chlorella vulgaris	96 h	EC 50		1.27	Yan, X. et al. 1999	2
Chlorella protothecoides (heterotrophic)	24 h	EC 50		2.53	Yan, X. et al. 1999	2
Chlorella protothecoides (autotrophic)	96 h	EC 50		0.85	Yan, X. et al. 1999	2
Chlorella fusca		EC 50		0.536	Hutchinson et al, 1980	3
Chlorella pyrenoidosa	24 h	NOEC		0.030	Oris et al, 1984	2
Dunaliela biocula, Phaeodactylum tricornutum and *Isochysis galbana*					Jensen et al., 1984	

* by Klimisch et al, 1997

3.2.1.1.4 Water plants

Photosynthetic activity was monitored both in vivo and in vitro by measuring chlorophyll α (Chl α) fluorescence, carbon fixation, and electron transport in duckweed *Lemna gibba*. In simulated solar radiation, inhibition of photosynthesis was more rapid with photomodified anthracene than with intact anthracene, and intact anthracene appeared to only inhibit photosynthesis following its photomodification. The primary site of action of phomodified anthracene was found to be electron transport at or near photosystem I(PSI). This was followed by inhibition of photosystem II (PSII) (Huang et al., 1997a,b,c). The toxicity of 16 PAHs to the duckweed *Lemna gibba* was measured as inhibition of leaf production in simulated solar radiation (a light source with a spectrum similar to that of sunlight). The photomodification rates of the PAHs showed a moderate correlation to toxicity. The median inhibitory concentration (IC50) was approximately 2mg/l, which is higher than the median effective concentration (EC50) for growth inhibition of *L. gibba* for either intact or photomodified anthracene (1.5 and 0.5mg/l respectively). Photomodified anthracene had a median inhibiitory time (IT50) of 2h (faster IT50=1.1 h). (Validity: 2)

The ability of a commercial (Aldrich Chemical Co.) humic acid (AHA) to ameliorate the photo-induced toxicity of polycyclic aromatic hydrocarbons (PAHs) was examined using *Lemna gibba*. Plants were exposed to anthracene and benzo(a)pyrene both with and without AHA and grown under visible light as well as lighting that simulates relative abundances of UV-A and UV-B in natural sunlight (SSR). Modest additions of 1.6 mg/l AHA were sufficient to ameliorate the photo-induced toxicity of 2 mg/l anthracene by improving growth rates to nearly 50% of controls and inducing minor recovery from complete chlorosis in the most highly affected plants. The protective effects of AHA on anthracene toxicity increased linearly with increases in AHA concentrations up to 6.2 mg/l. Slopes of these relationships changed in the presence of UV light relative to visible light treatments; thus UV changed the extent to which AHA mediates PAH toxicity. However, the net effect was still for AHA to

ameliorate PAH photo-induced toxicity even though UV has the potential to photooxidize AHA and enhance the production of potentially toxic reactive oxygen species from AHA photosensitization. (Gensemer, R.W. et al.1998) (Validity: 4)

The influence of a commercial humic acid (AHA) on the development of anthracene photoinduced toxicity to the duckweed *Lemna gibba* was examined using both room- and low-temp. (77°K) chlorophyll fluorescence assays. Plants were exposed to 2 mg liter-1 anthracene both with and without 6.2 mg/l of AHA and grown under either visible light or simulated solar radiation that mimics the natural abundance of UV radiation. Exposure periods ranged from 1 to 48 h to examine temporal changes in chlorophyll degradation and chlorophyll a fluorescence induction in response to these light and HA treatments. The onset of anthracene photoinduced toxicity followed a definite sequence; chlorophyll a fluorescence induction parameters (Fv/Fm, and t1/2) responded earliest to anthracene exposure, with observable chlorophyll degradation requiring up to 24 to 48 h. Of these, t1/2 was the most sensitive, with significant inhibition apparent within 1 h of exposure. Throughout the entire 48-h exposure, 6.2 mg/l AHA ameliorated the photoinduced toxicity of anthracene, in terms of both chlorophyll degradation and Fv/Fm inhibition. In contrast, AHA delayed the complete inhibition of t1/2 by only 1 to 24 h rather than permanently protecting the plants from anthracene damage to PS2. This suggests that AHA may slow, but not prevent, the entrance of either intact anthracene or its photooxidized byproducts under these exposure conditions. (Gensemer, R.W. et al.1999) (Validity : 4)

Huang et al (1995, 1997) demonstrated that simulated solar radiation, with a fluence rate of only 40 mumol/sec, increased polycyclic aromatic hydrocarbon toxicity to the duckweed *Lemna gibba* and that polycyclic aromatic hydrocarbon photomodified in simulated solar radiation (generally oxygenation of the ring system) are more toxic than the parent compounds. It is not known, however, to what extent toxicity of poly aromatic hydrocarbons can increase due to photomodification. Thus, natural sunlight, which has a high fluency rate (approximately 2000 µmol/sec), was used to photomodify anthracene and toxicity examined on growth inhibition of *Lemna gibba* and measured as the rate of production of new leaves over and 8 day period. Next the polycyclic aromatic hydrocarbons were photomodified in sunlight prior to incubation with the plants. The half-lives of the polycyclic aromatic hydrocarbons in sunlight ranged from 12 minutes to 30 hours. The degree that polycyclic aromatic hydrocarbon toxicity increased following photomodification in sunlight was probed. The mixtures of photomodified chemicals that were derived from each polycyclic aromatic hydrocarbon in sunlight were applied of *Lemna gibba* and growth inhibition under 100 mumol/m^{-2} . sec^{-1} of simulated solar radiation was determined. (Validity: 2)

Table 3.41 : Toxicity in water plants

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
Duckweed (*Lemna gibba*)		IC50 EC50 IT50 (1.1 h)	Inhibition of growth	0.002 1.5	Huang et al., 1997a,b,c	2
Duckweed (*Lemna gibba*)	20 d	IC_{50} (exposed to sunlight)	Inhibition of growth	0.2	Huang et al, 1995, 1997	2
Duckweed (*Lemna gibba*)	8 d	LOEC	Inhibition of growth	0.06	Huang et al, 1995,	2

Species	Exposure duration	Endpoint	Effect	Concentration (mg/l)	References	Validity*
					1997	
Duckweed (*Lemna gibba*)	8 d	EC_{50}	Inhibition of growth	0.8	Huang et al, 1995, 1997	3
Duckweed (*Lemna gibba*)	8 d	LOEC	Inhibition of growth	0.01	Huang et al, 1995, 1997	2

* by Klimisch et al, 1997

3.2.1.1.5 Microorganisms

Toxicity bioassays furnish reasonable estimates concerning the biological impacts of sediment contaminants, but provide no indications as the cause(s) of the observed toxic responses. The Microtox® toxicity assay utilizes freeze-dried cultures of the marine bacterium *Photobacterium phosphoreum* and is based on the inhibition of bioluminescence by toxicants. The results of several studies of pure compounds and complex chemically mixtures revealed that Microtox® generally agreed with standard fish and invertebrate bioassays. In sediment types the most toxic sediment extract was collected downstream from the abandoned creosote tank site and was visibly saturated with creosote and exhibited an EC50 of 0.03mg/ml. Statistical analysis using Spearman's procedure showed that concentrations of total PAHs and Microtox® toxicities of sediment extracts were significantly associated. Anthracene correlated with extract toxicity (r_s= -0.1984, p=0.043). EC50 for anthracene was estimated at 33.4mg/l. (Jacobs, et al., 1993). (Validity: 3)

Methodology was developed by El-Alawi et al (2002) for measuring photoinduced short-term toxicity of an important group of contaminants, polycyclic aromatic hydrocarbons, using the luminescent bacterium *Vibrio fischeri*. The sort-term assay did not reveal photoinduced toxicity for any of the test chemicals. However, photoinduced toxicity was apparent in the long-term assay, indicating that sort-term assays may be opaque to this key mechanism of PAHs toxicity. (El-Alawi et al., 2002) (Validity: 3)

Paramecium caudatum (protozoan) exposed to 0.1 mg/l of anthracene for 60 minutes, exhibited a 90% lethal photodynamic response (Epstein et al., 1993). (Validity: 3)

Photoinduced toxicity test of anthracene in *Paramecium aurelia* showed no lethal effect after 6 h exposure in the dark. After exposure to sun light, survival concentrations was:

0.1mg/l for 15min: 90%
0.1mg/l for 30min: 27%
0.1mg/l for 45min: 0%
1mg/l for 15 min: 0%

Test temperature was 20 (+/-2) degree C and irradiation with sunlight becomes on sunny days between 9.30 and 11.30 a.m. Prior to sun light exposure there was a 6 h period in the dark (Joshi P.L. and Misra R.B. 1986). (Validity: 3)

Static growth rate of *Escherichia coli* measured in logarithm phase. Anthracene inhibited bacterium growth at concentrations of 0.1 to 10 µmol/l. (Hass, et. al., 1975) (Validity: 3)

Table 3.42 : Toxic effects of anthracene in microorganisms

Species	Temp (°C)	pH	Exposure duration	Endpoint	Effect	Conc (mg/l)	References	Validity *
Marine bacterium (*Photobacterium phosphoreum*)				Microtox® toxicity assay	EC_{50}	33.4	Jacobs, et al., 1993	3
Marine bacteria (*Vibrio fischeri*)			18 h	Inhibition of growth	EC_{10}	0.023	El-Alawi et al, 2001	3
Marine bacteria (*Vibrio fischeri*)	20	7.2 +/- 0.1	15 min	Inhibition of luminescence	EC_{50} (incubated in darkness)	17.07	El-Alawi et al, 2002	3
Marine bacteria (*Vibrio fischeri*)	20	7.2 +/- 0.1	15 min	Inhibition of luminescence	EC_{50} (incubated in simulated solar radiation)	16.13	El-Alawi et al, 2002	3
Marine bacteria (*Vibrio fischeri*)	20	7.2 +/- 0.1	18 hour	Inhibition of growth	EC_{50} (incubated in darkness)	85.78	El-Alawi et al, 2002	3
Marine bacteria (*Vibrio fischeri*)	20	7.2 +/- 0.1	18 hour	Inhibition of growth	EC_{50} (incubated in simulated solar radiation)	0.13	El-Alawi et al, 2002	3
Paramecium caudatum			1 h	Mortality	90% lethal conc	0.1	Epstein et al., 1993	3
Paramecium aurelia	20		15min 30min 45min 15 min	Mortality	90% lethal conc 27% lethal conc 0% lethal conc 0% lethal conc	0.1 0.1 0.1 1	Joshi P.L. and Misra R.B. 1986).	3

* by Klimisch et al, 1997

3.2.1.1.6 Amphibians

Anthracene showed phototoxicity on late embrionic stages of *Rana pipiens*, with an LC50 of 0.065 ppm after 30 min o exposure and 0.025 ppm after 5 h. (Kagan et al., 1984). Values in ppm are not useful to calculate PNEC. That's why we check as valid or not valid the same study of Kagan et al, 1984. (Validity: 2 or 3)

Table 3.43: Toxic effects of antracene in amphibia

Species	Exposed period	LC50 (µg/l)	References	Validity*
Rana pipiens	24 h	110	Kagan et al., 1984	2
Rana pipiens	5 h	25	Kagan et al., 1984	2
Rana pipiens	30 min	65	Kagan et al., 1984	2

* by Klimisch et al, 1997

3.2.1.2 Calculation of Predicted No Effect Concentration (PNEC)

For most existing chemicals, the pool of data from which to predict ecosystems effects is limited. In these circumstances empirically derived assessment factors are used to calculate a predicted no effect concentration (PNEC). The PNEC is the level below which the probabilities suggest that an adverse environmental effect will not occur. It is not intended to be a level below, which the chemical in considered as safe. The PNEC is calculated dividing the lowest LC_{50} (lethal concentration) or NOEC value by the appropriate assessment factor. In establishing the size of these assessment factors, a number of uncertainties must be addressed to extrapolate from single species laboratory data to a multi-species ecosystem. These may be summarized as follows (TGD 2003):

Assessment factors

Available valid data	Assessment factor
At least one short-term LC_{50} from each of three tropic levels of the 'base-set' (fish, Daphnia, algae)	1000
One long-term NOEC (either fish or Daphnia)	100
Two long-term NOECs from species representing two tropic levels (fish and/or Daphnia and/or algae)	50
Long-term NOECs from at least three species (normally fish, Daphnia and algae) representing three trophic levels	10
Field data or model ecosystems	Reviewed on an individual basis

Toxicity data for anthracene are available for microorganisms, aquatic plants, aquatic invertebrates and fishes under acute conditions and chronic toxicity studies. However, most of these studies are photoinduced studies. Some concentrations are calculated and some others are measured. In some cases effect or not effect concentrations are higher than water solubility of anthracene. As anthracene has a phototoxic response, the studies used solar radiation should be used in PNEC derivation. Studies using UV radiation were not used in PNEC derivation.

Long- and short-term studies for aquatic plants and algae gives a wide range of values. However, the lowest EC50 value is 3.9µg/l for *Sellanastrum capricornutum* (Gala and Giesly, 1992), and NOEC 3µg/l for *Chlorella pyrenoidosa* (Oris et al, 1984) but under UV radiation.

For aquatic inverterbates, photoenhanced effects of anthracene exposure on reproduction of *Daphnia magna* over a 21-d period reported by Foran J.A. et al (1991) showed a NOEC in UV exposure at 1.9-2.2μg/l. NOEC without UV exposure detected at 2.2μg/l.
In fishes, NOEC detected at 1,2μg/l for 24 h for *Lepomis macrochirus* as the lowest value (Oris and Giesy, 1986). LC50s are always higher, with a lowest limit of 2,78μg/l for *Lepomis macrochirus* in the presence of simulated sunlight by Oris and Giesy (1985).

Comparison of the results for Daphnia magna (the most sensitive species for phototoxicity) reproduction from experiments with UV radiation, revealed that the NOEC-values from these radiated experiments are in the same order of magnitude as the unradiated test result. Taking into account all NOEC-values for reproduction of Daphnia magna, a geometrical mean of 1.9μg/l can be derived and applying an assessment factor 10 the $PNEC_{apuatic}$ becomes 0.19μg/l.

Anthracene is very phototoxic and toxic effects (LC50s) are observed at concentrations lower or equal to the lowest chronic effect concentrations. These acute effects are observed when organisms exposed to anthracene are irradiated by a source of ultraviolet radiation for a relatively short period of time (e.g. half an hour) . The strongest effects are observed for natural sunlight (e.g. Allred & Giesy, 1985). In the study by Allred & Giesy (1985), adult *Daphnia pulex* were exposed to anthracene in the dark for 24 hours. Then they were exposed to full sunlight for half an hour. A dose-response relationship can not be easily determined, because only one exposure concentration does not result in 100% effects . As the phototoxic effects can be observed after a short period of exposure, the long-term NOEC will be nearly identical to the short term NOEC or effect concentrations. This acute study with *Daphnia pulex* (Allred & Giesy, 1985) should be considered to be a worst-case situation for light intensity, because of the steepness of the dose-response relationship.The LC50 is estimated to be 1.0 μg/L . Therefore, an assessment factor of 10 to this LC50 is considered to be appropriate.
The PNEC derived from the above mentioned study, for anthracene in fresh water, is 0.1 μg/L. This PNEC for fresh water species is also suitable for marine aquatic species, because marine crustaceans seem to be subject to phototoxicity as well as fresh water species. From the limited data the sensitivity of marine species is comparable with that of fresh water species.

Calculation of PNEC for microorganisms
In Jacobs et al (1993) publication, a value of EC50 33.4mg/l is mentionedThis value is 1.000 times higher than anthracene solubility in water. A PNEC value of 3.34mg/l, from the above mentioned EC50 with an assessment factor of 10, could be derived. However, as the bacterium *Photobacterium phosphoreum* is a marine species it can not be considered as reliable.

No standard studies on the toxicity of anthracene on micro-organims of sewage treatment plants are available. Toxicity studies for the different PAHs are only available for the bacteria *Vibrio fischeri* (El-Alawi *et al*., 2001; 2002), and gives. EC_{50} for inhibition of luminescence 17.92 ± 1.22mg/l According to the EU TGD (2003), these results are however not feasible for the risk assessment, as *Vibrio fischeri* is a saltwater species.
Consequently, a $PNEC_{microorganisms}$ in STP can not be derived. Based on the water solubility of the substance (section 1.3), it can however be assumed that the PNEC values will be in the range of μg/l or higher. (CTPHT RAR, EC, 2008).

3.2.1.3 Toxicity test results for sediment organisms

Two sediment studies are available. The 28-d LC50 for *Chironomus riparius* (Bleeker et al., 2003) is 26 mg/kg_{dw}, recalculated to a sediment with an organic carbon content of 10%. It should be noted that although this value is an LC50, the exposure duration is rather chronic than acute. From the raw data, the LC10 is estimated to be 14 mg/kg_{dw}, recalculated to a sediment with an organic carbon content of 10%. At the lowest tested concentration significant effects on emergence time were still observed, however, only in females. For sediment with 10% organic carbon this NOEC will be lower than 21 mg/kg_{dw}. Because emergence ratio and survival are directly coupled and the effects on the emergence time are much less at 21 than at 29 mg/kg_{dw}, the EC10 of 14 mg/kg_{dw} can be considered as a chronic endpoint. A10-d LC50 was found for *Hyalella azteca* (Hatch & Burton, 1999). The used sediment has an organic carbon content of 0.39%. The LC50 recalculated to a sediment with 10% organic carbon is 85 mg/kg_{dw}.

3.2.1.4 Calculation of Predicted No Effect Concentration (PNEC) for sediment organisms

In the absence of data on the toxic effects of anthracene on sediment dwelling organisms it is proposed that a sediment partition method be used to calculate a $PNEC_{sediment}$. In using this approach the following assumptions are made:

- Sediment dwelling and aquatic organisms are equally sensitive to the chemical.
- The concentration in sediment, interstitial water and benthic organisms are at a thermodynamic equilibrium. The concentration in any of these phases can be predicted using the appropriate partition coefficient.
- The sediment/water partition coefficient can either be measured or derived on the basis of a generic partition method from separately measured characteristics of the sediment and the properties of the chemical.

The $PNEC_{sediment}$ is given by the following equation:

$$PNEC_{sed} = \frac{K_{susp\text{-}water}}{RHO_{susp}} \cdot PNEC_{water} .1000$$

Where:

$K_{susp\text{-}water}$ = suspended matter-water partition coefficient (151 m^3/m^3)

RHO_{susp} = bulk density of suspended matter (1150kg.m^{-3})

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}.

For anthracene the PNEC for aquatic organisms is 0.12 µg/l. This gives a $PNEC_{sediment}$ of 11,9 µg/kg using EUSES.

Based and agreed on Coal Tar Pitch RAR, for the risk assessment two (semi)chronic LC50s and one chronic LC10 are available. With an assessment factor of 100 for the lowest NOEC, a PNEC of 0.14 mg/kg_{dw} is derived.
For marine sediment species no data are available. The PNEC for marine sediment is derived by applying an assessment factor of 1000 to the NOEC, which results in a PNEC of 14 $\mu g/kg_{dw}$.

3.2.2 Terrestrial compartment

For soil data are available for annelida, macrophyta and collembola. The lowest usable endpoint is for reproduction of *Folsomia fimetaria*.

3.2.2.1 Toxicity test results

3.2.2.1.1 Plants

The toxicity of polycyclic aromatic hydrocarbons is known to be enhanced by light via photosensitization reactions (production of active oxygen) and photomodification of the chemicals (e.g., oxidation) to more toxic compounds. Anthracene toxicity in particular has been found to increase dramatically following photomodification. (Mallakin, et al., 1999)

In most tests with macrophyta no effects are observed either Mitchell et al., 1988). Only growth of seedlings of *Avena sativa* has a 14-d EC50 of 51 mg/kg_{dw} (Mitchell et al., 1988) and the NOEC for shoot and root growth of *Lolium perenne* exposed for 40 days (Leyval & Bunet, 1998) appeared to be smaller than 133 mg/kg_{dw}, both values recalculated to a soil with 2% organic carbon. The latter study (Leyval & Bunet, 1998) was performed with moderate visible light (PAR 400-700 nm at 130 $\mu mol/m^2/s$). At the lowest concentration 22 to 41% reduction in growth was observed. From the presented data at the three tested concentrations a reliable EC10 could not be derived. In the study by Mitchell et al., (1988), it can be deduced from the figure for percentage emergence of seeds that the LC10 for *Avena sativa* should be significantly lower than 170 mg/kg_{dw}. For the more sensitive effect of growth only the EC50 is presented. From the figure for time of emergence it is also obvious that for four plant species the NOEC lies below 17 mg/kg_{dw}.

Photoinduced toxicity of anthracene to the foliar regions of *brassica napus* (canola) and *Cucumbis sativus* (cucumber) has been estimated in simulated solar radiation. The levels of chemicals required for toxicity were similar to previous observations for *L. gibba* and *B. napus* root by Greenberg et al., (1993), Huang et al., (1993) and Ren et al., (1996). The shot weight of the plants exposed to 2mg/l anthracene was 75% of the control, while at 8mg/l it was only 40% of the control. This increase in toxicity was significant ($p<0.05$). The tested chemicals were chronically toxic to cucumber and the effects increased with doses. (Huang et al., 1996)

Several pure components of tar were tested to determinate which component damage plants, especially potatoes. When anthracene evaporates, it forms small crystals on the epidermis of the leaf; therefore, anthracene was mixed with glycerol to form small droplets on the leaf surface. Injuries occurred with anthracene, but only when the plants were placed in direct

sunlight after fumigation. Anthracene can photochemically form peroxides or the corresponding quinones in sunlight. When fumigated plants were placed behind glass wall absorbing u.v. radiation, no injuries occurred, although 5 weeks afterwards, the fluorescence microscope still indicated droplets on the leaves (Hallbwachs G. 1968)

The effect of polyaromatic hydrocarbons (PAHs) on mycorrhizal colonization in polluted soil, and the effect of *Arbuscular mycorrhizal* (AM) fungi on plant growth in these soils has been studied. Leek (*Allium porrum L.*), maize (*Zea mays L.*), ryegrass (*Lolium perenne L.*), and clover (*Trifolium subterraneum L.*) were grown in pots contg. a soil artificially contaminated with increasing concns. of anthracene or mixed with an industrial soil polluted with PAHs. Mycorrhizal colonization by the indigenous AM population of the nonpolluted soil was not significantly affected by the addition. of anthracene up to 10 g/kg. However, mycorrhizal colonization of clover and leek decreased when the industrial soil was added to the nonpolluted soil, while maize and ryegrass colonization was not affected. (Leyval, C. et al;1998)

The effect of soil-incorporated copper, triallate, and anthracene on the emergence and early growth of three Australian native species (*Banksia ericifolia, Casuarina distyla,* and *Eucalyptus eximia*) and 3 crop species (*Avena sativa, Cucumis sativus,* and *Glycine max*), was assessed using OECD Test Guideline 208. The crop species are sensitive species used in overseas phytotoxicity testing, and their responses were compared with those of the native species. Seeds were grown in pots in a glasshouse in a sandy loam soil at the chem. concns. of 0, 10, 100, 1000, and 2000 mg kg-1. LC50 and EC50 values were detected for each species. The most sensitive species was the monocotyledon *A. sativa*, whereas among the five dicotyledons *C. distyla* was most sensitive. All three chems. delayed emergence and affected seedling growth. The conditions of the OECD Test Guideline can be met under Australian conditions, but the Guideline requires modification for use with Australian native species. The results were: (Mitchell R.L. et al.,1988) (Validity: 1)

Table 3.44: Toxic effects of anthracene in terrestrial plants (Mitchell R.L. et al.,1988)

Species	Endpoint	Method	LC50 (mg/kg soil)	EC50* (mg/kg soil)
Avena sativa	emergence	OECD guide line 208	385-665	20-45
Casaurina distyla	emergence	OECD guide line 208	>1000	>1000
Eucalyptus emixia	emergence	OECD guide line 208	>1000	>1000
Cucumis sativus	emergence	OECD guide line 208	>1000	225-1655**
Glycine maxima	emergence	OECD guide line 208	>1000	>1000
Banskia ericifolia		OECD guide line 208	>1000	>1000

* post-emergence seedling growth ** air-dried soil

3.2.2.1.2 Earthworms

Oligochaete *Lumbriculus variegates* has been tested to binary mixture of polycyclic aromatic hydrocarbons (Erickson et al., 1999). For anthracene, the mean $\pm$SD percentages chemical remaining in the organisms for all treatments was 40.8$\pm$2.8 after 24h and 17.6$\pm$1.8 after 96h. After 96h of elimination under UV exposure, test animals contained significantly less chemical than animals held in dark. For anthracene, concentration were below the level of quantification for all sample except one. No mortality was observed on oligochaetes exposed to anthracene or binary mixture of PAHs for 96 h. However, there was significantly mortality

of oligochaete exposed to anthracene, either singly or in combination, upon subsequent exposure to UV light. Used concentration were 2.1, 7.1, 16 and 31.9 µg/l.
For the tested annelid *Enchytraeus crypticus*(Bleeker et al., 2003), no effects were observed in the highest concentration, i.e. NOEC ≥780 mg/kg$_{dw}$, reculculated to a soil with 2% organic carbon.

3.2.2.1.3 Other terrestrial organisms

The acute toxicities of polycyclic aromatic hydrocarbons (PAH) in cricket *Acheta domesticus* nymphs and adults were low when administered orally or topically. Anthracene produced LD50s >580 µg/g. Chronic ingestion of anthracene caused mortality significantly higher than that of controls. (Walton, B. T. 1980)

LC50 of *Agelaius phoeniceus* detected after feed uptake within 18 h and a single oral dose in propylenglycole after 2-6 weeks of settling down. LC50 was >111mg/kg bw (Schafer et al., 1983).

The lowest usable endpoint is for reproduction of *Folsomia fimetaria*.

Toxicity data include the effects of 16 PAHs on the survival and reproduction of the soil-dwelling springtail *Folsomia fimetaria*. The results show that anthracene significantly affected the survival or reproduction of the test organisms. Threshold values for the toxicity of anthracene could be expressed as pore-water concentration by the use of reported organic carbon-normalized soil-pore-water partitioning coefficients (K_{oc} values). A narcotic mode of toxic action for most substances has been suggested.. The EC10 recalculated to a soil with 2% organic carbon is 6.3 mg/kg$_{dw}$ (Sverdrup et al., 2002).

Effects were not observed in the related species *Folsomia candida* at the highest tested concentration (Bleeker et al., 2003). To explore toxicity of PACs to soil organisms the springtail *Folsomia candida* and the enchytraeid worm *Enchytraeus crypticus* were exposed to spiked natural soil (LUFA 2.2) for 28 days in a similar set-up for both test species. For all PACs tested *E. crypticus* was less sensitive than *F. candida*, but again pore water appears to be the main exposure route for both species. ~~When toxicity was expressed as pore water concentrations toxicity for both soil organisms was well described by the relationship between acute LC50values for *C. riparius* and log *Kow*.~~

3.2.2.2 Calculation of Predicted No Effect Concentration (PNEC)

The same assessment factors are used for the terrestrial systems as for the aquatic systems (TGD, 2003)

.Only few data on earthworms are available for terrestrial compartment. However, there are enough data from plants according standard tests (OECD guide line 208) and these data could be used to calculate PNEC. As 20mg/kg is the lowest EC50 value from *Avena sativa*, a PNEC of 20µg/kg derive using an assessment factor of 1000.

The PNEC for the terrestrial compartment has also been calculated using the equilibrium partitioning method in the Technical Guidance document. The PNEC for terrestrial compartment using EUSES is 10.9µg/kg using the PNEC calculated for freshwater. This value is not so much higher than PNEC from studies.

The effect concentrations from chronic studies with macrophyta are one order of magnitude or even less higher than the EC10 for *Folsomia fimetaria*. Because it can not be excluded that the NOEC or EC10 for some plant species is lower than that of *Folsomia fimetaria*, an assessment factor of 50 will be applied to the lowest effect concentration of the two remaining trophic levels, which is the EC10 for *Folsomia fimetaria*. **The PNEC for soil then becomes 130 µg/kg$_{dw}$.**

3.2.3 Atmosphere

It is not possible to calculate a PNEC for anthracene in the atmosphere although no observed effect concentrations for inhalation toxicity are available.

Chemical processes including ozone and hydroxide radical and photochemical reaction degrade atmospheric anthracene. The degradation of vapour phase atmospheric anthracene is expected to be faster than particle sorbed anthracene. The atmospheric half-life of anthracene may vary from hours to days. However anthracene is not thought to contribute to global warming, stratosphere ozone depletion or acidification.

Since the atmospheric lifetime is short and anthracene does not contain chlorine or bromine substituents, anthracene will not contribute significantly to stratospheric ozone depletion.

3.2.4 Secondary poisoning

Anthracene exhibits low acute systemic toxicity in animals after oral exposure, with LD50 of 8.12 g/kg in the rat (Mellon Institute, 1977). Repeated dose systemic toxicity data from rats (life-time exposure) and mice (90-day exposure) also suggest low toxic potential by the oral route. No specific targets are revealed by these studies, and a small increase in ovary weight in mice receiving 500 mg/kg/day for 90 days is not considered to have toxicological significance. Thus values of oral NOEAL's of 50 mg/kg (rat) and 1,000 mg/kg (mouse) have been derived. Although the latter value is derived from 90-day, rather than life-time exposure, it is considered more reliable as it was based on a more recent and better conducted study and for this reason it will be adopted for risk assessment.

With 50% systemic absorption, an oral NOAEL of 1,000 mg/kg corresponds to a daily body burden of 500 mg/kg/day. As no data suitable for estimating limit values for systemic toxicity after inhalation or dermal exposure are available, in subsequent paragraphs this value of the body burden will be employed as a limit value corresponding to a NOAEL for systemic exposure, and compared with the body burdens arising from exposure from other routes in order to derive the corresponding MOS values.These data might not necessarily reflect the lowest tolerated exposure levels of the ecological relevance of the adverse effect and might therefore the potential risk is underestimated and questionable.

Using this NOAEL, a conversion factor of 8.3 (to convert to mg/kg bw/day to mg/kg food for mice) and an assessment factor of 90 (in accordance with the TGD), a PNEC of ≥92 mg/kg food can be estimated. . In the absence of reprotoxicity data for anthracene, such a high PNEC might not be adequately protective.

. Summary of PNECs

PNEC		AF	Species
fresh water (μg/l) PNEC marine water (μg/l) fresh water sediment (μg/kgdw)	0.1 0.1 140	10 10 100	*Daphnia pulex acute* *D. pulex acute* *C. riparius*
marine sediment (μg/kgdw)	14	1000	*C. riparius*
soil ($\mu g/kg_{dw}$) microorganisms (μg/l)of STP	130	50	*F. fimetaria*

3.3 RISK CHARACTERISATION [5]

The production of anthracene does fall under this ESR risk assessment which considers only pure anthracene, supplied under CAS Number 120-12-7. The scenarios cover the production of pure anthracene and the use (formulation) in pyrotechnics. All other values presented in the exposure assessment are for informative purposes only.

As the site-specific data did not refer only to the releases of the production of pure anthracene, but they included as well releases of other processes at the same site, the measured and calculated data provided an overestimation of the releases to water from the anthracene production process. Therefore the risk assessment for the production of anthracene can be consisered a worst case assessment.

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.

3.3.1 Aquatic compartment (incl. sediment)

Using 0.00011 ng/l ή 0.1×10^{-6} μg/l, (chapter 3.1.4.1.1), as a PEC water and 0.1 μg/l as the PNEC a PEC/PNEC ratio <1 is found, which does not indicate concern.
PEC/PNEC ratios derived using local PECs calculated from site-specific data or default values given in the Technical Guidance Document are given in next Table.

Table 3.45 : PEC/PNEC ratios for water

Process	PEClocal, surface water μg/l	PEC/PNEC
Production of anthracene	0.1×10^{-6}	<<1
Production of pyrotechnics (form.)	3.62×10^{-2}	<<1
Regional	1.22×10^{-5}	<<1
Continental	1.05×10^{-6}	<<1

Consequently :

Conclusion (ii) applies to production of anthracene. No further information is considered nessesary.

Conclusion (ii) applies for production of pyrotechnics.

[5] Conclusion (i) There is a need for further information and/or testing.
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 being applied already.
Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

Sediment
The PNEC for sediment from freshwater areas was calculated based on studies as 140 µg/kg. This can be compared with measured levels of anthracene in sediments.
As it has already been mentioned in chapter 3.4.1., based on the new site specific information, PEC local (sediment) is calculated as 0.00033 $\mu g/kg_{dwt}$ (Table 3.35 of CTPHT RAR version 9 (Jan 2006) .

Table 3.46 : PEC/PNEC ratios for sediment

Process	$PEC_{sediment}$ µg/kg	PEC/PNEC
Production of anthracene	0.00033	<<1
Production of pyrotechnics (form.)	107	<1
Regional	7.06×10^{-2}	<<1
Continental	6.05×10^{-3}	<<1

Conclusion (ii) applies to production of anthracene. No further information is considered nessesary.

Conclusion (ii) also applies for production of pyrotechnics.

Microorganisms
PNEC for microorganisms of STP can not be derived in the absence of data. However, based on the assumption that the PNEC has to be in the µg/l range or higher, it is not expected the calculated concentration for Anthracene in the production site (0.018 ng/l) will pose a risk for micro-organisms in a STP (**conclusion ii**), (see also the EC risk assessment report on coal tar pitch, high temperature (EC 2008).

Conclusion (ii) applies for the risk assessment for microorganisms.

3.3.2 Terrestrial compartment

PNEC for the terrestrial compartment has been calculated based on the studies for terrestrial organisms. This gives 130µg/kg. According to the CTPHT RAR local PECs are found as 51 ng/kg_{dwt} for agricultural soil and 100 ng/kg_{dwt} for grassland . These values give a PEC/PNEC ratio of <<1. In uncontaminated soils, anthracene levels are measured in ppt and so mean values of a PEC/PNEC ratio is < 1. No values are available for locations close to the production and use sites of anthracene. 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 anthracene are given in next Table with PNEC of 130µg/kg using EUSES.

Table 3.47 : PEC/PNEC ratios for soil

Process	PEC_{soil} µg/kg	PEC/PNEC
Production of anthracene	51 (agricultural soil) $\times 10^{-3}$ 100 (grassland) $\times 10^{-3}$	<<1 <<1
Production of pyrochnics (form.)	32.1	<1

Process	PEC_{soil} µg/kg	PEC/PNEC
Regional	2.31×10^{-3}	<<1
Continental	1.52×10^{-4}	<<1

Conclusion (ii) applies to production of anthracene. No further information is considered nessesary.

Conclusion (ii) applies for production of pyrotechnics.

3.3.3 Atmosphere

Due to the lack of data, no PNEC has been established for the atmospheric compartment. Moreover, the modelled air concentration can be considered as a conservative prediction. A more accurate measure of air concentration is difficult to be made with the generic tools available and can only be obtained by local measurements taking into account the site-specific conditions. See also the EC risk assessment report on coal tar pitch, high temperature (EC 2008. In the risk assessment for man indirectly exposed to the environment the exposure to air concentrations in the vicinity of the production plant is considered. Since conclusion ii) comes up for the risk assessment for man indirectly exposed to the environment, a conclusion ii) can also be qualitatively taken for the atmospheric compartment.

Conclusion (ii) applies for the risk assessment for the atmospheric compartment

3.3.4 Secondary poisoning

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A tentative PNEC of ≥92 mg/kg food can be estimated, using a NOAEL: 1000 mg/kg/day based on 90 d mice study (see EHC 202 report from the WHO, 1998), as it has already been mentioned in chapter 3.2.4. Although this high NOAEL and the corresponding high PNEC do not result any risks, a literature search on the reprotoxicity of PAHs done by RIVM concluded that as there is no reprotoxicity data for anthracene, such a high PNEC might not be adequately protective. Furthermore, the ecological relevance of the adverse effect on which this high NOAEL is based, might also be questionable, e.g.heamatological effect and increased ALAT activity.

It should be noted that as anthracene is characterised a vPvB and PBT substance risk management measures must be implemented. These measures could also affect the risk for secondary poisoning The most effective measures will be taken to minimise the emission of anthracene to the environment which automatically will reduce the risk for secondary poisoning. Therefore, it is not considered necessary to perform a full risk assessment for secondary poisoning..

3.3.5 Summary of PBT Assessment

According to Mackay et al. (1992), half lifes of anthracene are found 300-1000 hours for water, 125-420 days for soil and 420-1200 days for sediments. Furthermore, “anthracene fulfils the vP criterion based on simulation study resulting in a dt50 of 95-141 d in the water amd 57-210 d in the sediment (Lee et al., 1983).

Anthracene is not readily biodegradable according to the MITI I test (MITI 1992). Although such mass balance problems like Industry states would exist in ready-tests, it does not remove the fact that anthracene is not degraded in STP and thus finds its way to the environment, where the biotic degradation is even slower.

Anthracene also fulfils the vB criterion as there are valid studies with fish which provide “at least the lower BCF of 4550 (De Voogt, et al. 1991), that refers to the “parent compound” as well as the BCF of 6760 (De Maagd 1996, Table 3.20).

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Since there are valid studies for fish (Hall & Oris, 1991, Table 3.37) and aquatic invertebrates (daphnia Table 3.39) with chronic NOEC<, 0.01 mg/l (T-trigger) without UV light, anthracene clearly meets the T-criterion.

Therefore it can be concluded that anthracene meets the vP, vB and T criteria and hence is considered **as a vPvB and PBT substance.** Consequently, there is a need for limiting the risks; risk reduction measures that are already being applied shall be taken into account. **(Conclusion iii).**

4 RESULTS

CAS No.: **120-12-7**
EINECS No.: **240-371-1**
IUPAC Name: **Anthracene**

ENVIRONMENT

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

(X) 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

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

Conclusion iii) is reached, because:

- The substance meets the PBT and vPvB criteria. There is a need to minimize the emissions of anthracene into the environment.

Conclusion (ii) is reached, because:

- There is no concern identified for the aquatic, terrestrial and atmospheric compartment based on the PEC/PNEC approach for production of anthracene and the formulation of anthracene into pyrotechnics.

5 REFERENCES

REFERENCES
Chapter 1, 2

1. Andersson K., Levin J.-O. & Nilsson C.-A. (1983) Sampling and analysis of particulate and gaseous PAH's from coal-tar sources in the working environment. Chemosphere 12, 197-204
2. Clar E. (1964) Polycyclic Hydrocargbons, Academic Press, vol. 1., 288-307
3. Collin G. & Zauder M. (1982) Tar and Pitch. In: Ullmann's Encyclopedia of Technical Chemistry.
4. Collin G. and Höke, H., (1985) Anthracene. In: Ullman's Encyclopedia of Industrial Chemistry, 5th Ed., vol. 2.
5. Danish EPA (1995) Human health and environmental concerns of creosote. Danish EPA, Copenhagen.
6. Dean J. (1979) Lange's Handbook of Chemistry, 12th ed., Dean J. (ed.)
7. Franck H.-G. & Stadtlhofer, J.W. (1987) Industrial Aromatic Chemistry, Raw Materials, Processes and Products.
8. Geyer, H., Politzki G. and Freitag D. (1984) Prediction of ecotoxicological behaviour of chemicals: relationship between n-octanol/water partition coefficient and bioaccumulation of organic chemicals by alga chlorella. Chemosphere 13, 269-284.
9. Hashimoto Y., Tokura K., Kishi H. and Strachan W.M.J. (1984) Prediction of seawater solubility of aromatic compounds. Chemosphere 13, 881-888.
10. Hausigk D. and Koelling G. (1968) Űber racktionen von partiell hydrierten mehrkernigen aromatischen verbindungen mit 1,4-dichlor-butan. Chem. Ber. 101, 469-472.
11. IARC (1983a) International Agency for Research on Cancer Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Humans. Vol. 32, Bitumens, coal-tars and derived products, shale-oils and soots, Part 1.
12. Ingram L.L. Jr., McGinnis, G.D., Gjovik, K.R. et al. (1982) Migration of creosote and its components from treated piling sections in marine environments. Proceedings of the American Wood Preservation Association, 78, 120-128
13. Jandris L.J. and Forcė R.K. (1983) Determination of polycyclear aromatic hydrocarbons in vapor phases by laser-induced molecular fluorescence. Anal. Chim. Acta 151, 19-27.
14. Karickhoff S.W. et al. (1979), Sorption of hydrophobic pollutants on natural sediments. Water Res. 13, 241-248.
15. Kleffner H.W., Talbiersky J. and Zander M. (1981), Simple scheme for the estimation of concentrations of polycyclic aromatic hydrocarbons in tars. Fuel 60, 361-362. Cited in IARC (1983a)
16. Lang, K.F. and Eigen I. (1967), Forstschr. Chem. Forsch. 8, 91-141
17. Lax E. and Synowietz C. (1964) Taschenbuch für Chem. und Physicker. Lax E. and Synowietz C. (eds.).
18. Lehman E., Auffarth, J. & Hager, J. (1984) Determination of selected polycyclic aromatic hydrocarbons in coal-tar, coal-tar pitch and tar products. Staub-Reinhalt. Luft 44, 452-455
19. Lide D.R. (1991) CRC Handbook of Chemistry and Physics, 72nd ed., Lide D.R. (ed.), CRC Press, Boca Raton
20. Marlich L.I. and Leukevitch Z.K. (1973) Capillary chromatograph as a method for the rapid determination of the main component so in coal tar and coal tar fractions. Zh. ana. Khim. 28, 1193-1198. Cited in IARC (1983a)
21. May W., Walik S.P., Miller M.M., Tewari U.B., Brown-Thomas J.M. and Goldgerg R.N. (1983) Solution thermodynamics of some slightly soluble hydrocarbons in water. J. Chem. Eng. Data 28, 197-200.
22. McNeil D. (1983) Tar and Pitch. In: Encyclopedia of Chemical Technology (Kirk R.E. and Othmer D.F., eds.), 3rd edn.
23. Nabert K. and Schoen G. (1963) Sickerheitstechnische Kennzahlen brennbarer Gase und Daempfe, 2nd ed.
24. Neff J.M. (1979) Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fates and biological effects (Neff, J.M., ed.), Applied Science Publisheres Ltd., London.
25. Nestler F.H.M. (1974) The characterization of wood-reserving creosote by physical and chemical methods of analysis (USDE Forest Service Research Paper FPL 195), U.S. Department of Agriculture
26. Novotny M., Strand J.W., Smith S.L., Wieslder D. and Schwende F.J. (1981) Compositional studies of coal tar by capillary gas chromatograph/mass spectrometry. Fuel 60, 213-220. Cited in IARC (1983a).
27. Sangster J. (1989) Octanol-water partition coefficients of simple organic compounds. J. Phys. Chem. Reference Data 18, 111-1129.
28. Sax N.I. (1975) Dangerous properties of industrial materials, van Nostrand Reinhold Company, New York.
29. Schwartz F.P. (1977) Determination of temperature dependence of solubilities of polycyclic aromatic hydrocarbons in aqueous solutions by a fluorescence method. J. Chem. Eng. Data 22, 273-277.

30. Sonnefeld W.J., Zoller W.H. and May W.E. (1983) Dynamic coupled-column liquid chromatographic determination of ambient temperature vapor pressures of polynuclear aromatic hydrocarbons. Anal. Chem. 55, 275-280.
31. Sorgo P.M. (1996) Report of the European Commission (DG III) on the justification of the Danish position concerning the Directive on creosote.
32. Staesse H.L. (1954) Fractional distillation of creosote and composition of preservatives used in the coopative creosote programme. Proc. American Wood Preservers' Association 50, 13-40
33. Whitehouse B.G. (1984) The effects of temperature and salinity on the aqueous solubility of polynuclear aromatic hydrocarbons. Marine Chem. 14, 319-332.
34. Yoshida K., Shigeoka T. and Yamauchi F. (1983), Relationship between molar refraction and n-octanol/water partition coefficient. Ecotox. Environ. Safety 7, 558-565.

REFERENCES
Chapter 3

1. Abernethy S., Bobra A.M., Shiu W.Y., Wells P.G. and Mackey D, Aquatic Toxicol. (AMST), Acute lethal toxicity of hydrocarbons and chlorinated hydrocarbons to two planktonic crustaceans the key role of water organism-water partritioning. (1986), 8(3), 163-174
2. Aitken, Michael D.; Nitz, David C.; Roy, Denise V.; kazunga, Chikoma. Slurry-phase bioremediation of contaminated soil from a former manufacturer-gas plant site. Report - Water Resources Research Institute of the University of North Carolina (1998), (320), i-xviii, 1-65.
3. Allred, P. M.; Giesy, J. P., Solar radiation-induced toxicity of anthracene to Daphnia pulex. Environ. Toxicol. Chem. (1985), 4(2), 219-26.
4. Ankley, G.T. 1996. Evaluation of metal acid volatile sulfide relationships in the prediction of metal bioaccumulation by benthic macroinvertebrates. Environmental Toxicology and Chemistry 15(12):2138-2146
5. Andersson, B. E.; Henrysson, T. Accumulation and degradation of dead-end metabolites during treatment of soil contaminated with polycyclic aromatic hydrocarbons with five strains of white-rot fungi. Applied Microbiology and Biotechnology (1996), 46(5/6), 647-652.
6. Arey, J, and Atkinson, R (2003). Photochemical reactions of PAHs in the Atmosphere. In Douben (ed.) PAHs: An Ecotoxicological Perspective., Wiley, 47-64
7. Atagana, H. I.; Haynes, R. J.; Wallis, F. M. Bioremediation of creosote-contaminated soils by microbial intervention. Editor(s): Wickramanayake, Godage B. Case Studies in the Remediation of Chlorinated and Recalcitrant Compounds, International Conference on Remediation of Chlorinated and Recalcitrant Compounds, 2nd, Monterey, CA, United States, May 22-25, 2000 (2000), 131-138.
8. Atkinson, R.; Baulch, D. L.; Cox, R. A.; Hampson, R. F., Jr.; Kerr, J. A.; Troe, J. Evaluated kinetic and photochemical data for atmospheric chemistry. Supplement III. J. Phys. Chem. Ref. Data (1989), 18(2), 881-1097.
9. Atkinson R., arey J (1994) . Atmospheric chemistry of gas-phase polycyclic aromatic hydrocarbons : formation of atmospheric mutagens. Env. Health. Perspect. 102, 117-126.
10. Bagg, John; Smith, J. David. Polycyclic aromatic hydrocarbons in clams, sediments, and seawater from the Great Barrier Reef region, Australia. Polynucl. Aromat. Hydrocarbons: Meas., Means, Metab., Int. Symp., 11th (1991), Meeting Date 1987, 89-95.
11. Banerjee, D. K.; Fedorak, P. M.; Hashimoto, A.; Masliyah, J. H.; Pickard, M. A.; Gray, M. R.A Monitoring the biological treatment of anthracene-contaminated soil in a rotating-drum bioreactor. Appl. Microbiol. Biotechnol. (1995), 43(3), 521-8.
12. Stefan Bertilsson[1] and Anneli Widenfalk[2] Photochemical degradation of PAHs in freshwaters and their impact on bacterial growth – influence of water chemistry, Hydrobiologia, (2002),Volume 469, Numbers 1-3, 23-32.
13. Biermann, Heinz W.; Mac Leod, Helene; Atkinson, Roger; Winer, Arthur M.; Pitts, James N., Jr. Kinetics of the gas-phase reactions of the hydroxyl radical with naphthalene, phenanthrene, and anthracene. Environ. Sci. Technol. (1985), 19(3), 244-8.
14. Bilyk, Andrzej; Traczewska, Teodora M. Anthracene and phenanthrene degradation by bacteria immobilized on activated carbon and zeolites. Environment Protection Engineering (1999), 25(3), 123-129.
15. Binet, P.; Portal, J. M.; Leyval, C. Dissipation of 3-6-ring polycyclic aromatic hydrocarbons in the rhizosphere of ryegrass. Soil Biology & Biochemistry (2000), 32(14), 2011-2017.
16. Bjoerseth, Alf; Knutzen, Jon; Skei, Jens. Determination of polycyclic aromatic hydrocarbons in sediments

and mussels from Saudafjord, W. Norway, by glass capillary gas chromatography. Sci. Total Environ. (1979), 13(1), 71-86.
17. Bowling, J. W.; Leversee, G. J.; Landrum, P. F.; Giesy, J. P. Acute mortality of anthracene-contaminated fish exposed to sunlight. Aquat. Toxicol. (1983), 3(1), 79-90
18. Breedveld, G. D.; Briseid, T.. In situ bioremediation of creosote-contaminated soil: column experiments. Appl. Biotechnol. Site Rem., [Pap. Int. Symp. In Situ On-Site Bioreclam.], 2nd (1994), Meeting Date 1993, 204-12.
19. Brown et al, as cited in Investigations of USEPA,-600/7-79-164, (1979)
20. Brubaker WW, Hites RA (1998) . OH reaction kinetics of polycyclic aromatics hydrocarbons and polychlorinated dibenzo-p-dioxins and dibenzofurans. J.Phys. Chem. 102, 915-921.
21. Callahan, Michael A et al, Water-related environmental fate of 129 priority pollutants. Vol 1,EPA-440/4, 79-029a, Washington DC: USEPA 1979, p. 96-4.
22. Catallo, W. J.; Portier, R. J. Use of indigenous and adapted microbial assemblages in the removal of organic chemicals from soils and sediments. Water Sci. Technol. (1992), 25(3), 229-37.
23. Chemical insp. Test Inst., Biodegradation and bioaccumulation data of existing chemicals based on the CSCL Japan, Japan Chem. Ind. Ecol.-Toxicol. And inform. Center, (1992), ISBN 4, 89074-101-1 p2-112.
24. MITI (1992) - Biodegradation and Bioaccumulation data of existing chemicals based on the CSCL Japan. Chemicals Inspection and Testing Institute. Japan. October 1992. ln.
25. Clement, L.E., et al, Accumulation, fractionation and release of oil by the intertidal clam Macona balthica. Marine biology (1980), 57, 41-50.
26. Clemente, A. R.; Falconi, F. A.; Anasawa, T. A.; Durrant, L. R. Degradation of aromatic pollutants by a non-Basidiomycete ligninolytic fungus. Editor(s): Alleman, Bruce C.; Leeson, Andrea. International In Situ and On-Site Bioremediation Symposium, 5th, San Diego, Apr. 19-22, 1999 (1999), 8 105-110.
27. Cocchieri, Renata Amodio; Arnese, Antonio; Minicucci, Anna Maria. Polycyclic aromatic hydrocarbons in marine organisms from Italian central Mediterranean coasts. Mar. Pollut. Bull. (1990), 21(1), 15-18.
28. Cody, T. E.; Radike, M. J.; Warshawsky, D., The phototoxicity of benzo[a]pyrene in the green alga Selenastrum capricornutum. Environ. Res. (1984), 35(1), 122-32
29. David, Bernard; Boule, Pierre. Phototransformation of PAHs adsorbed on SiO2 in aqueous suspension. Polycyclic Aromat. Compd. (1993), 3(SUPPL.), 127-33.
30. de Voogt, P.; Van Hattum, B.; Leonards, P.; Klamer, J. C.; Govers, H. Bioconcentration of polycyclic heteroaromatic hydrocarbons in the guppy (Poecilia reticulata). Aquat. Toxicol. (1991), 20(3), 169-94.
31. Dean-Ross, Deborah; Moody, Joanna D.; Freeman, James P.; Doerge, Daniel R.; Cerniglia, Carl E.A Metabolism of anthracene by a Rhodococcus species. FEMS Microbiology Letters (2001), 204(1), 205-211.
32. De Maagd, P.-J. (1996). Polycyclic aromatic hydrocarbons: Fate and effects in aquatic environment. Ph.D. thesis, Utrecht University, Utrecht, The Netherlands.
33. Denzer, S, Herrchen, M, Lepper, P, Müller, M, Sehrt, R, Storm, A and Volmer, J (1999). Rivised proposal for a list of priority substances in the context of the water framework directive (COMMPS procedure). Ref 98/788/3040/DEB/E1
34. Diamond, Stephen A.; Mount, David R.; Burkhard, Lawrence P.; Ankley, Gerald T.; Makynen, Elizabeth A.; Leonard, Edward N. Effect of irradiance spectra on the photoinduced toxicity of three polycyclic aromatic hydrocarbons. Environmental Toxicology and Chemistry (2000), 19(5), 1389-1396.
35. Djomo, J. E.; Garrigues, P.; Narbonne, J. F. Uptake and depuration of polycyclic aromatic hydrocarbons from sediment by the zebrafish (Brachydanio rerio). Environmental Toxicology and Chemistry (1996), 15(7), 1177-1181.
36. Dore, M.; Brunet, N.; Legube, B. Participation of various organic compounds in the evaluation of global pollution criteria. Trib. CEBEDEAU (1975), 28(374), 3-11. Erickson, R. J., Ankley, G.T., DeFoe, D.L., Kosian, P.A., Makynen, e.A., (1999).Additive toxicity of binary mixtures of photolitic polycyclic aromatic hydrocarbons to the oligochaete Lumbriculus variegatus. Toxicol. Appl. Pharmacol. 154, 97-105.
37. EC (2001). Ambient Air Pollution by Polycyclic Aromatic Hydrocarbons (PAH). Position Paper. Prepared by the Working Group on Polycyclic Aromatic Hydrocarbons. July, 2001.
38. El- Alawi,Yousef S., George D. Dixon., BruceM. Greenberg (2001), Effects of a pre-incubation period on the photoinduced toxicity of polycyclic aromatic hydrocarbons to the luminescent bacterioum *Vibrio fischeri*, Environ. Toxicity, 16,277-286.
39. El-Alawi, Y. S.; McConkey, B. J.; Dixon, D. G.; Greenberg,B. M. (2002), Ecotox Environ Safety.
40. Erickson, R. J., Ankley, G.T., DeFoe, D.L., Kosian, P.A., Makynen, e.A., (1999).Additive toxicity of binary mixtures of photolitic polycyclic aromatic hydrocarbons to the oligochaete *Lumbriculus variegatus*. Toxicol. Appl. Pharmacol. 154, 97-105.
41. Fang, M.; Wan, C. K.; Wong, J. W. C. Enhancement of PAHs degradation by nonionic surfactants in composting system. Editor(s): Magar, Victor S. International In Situ and On-Site Bioremediation Symposium, 6th, San Diego, CA, United States, June 4-7, 2001 (2001), 6 73-80.

42. Fang, M.; Wan, C. K.; Wong, J. W. C. Enhancement of PAHs degradation by nonionic surfactants in composting system. Editor(s): Magar, Victor S. International In Situ and On-Site Bioremediation Symposium, 6th, San Diego, CA, United States, June 4-7, 2001 (2001), 6 73-80.
43. Field, J. A.; Boelsma, F.; Baten, H.; Rulkens, W. H. Oxidation of anthracene in water/solvent mixtures by the white-rot fungus, Bjerkandera sp. strain BOS55, Appl. Microbiol. Biotechnol. (1995), 44(1-2), 234-40.
44. Foran J.A., Holst L.L. and Giesy J.P Effects of photoenhanced toxicity of anthracene on ecological and genetic fitness of Daphnia magna: A reappraisal. Environ. Toxicol. Chem. (1991), 10 (3),425-427.
45. Frank A.P., Landrum P.F. and Eadie B.J. (1986) - Polycyclic aromatic hydrocarbon : Rates ofuptake, depuration, and biotransformation by Lake Michigan (*Stylodrilus heringianus*).*Chemosphere,* 15, 317-330
46. Freitag, D. et al, (1984) In: Kaiser, K.L.E. (ed), QSAR in Environmental Toxicology. D. Reidel Publ. Co., Dordecht/Netherlands, 111-136.
47. Freitag, D.; Ballhorn, L.; Geyer, H.; Korte, F. Environmental hazard profile of organic chemicals. An experimental method for the assessment of the behavior of organic chemicals in the ecosphere by means of simple laboratory tests with carbon-14-labeled chemicals. Chemosphere (1985), 14(10), 1589-616.
48. Gala, William R.; Giesy, John P., Flow cytometric determination of the photoinduced toxicity of anthracene to the green alga Selenastrum capricornutum., Environ. Toxicol. Chem. (1994), 13(5), 831-40.
49. Gala, William R.; Giesy, John P., Photo-induced toxicity of anthracene to the green alga, Selanastrum capricornutum. . Aquat. Environ. Contam. Toxicol. (1992), 23(316-323)
50. Gala, William R.; Giesy, John P., Using the carotenoid biosynthesis inhibiting herbicide, Fluridone, to investigate the ability of carotenoid pigments to protect algae from the photo-induced toxicity of anthracene. Aquat. Toxicol. (1993), 27(1-2), 61-70.
51. Gardner, Wayne S.; Lee, Richard F.; Tenore, Kenneth R.; Smith, Larry W. Degradation of selected polycyclic aromatic hydrocarbons in coastal sediments: importance of microbes and polychaete worms. Water, Air, Soil Pollut. (1979), 11(3), 339-47.
52. Gensemer R W; Dixon D G; Greenberg B M Amelioration of the photo-induced toxicity of polycyclic aromatic hydrocarbons by a commercial humic acid ECOTOXICOLOGY AND ENVIRONMENTAL SAFETY (1998 Jan), 39(1), 57-64
53. Gensemer, Robert W.; Dixon, D. George; Greenberg, Bruce M., Using chlorophyll a fluorescence to detect the onset of anthracene photoinduced toxicity in Lemna gibba, and the mitigating effects of a commercial humic acid. Limnology and Oceanography (1999), 44(3, Pt. 2), 878-888.
54. Gerould, Sarah; Landrum, Peter; Giesy, John P. Anthracene bioconcentration and biotransformation in chironomids: effects of temperature and concentration. Environ. Pollut., Ser. A (1983), 30(3), 175-88.
55. Geyer, H.; Politzki, G.; Freitag, D. Prediction of ecotoxicological behavior of chemicals: relationship between n-octanol/water partition coefficient and bioaccumulation of organic chemicals by alga Chlorella. Chemosphere (1984), 13(2), 269-84.
56. Giesy, J. P. et al, 1982. Fates and biological effects of polycyclic aromatic hydrocarbons in aquatic systems. EPA report IAG (1982), EPA-78-D-X0290
57. Goodin, J. D.; Webber, M. D. Persistence and fate of anthracene and benzo(a)pyrene in municipal sludge treated soil. J. Environ. Qual. (1995), 24(2), 271-8.
58. Gramss, Gerhard; Voigt, Klaus-Dieter; Kirsche, Brigitta. Degradation of polycyclic aromatic hydrocarbons with three to seven aromatic rings by higher fungi in sterile and unsterile soils. Biodegradation (1999), 10(1), 51-62.
59. Greenberg, Bruce M.; Huang, Xiao-Dong; Dixon, D. George; Ren, Lisha; McConkey, Brendan J.; Duxbury, Cheryl L. Quantitative structure activity relationships for the photoinduced toxicity of polycyclic aromatic hydrocarbons to duckweed - A preliminary model. ASTM Spec. Tech. Publ. (1993), STP 1216(ENVIRONMENT TOXICOLOGY AND RISK ASSESSMENT: 2ND VOLUME), 369-78.
60. Grosser, Robert J.; Warshawsky, David; Vestal, J. Robei. Mineralization of polycyclic and N-heterocyclic aromatic compounds in hydrocarbon-contaminated soils. Environ. Toxicol. Chem. (1995), 14(3), 375-82.
61. Hall A.T., Taylor D.H., Oris J.T. & McGovern J.A. Effects of anthracene on reproduction and learning in fethead minnows. Neurotoxicol. Teratol. (1990), 12, 563
62. Hall, A. Tilghman; Oris, James T. Anthracene reduces reproductive potential and is maternally transferred during long-term exposure in fathead minnows. Aquat. Toxicol. (1991), 19(3), 249-64.
63. Hass, Bruce S.; Applegate, Howard G., Effects of unsubstituted polycyclic aromatic hydrocarbons on the growth of Escherichia coli. Chem.-Biol. Interact. (1975), 10(4), 265-8.
64. Hatch, A.C. and Burton, G.A. (1999) Photo-induced toxicity of PAHs to Hyalella azteca and Chironomus tentans: effects of mixtures and behaviour. Environmental Pollution 106, 157-167.
65. Herbes, S. E.; Schwall, L. R.. Microbial transformation of polycyclic aromatic hydrocarbons in pristine and petroleum-contaminated sediments. Appl. Environ. Microbiol. (1978), 35(2), 306-16.
66. Herbes, Stephen E.; Risi, George F.. Metabolic alteration and excretion of anthracene by Daphnia pulex.

Bull. Environ. Contam. Toxicol. (1978), 19(2), 147-55.

67. Holst, L.L. & Giesy, J.P. Chronic effects of the photoenhanced toxicity of anthracene on daphnia magna reproduction. Environ. Toxicol. Chem. (1989), 8, 933-944
68. Howard P.H., Boethling R.S., Jarvis W.F., Meylan W.M. and Michalenko E.M. (1991) -Handbook of environmental degradation rates. Chealsea, Michigan, Lewis Publisher, p 725,Printup HT Ed
69. Huang X D; Dixon D G; Greenberg B M, Increased polycyclic aromatic hydrocarbon toxicity following their photomodification in natural sunlight: impacts on the duckweed Lemna gibba L. G-3 ECOTOXICOLOGY AND ENVIRONMENTAL SAFETY (1995 Nov), 32(2), 194-200.
70. Huang, Jian; Liu, Xianagdong; Tang, Xuexi. Study of the anthracene and benzo[a]pyrene toxicity effect on marine microalgae. Qingdao Haiyang Daxue Xuebao (2000), 30(3), 499-502
71. Huang, Xiao Dong; Dixon, D. George; Greenberg, Bruce M. Impacts of UV radiation and photomodification on the toxicity of PAHs to the higher plant Lemna gibba (duckweed), Environ. Toxicol. Chem. (1993), 12(6), 1067-77.
72. Huang, Xiao-Dong; Babu, Tripuranthakam S.; Marwood, Christopher A.; Gensemer, Robert W.; Solomon, Keith R.; Greenberg, Bruce M. Inhibition of photosynthesis as an endpoint for the photoinduced toxicity of intact and photomodified PAHs. ASTM Special Technical Publication (1997), STP 1317(Environmental Toxicology and Risk Assessment: Modeling and Risk Assessment, (Sixth Volume)), 443-455.
73. Huang, Xiao-Dong; Dixon, D. George; Greenberg, Bruce M., Increased polycyclic aromatic hydrocarbon toxicity following their photomodification in natural sunlight: impacts on the duckweed Lemna gibba L. G-3, Ecotoxicol. Environ. Saf. (1995), 32(2), 194-200.
74. Huang, Xiao-Dong; Mcconkey, Brendan J.; Babu, T. Sudhakar; Greenberg, Bruce M. Mechanisms of photoinduced toxicity of photomodified anthracene to plants: inhibition of photosynthesis in the aquatic higher plant Lemna gibba (duckweed). Environmental Toxicology and Chemistry (1997), 16(8), 1707-1715.
75. Huang, Xiao-Dong; Krylov, Sergey N.; Ren, Lisha; Mcconkey, Brendan J.; Dixon, D. George; Greenberg, Bruce M., Mechanistic quantitative structure-activity relationship model for the photoinduced toxicity of polycyclic aromatic hydrocarbons: II. An empirical model for the toxicity of 16 polycyclic aromatic hydrocarbons to the duckweed Lemna gibba L. G-3. Environmental Toxicology and Chemistry (1997b), 16(11), 2296-2303.
76. Huang, Xiao-Dong; Zeiler, Lorelei F.; Dixon, D. George; Greenberg, Bruce M., Photo-induced toxicity of PAHs to the foliar regions of Brassica napus (canola) and Cucumis sativus (Cucumber) in simulated solar radiation. Ecotoxicology and Environmental Safety (1996), 35(2), 190-197.
77. Huovinen, P. S.; Soimasuo, M. R.; Oikari, A. O. J.Photoinduced toxicity of retene to Daphnia magna under enhanced UV-B radiation, Chemosphere (2001), 45(4-5), 683-691.
78. Hutchinson T.C. Environ. Sci. Res., (1980), 16, 577-586
79. Iosifidou, Helen G.; Kilikidis, Stylianos D.; Kamarianos, Athanasios P. Analysis for polycyclic aromatic hydrocarbons in mussels (Mytilus galloprovincialis) from the Thermaikos Gulf, Greece. Bull. Environ. Contam. Toxicol. (1982), 28(5), 535-41.
80. Jacobs, Mark W.; Coates, John A.; Delfino, Joseph J.; Bitton, Gabriel; Davis, William M.; Garcia, K. Lizanne. Comparison of sediment extract Microtox toxicity with semi-volatile organic priority pollutant concentrations. Arch. Environ. Contam. Toxicol. (1993), 24(4), 461-8.
81. Japan chemical industry. Biodegradation and bioaccumulation data of existing chemicals based on the CSCL Japan, compiled under the supervision of chemical Produts Safety division, Basic Industries Bureau MITI,ed. By CITI, October 1992. Published by Japan Chemical Industry Ecology and Information Center.
82. Joshi P.L. and Misra R.B. Biochem. Biophys. Res. Comm. (1986), 139,79-84.
83. Kaestner, M.; Lotter, S.; Heerenklage, J.; Breuer-Jammali, M.; Stegmann, R.; Mahro, B. Fate of 14C-labeled anthracene and hexadecane in compost-manured soil. Appl. Microbiol. Biotechnol. (1995), 43(6), 1128-35.
84. Kagan J., Kagan P.A. and Buhse H. E. Light-dependent toxicity of alpha-terthienyl and anthracene toward late embryonic stages of Rana pipiens. Jr., J Chem Ecol, (1984), 10 (7), 1115-1122.
85. Kagan, Jacques; Kagan, Edgard D.; Kagan, Isabelle A.; Kagan, Peggy A.; Quigley, Susan. The phototoxicity of noncarcinogenic polycyclic aromatic hydrocarbons in aquatic organisms.. Chemosphere (1985), 14(11-12), 1829-34.
86. Kagi, R.; Alexander, R.; Cumbers, M. Polycyclic aromatic hydrocarbons in rock oysters: a baseline study. Int. J. Environ. Anal. Chem. (1985), 22(1-2), 135-53.
87. M. Kästner, S. Streibich, M. Beyrer, H. H. Richnow, and W. Fritsche, Formation of Bound Residues during Microbial Degradation of [^{14}C]Anthracene in Soil, Applied and Environmental Microbiology, May 1999, p. 1834-1842, Vol.65, No.5 0099-224.
88. Kim, Mi-Sun; Huh, Eun-Jee; Kim, Hyun-Kyung; Moon, Kwang-Woong. Degradation of polycyclic aromatic hydrocarbons by selected white-rot fungi and the influence of lignin peroxidase. Journal of

Microbiology and Biotechnology (1998), 8(2), 129-133.
89. Klimisch H. J., M. Andreae and U. Tillmann. A Systematic Approach for Evaluating the Quality of Experimental Toxicological and Ecotoxicological Data. Regulatory Toxicology and Pharmacology, (1997) 25, 1, 1-5
90. Landrum, P.F. and D. Scavia. 1983. Influence of Sediment on Uptake, Depuration, and Biotransformation by the Amphipod *Hyalella azteca*. Can. J. Fish. Aquat. Sci. 40: 298-305.
91. Landrum, P. F., W. R. Faust and B. J. Eadie. 1989. Bioavailability and toxicity of a mixture of sediment associated chlorinated hydrocarbons to the amphipod, Pontoporeia hoyi. Aquatic Toxicology and Hazard Assessment: 12th Volume, ASTM STP 1027. U. M. Cowgill and L. R. Williams, Eds., American Society for Testing and Materials, Philadelphia, PA pp. 315-329..
92. Landrum P F; Bartell S M; Giesy J P; Leversee G J; Bowling J W; Haddock J; LaGory K; Gerould S; Bruno M Fate of anthracene in an artificial stream: a case study. ECOTOXICOLOGY AND ENVIRONMENTAL SAFETY (1984 Apr), 8(2), 183-201.
93. Landrum, Peter F.. Uptake, depuration and biotransformation of anthracene by the scud Pontoporeia hoyi. Chemosphere (1982), 11(10), 1049-57.
94. Laughrey, Z. R.; Bear, E.; Jones, R.; Tarr, M. A. Sonochemical degradation of PAHs: mechanisms and influence of dissolved organic compounds. Department of Chemistry, Preprints of Extended Abstracts presented at the ACS National Meeting, American Chemical Society, Division of Environmental Chemistry (2000), 40(2), 66-67.
95. Leduc, R.; Samson, R.; Al-Bashir, B.; Al-Hawari, J.; Cseh, T. Biotic and abiotic disappearance of four polycyclic aromatic hydrocarbon (PAH) compounds from flooded soil under various redox conditions. Water Sci. Technol. (1992), 26(1-2, Water Qual. Int. '92, Pt. 1), 51-60.
96. Lee, Byung-Dae; Hosomi, Masaaki. Ethanol washing of PAH-contaminated soil and Fenton oxidation of washing solution. Journal of Material Cycles and Waste Management (2000), 2(1), 24-30.
97. Lee, Byung-Dae; Hosomi, Masaaki; Murakami, Akihiko. Fenton oxidation with ethanol to degrade anthracene into biodegradable 9,10-anthraquinone. A pretreatment method for anthracene-contaminated soil. Water Science and Technology (1998), 38(7), 91-97.
98. Lee,Cindy M.; Polynuclear aromatic hydrocarbons: Properties aand environmental fate. Environmental Engineering & Science, Clemson University, 1998.
99. Lee, Jong-Hyeon, Peter F. Landrum and Chul-Hwan Koh. Toxicokinetics and Time Dependent PAH Toxicity in the Amphipod *Hyalella azteca*. Environmental Science & Technology, 2002. Volume 36, No. 14, pp. 3124-3130
100. Lepper, P. (2002) Towards the Derivation of Quality Standards for Priority Substances in the Context of the Water Framework Directive. Identification of quality standards for priority substances in the field of water policy. Fraunhofer-Institute Molecular Biology and Applied Ecology. Final Report of the Study Contract No. B4-3040/2000/30637/MAR/E1
101. Leversee, G.J., et al, Effects of humic on polycyclic aromatic hydrocarbons: Accumulation by Daphnia magna and co-precipitation at estuarine salinities. Can. J. Fish. Aqua. Sci., Ocean Poll. Symp. (1982)
102. Leyval, C.; Binet, P. Effect of Polyaromatic hydrocarbons in soil on arbuscular mycorrhizal plants. Journal of Environmental Quality (1998), 27(2), 402-407.
103. Linder, Greg; Bergman, Harold L.; Meyer, Joseph S. Anthracene bioconcentration in rainbow trout during single-compound and complex-mixture exposures. Environ. Toxicol. Chem. (1985), 4(4), 549-58.
104. Lisowska, Katarzyna; Persson, Ingrid; Rumijowska, Anna; Parniewski, Pawel; Sedlaczek, Leon; Hahn-Haegerdal, Baerbel. The degradation of anthracene by a Pseudomonas sp. and Pseudomonas mendocina at low temperature. Int. Symp. Environ. Biotechnol., Three-Day Symp., 2nd (1994), 155-7.
105. Liu, D.; Maguire, R. J.; Pacepavicius, G. J.; Nagy, E. Microbial degradation of polycyclic aromatic hydrocarbons and polycyclic aromatic nitrogen heterocyclics. Environ. Toxicol. Water Qual. (1992), 7(4), 355-72.
106. Lors, Christine; Mossmann, Jean-Remi; Barbe, Pascal; Laboudigue, Agnes. Investigation of PAHs intrinsic degradation in soils of former coke facility sites. Editor(s): Leeson, Andrea. International In Situ and On-Site Bioremediation Symposium, 6th, San Diego, CA, United States, June 4-7, 2001 (2001), 2 129-136.
107. Lyman, Warren J.; Reehl, William F.; Rosenblatt, David H. Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds. (1990), unpaged.
108. Mackay D., Shiu W. and Ma K.. (1992). Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals. Vol. II : polynuclear aromatic hydrocarbons, polychlorinated dioxins and dibenzofurans. Lewis Publishers, Chelsea, Mi. pp. 597.
109. Mallakin. A. et al (1999) Impacts of Structural Photomodification on the Toxicity of Environmental Contaminants: Anthracene Photooxidation Products. Ecotoxicology and Environmental Safety, 43, 204-212.
110. Malaney G.W. and Mc Kinney R.E., Water Sew Works, (1966), 113, 302-309.
111. Maliszewska-Kordybach, Barbara. The relationship between the properties of PAHs and the rate of their

disappearance from soils. Toxicological and Environmental Chemistry (1998), 66(1-4), 47-52.
112. Mallakin, Ali; McConkey, Brendan J.; Miao, Goubin; McKibben, Brian; Snieckus, Victor; Dixon, D. George; Greenberg, Bruce M., Impacts of structural photomodification on the toxicity of environmental contaminants: anthracene photooxidation products. Ecotoxicology and Environmental Safety (1999), 43(2), 204-212.
113. Marsili, L.; Fossi, M. C.; Casini, S.; Savelli, C.; Jimenez, B.; Junin, M.; Castello, H. Fingerprint of polycyclic aromatic hydrocarbons in two populations of southern sea lions (Otaria flavescens). Chemosphere (1997), 34(4), 759-770.
114. McCarthy John F., Braulio D. Jimenez and Thelberstine Barbee. Effect of dissolved humic material on accumulation of polycyclic aromatic hydrocarbons: Structure-activity relationships. *Aquatic Toxicology*, *7, 1-2*, *October 1985*, *15-24*
115. McCloskey, John T.; Oris, James T. Effect of water temperature and dissolved oxygen concentration on the photo-induced toxicity of anthracene to juvenile bluegill sunfish (Lepomis macrochirus). Aquat. Toxicol. (1991), 21(3-4), 145-56.
116. McCloskey, John T.; Oris, James T., Effect of anthracene and solar ultraviolet radiation exposure on gill ATPase and selected hematologic measurements in the bluegill sunfish (Lepomis macrochirus). Aquat. Toxicol. (1993), 24(3-4), 207-17.
117. McFall, Jo Ann; Antoine, Shelley R.; DeLeon, Ildefonso R. Base-neutral extractable organic pollutants in biota and sediments from Lake Pontchartrain. Chemosphere (1985), 14(10), 1561-9.
118. McKinney, Richard A.; Pruell, Richard J.; Burgess, Robert M. Ratio of the concentration of anthraquinone to anthracene in coastal marine sediments. Chemosphere (1999), 38(10), 2415-2430.
119. McNally, Dan L.; Mihelcic, James R.; Lueking, Donald R. Biodegradation of Three- and Four-Ring Polycyclic Aromatic Hydrocarbons under Aerobic and Denitrifying Conditions. Environmental Science and Technology (1998), 32(17), 2633-2639.
120. Mekenyan OG., Ankley G.T., Veith G.D. and Call D.J. QSARs for photoinduced toxicity: I. Acute lethality of polycyclic aromatic hydrocarbons to Daphnia magna. Chemosphere (1994), 28 (3), 567-582.
121. Mellon Institute (1977) Range Finding Toxicity Tests on Rabbits and Rats, EPA Doc. No. 86-870001568, Fiche No. OTS0516149
122. Meulenberg, Rogier; Draaisma, Rene B.; de Ridder, Edwin Moison Erik J.; Rijnaarts, Hubb H. M.; Field, Jim A. Sequential anthracene degradation by Bjerkandera BOS55 and bacteria. In Situ and On-Site Bioremediation, Papers from the International In Situ and On-Site Bioremediation Symposium, 4th, New Orleans, Apr. 28-May 1, 1997 (1997), 2 487-492.
123. Mitchell R.L., Burchett M.D., Pulkownik A. and McCluskey L. Effects on environmentally hazardous chemicals on the emergence and early growth of selected Australian plants. Plant Soil (1988), 112(2), 195-200
124. Moore, M. N.; Lowe, D. M.; Fieth, P. E. M., Lysosomal responses to experimentally injected anthracene in the digestive cells of Mytilus edulis. Mar. Biol. (Berlin) (1978), 48(4), 297-302.
125. Mueller, James G.; Middaugh, Douglas P.; Lantz, Suzanne E.; Chapman, Peter J. Biodegradation of creosote and pentachlorophenol in contaminated groundwater: chemical and biological assessment. Appl. Environ. Microbiol. (1991), 57(5), 1277-85.
126. Mulkins-Phillips, G. J.; Stewart, James E. Effect of environmental parameters on bacterial degradation of Bunker C oil, crude oils, and hydrocarbons. Appl. Microbiol. (1974), 28(6), 915-22.
127. Munoz, M. J.; Tarazona, J. V. Synergistic effect of two- and four-component combinations of the polycyclic aromatic hydrocarbons: phenanthrene, anthracene, naphthalene and acenaphthene on Daphnia magna. Bull. Environ. Contam. Toxicol. (1993), 50(3), 363-8.
128. Naes, Kristoffer; Knutzen, Jon; Berglind, Lasse. Occurrence of PAH in marine organisms and sediments from smelter discharge in Norway. Sci. Total Environ. (1995), 163(1-3), 93-106.
129. Neman L.M. & Johnson E.M., Phototoxicity prescreening with hydra: pleliminary feasibility studies. Toxicologist (1991), 11(1), 281
130. Niimi, A. J.; Palazzo, V.. Biological half-lives of eight polycyclic aromatic hydrocarbons (PAHs) in rainbow trout (Salmo gairdneri). Water Res. (1986), 20(4), 503-7
131. Nishihara, T.; Hasebe, S.; Nishikawa, J.; Kondo, M. Biodegradation of aniline, anthracene, chlornitrophen, fenitrothion and linear alkylbenzene sulfonate in pond water. Journal of Applied Microbiology (1997), 82(4), 441-447.
132. Ogata, Masana; Fujisawa, Kuniyasu; Ogino, Yasuo; Mano, Emiko. Partition coefficients as a measure of bioconcentration potential of crude oil compounds in fish and shellfish. Bull. Environ. Contam. Toxicol. (1984), 33(5), 561-7.
133. Oris, J. T.; Giesy, J. P.; Allred, P. M.; Grant, D. F.; Landrum, P. F., Photoinduced toxicity of anthracene in aquatic organisms: an environmental perspective. Stud. Environ. Sci. (1984), 25(Biosphere: Probl. Solutions), 639-58.
134. Oris, James T.; Giesy, John P., Jr., Photoinduced toxicity of anthracene to juvenile bluegill sunfish

(Lepomis macrochirus Rafinesque): photoperiod effects and predictive hazard evaluation. Environ. Toxicol. Chem. (1986), 5(8), 761-8.
135. Oris, James T.; Giesy, John P., Jr., The photo-induced toxicity of polycyclic aromatic hydrocarbons to larvae of the fathead minnow (Pimephales promelas). Chemosphere (1987), 16(7), 1395-404.
136. Oris, James T.; Giesy, John P., Jr.. The photoenhanced toxicity of anthracene to juvenile sunfish (Lepomis spp.). Aquat. Toxicol. (1985), 6(2), 133-46.
137. Park, Kap S.; Sims, Ronald C.; Dupont, R. Ryan; Doucette, William J.; Matthews, John E.Fate of PAH compounds in two soil types: influence of volatilization, abiotic loss and biological activity. Environ. Toxicol. Chem. (1990), 9(2), 187-95.
138. Pelletier Marguerite C., Robert M. Burgess, Kay T. Ho, Anne Kuhn, Richard A. McKinney and Stephan A. Ryba., Phototoxicity of individual polycyclic aromatic hydrocarbons and petroleum to marine invertebrates larvae and juvenils. Environ. Toxicol. And Chemistry, (1997), 16, 10, 2190-2199.
139. Pereira, Wilfred E.; Hostettler, Frances D.; Rapp, John B. Bioaccumulation of hydrocarbons derived from terrestrial and anthropogenic sources in the asian clam, Potamocorbula amurensis, in San Francisco Bay estuary. Mar. Pollut. Bull. (1992), 24(2), 103-9.
140. Qiang, Z.; Chang, J. H.; Huang, C. P.; Cha, D. Oxidation of selected polycyclic aromatic hydrocarbons by the Fenton's reagent: effect of major factors including organic solvent. ACS Symposium Series (2001), 778(Nuclear Site Remediation), 187-209.
141. Raghukumar, C.; Vipparty, V.; David, J. J.; Chandramohan, D. Degradation of crude oil by marine cyanobacteria. Applied Microbiology and Biotechnology (2001), 57(3), 433-436.
142. Rainio, Kristiina; Linko, Reino R.; Ruotsila, Leena. Polycyclic aromatic hydrocarbons in mussel and fish from the Finnish Archipelago Sea. Bull. Environ. Contam. Toxicol. (1986), 37(3), 337-43.
143. Rao, Venkateswara R.; Mitz, Stephen V.; Hadden, Charles T.; Cornaby, Barney W. Distribution of contaminants in aquatic organisms from East Fork Poplar Creek. Ecotoxicol. Environ. Saf. (1996), 33(1), 44-54.
144. Reilley, K. A.; Banks, M. K.; Schwab, A. P. Dissipation of polycyclic aromatic hydrocarbons in the rhizosphere. J. Environ. Qual. (1996), 25(2), 212-19.
145. Ren, Lisha; Zeiler, Lorelei F.; Dixon, D. George; Greenberg, Bruce M., Photoinduced effects of polycyclic aromatic hydrocarbons on Brassica napus (Canola) during germination and early seedling development. Ecotoxicol. Environ. Saf. (1996), 33(1), 73-80.
146. Reutgerswerke AG Unpublished data. (1991)
147. Roberts M.H., Hargis W.J.Jr., Strobel C.J. and De Lisle P.F., Acute toxicity of PAH contaminated sediments to the estuarine fish, Leiostomus xanthurus. Bull. Environ. Contam. Toxicol. (1989), 42, 142-149.
148. Rockne, Karl J.; Stensel, H. David; Herwig, Russell P.; Strand, Stuart E. PAH degradation and bioaugmentation by a marine methanotrophic enrichment. Bioremediation Journal (1998), 1(3), 209-222.
149. Rostad, Colleen E.; Pereira, Wilfred E.. Creosote compounds in snails obtained from Pensacola Bay, Florida, near an onshore hazardous-waste site. Chemosphere (1987), 16(10-12), 2397-404.
150. Roubal, William T.; Collier, Tracy K.; Malins, Donald C. Accumulation and metabolism of carbon-14 labeled benzene, naphthalene, and anthracene by young coho salmon (Oncorhynchus kisutch). Arch. Environ. Contam. Toxicol. (1977), 5(4), 513-29.
151. Sanders, M.. Distribution of polycyclic aromatic hydrocarbons in oyster (Crassostrea virginica) and surface sediment from to estuaries in South Carolina. Arch. Environ. Contam. Toxicol. (1995), 28(4), 397-405.
152. Sanseverino, John; Applegate, Bruce M.; King, J. M. Henry; Sayler, Gary S. Plasmid-mediated mineralization of naphthalene, phenanthrene, and anthracene. Appl. Environ. Microbiol. (1993), 59(6), 1931-7.
153. Sasaki K., (1978) In: Aquatic pollutants: Transformation and biological effects (Hutzinger O. et al., eds.), Oxford, Pergamon Press.
154. Saxe, Jennifer K.; Allen, Herbert E.; Nicol, Gordon R. Fenton oxidation of polycyclic aromatic hydrocarbons after surfactant-enhanced soil washing. Environmental Engineering Science (2000), 17(4), 233-244.
155. Schafer Jr. E.W. et al. Arch. Environ. Contam. Toxicol. (1983) 12, 335-382.
156. Schutt, Wendy S.; Feng, Kesheng; Bousman, Kenneth S.; Gao, Ning; Li, Yuzhuo. Photoinduced degradation of environmental pollutants in the presence of O2-free ozone. Journal of Advanced Oxidation Technologies (1997), 2(3), 401-407.
157. Schwall, L. R.; Herbes, S. E. Methodology for the determination of rates of microbial transformation of polycyclic aromatic hydrocarbons in sediments. ASTM Spec. Tech. Publ. (1979), (STP 673, Methodol. Biomass Determinations Microb. Act. Sediments), 167-83.
158. Schwartz F.P. (1977) Determination of temperature dependence of solubilities of polycyclic aromatic hydrocarbons in aqueous solutions by a fluorescence method. J. Chem. Eng. Data 22, 273-277.
159. Sigman, Michael E.; Zingg, S. P.; Pagni, Richard M.; Burns, John H.Photochemistry of anthracene in

water. Tetrahedron Lett. (1991), 32(41), 5737-40.
160. Sims, R. C.; Overcash, M. R. Fate of polynuclear aromatic compounds (PNAs) in soil-plant systems. Residue Rev. (1983), 88 1-68.
161. Slooff W, Janus JA, Matthijsen AJCM, Montizaan GK and Ros JPM (1989). Integrated Criteria Document PAHs. National Institute for Public Health and the Environment, RIVM, Bilthoven, the Netherlands. RIVM report nr. 758474011.SRC (2004). Interactive PhysProp Database at URL: Demohttp://www.syrres.com/esc/physdemo.htm. Syracuse Research Corporation (SRC).
162. Smith S.B. et al. Great Lakes Res., (1988), 14, 394-404
163. Smith, John R.; Neuhauser, Edward F.; Middleton, Andrew C.; Cunningham, James J.; Weightman, Robin L.; Linz, David G. Treatment of organically contaminated groundwaters in municipal activated sludge systems. Water Environ. Res. (1993), 65(7), 804-18.
164. Sonnefeld W.J., Zoller W.H. and May W.E. (1983) Dynamic coupled-column liquid chromatographic determination of ambient temperature vapor pressures of polynuclear aromatic hydrocarbons. Anal. Chem. 55, 275-280.
165. Southworth, G. R. 1979. Transport and transformation of anthracene in natural waters. *In* : L.L. Marking and R.A. Kimerle (eds.) Aquatic Toxicology. ASTM STP 667. Amer. Soc. Test. Mat., Philadelphia, Pa. p. 359-380
166. Southworth, G. R.; Beauchamp, J. J.; Schmieder, P. K. Bioaccumulation potential of polycyclic aromatic hydrocarbons in Daphnia pulex. Water Res. (1978), 12(11), 973-7.
167. Southworth, George R.. The role of volatilization in removing polycyclic aromatic hydrocarbons from aquatic environments. Bull. Environ. Contam. Toxicol. (1979), 21(4-5), 507-14.
168. Spacie, Anne; Landrum, Peter F.; Leversee, Gordon J. Uptake, depuration, and biotransformation of anthracene and benzo[a]pyrene in bluegill sunfish. Ecotoxicol. Environ. Saf. (1983), 7(3), 330-41.
169. Srivastava, V. J.. The effect of moderate coal cleaning on microbial removal of organic sulfur: technical report, September 1, 1990 - November 30, 1990. Report (1990), (DOE/PC/90176-T3; Order No. DE91010502), 11 pp.
170. Sverdrup, Line E.; Nielsen, Torben; Krogh, Paul Henning. Soil Ecotoxicity of Polycyclic Aromatic Hydrocarbons in Relation to Soil Sorption, Lipophilicity, and Water Solubility. Environmental Science and Technology (2002), 36(11), 2429-2435.
171. Tabak, Henry H.; Quave, Stephen A.; Mashni, Charles I.; Barth, Edwin F. Biodegradability studies with organic priority pollutant compounds. J. - Water Pollut. Control Fed. (1981), 53(10), 1503-18.
172. Tang, Jixin; Alexander, Martin. Mild extractability and bioavailability of polycyclic aromatic hydrocarbons in soil. Environmental Toxicology and Chemistry (1999), 18(12), 2711-2714.
173. Tang, Jixin; Carroquino, M. J.; Robertson, B. K.; Alexander, Martin. Combined Effect of Sequestration and Bioremediation in Reducing the Bioavailability of Polycyclic Aromatic Hydrocarbons in Soil. Environmental Science and Technology (1998), 32(22), 3586-3590.
174. Thomas, Robert E.; Rice, Stanley D.. Metabolism and clearance of phenolic and mono-, di-, and polynuclear aromatic hydrocarbons by Dolly Varden char. Physiol. Mech. Mar. Pollut. Toxic., [Proc. Symp. Pollut. Mar. Org.] (1982), Meeting Date 1981, 161-76.
175. Trapido, Marina; Veressinina, Yelena; Advanced oxidation processes and ozone treatment of anthracene in aqueous solutions. Eesti Tead. Akad. Toim., Keem. (1994), 43(2), 61-7.
176. USEPA, Health and environmental effects profile of anthracene, (1987), p.iv ECAO-CIN p230.
177. Uthe, John F.; Musial, Charles J. Intercomparative study on the determination of polynuclear aromatic hydrocarbons in marine shellfish tissue. J. - Assoc. Off. Anal. Chem. (1988), 71(2), 363-8.
178. Verschueren K., Handbook of environmental data on organin chemicals. 3rd ed. New York, NY, Van Nostrand Reinhold Co. (1996),
179. Walton, Barbara T. Influence of route of entry on toxicity of polycyclic aromatic hydrocarbons to the cricket (Acheta domesticus). Bull. Environ. Contam. Toxicol. (1980), 25(2), 289-93.
180. Weinstein, John E.; Polk, Kevin D., Phototoxicity of anthracene and pyrene to glochidia of the freshwater mussel Utterbackia imbecillis. Environmental Toxicology and Chemistry (2001), 20(9), 2021-2028.
181. Wild, SR, Berrow, ML, and Jones, KC (1991) The persistence of polynuclear aromatic hydrocarbons (PAHs) in sewage sludge amended agricultural soils. Environ. Pollut., 72, 141-157
182. Wild, SR, and Jones, KC (1995) Polynuclear aromatic hydrocarbons in the United Kingdom environment: a preliminary source inventory and budget. Environ. Pollut., 88(1), 91-108
183. Willumsen, B.; Gray, M. R.; Dudas, M. J. Biological degradation of anthracene in soil after sorption from non-aqueous phase liquids. Environmental Technology (1997), 18(7), 755-762.
184. Yan, Xue; Yan, Guo-an; Sheng, Guo-xing; Peng, Jing-liang., Toxicological response of Chlorella to anthracene under different nutrient conditions. Wuhan Daxue Xuebao, Ziran Kexueban (1999), 45(6), 845-848.
185. Zepp R.G. & Schlotzhauer P.F. Photoreactivity of selected aromatic hydrocarbons in water, in Jones P.W. and Leber (Eds.), Polynuclear aromatic hydrocarbons, Ann Arbor Science Publishers. Ann Arbor, MI,

(1979)

186. Zingg, S. P.; Sigman, Michael E. Influence of an silicon dioxide/cyclohexane interface on the photochemistry of anthracene. Photochem. Photobiol. (1993), 57(3), 453-9.

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
bw	body weight / *Bw*, *b.w.*
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
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
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)
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
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

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)

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Environment and quality of life series

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The report provides the comprehensive risk assessment of the substance anthracene. It has been prepared by Greece 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 man 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.

The substance is considered to be a PBT and vPvB substance. Therefore a strategy to reduce the emissions into the environment will have to be considered under REACH. There is no concern identified for the aquatic, terrestrial and atmospheric from the production of anthracene and the formulation into pyrotechnics based on the PEC/PNEC approach.

European Chemicals Bureau

European Union Risk Assessment Report
anthracene

CAS: 120-12-7
EC: 204-371-1

PL-3
78

Institute for Health and Consumer Protection

European Chemicals Bureau

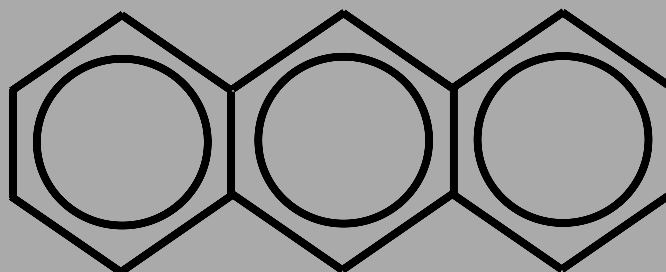
Existing Substances

European Union Risk Assessment Report

CAS No: 120-12-7

EINECS No: 204-371-1

anthracene
part II – human health



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The mission of the IHCP is to provide scientific support to the development and implementation of EU polices related to health and consumer protection. The IHCP carries out research to improve the understanding of potential health risks posed by chemical, physical and biological agents from various sources to which consumers are exposed.

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European Union Risk Assessment Report

ANTHRACENE

Part II – Human Health

CAS No: 120-12-7

EINECS No: 204-371-1

RISK ASSESSMENT

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ANTHRACENE

Part II – Human Health

CAS No: 120-12-7

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RISK ASSESSMENT

Final Report, 2007

Greece

The scientific assessments in this Report have been prepared by the National Hellenic Research Foundation (Unit of Environmental Toxicology), under contract with the Rapporteur.

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Date of Last Literature Search: 2003
Review of report by MS Technical Experts finalised: 2005
Final report: 2007

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 Health and Environmental Risks (SCHER) 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 and confirmed in the Johannesburg Declaration on Sustainable Development at the World Summit on Sustainable Development, held in Johannesburg, South Africa in 2002.

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.

Roland Schenkel	**Mogens Peter Carl**
Director General	Director General
DG Joint Research Centre	DG Environment

[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:	120-12-7
EINECS No:	204-371-1
IUPAC Name:	anthracene
Synonyms:	Paranaphthalene, p-naphthalene

This Risk Assessment Report assesses the risks to human health associated with the production and use of the isolated commercial product anthracene. Anthracene is also found as part of complex mixtures in coal tar and products derived there from, as well as in the products of incomplete combustion of organic matter. According to Council Regulation 793/93 non-isolated anthracene is outside the scope of the present Report. However, estimates of the exposures and risks associated with such mixtures are included for illustrative purposes.

Environment

(to be added later)

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.

This conclusion applies to the induction of dermal phototoxicity as a result of occupational dermal exposure during the production of anthracene from anthracene oil, the packaging of anthracene, and the manufacture of pyrotechnics.

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

This conclusion applies to

- measured exposure data for workers involved in anthracene production from anthracene oil, anthracene packaging and anthracene use in the manufacture of pyrotechnics;
- testing to determine the reproductive and developmental toxicity of anthracene.

This substance has not been fully tested for reproductive toxicity and consequently this risk assessment does not evaluate the risks to any human populations for this endpoint. The need for a developmental toxicity study to fill this data gap has been identified following OECD 414 (Prenatal developmental toxicity study). However, this risk assessment describes the situation in the EU in 2003, in which there is only one production site in the EU, with 99% of production volume exported outside the EU and only a very minor use in pyrotechnics. There are no consumer exposures to the commercially-produced substance and human environmental exposures are very low. The potential for worker exposure using modelled estimates is low, and limited measured data and control measures, known to be applied at the production site, indicate that the model predictions are probably over-estimates.

On this basis, and taking into account that a) the developmental toxicity of PAHs is at least partly dependent on binding to the Ah receptor, and b) anthracene does not show such binding to any significant extent, the Technical Meeting agreed that there may be grounds on exposure

considerations to waive the requirement on the producer to generate such a study, as long as the exposure situation did not change. Further measured exposure data for workers involved in anthracene production from anthracene oil, anthracene packaging and anthracene use in the manufacture of pyrotechnics, would of course increase the level of confidence in this decision. The situation should be closely monitored and if there are any indications that production and use patterns are changing the potential for increasing exposure should be reconsidered and the need to request a developmental toxicity study revisited.

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 the induction of systemic toxicity as a result of exposure during the production of anthracene from anthracene oil, the packaging of anthracene, and the manufacture of pyrotechnics.

Consumers

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.

Humans exposed via the environment

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.

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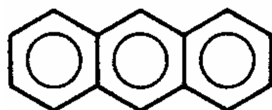
1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS No: 120-12-7
EINECS No: 204-371-1
IUPAC Name: Anthracene
Common Name: Anthracene
Synonyms: Paranaphthalene, p-naphthalene
Molecular formula: $C_{14}H_{10}$
Molecular mass: 178.24
Structural formula:



1.2 PURITY/IMPURITIES, ADDITIVES

1.2.1 Purity/impurities

Technical grade anthracene is approximately 97% pure, the main impurities being the following: phenanthrene (1.0%); carbazole (1.0%); naphthothiophene (0.4%); dibenzo[b,c]thiophene (0.3%); acridine (0.2%); acetophenone (0.4%).

Higher-grade anthracene can be obtained by treatment with air to oxidise 9,10-dihydroanthracene to anthracene, and by further recrystallisation, leading to a product with a low nitrogen (carbazole) content. Anthracene can generally be further separated from the higher boiling carbazole (b.p. 354°) by further distillation with a lower-boiling hydrocarbon fraction as reflux medium, or by azeotropic distillation with ethylene glycol. Azeotropic distillation is also used to separate the anthracene-accompanying tetracene (naphthacene) and to obtain very pure anthracene which is used for scintillation counting.

1.3 PHYSICO-CHEMICAL PROPERTIES

1.3.1 Physical state at STP

Anthracene is a colourless crystalline solid, with violet fluorescence.

1.3.2 Melting point

A value of 218°C is reported in IUCLID (Lang and Eigen (1967), in Merck Index (1983), in Ulmann's Encyclopaedia (Collin and Höke, 1985) and in Clar (1964). A closely similar value (217°C) is reported in ECDIN as well as in Sax (1975). IUCLID also reports lower values of 214°C (Hausigk and Koelling, 1968) and 216.4^{o}C (Karcher et al., 1985), while an additional value of 216.2^{o}C is reported in ECDIN. The value of 218°C is adopted in the present Report as the highest value which represents the highest purity material.

1.3.3 Boiling point

Two values (340°C and 342°C) are reported in IUCLID and in several other publications (e.g. 340°C in Lang and Eigen, 1967; 342°C, Lax and Synowietz, 1964). The value of 340°C is reported in CRC (1987) as "corrected", while in ECDIN the value of 339.9^{o}C is reported as the value at 760 mm Hg. Consequently the value of 340°C is adopted in the present Report.

1.3.4 Relative density

IUCLID gives values of 1.252 (25/4°C) (Collin and Höke, 1985) and 1.283 (25/4°C) (Lide, 1991). The same value is given in Lax and Synowietz (1964); while a value of 1.25 (27/4°C) is also reported in ECDIN and in Dean (1979). The value of 1.252 (25/4°C) is adopted in the present Report.

1.3.5 Vapour pressure

Three values are given in IUCLID. Sonnefeld et al. (1983) obtained a value of $8.0 \cdot 10^{-4}$ Pa (25°C) using a method closely similar to one of those recommended by Dir. 67/548/EEC. Their system was based on dynamic coupled-column liquid chromatography that is direct coupling of a gas saturation system to HPLC, used at the ambient temperature range. Commercially available anthracene was employed (purity > 98%). However, through the provisions of the system (pre-washing/conditioning of the saturation column) the measurements concerned pure anthracene.

Jandris and Forcė (1983) measured the vapour pressure of anthracene by analysing the vapour concentration using laser-induced molecular fluorescence. The material employed was 99.9% pure. Fluorescence intensity data over the range 25-150°C were obtained and employed to calculate first the vapour pressure at 101°C, which was then extrapolated to 25°C, yielding a value of $9.1 \cdot 10^{-9}$ atm ($9.2 \cdot 10^{-4}$ Pa).

A third value of 0.026 Pa, is reported in IUCLID citing a secondary reference (Neff, 1979) where no information on the method employed is given.

In the present report, the value of $8.0 \cdot 10^{-4}$ Pa (25°C) is adopted because the method employed by Sonnefeld et al. (1983) is closer to those recommended in Dir. 67/548/EEC.

1.3.6 Water solubility

Among the values reported in IUCLID, the value of 0.041 mg/l (25°C) (Schwartz, 1977) is adopted in the present Report, having been determined at 25°C by direct analysis of a saturated solution using UV and fluorescence analytical methodology. Other values reported in IUCLID are 0.032 mg/l (20°C) (Hashimoto et al., 1984), 0.037 mg/l (22°C) (May et al., 1983) and 0.044 mg/l (25.3^{o}C, but using tap water) (Whitehouse, 1984). A value of 0.073 mg/l (25°C) (Mackay et al., 1977), measured fluorimetrically after extraction of a saturated solution with cyclohexane, was not selected as it was reported in only one study, while the selected value is close to those reported by all other studies.

Values of the solubility of anthracene in saline water are also reported in IUCLID, including 0.0324 mg/l (25.3^{o}C, salinity of 36.5 o/oo; measured by dynamic column liquid

chromatography) (Whitehouse, 1984) and 0.021 mg/l (20°C, salinity of 35 o/oo; measured after extraction of a saturated solution with benzene) (Hashimoto, 1984).

1.3.6.1 Solubility in other solvents

Anthracene is reported to be slightly soluble in benzene, chloroform and carbon bisulfide and less soluble in ether and alcohol, but no quantitative information in given (Collin and Höke, 1985).

1.3.7 Partition coefficient

IUCLID (1995) gives a compilation of measured and calculated values for $logP_{ow}$ in the range of 3.45-4.8, as reported by Sangster (1989). Among the values reported, two, 4.50 and 4.54, were based on direct measurement (shake-flask), the latter in particular being measured at 25°C (Karickhoff et al., 1979). Yoshida et al. (1983) derived by calculation, using equations related to molar refraction, the values 4.21 and 4.71, and compared them to a measured value of 4.50. In another study, Geyer et al., (1984) a value of 4.54 was reported, derived from measurements carried out in accordance with the Guidelines of Directive 67/548/EEC (OECD method 107). For this reason the value of 4.54 is adopted in the present Report.

1.3.8 Flash point

A single value of 121°C (close cup) is given in IUCLID derived from Sax (1992).

1.3.9 Autoflammability

A single value of 540°C at 1,013 hPa (1 Atm.) is given in IUCLID, derived from Nabert and Schoen (1963).

1.3.10 Explosivity

Information given in IUCLID, derived from Nabert and Schön (1963), indicates a low explosion limit of 45 g/m^3 (20°C, 1 atm) or 0.6% by volume, while no high explosion limit is given. It is also indicated that dust is possibly explosive.

1.3.11 Oxidising properties

Anthracene is not an oxidising agent.

1.3.12 Summary of physicochemical properties

Table 1.1 Summary of physico-chemical properties of anthracene

Property	Value	Reference
Melting point	218°C	Lang and Eigen, 1967, Merck Index, 1983, Collin and Höke, 1985; Clar, 1964
Boiling point	340°C at 1013 hPa (1 Atm.)	Lang and Eigen, 1967; CRC, 1987
Relative density	1.252 at 25°C	Collin and Höke, 1985
Vapour pressure	8.0 · 10-4 Pa at 25°C	Sonnefeld et al., 1983
Water solubility	0.041 mg/l at 25°C	Schwarz, 1977
Partition coefficient (log Pow)	4.54 at 20°C	Karickhoff et al., 1979; Geyer et al., 1984
Flash point	121°C	Sax, 1992
Autoflammability	540°C at 1013 hPa (1 Atm.)	Nabert and Schön, 1963

1.4 CLASSIFICATION

Anthracene has not been classified in the context of Directive 67/548/EEC concerning dangerous substances.

<u>Classification proposed on the basis of the current RAR:</u>

X_i
R38 Irritating to skin
S37 Wear suitable gloves

2 GENERAL INFORMATION ON EXPOSURE

2.1 GENERAL COMMENTS ON RELEASES AND EXPOSURE

Releases of and exposure to anthracene can occur during the production and use of anthracene and anthracene-containing products. Anthracene is produced from light anthracene oil (a fraction of coal tar distillation) and its use is restricted to the industrial production of an aldehyde (which will cease operating in the EU as of 2003), the manufacture of pyrotechnics and in scientific research laboratories.

Several products containing anthracene as part of complex mixtures but not involving addition of isolated commercial anthracene, such as coal tar itself, coal tar-containing products (paints, waterproof membranes etc.) and creosote, are outside the scope of the present Report (Council Regulation 793/93). Nevertheless, all these products may affect, through anthracene releases during their production and use, the background environmental concentrations.

Background environmental concentrations of anthracene can also be affected by releases arising from incomplete combustion of organic matter, as occurring during fossil fuel combustion or in various workplaces (e.g. carbon anode/graphite, silicon carbide, aluminium, iron and steel production plants and others).

2.2 PRODUCTION

Anthracene is present in coal tar, from where it can be recovered efficiently. Hence recovery from coal tar and, in particular, from anthracene oil (one of coal tar's distillate fractions), constitutes the basis for the industrial production of anthracene. In view of the importance of coal tar distillation in anthracene production, and because the production and use of certain coal tar distillation products constitute sources of human exposure to anthracene, coal tar and its distillation products are discussed in some detail below.

2.2.1 Coal tars

Coal tars are by-products of the destructive distillation of coal, called carbonisation or coking. The composition and properties of a coal tar depend mainly on the temperature of carbonisation and, to a lesser extent, on the nature of the coal used as feedstock. Coal tars are complex mixtures of hydrocarbons, phenols and heterocyclic (oxygen-, sulphur- and nitrogen-containing) compounds. Probably as many as 10,000 compounds are actually present in coal-tars, of which over 400 have been identified. Two main classes of coal tars are recognised, depending on the temperature of carbonisation, namely high-temperature ($> 700^{o}C$) and low-temperature coal-tars ($< 700^{o}C$), which differ significantly in their chemical composition. The anthracene content of high-temperature coal tars (low-temperature ones contain negligible amounts of anthracene) has been reported by several sources as shown in **Table 2.1**.

Table 2.1 Anthracene content of different coal tar types

McNeill, 1983		Kleffer et al., 1981		Novotny et al., 1981		Marlich and Leukevitch, 1978		Collin and Höke, 1985	
Coal tar type	**Content**	**Coal tar type**	**Content**	**Coal tar type**	**Content**	**Coal tar type**	**Content**	**Coal tar type**	**Content**
coke-oven (UK)	1%	High tempe-rature	1.5%	coke-oven	5.5%	High tempe-rature	1.1%	High tempe-rature	1.5%
coke-oven (DE)	1.8%								
coke-oven (US)	0.75%								
CVR (UK)	0.26%								
low tempe-rature	0.06%								
Lurgi	0.32%								

Based on the above figures as well as figures from other sources, a value of 1.5% is adopted in the present Report as a representative level for the anthracene content of high temperature coal tars.

2.2.1.1 Coal-tar distillation products (tar oils)

Coal tar distillation is conducted at 10 distillation plants in Europe (1 each in Germany, Belgium, France, the Netherlands, Italy, Denmark, and 2 each in the U.K. and Spain) (Betts, 2000). The amounts of coal-tar produced and distilled in the EU during 1997-1999 are given in **Table 2.2** (the 1998 and 1999 production figures do not include data for Germany) (Betts, 2000).

Table 2.2 Volumes of coal-tar production and distillation in the European Union

Year	Produced (tonnes)	Distilled (tonnes)
1997	1,266,000	1,799,000
1998	1,109,000	1,810,000
1999	825,000	1,767,000

The important common distillation fractions and residues obtained from coal tar by high-temperature processes, including light anthracene oil (the starting material for anthracene production), are shown in **Table 2.3**.

Anthracene oil (distillation temperature 300-450°C) is a semisolid, greenish brown crystalline material. It is obtained in two fractions from the primary distillation of coal tars. The lower-boiling fraction (light anthracene oil) has a high content of phenanthrene, anthracene and carbazole. The higher-boiling fraction (heavy anthracene oil) has a high content of fluoranthene and pyrene (IARC, 1983a; Collin and Zauder, 1982). Light anthracene oil, which makes up

about 20% of coal tar and usually has an anthracene content of 6-7%, is used as the starting material for the production of pure anthracene (Franck and Stadelhofer, 1987; Collin and Höke, 1985). The value of 6% as the anthracene concentration in light anthracene oil is adopted in the present Report and taken forward to risk characterisation.

Table 2.3 Primary distillation fractions and residues obtained from high temperature coal tar distillation

Coal tar fraction		Dist. range	Main components
a	light oil/overheads	<180°C	Mainly toluene, xylene, benzene and indene-naphthalene
b	carbolic oil	180-205°C	Mainly higher alkylbenzenes, phenol and alkylphenols, indene, xylene and naphthalene
c	naphthalene oil	200-230°C	Mainly naphthalene and methylnaphthalene
d	creosote oil/wash oil	230-290°C	Mainly alkynaphthalenes, naphthalene, diphenylacena-phthene, fluorene, plus some higher phenols
e	light anthracene oil	260-310oC	Mainly anthracene, phenan-threne and carbazole, with small amounts of fluorene and pyrene; this fraction is used for anthracene recovery
f	heavy anthracene oil/base oil	>310°C	Mainly polynuclear aromatic compounds of higher molecular weight
g	medium-soft pitch	residue	40-50% polynuclear aromatic compounds of 4-7 rings

2.2.1.2 Creosote

While according to IARC the term “creosote oil” refers to one of the coal-tar distillation fractions (distillation temperature 230-290°C), this term is commonly used to describe the material used for timber impregnation and is made up of a blend of several coal-tar distillation fractions. It is emphasised here that creosote blending does not involve the addition of pure anthracene.

Creosote, in its best known and most commonly used form, is a mid-heavy distillate of coal tar, boiling range 200-400°C, with varying composition because of the different blending procedures employed in its production. It is generally described as a yellow-dark-green-brown oily liquid consisting of aromatic hydrocarbons (including anthracene, naphthalene and phenanthrene derivatives), some tar acids (phenol, cresols and xylenols) and tar bases (e.g. pyridine and lutidine derivatives) (IARC, 1983a; McNeill, 1983). About 160-200 compounds are present in creosote (IARC, 1983a; Nestler, 1974). However, only a limited number (about 30) have been identified, 20 of which are present at levels exceeding 1% and make up the major portion of creosote. Polycyclic aromatic hydrocarbons (mostly unsubstituted) generally account for 75-85% of creosote (Lorenz and Gjovik, 1972). The maximum benzo[a]pyrene content of WEI Grade A creosote is 500 ppm and that of WEI Grades B and C 50 ppm (WEI, 2000).

The anthracene content of some creosotes is given in **Table 2.4**. The anthracene content of “impregnation oil” is said in additional reports to vary from as low as 0.16% to as high as 7% (Lehman et al., 1984; Danish EPA, 1995; Ingram, 1982). In the EU, creosote is generally manufactured to grades specified by the West European Institute for Wood Preservation (WEI). Three creosote types are specified, based on their density, the distillation ranges and other physicochemical characteristics, without any reference to their anthracene content.

Table 2.4 Anthracene content of some creosotes

Component	Creosote type			
	A probably a mixture (classes 3, 4, 5 and 6) (Staesse, 1954)	B classes 3, 4, 5 and 6 (Staesse, 1954)	C average of 9 creosote oil samples (class 3) (Nestler, 1974, Staesse, 1954)	D typical creosote (classes 3, 4, 5 and 6) used for the impregnation of railway sleepers (Andersson et al., 1983)
anthracene	2.0	*	1.5	7.0

* The anthracene content is included in that of phenanthrene (17.4%)

Recent information gives the following picture regarding the anthracene content of creosote preparations:

a) Analyses carried out by WEI for type A and B creosotes show anthracene contents of 1.1% and 1.5%, respectively (Betts, 2000). According to the same source, other analyses gave 1.7% (AWPA P1-65) and 1.3% (P1/P13 Industry composite Test Material).

b) Analyses carried out by the Swiss independent institution EMPA indicate the following concentrations: For two creosote companies, anthracene concentrations of 0.9% and 0.8% (WEI type A), 0.55% and 0.7% (WEI type B) and 1.36% and 1.05% (WEI type C). These figures are in broad agreement with the results of analysis carried out by one of the above companies indicating contents of 0.7% (WEI type A), 0.45% (WEI type B) and 0.8% (WEI type C) (Höke, 2000).

c) The UK Health and Safety Executive have measured the anthracene content of 3 creosote products available to amateur users and found them to depend on the colour of the product as follows (HSE ECOS, unpublished):

Golden Brown	0.07%
Dark Brown	9.9%
Dark Brown	11.6%

Finally, anthracene concentrations in creosote lie at a maximum of 1.5%. A higher content would create problems with crystallisation and workability of the oil (WEI, 2000).

In the present Report the typical anthracene content of commercial creosote is assumed to be 1.5%.

2.2.2 Anthracene production from coal tar

According to information provided by the only European manufacturer of anthracene (operating in Germany), the procedure employed for its production involves, as feedstock, light anthracene oil containing about 6% anthracene (Frank and Stadlhofer, 1987). In the present Report 6% is taken as the anthracene content of light anthracene oil used for the production of anthracene. **Table 2.5** shows a typical composition of this fraction.

Table 2.5 Composition of light anthracene oil (%) (from Frank and Stadlhofer, 1987)

Component	%
Dimethylnaphthalenes	0.7
Acenaphthenes	3.1
Dibenzofuran	4.0
Fluorene	7.7
Methylfluorene	10.6
Dibenzothiophene	1.9
Anthracene	5.8
Phenanthrene	18.8
Carbazole	3.8
Methylphenanthrenes	12.3
Fluoranthenes	8.0
Pyrene	4 1
Other aromatics	19.2

Anthracene is recovered from anthracene oil by the combined application of crystallisation and distillation (vacuum distillation), while the product is further refined by recrystallisation. **Figure 2.1** shows the flow diagram for the recovery of pure anthracene from anthracene oil.

The first stage in the production of anthracene is the recovery of a concentrate (25-30% anthracene content) by crystallisation, which can be carried out in two stages to increase the yield. The final crystallisate, known as "anthracene cake" or crude anthracene, is generally concentrated to around 50% by vacuum distillation. Subsequent refining to yield anthracene of purity over 95% is normally achieved by recrystallisation. The quality of the anthracene thus obtained is of technical grade with a typical composition shown in **Table 2.6.**

Figure 2.1 Flow diagram for the recovery of anthracene from anthracene oil (from Frank and Stadlhofer, 1987)

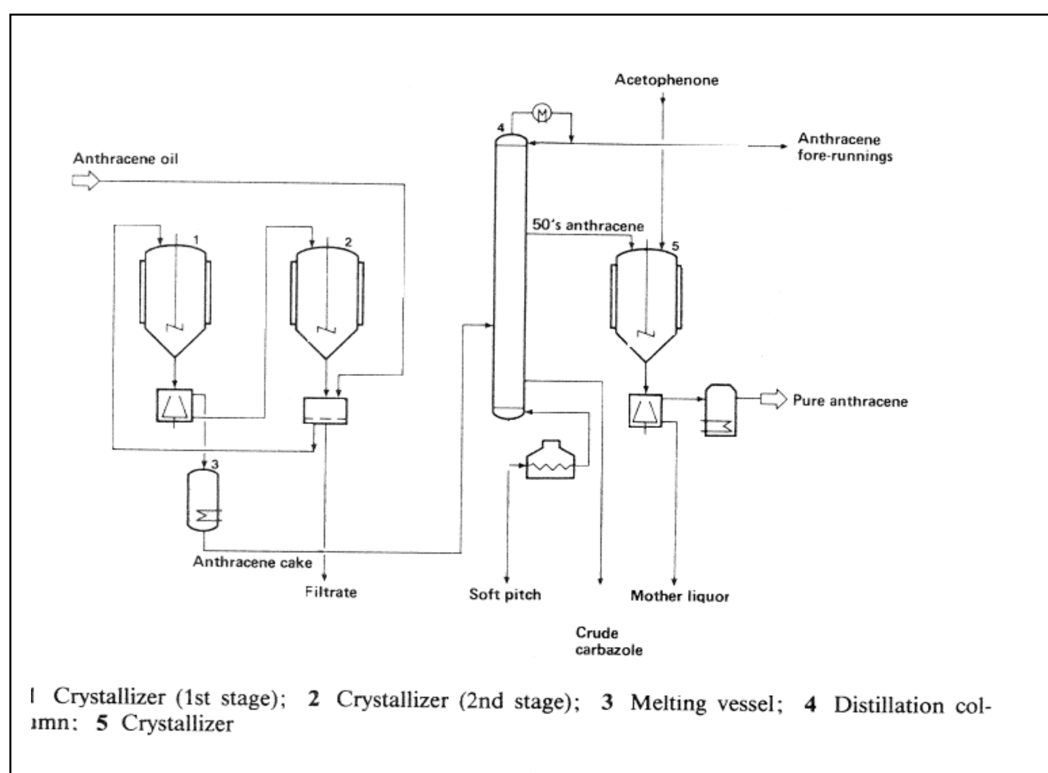


Table 2.7 shows the amounts of anthracene produced in the EU during recent years (Höke, 2000 and 2002). These data indicate that production of pure anthracene dropped to around 1,000 tpa or less during recent years. Approximately 99% of the 1999 production was exported to outside the EU. No importation of anthracene into the EU appears to take place.

Table 2.6 Typical composition of technical grade anthracene (%)

Component	%
Anthracene	97.0
Phenanthrene	1.0
Carbazole	1.0
Naphthothiophene	0.4
Dibenzo[b,c]thiophene	0.3
Acridine	0.2
Acetophenone	0.4

Table 2.7 Anthracene production volumes in Europe

Year	Production (tonnes)*					
	Pure	40% (liquid) (approx.)	50% (liquid) (approx.)	50% (solid) (approx.)	Total 50% (approx.)	Total crude (approx.)
1987	8,000					
1989	7,500					
1990	1,300					
1991	1,500	-	5,600	-		5,600
1992	3,600	40	420	280	700	740
1993	2,100	600	2,080	1,480	3,550	4,150
1994	2,100	400	2,890	2,490	5,380	5,780
1995	1,900	70	899	480	1,280	1,350
1996	1,800	-	1,030	-	1,030	1,030
1997	1,700	-	780	-	780	780
1998	1,600	-	1,400	-	1,400	1,400
1999	550	-	930	660	1,590	1,590
2000	1,190	-	600	1,330	1,930	1,930
2001	1,150	-	-	400	400	400

* The crude anthracene figures refer to amounts in addition to those of the pure product (Höke, 2000 and 2002).

2.3 USES OF ANTHRACENE AND ANTHRACENE-CONTAINING PRODUCTS

Until recently, the main uses of anthracene which could give rise to releases were two specific types of chemical synthesis. As discussed in the next section, these processes have stopped in recent years. The only known remaining use of anthracene relates to the use of small amounts of anthracene in pyrotechnics and in scientific research laboratories.

Other products containing anthracene are creosote, tar paints, waterproof membranes and related products containing coal tar distillates. These products contain anthracene as part of a complex mixture and do not involve addition of pure anthracene.

Another potential source of exposure to anthracene, which no longer exists and therefore does not need to be considered here, is related to the use of anthracene oil and coal tar in cosmetics products such as soaps, lotions, oils, shampoos and gels. The use of anthracene oil for such purposes was prohibited by Directive 76/768/EC, while more recently Directive 97/45/EC prohibited the use of coal tar in these products.

2.3.1 Uses of anthracene

According to the latest information available, practically all consumption of anthracene in the EU, which until recently was carried out by 2 main industrial users, has now stopped and almost all anthracene produced in Europe is exported. Figures on anthracene consumption during recent years are shown in **Table 2.8**.

Most of the material used by one of these users (User 1, **Table 2.8**) went to the manufacture of anthraquinone. However, production of anthraquinone at this plant has stopped since the end of 1998. User 2 used the quantities of anthracene indicated below for chemical synthesis of anthracene-9-aldehyde. According to information recently provided by this user, as of 2003 this use of anthracene will cease and anthracene-9-aldehyde will no longer be produced (Höke, 2003). Consequently neither of these processes need be further discussed in the present Report. However, in view of the use of anthracene in synthesis of anthracene-9-aldehyde until very recently, this process will be considered in the following Sections for illustrative purposes only.

Table 2.8 Anthracene consumption volumes in Europe

Year	Consumption within EU (tonnes)			Production (pure anthracene; tonnes, from Table 2.7)
	User 1	User 2	Total	
1995	1,937	n.d.	1,937	1,900
1996	1,330	n.d.	1,330	1,800
1997	1,679	19.2	1,699	1,700
1998	1,675	13.5	1,689	1,600
1999	none	6.8	6.8	550
2000	none	none	None	1,190
2001	none	none	None	1,150
2002	none	7.0	7.0	no data available

Small quantities (approximately 0.2 tonnes per year) of anthracene are sold to a company operating in the EU for the manufacture of pyrotechnics.

Small amounts of anthracene are also used in scientific research laboratories. In accordance with Council Directive 79/831, this type of use does not come under the terms of the present Report and for this reason it will not be discussed further.

In conclusion, practically no use of anthracene takes place in Europe. Consequently only the limited use of anthracene for the production of pyrotechnics will be considered in the context of this Report.

2.3.2 Uses of anthracene-containing products

2.3.2.1 Uses of creosote

Creosote is used almost exclusively in wood impregnation. Recent estimates put the amount of creosote used in the EU at approximately 107,000 tpa (Sorgo, 1996). There are 9 bulk wood impregnation plants in the EU (WEI, 2000).

The marketing and use of creosote in the EU are strictly regulated by Directive 2001/90 (adapting to technical process Annex I of Directive 76/769 concerning the restriction of the marketing and use of certain dangerous substances and preparations). This Directive does not permit the use of creosote for wood treatment. By derogation, it permits the use of special creosote (containing < 0.005% benzo[a]pyrene and < 3% water-extractable phenols) only in industrial installations or by professionals for in situ re-treatment. In addition, this kind of creosote may be place on the market only in containers of capacity ≥ 20 l, and may not be sold to consumers. Apart from other labelling restrictions, the packaging of this creosote should mention “For use in industrial installations or professional treatment only”. It is concluded from this that creosote cannot be used by consumers.

2.3.2.2 Tar paints, waterproof membranes and related products containing coal tar distillates

Coal tar and its distillates are used in some specialist paints, damp-proofing materials, waterproof membranes, coal tar epoxy paints and coal tar poly-urethane sealers. It is understood that tar paints are no longer used in Germany and that Scandinavian countries are moving away from them. Coal tar paints usually contain 0.5% anthracene, while the anthracene content of other products seems to be below 0.5% (IARC, 1983a).

No information on the number of plants or the production volumes of these products in Europe is available.

2.4 RELEASES OF ANTHRACENE DURING COMBUSTION AND RELATED INDUSTRIAL PROCESSES

Anthracene is produced during incomplete combustion of organic matter. Therefore it is emitted as a component of vehicle exhaust gases, as well as during various industrial processes such as carbon anode/graphite, silicon carbide, aluminium, iron and steel production plants and others.

2.5 SUMMARY OF INFORMATION ON RELEASES DURING PRODUCTION AND USE OF ANTHRACENE AND ANTHRACENE-CONTAINING PRODUCTS

Environmental releases of anthracene can arise mainly through the production and use of anthracene itself, during the production and use of anthracene-containing coal-tar distillates, and during combustion processes. The production of anthracene has been declining significantly during recent years in Europe, ranging around approximately 1,000 tonnes/per annum since 1999. Of this production, 99% was exported to outside the EU. On the other hand, more than 1.7 million tonnes of coal-tar, containing over 25,000 tonnes of anthracene, were distilled in Europe during 1999. Thus the production and use of coal-tar derivatives (especially creosote, 107,000 tpa used for wood treatment in Europe) represents a significantly greater potential source of environmental release of anthracene. In 1998, anthracene production in Europe involved 1,600 tonnes of pure anthracene plus approximately 1,400 tonnes of crude (50%), making a total of 2,300 tonnes of anthracene. In 1999, 550 tonnes of pure anthracene were produced. Taking 1.5% as the typical anthracene content of coal-tar, these quantities would have been derived from the distillation of 153,000 and 37,000 tonnes of crude-coal tar, respectively, corresponding to 8.5% and 2.1% of the total amount of coal-tar distilled in Europe during these two years. Thus, the distillation of coal tar for the ultimate purpose of production of anthracene contributes to fewer than 10% and appears to be following a decreasing trend, and should be seen in this context.

2.6 EXISTING LEGISLATIVE CONTROLS CONCERNING ANTHRACENE AND ANTHRACENE-CONTAINING PRODUCTS

Classification

Anthracene

Anthracene has not been classified in the context of Directive 67/548/EEC concerning dangerous substances.

Anthracene oils

All kinds of anthracene oils (different distillation fractions) are classified according to Directive 94/69 (21st adaptation to technical progress of Directive 67/548/EEC) as

Carcinogen, category 2; R45 - May cause cancer

Labeling: T R45; S53 (Avoid exposure – obtain special instructions before use); S45 (In case of accident or if you feel unwell, seek medical advice immediately - show the label where possible)

Such classification may be considered as not necessary, depending on benzo[a]pyrene content.

Creosote and creosote oils

All types of creosote and creosote oils are classified according to Directive 67/548/EEC as

carcinogen, category 2; R45

labeling T, R45, S53, S45

Such classification may be considered as not necessary, depending on benzo[a]pyrene content.

Occupational Exposure

Directive 91/332 sets a limit value for occupational exposure to “coal tar volatiles” of 0.2 mg/m^3 (8-hour TWA).

Marketing and uses

Creosote

Directive 2001/90 (7th Adaptation to technical progress of Annex I of Directive 76/769) sets strict restrictions on the marketing and use of creosote. Creosote may not be used in the treatment of wood except, by derogation, in the following case:

It may be used for wood treatment in industrial installations, or by professionals covered by Community legislation on the protection of workers for in situ re-treatment, only if it contains < 0.005% benzo[a]pyrene and < 3% water-extractable phenols. Such creosote may be placed on the market only in packaging of capacity ≥ 20 l, and it may not be sold to consumers. It must also be specially labelled as “For use in industrial installations or professional treatment only”.

Further provisions restrict the use of creosote-treated wood: The use of wood treated in industrial installations or by professionals as described above, which is placed on the market for the first time or retreated in-situ, is permitted for professional and industrial use only, e.g. on railways, in electric power transmission and telecommunications, for fencing, for agricultural purposes (e.g. stakes for tree support) and in harbours and waterways.

The restrictions concerning creosote-treated wood do not apply to wood which was treated with creosote before this Directive came into operation and which is placed on the second-hand market for re-use. However, such wood may not be used:

- inside buildings, whatever their purpose,
- in toys,
- in playgrounds,
- in parks, gardens, and outdoor recreational and leisure facilities where there is a risk of frequent skin contact,
- in the manufacture of garden furniture such as picnic tables,
- for the manufacture and use and any re-treatment of:
- containers intended for growing purposes,
- packaging that may come into contact with raw materials, intermediate or finished products destined for human and/or animal consumption,
- other materials which may contaminate the products mentioned above.

Cosmetics

According to Directive 76/768 concerning cosmetics, the use of “anthracene oil” is not permitted, while according to Directive 97/45 (adapting to technical progress Directive 74/768) the use of “crude and refined coal tars” is also not permitted.

3 ENVIRONMENT

(to be added later)

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

There is a scarcity of measured data on exposures to anthracene. For this reason, in the present Report it was necessary to resort frequently to the use of EASE modelling in order to estimate exposures to anthracene. In applying the EASE model, in addition to the various exposure conditions which are stated analytically in each case, it has been assumed that the processes which result in human exposure (e.g. loading, unloading and cleaning in workplaces) take place at room temperature. It is noted that the output of EASE for inhalation exposure to anthracene is unaffected by the input temperature in the range of values below 120°C and changes above this temperature because the vapour pressure then begins to exceed 1 Pa. In this context it is also important to note that EASE tends to overestimate significantly the vapour concentrations of substances of low volatility such as anthracene. Therefore, when vapour concentrations higher than the concentration of saturated anthracene vapours at room temperature (approximately 60 $\mu g/m^3$) were estimated by EASE, they were rejected as an overestimate and only the latter value was carried forward to risk characterisation.

The exposure estimates provided by EASE are 8-hour TWA. Since no information on the duration of specific tasks, likely to be associated with the bulk of exposures, is available, it was not possible to derive estimates of short-term exposures, and for this reason all exposures have been treated as referring to 8-hour TWA.

A complication which may affect the estimation of modelled exposures relates to the fact that the proportion of anthracene in the vapour phase of mixtures such as coal tar and creosote has been assumed to be the same as that in the liquid phase. In reality, the composition of the vapour phase will depend on the temperature of the process concerned.

4.1.1.2 Occupational exposure

4.1.1.2.1 Occupational Exposure during manufacture of anthracene

Exposure to anthracene can take place during coal-tar distillation for the production of anthracene oil, starting material for the production of anthracene. Although this process is not part of anthracene production per se, it is considered here for illustrative purposes.

As far as occupational exposure during anthracene manufacture itself is concerned, it can occur at two stages:

a. manufacture of anthracene from anthracene oil

b. anthracene packaging

Coal-tar distillation and production of anthracene oil

There exist 10 coal-tar distillation plants in Europe. It is expected that in such plants there is exposure of workers to anthracene because of the anthracene content of coal tar (about 1.5%). The total number of exposed employees in Europe is estimated to be no higher than 100.

Worker exposure during coal tar distillation may involve inhalation of vapours and dermal contact which may occur during tank filling, sampling, routine cleaning and maintenance operations. No measured data on occupational exposure to anthracene have been reported. Calculations using the EASE model (non-dispersive use, local exhaust ventilation, direct handling / incidental contact, i.e. once per shift), predict an air concentration of 0-0.1 ppm and a dermal exposure of 0-0.1 $mg/cm^2/day$. For an anthracene content of 1.5% these values correspond to anthracene exposures of 0-0.0015 ppm (0-11 $\mu g/m^3$) and to 0-1.5 $\mu g/cm^2/day$. Actual exposures may be lower to the extent to which personal protective equipment is in use. However, for the purposes of the present Report, the upper values of the predicted ranges are taken forward to risk characterisation.

Manufacture of anthracene from anthracene oil

As already discussed (see Section 2.2.2), anthracene is prepared from anthracene oil by the combined application of crystallisation and vacuum distillation. Limited information regarding exposure during the production of anthracene from anthracene oil is available from the single EU plant which produces pure anthracene:

Manufacture is carried out normally in a closed/encapsulated system, and all tanks and apparatus involved (including the centrifuge used during crystallisation) are connected with a waste air treatment plant. The distillation columns are opened usually every second year for approximately one week. Prior to being opened they are washed and their temperature when opened is below 40^oC.

Approximately 12 employees are involved in anthracene production; all men aged 20-55. Occupational exposure (dermal or by inhalation) arises through activities such as sampling, loading, cleaning and maintenance. Worker protection includes eye and skin protection, while respiration protection is also used whenever necessary. Air sampling to determine actual occupational exposure is not normally carried out, as it is assumed unnecessary in view of the general exposure controls in place. However, limited measurements concerning exposure during two stages of anthracene production have been made available (Höke, 2003):

a) One of eight workers involved in the production of crude anthracene (“anthracene cake”, see Section 2.2.2) was monitored for personal exposure to anthracene during a single 8-hour shift, during which anthracene oil was cooled to form crystals which were removed by filtration and purified by centrifugation. This worker was involved in the filling, emptying and cleaning of vessels and the sampling and loading onto railcars of the crude anthracene thus produced. Gloves and safety glasses were worn. For the purpose of exposure monitoring, a personal pump was carried for the collection of airborne dust (no information on the size of the dust collected was given), and the associated polycyclic aromatic hydrocarbons collected were eluted and analysed by HPLC. The 8-hour time-weighted average concentration of anthracene thus measured was 0.5 $\mu g/m^3$.

b) Personal exposure to anthracene during the subsequent stage of the production process (refining of the crude material) was conducted once during an 8-hour shift in a single worker. During this process, crude anthracene was transferred from railcars to a melting system before liquid storage, distillation and further refining. The activities involved

included filling, emptying and cleaning of vessels and loading of the product only railcars. Exposure monitoring again involved collection of airborne dust with a personal pump and HPLC analysis of the associated polycylic aromatic hydrocarbons. The 8-hour time-weighted average concentration of anthracene measured was 0.78 μg/m^3.

The above measurements refer to concentrations of particulate anthracene (anthracene dust or anthracene bound to airborne particulates) and give no indication of the concentration of anthracene vapour. Based on its physicochemical characteristics, airborne anthracene would be expected to exist primarily in the vapour phase. Indeed, analysis of the vapour and the particulate phase of ambient atmospheric polycyclic aromatic hydrocarbons confirmed that 78-98% of anthracene is in the vapour phase (Cautreels and van Cauwenberghe, 1978; Thrane and Mikalsen, 1981). In a study involving measurement of personal occupational exposure to particulate and vapour PAH in plants handling coal tar or creosote-impregnated wood, the following concentrations of airborne anthracene were found (only single, "typical" values are reported) (Andersson et al., 1983):

	Vapour (mg/m^3)	Particulate (mg/m^3)
Coke oven battery top of coke plant	55	<1.0
Handling of creosote-impregnated railroad ties		

A significant predominance of anthracene presence in the vapour, rather than the particulate phase, mostly based on limited numbers of measurements, was also reported in plants carrying out silicon carbide manufacture, basic metal manufacture and iron and steel production (these data are discussed further in Section 4.1.1.2.4). The fraction of anthracene found in the particulate phase, as a percentage of the total airborne amount, reported in various work environments is summarised in **Table 4.1** (overleaf). It is clear that, even if the representativeness of some of these data is limited by the small numbers of measurements, in most cases airborne particulate anthracene constitutes a small fraction of the total airborne concentrations.

Based on the figures in **Table 4.1**, the concentrations of total airborne anthracene during anthracene production will be assumed to be 10 fold higher than the measured particulate concentrations, i.e. 5 μg/m^3 for the preparation and 7.8 μg/m^3 for the refining of crude anthracene. It is also assumed that all this material is respirable.

While the above estimates provide an indication of the order of magnitude of the airborne exposures during anthracene production, their representativeness is limited by the fact that the measurements on which they are based come from monitoring only one worker, and on a single occasion, for each of the two activities considered. For this reason, an estimate of the expected exposures is also obtained by modelling.

Table 4.1 Proportion of anthracene in airborne particulate phase

Activity	Vapour µg/m³	Particulate µg/m³	Particulate as % of total	Ref.
coke plant	55	<1.0	<2	Andersson et al., 1983
handling of creosote-impregnated railroad ties	13.0	4.7	27	
silicon carbide plant	3.5	0.13	4	Norway Competent Authorities
aluminium reduction plants	12.3	0.74	5	Bjorseth et al, 1978a
	3.90	0.1	3	
	2.7	0.13	5	Andersson et al., 1983
basic metals manufacture	0.99	0.49	33	Norway Competent Authorities
	0.42	0.23	33	
iron and steel processing	13.9	0.04	<1	
	0.29	0.41	59	

Calculations using the EASE model to the two processes for which the above exposure measurements were carried out (non-dispersive use, local exhaust ventilation, direct handling / incidental contact, i.e. once per shift) predict an air concentration of 0-0.1 ppm (vapour), no exposure to anthracene dust, and a dermal exposure of 0-0.1 mg/cm²/day. For the first stage (production of crude anthracene from anthracene oil containing 6% anthracene) these values correspond to anthracene exposures of 0-0.006 ppm (0-44 µg/m³) and to 0-6 µg/cm²/day. For the second stage (anthracene refinement from crude anthracene with an upper limit of anthracene content of 30%), the modelled exposures correspond to 0-0.03 ppm (0-222 µg/m³) and 0-30 µg/cm²/day, respectively. In view of the use of personal protective equipment as stated by the manufacturer, actual exposure is likely to be on the low side of the modelled range.

For clarity of the comparisons, the total airborne concentrations (in µg/m³) estimated from the measured data and EASE, are tabulated below:

	Production of crude anthracene	Refining of anthracene
Estim. From measured data	5	7.8
Estim. By EASE	0-44	0-222

It can be seen that in the case of production of crude anthracene the two types of exposure estimates are of similar order of magnitude, while in the case of anthracene refining the EASE estimate is 1-2 orders of magnitude higher than that obtained from the measured data. This difference reflects the conservative assumptions of the exposure scenarios employed and the limitations of EASE in estimating the vapour concentrations of low-volatility chemicals. While it is likely that the measured data reflect more closely the true exposure levels, in view of their limitations both they and the modelled data (with a maximum of 60 µg/m³ for vapour concentration) will be taken forward to risk characterisation.

Anthracene packaging

Following anthracene manufacture, the final product is packaged manually in paper packages of 25 kg and stored in the dark. No information on measured exposures is available, and, according to information given by the manufacturer, no protection measures are taken during transport and storage of the material.

Calculations using the EASE model (non-dispersive use, local exhaust ventilation, direct handling / intermittent contact, i.e. 2-10 times per shift, non-fibrous, non-aggregating dust, dry manipulation, low tendency to become airborne) predict an air concentration of 2-5 mg/m^3 dust and 0-0.1 ppm (0-741 µg/m^3) vapour, and a dermal exposure of 0.1-1 mg/cm^2/day. The dust particles are likely to be largely of non-respirable size which will be trapped in the nasal region and be carried to the gastrointestinal tract by mucociliary clearance, giving rise to systemic exposure via oral absorption. Actual exposure is likely to be on the low side of this range to the extent to which personal protective equipment is employed during packaging. By analogy with the conclusions drawn in the previous section, it is considered likely that the inhalation exposures predicted by EASE may overestimate the true exposures.

4.1.1.2.2 Occupational exposure during uses of anthracene

Production of anthracene-9-aldehyde

Quantities of anthracene corresponding to 6.8 tonnes in 1999 and 7 tonnes in 2002 were used for chemical synthesis of anthracene-9-aldehyde by one EU manufacturer. No such use was made in 2000 and 2001, and, starting in 2003, the processes will no longer be used. While consideration of this process is consequently not strictly necessary for the purpose of the present Report, in view of its use until very recently it will be examined for illustrative purposes.

According to information provided by the manufacturer, this process was carried out on 20 occasions in 1999, involving approximately 350 kg of anthracene each time and the process lasted 2 hours. The process was carried out in a closed vessel which was filled with anthracene, in the form of lumps, with very little dust formation, via the open manhole at room temperature and under local exhaust ventilation. Subsequently the vessel was closed and reaction took place in a solvent. According to the same source of information, exposure of workers, who were properly protected, was minimal. The number of workers exposed to anthracene in this way was 10-15 in all.

No data on occupational exposure to anthracene during its use in the synthesis of anthracene-9-aldehyde is available. Calculations with the EASE model (non-dispersive use, local exhaust ventilation, direct handling/incidental contact, i.e. 1 time per shift, granular particles) predict an airborne concentration of 0-0.1 ppm (0-741 µg/m^3) and dermal exposure of 0-0.1 mg/cm^2/day. Because the upper end of the latter range exceeds the saturated vapour concentration at room temperature (approximately 60 µg/m^3), the latter value is taken forward to risk evaluation. Actual exposure is likely to be on the low side of this range to the extent to which personal protective equipment is employed.

Production of pyrotechnics

Approximately 0.2 tpa of anthracene are used for the manufacture of pyrotechnics in one plant in Europe. No information on the manufacturing process or related exposures is available.

Calculations using the EASE model (non-dispersive use, local exhaust ventilation, direct handling / intermittent contact, i.e. 2-10 times per shift, non-fibrous, non-aggregating dust, dry manipulation, low tendency to become airborne) predict an air concentration of 2-5 mg/m^3 dust and 0-0.1 ppm (0-741 µg/m^3) vapour, and a dermal exposure of 0.1-1 mg/cm^2/day. The upper end of the modelled vapour concentration range exceeds the saturated vapour concentration at room temperature (approximately 60 µg/m^3) and must be rejected as an overestimate. The dust particles are likely to be largely of non-respirable size which will be trapped in the nasal region and be carried to the gastrointestinal tract by mucociliary clearance, giving rise to systemic exposure via oral absorption. For the purposes of this Report, the higher end of the modelled ranges (with a maximum of 60 µg/m^3 for vapour concentration) is taken forward to risk characterisation.

4.1.1.2.3 Occupational exposure to anthracene via creosote

Exposure to anthracene arising from the manufacture of creosote or creosote-containing products does not strictly come under the terms of the present Report. However, it is considered for illustrative purposes.

Occupational exposure to anthracene present in creosote may occur mainly during the blending and packaging of creosote, treatment of timber at bulk impregnation plants, wood brushing and various other minor uses. Exposure is expected to occur through inhalation and the dermal route.

Creosote blending

Ceosote blending involves the mixing of various fractions of coal-tar distillation which may contain greater or smaller amounts of anthracene. In the EU, creosote blending takes place in 10 tar distillation plants. In the UK tar distillation plants, the number of affected workers is of the order of 2-3 per plant, suggesting that the total number of such workers in Europe may reach 20-30. Additional exposure may take place in an unknown number of small plants which blend purchased coal tar distillates.

No data on occupational exposure to anthracene during creosote blending are available. Calculations using the EASE model (non-dispersive use, local exhaust ventilation, direct handling / intermittent contact, i.e. 2-10 times per shift) predict an air concentration of 0-0.1 ppm and a dermal exposure of 0.1-1 mg/cm^2/day. For an anthracene content of 1.5% these values correspond to anthracene exposures of 0-0.0015 ppm (0-11 µg/m^3) and to 1.5-15 µg/cm^2/day. Actual exposure is likely to be on the low side of this range to the extent to which personal protective equipment is employed during packaging. However, for the purposes of this Report, the higher end of these ranges is taken forward to risk characterisation

Creosote packaging

Creosote is delivered to packaging plants by road tankers through a piping system and it is packaged in containers for sale. The packaging operation is usually automatic, except for small companies where the procedure is manual and where some exposure can occur. Short-lived exposure is also likely during special, short duration, intermittent tasks related to tanker delivery, removal of drip trays, transfer of wastes or maintenance. According to information available, there are 10 plants packaging creosote in the UK, with 3-4 affected workers in each. The total number of such plants in Europe is not known.

No data on occupational exposure to anthracene during creosote packaging are available. Calculations using the EASE model (non-dispersive use, local exhaust ventilation, direct handling / intermittent contact, i.e. 2-10 times per shift) predict an air concentration of 0-0.1 ppm and a dermal exposure of 0.1-1 mg/cm^2/day. For an anthracene content of 1.5% these values correspond to anthracene exposures of 0-0.0015 ppm (0-11 µg/m^3) and to 1.5-15 µg/cm^2/day. Actual exposure is likely to be on the low side of this range to the extent to which personal protective equipment is employed during packaging. However, for the purposes of this Report, the higher end of these ranges is taken forward to risk characterisation.

Timber impregnation

There are 9 bulk timber impregnation plants in Europe. From the number of affected workers (three to four) known to exist in some of them, a total number of 30-40 for Europe may be estimated.

Measured data on anthracene exposure through creosote timber impregnation are limited:

a) A study of occupational exposure to creosote was carried out at two bulk impregnation plants and also during the handling of creosote-treated wood (stevedores, railway sleepers, welding on track) in Finland (Heikkila et al., 1987). In the context of this study, particulate polycyclic aromatic hydrocarbons were identified and personal exposures quantified. No data on the presence of anthracene in the vapour phase are given. The total concentrations of airborne particulate PAH's (including anthracene) found in the timber impregnation plants varied between 0.2 and 46 µg/m^3. As regards more specific information on anthracene, the following mean exposure values (without information on ranges) were given:

   Impregnation plant workers (18 samples): 1 µg/m^3
   Openings (impregnation plants) (2 samples): 19 µg/m^3
   Cleaning chambers (impregnation plants) (3 samples): 6 µg/m^3
   Switch element assembly (railways) (8 samples): 0.5 µg/m^3
   Manual metal arc welding (3 samples): 1.8 µg/m^3

   Thus, for the activity associated with maximum exposure to anthracene in impregnation plants (openings), the mean air concentration was 19 µg/m^3.

b) In another study conducted in the Netherlands (van Rooij et al., 1993), concerning wood creosoting plants, inhaled pyrene concentrations were found to be in the range 0.3-3 µg/m^3, while average dermal exposure to pyrene was estimated at 0.6 mg/day (range 0.2-1.5 mg/day). Taking into account that the proportion of pyrene in creosote was 3.4% and that of anthracene is 1.5%, and assuming a similar composition of emitted vapours (the vapour pressures at 25°C of pyrene and anthracene are $0.6 \cdot 10^{-3}$ Pa and $0.8 \cdot 10^{-3}$ Pa, respectively (Sonnefeld and Zoller, 1983)), the corresponding exposures to anthracene can be approximately estimated at 0.1-1 µg/m^3 (inhalation) and 265 µg/day (range 88-662 µg/day) (dermal). Assuming that dermal exposure arises mainly through contact with the hands and forearms (surface area 1,980 cm^2 - EASE default), the latter figure would correspond to an average dermal dose rate of 134 ng/cm^2/day (range 44-334 ng/cm^2/day). To the extent to which the exposed areas were in fact lower, the estimated dose rates would be correspondingly higher.

   Calculations using the EASE model (non-dispersive use, local exhaust ventilation, direct handling / intermittent contact, i.e. 2-10 times per shift) give an air concentration of 0-0.1 ppm and a dermal exposure of 0.1-1 mg/cm^2/day. For an anthracene content of 1.5%

these values correspond to anthracene exposures of 0-0.0015 ppm (0-11 μg/m^3) and 1.5-15 μg/cm^2/day. These estimates are in fair agreement with the abovementioned measured values. For the purpose of risk characterisation, the upper range of the measured data will be utilised (inhalation exposure: 19 μg/m^3; dermal exposure: 334 ng/cm^2/day).

Creosote brushing

Although according to Directive 2001/90 creosote brushing of wood is not expected to occur, exposure of professionals in the context of in situ re-treatment of wood may still occur. Such exposure is expected to occur via dermal contact. There are no measured data available on occupational exposure to creosote via its use for wood brushing. However, an indication of the levels of dermal exposure may be obtained by reference to the results of 2 studies on amateur volunteers.

a) In a study by Garrod et al. (2000), volunteers were monitored during the use of commercial preservative or anti-foulant formulations (not related to creosote or anthracene) for brushing wooden fences and panels. Gauze pads were fixed on top and underneath their clothing (to allow estimation of through-clothing penetration), while, when gloves were worn on the hands, additional cotton sampling gloves were worn underneath to allow estimation of through-glove penetration. The most relevant data reported in this study are (actual numbers of measurements are not given):

   i) dermal exposure to the formulation:
   body, potential exposure (amount deposited on clothes) 5 mg/min (median), 63.3 mg/min (upper limit);
   gloved hand (actual skin exposure), 0.015 mg/min (median), 3.2 mg/min (upper limit);
   bare hand, 3.5 mg/min (median), 56.2 mg/min (upper limit)

   ii) penetration through coveralls to the body: 10% (median), 67% (upper limit)

   Applying these data to the case of application of creosote by professionals (anthracene content 1.5%; exposure 8 hours per day), the corresponding skin exposures would be:

- actual body exposure (10% penetration through the clothes; 5,690 cm^2 area of the trunk): 0.6 μg/ cm^2/day (median), 8.0 μg/ cm^2/day (upper limit);
- gloved hand (420 cm^2 per hand): 0.26 μg/cm^2/day (median), 54.9 μg/cm^2/day (upper limit);
- bare hand: 60 μg/ cm^2/day (median), 963 μg/ cm^2/day (upper limit)

b) In the study of Roff (1997), amateur volunteers, wearing different types of clothing, were asked to conduct, for 0.5 or 1 hour, brushing of wooden fences with spirit- or water-based woodworm fluids which contained a colourless fluorescent dye. Body exposure was measured using a fluorescence monitor-based system. Highest contamination was found on the bare hands, while clothing was found to provide a major protective effect. The worst case conditions led a predicted maximal value of total dermal exposure of 5.5 ml after 1 hour brushing, of which 75% (i.e. 4.1 ml) is concentrated on the bare hands. Applying these figures to creosote containing 1.5% anthracene leads to an estimated dermal exposure of the bare hands of 61.5 mg. For a skin area (two hands) of 840 cm^2, this corresponds to 585 μg/cm^2/day. This value compares favourably with the worst-case estimate derived from the Garrod et al. (2000) study (bare hand, upper limit) of 963 μg/cm^2/day.

   In using the above data to obtain an estimate of professional exposure it would be reasonable to expect professionals to be more careful than amateurs and make full use of

protective equipment (gloves, coverall). In this context it should be appreciated that, even though the upper limits of dermal exposure estimated from the data of Garrod et al. (2000) (i.e. 8.0 μg/cm^2/day through the trunk and 54.9 μg/cm^2/day through the gloved hand), being based on measured values, will be taken through to risk characterisation, they must be considered as overestimations.

Calculations using the EASE model (non- dispersive use, dilution ventilation and direct handling, extensive contact, i.e. more than 10 times per shift) predict a dermal exposure of 1-5 mg/cm^2/day. For an anthracene content of 1.5%, these values correspond to an anthracene exposure of 15-75 μg/cm^2/day. The modelled range of dermal exposure is comparable with the worst-case estimates obtained above from the Garrod et al. (2000) study.

4.1.1.2.4 Occupational exposure from other industrial sources

Occupational exposure to anthracene in the context of industrial activities not related to the production or use of anthracene itself. Such exposures do not strictly come under the terms of the present Report. However, they are considered for illustrative purposes.

Most reported data on occupational exposures to PAH in relevant occupations focus on benzo[a]pyrene, while data specific to anthracene are limited (the number of measurements in each case being unspecified in published reports, but often by implication they appear to be single measurements), permitting an estimation of the order of magnitude of exposure but giving little or no indication of the variance of the exposures. Therefore it is not possible to estimate the 90th percentiles or other measures of the upper limits of exposure.

Processing of coal and related activities

Geometric mean values for vapour plus particulate airborne anthracene concentrations of 18.61 μg/m^3 (stationary sampling) and 0.073 μg/m^3 (personal sampling) have been reported for a coke plant and bitumen paving plant, respectively (Bjorseth et al, 1978b). No indication of the range of values measured is given.

Average concentrations of anthracene in 5 static and 7 personal air samples for workers employed at a needle coke plant, during normal operation, were 0.66 μg/m^3 and 0.03 μg/m^3, respectively (Boogaard and Vansittetr, 1995).

In a study involving measurement of personal occupational exposure to particulate and vapour PAH in coal tar-handling plants, the following concentrations of airborne anthracene were reported (only single, “typical” values are reported) (Andersson et al., 1983):

	Vapour (mg/m^3)	Particulated (mg/m^3)
Coke oven battery top of coke plant	55	< 1.0
Handling of creosote-impregnated railroad ties	13.0	4.7

B) Carbon anode/graphite plants

In a plant producing carbon anodes for aluminium electrolysis, total airborne anthracene exposure, as measured by personal monitoring) was as follows for workers employed on different tasks (Petry et al, 1996b):

forming section: 0.83-1.49 µg/m^3
paste plant: 2.76-5.51 µg/m^3
store section (broken green anodes and burned anodes): 0.55-1.14 µg/m^3
forming section: 0.57-0.76 µg/m^3
store section (coke) paste plant: 0.77-1.60 µg/m^3
all worksites: 0.42-1.39 µg/m^3

According to another study, the mean (geometric) airborne concentrations of total airborne anthracene were 0.894 µg/m^3 and 0.042 µg/m^3 in a carbon anode and a graphite plant, respectively (Petry et al, 1996a).

C) Silicon carbide plants

The average workplace air concentration of anthracene in the particulate fraction in a silicon carbide and refractory brick industries was reported as 12 µg/m^3 by Lesage et al, (1987), while the geometric mean total airborne concentration in a silicon carbide plant was reported as 0.006 µg/m^3 by Petry et al. (1996a).

In the furnace area of 2 silicon carbide process plants (plant A and plant B), airborne anthracene concentrations (as measured using personal monitors) were as follows for workers employed on different tasks (Petry et al, 1996a):

foreman, 14-20 ng/m^3 and 8-55 ng/m^3
crane operator, 9-16 ng/m^3 and 4-57 ng/m^3
oven workers operating in close proximity to the oven: 14-20 ng/m^3 and 40-249 ng/m^3
oven workers whose tasks did not involve direct work close to the oven site: 3-15 ng/m^3 and 75-200 ng/m^3.

Anthracene in the air of 2 silicon carbide plants had concentrations in the range 0.01-0.85 µg/m^3 in the gaseous phase and 0.01-139 µg/m^3 in the particulate phase (Dufresne et al, 1987). Stationary air samples collected near the ovens of 2 silicon carbide process plants contained anthracene at concentrations ranging from <1 to 7 ng/m^3 and 100-196 ng/m^3, respectively (Petry et al, 1996a).

Measurements of airborne anthracene in a silicon carbide production plant in Norway gave the following results (Norway CA):

Task	N	Vapour (mg/m^3)		Particulate (mg/m^3)	
		Mean	Range	Mean	Range
Electrolysis cellwork (working conditions: 4 “normal”, 1 “worse”	5	3.5	0.4 – 8.6	0.13	03 – 0.32
Unknown welding	1			3.6	
	1			1.1	

D) Aluminium and other metals production

Anthracene was detected in the air of an aluminium reduction plant: 61% of 28 particulate samples contained anthracene at concentrations ranging from non-detectable to 2.8 µg/m^3

(average 0.74 µg/m^3) and 94% of 18 gaseous samples contained anthracene at concentrations ranging from non-detectable to 32.6 µg/m^3 (average 12.3 µg/m^3) (Bjorseth et al., 1978a).

Anthracene was detected in the workplace atmosphere of an aluminium plant in Norway, at a concentration of 4 µg/m^3 (Becher and Bjorseth, 1985). In another similar study, the amount of anthracene in the gaseous phase was found to be 3.90 µg/m^3, while it was 0.1 µg/m^3 in the particulate phase (Bjorseth et al, 1978a).

"Typical" levels of personal exposure in the pot-room of a Sonderberg aluminium plant were reported as 2.7 µg/m3 (vapour) and 0.13 µg/m3 (particulate) (Andersson et al., 1983).

Measurements of airborne anthracene in a metal manufacture plant in Norway gave the following results (Norway CA):

Task	N	Vapour (mg/m^3)		Particulate (mg/m^3)	
		Mean	Range	Mean	Range
Basic Metals					
Electrolysis	5	0.99	0.48-1.65	0.49	0.03-1.10
Welding	2	0.42	0.17-0.66	0.23	-

Measurements in the context of the same study in the manufacture of "other inorganic basic metals" gave exposures to particulate anthracene of 256 µg/m^3 for one electrolysis worker and traces for another two.

A mean value of 0.040 µg/m^3 was reported for airborne anthracene in a metal recycling plant (Petry et al, 1996a).

E) Iron and steel processing

Measurements of airborne anthracene in an iron and steel processing plant in Norway gave the following results (Norway CA):

Task	N	Vapour (mg/m^3)		Particulate (mg/m^3)	
		Mean	Range	Mean	Range
Electrolysis	3	13.9	1.8-22	0.04	0.01-0.06
Pot making	1			273	
Welding	2	29		0.41	0.26-0.56

Table 4.2 summarises the available data and presents the typical and maximum levels reported in the studies described above. Within the limits of confidence permitted by the fragmentary nature of the available information, it can be seen that highest concentrations have been found in silicon carbide and in iron and steel plants.

Table 4.2 Occupational exposure from other industrial sources – Summary (concentrations in µg/m³)

Type of industry	Typical range	Maximum levels*	Form	Reference
coal processing and related activities	< 20	52	Total	Bjorseth et al., 1978a
Carbon anode/graphite	< 1.5	5.51	Total	Petry et al., 1996a
Silicon carbide	< 1	139	Particulate	Dufresne et al., 1987
Aluminium	< 20	35.4	Particulate	Bjorseth et al., 1979
Iron and steel	Insuf. data	273	Particulate	Norway CA

* Sum of maximal vapour plus particulate concentrations, where available

4.1.1.2.5 Occupational exposure from consumer products

Occupational exposure to anthracene can occur during the professional use of coal tar-derived paints and related products. Various types of coal tar paints, as well as damp-proofing materials (usually emulsions of rubber and coal-tar pitch), containing 0.5% or less anthracene, find use in various situations including painting of walls and floors or other surfaces, and are normally used in open spaces.

Use of coal tar paints and related products

There are no measured data available on occupational exposure to coal tar paints and related products. A rough indication of exposures may be obtained if it is assumed that the arguments presented in the previous section to derive exposures during creosote brushing from the Garrod et al. (2000) data also apply to the present case. In this way, taking into account that the anthracene content is approximately 0.5%, the upper limits of the extrapolated exposures would be 2.7 µg/cm^2/day through the trunk and 18.3 µg/cm^2/day through the gloved hand.

Calculations using the EASE model (non-dispersive use, dilution ventilation and direct handling, extensive contact, i.e. more than10 times per shift) predict a dermal exposure of 1-5 mg/cm^2/day. For an anthracene content of 0.5%, these values correspond to an anthracene exposure of 5-25 µg/cm^2/day. The modelled range of dermal exposure is comparable with the worst-case estimates extrapolated from the data of the Garrod et al. (2000) study.

4.1.1.3 Consumer exposure

No consumer exposure is expected to occur to commercial anthracene (normal use of pyrotechnics containing anthracene is not expected to lead to significant exposure).

Consumer exposure can occur only during the use of coal tar-derived paints and related products. As already stated, the only significant exposure expected is that through the skin. No measured data for such exposures are available. However, a rough indication of the magnitude of dermal exposure may be obtained if it is assumed that the arguments presented in the previous section to derive exposures during creosote brushing from the data of Garrod et al. (2000) also apply to the present case. Recalling that the upper limit of dermal exposure through the bare hands (it is assumed as a worst case that consumers will work with bare hands) was 56.2 mg/min, and 6.3 mg/min through the trunk, and taking 2.5 hours as a typical time of exposure per day for amateurs, the resulting exposures can be estimated as 100 µg/cm^2/day through each hand and 0.8 µg/cm^2/day through the trunk. These values will be taken forward to risk characterisation.

4.1.1.4 Exposure of the general population via the environment

Exposure of the general population via the environment may occur by the following ways:

a) via inhalation of air polluted with anthracene: Measurements of anthracene concentration in polluted western European cities indicated maximal concentrations (vapour plus particulate) typically in the range below 10 ng/m^3, and a maximum reported level of 34 ng/m^3 (Broman et al., 1991; Smith and Harrison, 1996; Masclet et al., 1988). Incomplete combustion of fossil fuels seems to be the main source of this pollution, while the contribution of industrial activities related the production and use of anthracene or anthracene-containing products is minimal. Concentrations tend to be near the higher end of the range observed during the winter months, whereas they tend to be substantially lower during the summer period. The maximum reported value of 34 ng/m^3, taken to reflect regional exposure, would be expected to contribute 680 ng (9.7 ng/kg) to the daily intake of the general population. It is emphasised once more that only a small fraction of this exposure would be attributable to activities related to the production and use of isolated anthracene.

   No measured data exist regarding atmospheric concentrations of anthracene in the vicinity of the single European anthracene production plant. Application of standard EUSES modelling, and taking air emissions as 25 kg/year (Höke, 2000), leads to a calculated annual average PEC_{local} for anthracene production of 0.1 ng/m3, which would correspond to a daily intake by inhalation of 2 ng (0.03 ng/kg).

b) via intake of drinking water and food. Measured data on drinking water concentrations show that such concentrations are usually below the limit of detection, with detectible levels being in the range below 10 ng/l, and the highest value reported is 30 ng/l (Piet and Morra, 1983; Kvesheth and Sortland, 1982). Thus drinking water consumption is expected to contribute no more than 60 ng to daily intake of anthracene, with only a small fraction of this exposure arising from activities related to the production and use of isolated anthracene. Application of standard EUSES modelling leads to an estimated $PEC_{local,\ surface\ water}$ for anthracene production of 12.9 ng/l which, even if it corresponded to drinking water levels, would contribute 25.8 ng (0.4 ng/kg) to the daily intake.

As regards anthracene intake through the diet, there are no data sufficient to permit the proper estimation of human daily intake. Anthracene is found in relatively large concentrations in smoked food or in food which has been cooked on open fire or broiling. Residues of anthracene have been reported in:

- broiled meats at 4.5—7 µg/kg (US EPA, 1987; Fazio and Howard, 1983; Maga, 1986),
- smoked meats and meat products at 2-20 µg/kg (Lo and Sandi, 1978),
- vegetable oils at concentrations typically of the order of 10 µg/kg or less (Menichini et al., 1991; IARC, 1983b; Santodonato et al., 1981; Speer et al., 1990; Hopia et al., 1986), and
- vegetables up to about 0.2 µg/kg (Wickström et al., 1986).

Taking 10 µg/kg as a conservative, maximal concentration for foodstuffs, and a consumption of 1 kg of food per day, a maximum daily human intake of 10 µg anthracene would be via the diet. This estimate is supported by the results of a calculation using data on food intake from the Dietary and Nutritional Survey of the British population which resulted in an estimated mean anthracene intake of 45 ng/kg/day, i.e. 3.1 µg/day (Health and Safety Executive (UK), 2002). Hence a maximum daily intake via food of 10 µg will be adopted.

The above estimates show clearly that exposure via food dominates exposure of the general population via the environment, and a maximal value of 10 µg (143 ng/kg) for oral daily intake via food and water will be taken forward to risk assessment.

4.1.1.5 Combined exposure

Given the extremely low levels of exposure of the general population via the environment, relative to occupational exposures, and the very limited opportunity of consumer exposure (only via one product), it was not considered useful to produce a combined exposure assessment.

4.1.1.6 Exposure assessment – conclusions and summary

The ranges of exposure levels estimated for the various scenarios related to the production and current uses of anthracene, and the maximal values taken forward to risk assessment, are summarised in **Table 4.3**. The corresponding figures estimated for exposures related to other activities, which do not come under the terms of the present Report but are discussed for illustrative purposes, are summarised in **Table 4.4**.

Occupational exposure

Occupational exposure to anthracene can occur during the manufacture of anthracene from anthracene oil and in the context of the use of anthracene for the manufacture of pyrotechnics. Measured data on these exposures are limited, and in most cases EASE has been used to obtain modelled values which are taken forward to the risk characterisation section. Because in some cases the air concentrations predicted by EASE exceed the concentration of saturated vapours of anthracene at room temperature (a consequence of the overestimation to which EASE leads for compounds of relatively low volatility), the figure of 60 $\mu g/m^3$ (concentration of saturated anthracene vapour at room temperature) has been taken forward to risk characterisation. It is likely that even this figure represents a significant overestimation of the true vapour concentrations.

Additional occupational exposures arise during the production of anthracene oil from coal tar, as a result of the production and use of products which are based on tar distillates and contain anthracene as part of a complex mixture (e.g. creosote and products containing coal tar), as well as in the context of workplace activities where organic material is incompletely combusted. For most of these exposure situations, there are no measured exposure data available, and for this reason modelling had to be employed to estimate exposure by inhalation and dermal exposures.

Consumer exposure

No exposure from products containing commercial anthracene is expected. Consumer exposure to anthracene can come through the use of products based on coal tar (which contains anthracene), used for painting and damp-proofing. No measured data are available on the exposure which such uses entail, and the estimated exposures are taken to be similar to those obtained for the corresponding professional uses. These are calculated by the EASE model. However, it is recognised that such exposures in the case of consumers will be much more limited in frequency and duration.

<u>Exposure of the general population via the environment</u>

Exposure of the general population to anthracene via the environment can come from the inhalation of polluted air and the consumption of polluted water and food. The contribution of activities directly related to the production and use of anthracene to this exposure is probably very limited.

Table 4.3 Exposures related to production and current uses of anthracene

Type of exposure		Measured exposure (maximum values)	Modelled exposure			Values taken forward to risk characterisation	
Manufacture of anthracene			**EASE scenario**	**Air concentration**	**Dermal exposure**	**Air concentration**	**Dermal exposure**
Anthracene production	**Preparation of crude anthracene**	0.5 μg/m³ (particulate)	non-dispersive use; LEV; direct handling/incidental contact	0-44 μg/m³ vapour	0-6 μg /cm²/day	5 μg/m³ (respirable) Modelled: 44 μg/m³ (vapour)	6 μg /cm²/day
	Purification of crude anthracene	0.78 μg/m³ (particulate)	non-dispersive use; LEV; direct handling/incidental contact	0-222 μg/m³ * vapour	0-30 μg /cm²/day	7.8 μg/m³ (respirable) Modelled: 60 μg/m³ # (vapour)	30 μg /cm²/day
Anthracene packaging		none	non-dispersive use; LEV; direct handling/intermittent contact; non-aggregating dust; dry manipulation; low TBA	2-5 mg/m³ dust 0-741 μg/m³ vapour *	100-1000 μg/cm²/day	5 mg/m³ dust 60 μg/m³# vapour	1000 μg/cm²/day
Manufacture of pyrotechnics		none	non-dispersive use; LEV; direct handling/intermittent contact; non-aggregating dust; dry manipulation; low TBA	2-5 mg/m³ dust 0-741 μg/m³ vapour *	100-1000 μg/cm²/day	5 mg/m³ dust 60 μg/m³# vapour	1000 μg/cm²/day
Exposure of the general population through the environment		10 μg/day (oral) 680 ng/day (inhalation)	N/A	N/A	N/A	143 ng/kg/day (oral) 9.7 ng/kg/day (inhalation)	

upper limit rejected as higher than concentration of saturated vapour at room temperature
\# concentration of saturated vapour at 25°C

Table 4.4 Exposures to anthracene from activities not related to production and current uses

Type of exposure	Measured data (max. values)	EASE scenario	Modelled exposure		Values taken to risk characterisation	
			Air concentration	Dermal exposure	Air concentration	Dermal exposure
Coal-tar distillation	none	non-dispersive use; LEV; direct handling/incidental contact	0-11 µg/m³ vapour	0-1.5 µg /cm²/day	11 µg/m³ vapour	1.5 µg /cm²/day
Use of anthracene in chemical synthesis	none	non-dispersive use; LEV; direct handling/incidental contact	0-741 µg/m³ vapour *	0-100 µg/cm²/day	60 µg/m³ vapour #	100 µg/cm²/day
Occupational exposure via creosote						
Creosote blending	none	non-dispersive use; LEV; direct handling/intermittent contact	0-11 µg/m³ vapour	1.5-15 µg/cm²/day	11 µg/m³ vapour	15 µg/cm²/day
Creosote packaging	none	non-dispersive use; LEV; direct handling/intermittent contact	0-11 µg/m³ vapour	1.5-15 µg/cm²/day	11 µg/m³ vapour	15 µg/cm²/day
Timber impregnation	19 µg/m³ (particulate); 334 ng/cm²/day	non-dispersive use; LEV; direct handling/intermittent contact	0-11 µg/m³ vapour	1.5-15 µg/cm²/day	19 µg/m³ particulate	334 ng/cm²/day

Upper limit rejected as higher than concentration of saturated vapour at room temperature
Concentration of saturated vapour at 25ºC

Table 4.4 continued overleaf

Table 4.4 continued Exposures to anthracene from activities not related to production and current uses (continued)

Type of exposure	Measured data (max. values)	EASE scenario	Modelled exposure		Values taken to risk characterisation	
			Air concentration	Dermal exposure	Air concentration	Dermal exposure
Creosote brushing	8.0 µg/cm²/day through the hands and 54.9 µg/cm²/day through the trunk (indirect estimates)	non-dispersive use; dilution ventilation and direct handling; extensive contact	none	15-75 µg/cm²/day	none	8.0 µg/cm²/day through the hands; 54.9 µg/cm²/day through the trunk; 75 µg/cm²/day (EASE)
Occupational exposure from other industrial sources						
Coal processing & related activities	52 µg/m³ (total)	N/A (not applicable)			52 µg/m³ (total)	-
Carbon anode/graphite	5.51 µg/m³ (total)	N/A			5.51 µg/m³ (total)	-
Silicon carbide	139 µg/m³ (particulate)	N/A			139 µg/m³ (particulate)	-
Aluminium & other metals	256 µg/m³ (particulate)	N/A			35.4 µg/m³ (particulate)	-
Iron and steel	273 µg/m³ (particulate)	N/A			22 µg/m³ (particulate)	-
Occupational exposure from consumer products						
Use of coal tar paints and related products	18.3 µg/cm²/day through the hands and 2.7 µg/cm²/day through the trunk (indirect estimates)	non-dispersive use; dilution ventilation and direct handling; extensive contact	none	5-25 µg/cm²/day	none	18.3 µg/cm²/day through the hands and 2.7 µg/cm²/day through the trunk; 25 µg/cm²/day (EASE)
Consumer exposure						
Use of coal tar paints and related products	100 µg/cm²/day through the hands and 0.8 µg/cm²/day through the trunk (maximum, indirect estimate)	N/A	none	N/A	none	100 µg/cm²/day through the hands and 0.8 µg/cm²/day through the trunk

4.1.2 Effects assessment: Hazard identification and dose-response relationships

Anthracene belongs to the group of (homocyclic) polycyclic aromatic hydrocarbons (PAH), a group of compounds which includes many powerful genotoxic and carcinogenic agents (IPCS, 1998). Most studies of the toxicology of PAH have been carried out with compounds other than anthracene, and indicate that PAH are in general absorbed through the lung, the gastrointestinal tract, and the skin. Once absorbed by any route, they are widely distributed in the body and are found in almost all internal organs, particularly those rich in lipids. They can cross the placenta and have been detected in fetal tissues.

The metabolism of PAH is complex, and involves mainly conversion via intermediate epoxides to phenols, diols, and tetrols, which can subsequently form phase II conjugates (esters with sulfuric or glucuronic acids or with glutathione). Metabolites and their conjugates are excreted via the urine and feces, but conjugates excreted in the bile can be reabsorbed after being hydrolysed by enzymes of the gut flora. After inhalation or intratracheal instillation of PAH, the largest part of metabolites was recovered in the feces, suggesting significant hepatobiliary recirculation following pulmonary absorption. PAH do not persist in the body and their turnover is rapid (IPCS, 1998).

The molecular basis of the genotoxicity and carcinogenicity of PAH has been extensively investigated, and the ability to undergo metabolism to a bay-region diol epoxide is believed to constitute an important structural feature of it. It is important, from this point of view, to note that the anthracene molecule does not contain a bay region.

4.1.2.1 Toxicokinetics, distribution and metabolism

Anthracene is a photosensitive substance and needs to be protected from light during experimental handling. In many of the reported studies it is not explicitly clarified whether suitable precautions had been taken. When such precautions were described they are mentioned in the text below.

4.1.2.1.1 Studies in animals

In vivo studies

Inhalation

No information is available on the absorption, metabolism or excretion of anthracene after inhalation exposure. However, based on data obtained with other polycyclic aromatic hydrocarbons, it can be anticipated that the degree and rate of anthracene uptake and distribution following inhalation can be affected significantly by its physical form, i.e. whether it is in a vapour, aerosol or particulate form or whether it is adsorbed to solid particles (IPCS, 1998; Montizaan et al., 1989). Lung clearance of PAH is significantly slower when they are particulate or particulate-bound (half-life of the order of days) than when they are in a vapour or dissolved form (usually half-life of the order of hours), and depends on the size of the particles and the PAH-to-carrier weight ratio. The degree of penetration of particulate or particle-bound PAH into the lung depends on the size of the particles. Inhaled particles with aerodynamic diameter greater

than 10-20 µm can be intercepted in the nasopharynx and the tracheobronchial part of the lung, while particles < 2.5 µm are respirable, i.e. they can reach the lung alveoli. Depending on the size of different particles, the mucociliary system can contribute to different degrees to their clearance, in turn affecting the degree of elution and absorption of the particulate-adsorbed PAH. For the particle-bound PAH found in the ambient air, the overall degree of absorption through the lungs has been estimated at about 20% (Montizaan et al., 1989). The corresponding figure for other situations (e.g. industrial sites of anthracene production and handling) will depend on the size distribution of the corresponding particulates, for which no information is available.

The lung clearance kinetics of anthracene was examined in rats after intratracheal instillation (Bond et al., 1985). Twenty-four female F344/Crl rats received 1 nmole (178 ng, ~1.2 ng/kg) 9-^{14}C-anthracene (suspended in 250 µl of a vehicle consisting of 10% DMSO in 0.9% saline) by a single intratracheal instillation, and groups of 3 animals were killed 1, 3, 12, 24, 48, 72 and 96 hours later. The lungs were solubilised and the amount of radioactivity they contained measured by scintillation counting. Most (99.7%) of the radioactivity disappeared very rapidly (in less than 1 hour), while the remaining 0.3% was cleared much more slowly (half-life of 25.6 hours). It is not known whether mucociliary clearance contributed to the extremely rapid rate of the first phase. A qualitatively similar picture was reported in the same study for other PAH, and it is noted that broadly similar findings (biphasic and substantial lung clearance after intratracheal instillation or inhalation) have also been described for other PAH (e.g. benzo[a]pyrene) (Weyand and Bevan, 1986; Mitchell, 1982; reviewed by Montizaan et al., 1989). Nevertheless, the data from the Bond et al. (1985) study do not provide a clear answer to important questions such as whether all the material that disappeared from the lung was systemically absorbed and whether similar kinetics would hold after inhalation, or after different doses.

Oral

Information allowing the estimation of the net absorption of anthracene through the gastrointestinal epithelium is missing. In an old study in male white rats, groups of 4 animals were fed diets containing 0.2% or 1% anthracene for two 1-hour periods during the same day (total intake 270-830 mg; ~1-3 mg/kg), and feces were collected during the next 2 days (Chang, 1943). Gravimetric analysis of the feces, based on extraction with ether, saponification with alcoholic potassium hydroxide, and precipitation of the saponified material with water, indicated that an amount of saponifiable material (interpreted by the authors as corresponding to unchanged anthracene) equivalent to 53% and 83% of the administered doses, respectively, was excreted by this route. In the context of the same study, an aqueous starch suspension of 100 mg anthracene was administered by gavage to 2 male white rats. The “unchanged anthracene” found in feces collected during the next 3 days from each of the animals corresponded to 64% or 74% of the administered dose, respectively. The results of positive and negative control experiments reported in this study indicate that, at the level of dosing employed, the material detected in the feces was indeed related to the treatment. However, the crude (by modern standards) nature of the analytical method does not permit any conclusions on the net absorption of anthracene from the GI tract following oral ingestion, other than suggesting that it may not exceed 50%.

Because of their low water solubility, PAH can be physically adsorbed to the mucosal surfaces of the gastrointestinal tract. For this reason (as indicated by studies with PAH's other than anthracene - mainly benzo[a]pyrene), absorption and, hence, tissue concentrations tend to increase exponentially with dose (IPCS, 1998; Montizaan, 1989). For the same reason, systemic absorption of PAH may be aided by bile. To study the role of bile in the intestinal absorption of anthracene following oral intake, conscious rats with bile duct and duodenal catheters were given isotopically labelled anthracene (1 mg in 0.2 ml in corn oil, corresponding to 3.7 mg/kg), and the

recovery of radioactivity in bile and urine was measured (Rahman et al., 1986). One group of animals received via the duodenal catheter only anthracene solution, whereas a second group received the anthracene solution mixed with 0.5 mg bile, followed by 8 further doses of 0.5 ml bile at hourly intervals to simulate normal bile flow. Over the next 24 hours, samples of bile and urine were collected and the amount of radiolabel recovered was measured as an index of the efficiency of absorption. Irrespective of the presence or absence of bile, about two thirds of the radioactivity absorbed was found in the bile and one third in the urine. Cumulative recovery in the presence of bile was 75.55% of the administered dose, while in the absence of bile it was somewhat lower (53.65%), a finding attributed to the relatively low water solubility of anthracene and suggesting that bile-mediated micellar solubilisation facilitates the uptake process. While this study indicates that, over a period of 24 hours, at least 75% of orally administered anthracene is initially absorbed from the intestinal tract of rats with normal bile flow; in the absence of information on the amount of unmetabolised anthracene present in biliary secretions the net systemic absorption of anthracene via the intestine cannot be estimated. Nevertheless, the result of this study is not incompatible with that of Chang (1943) which suggested a net absorption of less than 50%.

In early studies of the metabolism of anthracene, (-)-1,2-dihydroxy-1,2-dihydroanthracene was found in the urine of rats fed a diet containing 4% anthracene (Boyland and Levi, 1935; 1936a; 1936b). While the free 1,2-dihydrodiol was the main metabolite found, additional metabolites detected included its glucuronic acid ester as well as 1-anthrylmercapturic acid. Rabbits treated in the same way excreted in the urine mainly the (+)-stereoisomer of the glucuronic acid ester.

In a more recent, detailed study, a group of 24 male Chester Beatty rats, was given for 3 weeks a diet containing 5% anthracene (Sims, 1964). Examination of metabolites in urine indicated that anthracene was converted to 1,2-dihydroxyanthracene and trans-1,2-dihydro-1,2,-dihydroxyanthracene, which were excreted mainly as sulphuric acid and glucuronic acid conjugates. An additional product of metabolism at the 1 and 2 positions of anthracene was N-acetyl-S-(1,2-dihydro-2-hydroxy-1-anthryl) cystein (a glutathione conjugation product), which was also found in the urine. Anthracene appears to undergo also metabolism at the 9- and 10-positions, as indicated by the detection of trans-9,10-dihydro-9,10-dihydroxy-anthracene as well as its further metabolites 2-hydroxy-9,10-anthraquinone, anthrone and conjugates of 9-hydroxy-, 9,10-dihydroxy- and 2,9,10-trihydroxyanthracene. A possible non-hepatic origin of the metabolites at the 9,10- position was suggested by the fact that *in vitro* metabolism of anthracene using rat liver microsomes led to the formation primarily of trans-1,2-dihydroxy-1,2-dihydroanthracene but not of metabolites at the 9,10-position (Akhtar et al., 1979).

Dermal

The rate of skin absorption of anthracene was examined in 55-day old mice of Strong/A strain (Bock and Burnham, 1961). 0.25 ml of a 1% solution of anthracene in a 99:1 mixture of benzene and mineral oil was applied on an area of shaved skin approximately 6 cm^2., resulting in an estimated dose of roughly 400 $\mu g/cm^2$. After periods of 10 minutes to 4 hours the animals were killed, the application site was cleaned with benzene and a piece of skin removed, homogenised, extracted with benzene and the extract analysed by spectrofluorimetry (no information is given on measures to protect the chemical from photodegradation). The skin concentration of anthracene increased rapidly, reaching its maximal value (10-15 µg/g wet weight of skin, representing roughly 0.2-0.3% of the applied dose) after approximately 1 hour and remained almost unchanged up to 4 hours post-application (no measurements were reported beyond this point). This steady-state concentration was not significantly affected by the sex of the animals. It

is noted that the use as a solvent of benzene, an efficient defattening agent, may have affected the absorption kinetics.

The cutaneous absorption of anthracene was also examined in female Sprague-Dawley rats that were administered a single topical skin application of ^{14}C-anthracene (14.2 µg, 9.3 µg/cm^2) dissolved in 71 µl 1:7 hexane:acetone. The solvent was removed with a stream of air immediately after application (no information is given on measures to protect the chemical from photodegradation) (Yang et al., 1986), and urine and feces were collected daily. Cumulative recovery of the applied radioactivity over a period of 6 days was 29.1% from urine and 21.9% from feces, while another 1.3% was found in tissues (mainly the liver and kidneys) collected when the animals were sacrificed at the end of this period. Absorption was fastest during the first 24 hours (20.1%), while, as indicated above, at 6 days it reached 52.3% and was still rising, albeit at a greatly decreased rate. This figure is compatible with the findings of the study of Bock and Burnham (1961), described in the previous section, which, based on an initial rate of increase of the tissue concentration of anthracene of 0.2-0.3% per hour, would suggest 50% absorption in 5-10 days.

The study of Yang et al. (1986) indicates that, at the relatively low dose used, the absorption rate through the rat skin is approximately 1% per hour. An approximately 2fold faster initial absorption rate, but similar (55.9%) cumulative skin absorption after 6 days, was described in the same report, using an *in vitro* system: To excised slices of rat skin, 350 µm thick, ^{14}C-anthracene was applied at the same concentration as used in the *in vivo* study (9.3 µg/cm^2), and the kinetics of penetration of radioactivity through the skin and into the receptor fluid of Franz-type diffusion cells measured.

Van Rooij et al. (1995) used an isolated blood-perfused pig ear system to study the skin penetration of PAHs applied in the form of coal tar. This test system is considered as a useful model for dermal absorption studies because of the morphological and functional similarity of pig skin to that of humans and because percutaneous absorption rates through the pig skin have been found to be comparable to those through the human skin. Coal tar containing 3.7% anthracene (other PAH present included phenanthrene, fluoranthene, fluorene and pyrene at levels ranging from 6.8% to 2.1%, and various heavier PAH at levels below 1%), was applied at 11 mg/cm^2 (corresponding to 407 µg/cm^2 anthracene) to a 24 cm^2 surface of a pig ear. The latter was perfused with blood for 250 min and blood concentrations of various PAHs measured at intervals. The mean absorption rate of anthracene was 19.6 ng/cm^2 per hour, corresponding to 0.005% of the applied dose per hour. This rate is much slower than the rates reported by the previously described *in vivo* and *in vitro* rat studies, a difference which can be ascribed primarily to the much higher dose employed in the van Rooij (1995) study where even after 200 minutes less than 0.2% of each PAH had been absorbed. In addition, the application of a PAH in the form of a complex mixture is known to increase significantly its dermal residence time (Dankovich et al., 1989). For these reasons the result of this study cannot be used to estimate the absolute value of the rate or degree of skin absorption of anthracene.

Sartorelli et al. (1999) examined the kinetics of absorption of various PAHs, including anthracene, through full-thickness monkey (Cercopithecus aetiops) skin using an *in vitro* static diffusion cell and saline solution with gentamycin sulphate and 4% bovine serum albumin as receptor fluid. Anthracene was applied at a dose of 15.1 nmol/cm^2 (2.7 µg/cm^2) as part of a mixture of 13 PAHs dissolved as a suspension in lubricating oil or dissolved in 30 µl of acetone. In the latter case, following evaporation of the solvent, a few drops of artificial sweat were applied to the residue on the skin surface No information is given on measures to protect the chemical from photodegradation. In the presence of artificial sweat, the anthracene absorption

rate reached a steady state corresponding to 0.35% of the applied dose per hour, while it was approximately 4fold slower when lubricating oil was used.

Other routes

Anthracene (0.4 µmol in 200 µl sesame oil – corresponding to approximately 0.5 µg/kg) was administered to male Sprague-Dawley rats by subcutaneous injection (Myers et al., 1988). The animals were killed 24 hours later and the tissue in contact with the anthracene removed, extracted with organic solvent and analysed by HPLC. The metabolites detected included 9-formylanthracene, 9-methylanthracene, 9-hydroxymethyl-10-methylanthracene, 9-hydroxymethyl-anthracene, 9,10-dimethyl-anthracene, and 9,10-dihydroxymethyl-anthracene. The detection of these metabolites indicates the operation of a pathway leading to methylation of positions 9 and 10 of anthracene, followed by further oxidative metabolism. The same metabolites were observed following *in vitro* incubation of anthracene (taking precautions to avoid photodegradation) with rat liver cytosol fortified with S-adenosylmethionine. In view of the weak tumour initiating activity of 9,10-dimethylanthracene (LaVoie et al., 1985), it was suggested that this type of biomethylation pathway (which was not reported after oral administration), may contribute to the induction of local sarcomas by subcutaneously applied anthracene.

Evidence of hepatobiliary recirculation was provided by the detection, after mild acid hydrolysis, of 1-anthrylglucuronic acid and free anthracene in the bile as well as in aqueous extracts of the duodenum and the intestine of mice (strain A) after intravenous administration of 0.5 mg of a colloidal suspension of anthracene (Harper, 1959). From this finding it was concluded that 2-hydroxy-1,2-dihydro-1-anthrylglucuronic acid is formed during the metabolism of anthracene.

In vitro studies

Two *in vitro* studies have already been mentioned: *In vitro* metabolism of anthracene with rat liver microsomes predominantly results in the formation of trans-1,2-dihydroxy-1,2-dihydroanthracene, with little evidence of metabolism at the 9,10-position (Akhtar et al., 1979). *In vitro* formation of the intermediate 1,2-epoxide during incubation of anthracene with purified cytochrome P450 from rat liver has been shown by van Blanderen et al. (1985). On the other hand, *in vitro* metabolism using rat liver cytosol (post-microsomal supernatant) fortified with S-adenosylmethionine gave metabolites derived from methylation at positions 9 and 10, as well as products of their further oxidative metabolism (Myers et al., 1988), the same metabolites being observed also after skin application.

In vitro metabolism of anthracene by microsomes from the liver and skin of New Zealand White rabbits leads primarily to the 1,2-dihydrodiol, as also observed with rat microsomes (Hall and Grover, 1987).

4.1.2.1.2 Studies in humans

In vivo studies

Five normal adult volunteers without cutaneous disease applied to their skin a 2% solution of crude coal tar in petrolatum, containing 190 mg/l anthracene (Storer et al, 1984). In total, 85 g of the solution were applied for 8-hour periods on two consecutive days. Organic extracts of blood collected after completion of the second application and subjected to gas chromatography and

mass spectrometry yielded evidence of anthracene adsorption in four of the five volunteers, with blood concentrations ranging 0.08-0.47 µg/l.

4.1.2.1.3 Summary

A limited study in human volunteers indicates that anthracene penetrates human skin but does not permit a quantitative estimation of the proportion of dermally applied anthracene which is systemically absorbed by humans. No data on the absorption of anthracene via the gastrointestinal tract or via the pulmonary system in humans are available.

In vivo studies with rats, and *in vitro* studies with rat and monkey skin, suggest that, at doses ranging from a few µg to a few hundreds of µg per cm^2, dermal absorption of anthracene occurs at a rate of 0.3-1% of the applied dose per hour.

Although at least 75% of orally administered anthracene is absorbed from the intestinal tract of rats in 24 hours, the available data do not allow the estimation of the net absorption after oral intake. Data from an old study suggest that net gastrointestinal absorption in rats may not exceed 50%.

Based on data from intratracheal instillation in rats, lung clearance appears to be practically 100% within 1 hour. No information on the extent of systemic absorption of anthracene after inhalation is available. Physical form (particulate, aerosol) and particle size are expected to be important determinants of the rate and degree of such absorption.

Analysis of urinary metabolites, as well as *in vitro* studies, suggest that the metabolism of anthracene after oral intake proceeds initially via epoxidation at the 1,2-position, followed by hydrolysis to the 1,2-dihydrodiol which undergoes further metabolism, mainly to glucuronic or sulphuric acid conjugates. There is also evidence of additional metabolic pathways, at least in rat skin, leading to methylation and oxidation of anthracene at the 9,10 positions. Although there is evidence that anthracene metabolites occur in the bile and the feces, no information on the amounts and nature of such metabolites is available.

In conclusion, the available studies leave significant questions regarding the absorption, distribution and metabolism of anthracene unresolved, including the degree of systemic absorption after inhalation or dermal exposure and the levels and nature of metabolites in the gastrointestinal tract. Although data on other PAH suggest that in general PAH do not persist in the body (IPCS, 1998), given the moderately high log Pow and the low water solubility of anthracene, its potential to accumulate in lipid-rich mammalian tissues also remains to be addressed.

4.1.2.2 Acute toxicity

4.1.2.2.1 Studies in animals

Inhalation

No information is available on the acute toxicity of anthracene after inhalation in animals.

Oral

Anthracene (40% suspension in 0.5% carboxymethylcellulose) was administered intragastrically, at a dose of 16 g/kg, to groups of 5 male and 5 female Wistar rats. No lethality was observed after 14 days of observation, indicating that the LD_{50} is > 16 g/kg (Grote, 1979a). The toxic effects observed included fatigue asthenia, hyperemia of the kidney, liver, heart and lungs, lipid changes in the liver and leukocytosis. A similar result was reported when anthracene was administered to mice (unspecified strain) at 17 g/kg (Nagornyi and Rodionov, 1969).

In another study, groups of 5 male Wistar strain rats were administered single doses of anthracene by gavage at dose levels of 5.0, 10.0 and 20.0 g/kg of body weight (Mellon Institute, 1977). Mortality was observed within 14 days of dosing in 4 animals receiving 10.0 g/kg and in all animals receiving 20.0 g/kg (no toxic symptoms were reported at the dose of 5.0 g/kg). The LD_{50} was calculated to be 8.12 g/kg of body weight (5.90-11.2, 95% confidence interval). Clinical observations included piloerection, sluggishness, prostration, rapid breathing, and bloody eyes. Gross necropsy evaluation revealed hemorrhages of the lungs; mottled livers with prominent acini and a burned white colour; spleens and kidneys pale and mottled; congested kidneys and adrenals; distended, chemical-filled and opaque stomachs; pink pylori; and distended, transparent, gas-filled, and yellowed intestines.

Dermal

An early study in which the ability of various compounds to suppress the sebaceous glands in mice was examined as a marker of skin carcinogenic potential reported anthracene to be negative (Bock and Mund, 1958).

Anthracene (purity 'Anthrazen reinst', dissolved at 0.4 g/ml in polyethylene glycol), was applied, at a single dose of 1,320 mg/kg, in the form of an occlusive patch to the shaved skin of Wistar rats (5 male, 5 female) for 24 hours, and the animals observed for 14 days. No deaths were observed, and it was reported that no local or systemic symptoms of toxicity or pathological findings were observed (although no details of the symptoms or changes looked for are given). This led to the conclusion that the dermal LD_{50} was greater than 1,320 mg/kg body weight (Worstmann, 1981).

Acute toxicity after dermal exposure was evaluated in a group of six male albino rabbits (strain not reported) receiving single occluded applications of anthracene at a dose level of 4.0 g/kg of body weight (Mellon Institute, 1977). The test article was held in contact with the intact skin (under polyethylene sheeting) for a 24-hour period. Mortality was not observed within 14 days of treatment; the LD_{50} was determined to be greater than 4.0 g/kg of body weight. Clinical observations included diarrhoea. Gross necropsy evaluation revealed congestion of the liver and spleen and pale and mottled kidneys. No further details are given in that report.

Intraperitoneal

The LD50 of anthracene after i.p. administration to mice (unspecified strain) was reported to be 430 mg/kg (Salamone, 1981).

Five mice were administered i.p. anthracene dissolved in olive oil, at a dose of 1,000 mg/kg. Sacrifice of 1 animal revealed that the oil had been fully resorbed after 15 days. The remaining 4 animals were without effects 5 months later (Shubik and Della Porta, 1957).

Administration i.p. to mice (unspecified strain) of anthracene at a dose of 1,000 mg/kg was reported in an old study, cited in a secondary reference, to cause a reduction of the growth rate from 4.7 g/day to 2.8 g/day (Elson et al., 1945).

In a study of the mechanism of control of cytochrome P450 expression, a single intraperitoneal injection of 300 mg/kg bw anthracene (dissolved in corn oil) to B6C3F1 mice was found to cause a 10-fold increase in the activity of hepatic microsomal methoxyresofurin O-deethylase (a cytochrome P4501a2-dependent activity) 24 hours later (Chaloupka et al., 1994). It also caused an increase in the mRNA levels of cytochrome P4501A2 by an Ah receptor-independent mechanism. No specific toxicological significance can be attached to these observations.

Other routes

According to a secondary report, 0.5 mg anthracene injected subcutaneously into rats decreased the anti-oxidative activity of the pancreas during the 25 days after injection. Pancreatic insular cells showed increases in the cell, nucleus, and nucleolus size (Clayton and Clayton, 1981).

In a test meant to compare the ability of carcinogenic and non-carcinogenic PAHs to affect the calcium ionophore A23187-induced activation of washed rabbit platelets, measured as biosynthesis of thromboxane B2, anthracene was reported to have an enhancing effect. Other (carcinogenic) PAH's (benz[a]anthracene, chrysene, benzo[a]pyrene, and benzo[ghi]perylene) had an inhibitory effect (Yamazaki et al., 1990).

4.1.2.2.2 Studies in humans

According to a report in a secondary source, which gives no other details, the acute symptoms of anthracene exposure include irritation of the upper airways, lacrymation, photophobia, oedema of the eyelids and conjuctival hyperemia (Volkova, 1983). Other effects described, which could not be associated with a specific route of anthracene exposure, include headache, nausea, loss of appetite, inflammation of the gastrointestinal tract, slow reactions and weakness. These symptoms are said to disappear within several days after cessation of contact.

No specific data are available on the acute toxicity of anthracene to humans.

4.1.2.2.3 Summary

The acute toxicity of anthracene after oral, dermal and i.p. administration is low. Its oral LD_{50} in the rat is 8.12 g/kg, while its dermal LD_{50} was greater than 1,320 mg/kg in the rat and greater than 4 g/kg in the rabbit. No tissue-specific acute toxic effects have been reported. No data on acute toxicity in humans are available.

4.1.2.3 Corrosivity and irritation

Anthracene is known to cause phototoxicity (photoirritation) in the presence of UV radiation (see Section 4.1.2.5). In this section reference is made to studies on the irritating potential of anthracene in the absence of UV radiation.

4.1.2.3.1 Studies in animals

Skin irritation

The induction of primary skin irritation by anthracene was tested in accordance with a method specified in the US Code of Federal Regulations (Title 16, Section 1500.41). To a 1 sq. inch area of intact or abraded skin (clipped free of hair) of 6 albino rabbits, 0.5 g of anthracene (reinst = purest), in the form of a 10% suspension in 0.5% carboxymethlcellulose, was applied under an occlusive gauze for 24 hours (Grote, 1979b). The skin condition was assessed for erythema/eschar and oedema formation, using the standard Draize scale, at the time of removal of the gauze as well as 48 hours later. The values for erythema/eschar formation at the two observation times for intact skin were added to those for abraded skin (4 values), as were the values for oedema formation at the two observation times for intact skin (4 values). The total of the 8 values was divided by 4 to give the primary irritation score. Very slight erythema and/or oedema was observed in five of the six rabbits, giving an overall irritation score of 0.79 and leading to its characterisation as “slightly irritating” according to the criteria of the method used. For classification as a skin irritant under present guidelines a score of 2 would be required. On the other hand, this testing method differs from that of Annex V of Dir. 92/69/EEC in a number of points, the most important of which is that it made use of a diluted formulation, rather than the pure substance.

In a study already described (see Section 4.1.2.2.1), anthracene (purity ’Anthrazen reinst’, 0.4 g/ml in polyethylene glycol), was applied in the form of an occlusive patch (5 · 7.5 cm) to the shaved skin of Wistar rats (5 male, 5 female) for 24 hours, at a mean dose of 300 mg (8 mg/cm^2), and the animals observed for 14 days (Worstmann, 1981). No erythema or oedema was observed at any time. The low dose employed in this study is noted.

In a short note, a substance described as “anthracene residues” was reported to have been applied to the inner surface of the ear of white New Zealand rabbits (one male and one female, 500 mg/animal), using an occlusive gauze, for 24 hours. Examination 7-days post exposure did not reveal evidence of “irritating activity” (Thyssen J, 1979). Although this report refers to using this test to look for irritating activity, it is noted that this method (as well as the analogous “Mouse Ear Swelling Method”) is normally used as a test for sensitisation. Furthermore, the “anthracene residues” employed in this study consisted of wastes of unknown composition from anthracene production (Höke, 2002). Therefore no conclusions regarding anthracene can be drawn from the study.

Recrystallised anthracene was applied to the skin of the ear of MNRI mice which were examined for evidence of irritation 24 hours later. The ID50 (dose causing irritation to 50% of the animals) was found to be 118 µg per ear (corresponding to 4.7 mg/kg or, assuming 1 cm^2 as the area of the treated area, 118 µg/cm^2) (Brune et al, 1978). No further information on the test method is given.

A quantity of 0.01 ml of 25% (w/v) suspension (described as “poor”) of anthracene in corn oil (2.5 mg) was applied to unclipped, uncovered intact belly skin of 5 rabbits (Mellon Institute, 1977). No irritation was reported to be present 24 hours later, but a marginal effect (“moderate capillary injection”) was found in one animal. No further details are given in the report. The low dose employed is noted.

Eye irritation

Eye irritation was tested in accordance with a method specified in the US Code of Federal Regulations, Title 16, Section 1500.42. Anthracene (reinst = purest) was administered at 100 mg

per animal to the conjunctival sac of six albino rabbits, and scoring for irritation according to the standard Draize criteria was done after 24, 48 and 72 hours. No effects on the cornea or the iris were observed in any of the tested animals. Slight to moderate redness of the conjunctiva appeared in 4 out of the 6 rabbits, while slightly increased secretion appeared in 1 animal, giving a total Draize irritation score of 1.0, leading to its characterisation as "non-irritant" according to the criteria of the method used. (Grote, 1979c). This test broadly fulfils the criteria of the Annex V of Dir. 92/69/EEC test for eye irritation, and leads to the conclusion that anthracene would be classified as "non-irritant" by the criteria of this method.

In a short note, a substance described as "anthracene residues" was reported to have been administered at 50 mg per animal to the conjunctival sac of white New Zealand rabbits (one male and one female) and the animals observed for 7 days. No evidence of eye irritation was found (Thyssen, 1979). For reasons given above (under Skin Irritation), no conclusions regarding anthracene can be drawn from this study.

Anthracene was instilled into the conjunctival sac of 5 rabbits, as powder (40 mg) or as a 25% suspension in corn oil (0.5 mg anthracene per eye) (Mellon Institute, 1977). Examination of the unstained eyes immediately, and with staining with 5% fluorescein 24 hours after later, did not reveal any corneal injury. No further details of the test procedure or the clinical findings are given in this report.

Corrosivity

The animal studies for skin irritation described above do not provide any evidence to suggest that anthracene is corrosive to the skin or eyes.

4.1.2.3.2 Studies in humans

According to secondary references, which give no further specific information, anthracene is a primary irritant, causing "possible mild irritation of skin, eyes, mucous membranes and the respiratory tract after exposure to anthracene fumes or dust during labour" (Montizaan et al., 1989) and irritation of the upper airways, lacrymation, edema of the eyelids and conjuctival hyperemia (Volkova, 1983). No conclusions can be drawn from these reports in view of their uninformative nature and the fact that they are based on observations in occupational settings which probably involve exposure to complex mixtures.

Skin disorders related to irritation and sensitisation ("occupational skin burns") are relatively common among workers exposed to coal tar and related products (Emmett, 1986; Riala et al., 1998). However, no studies specifically linking anthracene to these effects have been reported.

4.1.2.3.3 Summary

Anthracene has not been tested for skin irritating activity by a method conforming to the criteria of Annex V of Dir. 92/69/EEC. At a dose of 500 mg (as a 10% suspension) it caused slight erythema and oedema to the skin of rabbits. On the other hand, when applied on the skin of rats at a mean dose of 300 mg (as a 40% formulation) for 24 hours, no evidence of skin irritation was observed up to 14 days later. Finally, a report that at a dose of 118 $\mu g/cm^2$ it caused irritation to the skin of the ear of 50% of mice cannot be evaluated because of limited reporting. Although these reports do not provide convincing evidence that anthracene has skin irritating activity in the absence of UV light, none strictly fulfils the criteria of Annex V of Dir. 92/69/EEC. On the

other hand, in view of the strong skin phototoxicity potential of anthracene (see Section 4.1.2.5 below), and the proposal that it be classified as a skin irritant on this basis, no further testing of its skin irritating activity in the absence of UV light seems necessary.

Anthracene was negative in a test for eye irritation that closely resembled the corresponding method of Annex V of Dir. 92/69/EEC.

In view of the lack of evidence that anthracene causes skin irritation in the absence of light, no specific recommendation for further studies to examine the induction of lung irritation are necessary.

4.1.2.4 Sensitisation

4.1.2.4.1 Studies in animals

The ability to anthracene to induce contact sensitivity was tested by immunising adult female Hartley guinea pigs on each front foot pad with 125 µg of anthracene in the form of a 1:1 emulsion of a saline solution and complete Freund's adjuvant (Old et al., 1963). Two to three weeks later, each animal was tested for contact sensitivity by applying one drop of a serial two-fold dilution of anthracene (1 – 0.001%) dissolved in an acetone-olive oil mixture to the shaved ventral or dorsal skin. Skin induration and erythyma were examined 24 hours later. Anthracene was negative in this test, in contrast to the (carcinogenic) PAH benzo[a]pyrene, 3-methylcholanthrene and dimethylbenzanthracene which were positive.

4.1.2.4.2 Studies in humans

No information on the sensitising effects of anthracene (in the absence of light) in humans was found. Skin disorders related to irritation and sensitisation ("occupational skin burns") are relatively common among workers exposed to coal tar and related products (Emmett, 1986; Riala et al., 1998). However, no studies specifically linking anthracene to these effects have been reported.

4.1.2.4.3 Summary

There are no studies in humasn on the skin sensitisation potential of anthracene.

A limited report on a test for sensitising activity of anthacene in rabbits was negative. Anthracene has not been tested in animals for skin sensitising activity in accordance with Annex V of Dir. 92/69/EEC. On the other hand, in view of the strong skin phototoxicity potential of anthracene (see Section 4.1.2.5), and the proposal that it be classified as a skin irritant on this basis, no further testing of its skin sensitising activity in the absence of UV light seems necessary.

4.1.2.5 Phototoxicity

Under Commission Directive 2000/33/EC (27th adaptation to technical progress of Council Directive 67/548/EEC), Annex II ("B.41. Phototoxicity – *In vitro* 3T3 NRU phototoxicity text"), photoxicity is defined as a toxic response that is elicited after the first exposure of skin to certain

chemicals and subsequent exposure to light, or that is induced similarly by skin irradiation after systemic administration of a chemical. Photoirritation is defined in the same text as referring only to those phototoxic reactions which are produced at the skin after exposure to chemicals (topically or orally). These phototoxic reactions lead always to non-specific cell damage (sunburn like reactions). Finally, photoallergy is defined as an acquired immunological reactivity which does not occur on first treatment with chemical and light, and needs an induction period of one or two weeks before skin reactivity can be demonstrated.

Anthracene is a photodynamic compound which, in the presence of UV radiation, generates reactive oxygen species (singlet oxygen, superoxide anion) (Joshi and Pathak, 1984) and can elicit toxic effects. A possible mechanistic basis for anthracene's phototoxic effects may be related to its light-mediated interaction with, and modification of, cellular constituents.

Anthracene in combination with UV light has been shown to damage DNA, proteins and lipids. Although oxygen is believed to be important for the phototoxic effects of anthracene, there is also evidence that anthracene can also induce oxygen-independent effects on cellular components. Thus, irradiation of ^{14}C-labelled anthracene with UV light (wavelength > 292 nm) in the presence of calf-thymus DNA led to covalent binding of radioactivity to the DNA, which was not dependent on the presence of oxygen (Sinha and Chignell, 1983). The photoinduced covalent binding of anthracene to DNA *in vitro* and in monkey kidney and human skin epithelial cells in culture has also been demonstrated by the studies of Blackburn et al. (Blackburn et al., 1973; Blackburn and Tausig, 1975). On the other hand, the decrease of the thermal denaturation temperature of calf thymus DNA and the nicking of a circular plasmid (both reflecting induction of DNA strand breaks) by anthracene plus UV light were oxygen dependent, implying the involvement of reactive oxygen species.

A similar oxygen dependence was observed for the UV-induced covalent binding of anthracene to human serum albumin and the accompanying crosslinking of the same protein (Sinha and Chignell, 1983). Glutathione (a quencher of reactive oxygen species as well as electrophilic reactive intermediates) inhibited all the above effects regardless of the dependence or not on oxygen. Irradiation with light of wavelength > 320 nm of a solution containing a mixture of 19 aminoacids in the presence of anthracene was shown to cause modifications only of tryptophan (Schothorst et al., 1979). Similar treatment of a glutathione solution led to loss of -SH groups which, at low anthracene concentrations, was accompanied by formation of the glutathione dimer. The significance of such phenomena lies in the possibility that modification of proteins or peptides may give rise to the induction of an immune response or other toxic effects.

Finally, anthracene plus UV light cause lipid peroxidation in liposomes derived from rat liver microsomes (Sinha and Chignell, 1983). This oxidation reaction was not significantly inhibited by superoxide dismutase or catalase alone or in combination, suggesting that this reaction was not mediated by superoxide, hydroxide radicals or hydrogen peroxide.

4.1.2.5.1 Studies in animals and in *in vitro* cell cultures

In the context of a photocarcinogenicity study, Skh-1 hairless mice treated on their skin (size of painted area not specified) with 40 µl of a 0.01% solution of anthracene in methanol (approximately 4 µg anthracene) and subsequently irradiated with UV radiation (intensity and duration not specified) showed more severe skin inflammation than animals treated only with methanol and UV (Forbes et al., 1976).

Skh-1 hairless mice received a topical application to their backs (size of painted area not specified) of 20 µl of a 0.025% solution of anthracene in 20% pyrrolidone/ isopropyl alcohol 20:80 (approximately 5 µg anthracene), followed by topical irradiation with UV light (320-400 nm) for periods ranging from 100 to 2,000 seconds (total radiance delivered 1.3-26 kJ/m^2) (Argenbright et al., 1980a). A rapid and substantial onset of hyperemia was observed which was not accompanied by any change in permeability of vascular walls to plasma albumin. Hyperemia was prevented by the simultaneous application of histamine H1 and H2 receptor blockers, suggesting that it resulted from primary injury of dermal mast cells and consequent release of histamine which in turn acted to bring about vasodilation.

Analogous results were obtained in 9 white pigs (males and females, 9-15 kg) treated with 12.5 µg anthracene (50 µl of a 0.025% solution in pyrrolidone/ isopropanol 20:80) painted on an area of 6 cm^2. Forty-five minutes later the animals were exposed for 100 – 2,000 sec to UV radiation, receiving 2.6-52 kJ/m^2 of total radiance. Minimal erythema was observed within 2 minutes of irradiation and increased with radiation dose (no more information is given). A radiation dose-related increase of vascular permeability was also observed, which was mediated by histamine and serotonin receptors (Argenbright et al., 1980b).

The possibility that anthracene plus UV light may damage mast cell membranes, thus causing the release of inflammation-mediators, is supported by the observation that anthracene accumulates in the lysosomes of animal and human endothelial cells (Alison et al., 1966). It has been postulated that such accumulation may result, upon photoactivation, in membrane damage permitting the leakage of lytic enzymes and other chemicals which can initiate the inflammatory cascade. The ability of anthracene to cause light-mediated membrane damage is further indicated by its ability to induce photohemolysis (light-mediated lysis of erythrocytes) *in vitro*.

Hairless mice were treated on the skin with anthracene (2 applications of saturated solution, solvent and concentration unspecified) followed by 48 hours of continuous UV irradiation (intensity and wavelength unspecified) (Gloxhuber, 1970). Intense erythema was observed. No erythema was observed when the anthracene (100 mg/kg) was administered i.p., followed by 48 hours of UV irradiation. This finding is in contrast to that described in an abstract by Dayhaw-Barker et al. (1985), according to which oral administration of anthracene (50 mg/ml in corn oil) to mice by gavage, followed by UV irradiation of the skin for 1 hour, resulted in keratitis of the exposed skin. This effect was said to be less pronounced in animals receiving only UV radiation and absent from animals receiving only solvent.

The induction of erythema by combined treatment with anthracene and UV radiation, and the corresponding action spectrum, were investigated in guinea pigs by Kochevar et al. (1982). Groups of 6 Hartley strain female albino guinea pigs had 0.1 ml of a solution of anthracene in methanol (concentrations ranging 0.005 - 5 mM) applied to a site (2.5 · 2.5 cm) on their shaved and depilated back. Thirty minutes later the animals were irradiated with 20-40 W/m^2 UV irradiation (320 - 400 nm), receiving a total of 100 KJ/m^2 of radiance (the duration of UV exposure can be calculated as approximately 40-80 minutes). Twenty hours after irradiation, a dose-related increase in erythema was observed in all animals receiving both anthracene and UV irradiation (but not in any of those receiving only one of the two stimuli). Animals which had received 0.005 mM (corresponding to 14 ng/cm^2) anthracene showed no erythema, 0.05 mM being the lowest dose causing an effect. The action spectrum was found to broadly match the absorption spectrum of anthracene, showing activity in the range 340-380 nm and a maximum around 360 nm, as observed in corresponding studies with humans (Kaidbey and Nonaka, 1984). Thus for the induction of skin irritation by the combination of anthracene and 100 kJ/m^2 of UV light in the guinea pig the NOAEL is 14 ng/cm^2 and the LOAEL is 140 ng/cm^2

In another study, groups of 10 female guinea-pigs (Colworth Dunkin-Lartley) were treated on the hair-clipped dorsal skin with anthracene (10 µl of 0.01% solution in ethanol on a 14-mm diameter area; corresponding to 0.65 µg/cm^2) (Lovell and Sanders, 1992). Thirty minutes later they received a dose of 150 kJ/m^2 UVA (313-400 nm). Skin irritation symptoms were already observed during the irradiation period and were maximal at the first (4 hour post-irradiation) observation point, where a mean erythema score of 5.5 was obtained (score 4 corresponded to slight erythema and score 6 to definite erythema). The effects decreased thereafter, but were still detectible (score of 0.5; score 2 corresponding to faint trace of erythema) after 72 hours. A score of 0.1 was noted 4 hours after application of anthracene without irradiation and faded completely by 24 hours. Varying the UVA dose between 75 kJ/m^2 and 200 kJ/m^2 had no effect on the level of symptoms at 4 hours, but did affect slightly their rate of disappearance. No change in the effects was observed when the ethanol was replaced by acetone or dimethylacetamide-acetone-alcohol as a solvent. Varying the applied dose of anthracene between 0.001% and 1% indicated a minimum photoirritant concentration (at 150 kJ/m^2) of 0.003% (220 ng/cm^2), in broad agreement with the findings of the study of Kochevar et al. (1982).

Burnham and Rahman (1992) treated female C3H/HeN mice on their shaved dorsal skin (approximately 1 cm^2), or a similar area of cultured skin *in vitro*, with 25 µl of a solution of anthracene in 1:1 acetone/olive oil (5 µg/ml), corresponding to 125 ng/cm^2, for 2 hours. Subsequently they were received a dose of 20 kJ/m^2 UVA (365$\pm$10 nm) at 11 J/m^2/sec (over a 30 minute period). Forty hours later, the number of IA^k-expressing Langerhans cells as well as IA^k-negative but Thy-1-positive dendritic cells were enumerated by immunocytostaining and found to be significantly decreased. Depletion of these cells can result in a decreased immune response. No analogous effect was observed with a 10fold lower anthracene dose.

4.1.2.5.2 Studies in humans

According to a report (in a secondary source) which gives no other specific information, the phototoxic effects of anthracene in humans include acute dermatitis with symptoms of burning, itching and edema which are more pronounced in the exposed bare skin regions (Volkova, 1983). Prolonged exposure is said to give rise to pigmentation of the bare skin regions, cornification of its surface layers and telangioectasis. No conclusions can be drawn from this unspecific and limited report.

In 3 human volunteers, a 2% solution of anthracene in benzene was applied twice daily for 2 days on the skin of the forearms, prior to irradiation with long-wavelength UV light (340-380 nm). Urticarial reactions and burning were observed in all 3 subjects, symptoms which persisted for several days, with subsequent pigmentation. In 1 subject erythema also appeared, which persisted for a few days (Crow et al., 1961).

Kaidbey and Nonaka (1984) investigated the action spectrum and UV dose-response relationships governing the induction by anthracene of photoirritation in human volunteers. They applied 10 µl/cm^2 of a 0.25% solution of anthracene (at least 99% pure) (i.e. 25 µg anthracene/cm^2) in equal parts of 95% ethanol and benzene on the untanned backs of 6 Caucasian males of fair complexion. The application sites were covered with non-absorbent cotton cloth for 2 hours, and then uncovered again and allowed to air dry for 15 minutes. Subsequently they were irradiated with UV light of selected wavelength ranges (half-band width 6.6 nm) for different times. The threshold dose of radiation needed for the induction at each wavelength of a) immediate erythema, i.e. erythema localised to the area of exposure, appearing within a few minutes after irradiation and fading after 15 minutes, b) delayed erythema present 22-24 hours after irradiation and c) a weal-and-flare reaction appearing 5-10 minutes after

irradiation, were assessed. The effective wavelengths were in the range 340-380 nm, with maximum activity appearing at 360 nm for all three end-points, an action spectrum paralleling the absorption spectrum of anthracene. Of the three end-points examined, that appearing at the lowest radiation intensity was immediate, transient erythema (mean threshold dose 1.0 $\pm$ 0.6 kJ/m^2), followed by delayed erythema (mean threshold dose 1.9 $\pm$ 1.0 kJ/m^2), and by appearance of flare and wealing (mean threshold dose 2.8 $\pm$ 1.9 kJ/m^2). The observation of different thresholds for the various end-points suggests the existence of different cellular and molecular targets. It has been suggested that the flare and wealing effects may be related mainly to the release of inflammatory mediators from mast cells, as suggested by the fact these effects, but not immediate erythema, were prevented by prior injection of codeine, a mast cell degranulating agent.

A therapeutic use of anthracene phototoxicity to treat psoriasis has been reported in a conference abstract (Rispler and Urbanek, 1978). Forty µl of a 0.025% solution of anthracene (10 µg anthracene; solvent and size of treated area unspecified) were applied to the psoriatic plaques of patients, who were irradiated 1 hour later with long wavelength UV light or natural mid-day sunlight. Phototoxicity was induced by irradiation of the treated area with 0.1-1 J long wavelength UV (which gave rise to erythema and a stinging sensation). After 20-40 treatments the lesions were cleared for up to 1 year, with very little hyperpigmentation of the treated areas. While demonstrating a phototoxic effect of anthracene, this study gives no information on its ability to induce skin photoirritation or photosensitisation.

4.1.2.5.3 Summary

There are no standard validated methods for the assessment of phototoxicity *in vivo*. A standardised *in vitro* method for the assessment of phototoxicity has been defined under Annex II of Commission Directive 2000/33/EC, and added to Annex V of Commission Directive 67/548/EEC, based on the assessment of *in vitro* cytotoxicity in the presence of UV light. This method has not been employed to examine the phototoxicity of anthracene. However, Studies in humans, mice and guineapigs have amply demonstrated that, in combination with long-wavelength UV light (340 - 380 nm), anthracene can lead to photoirritation as defined under Commission Directive 2000/33/EC, with symptoms, in decreasing order of ease of appearance, of transient erythema, delayed erythema and flare-and-wealing. In guinea pigs, the highest amount of anthracene applied to the skin which, in combination with 100 kJ/m^2, did not give rise to erythema (NOAEL) was 14 ng/cm^2, while the corresponding LOAEL was 140 ng/cm^2.

In humans, a single skin application of 25 µg anthracene/cm^2 was sufficient to cause skin irritation after exposure to UVA radiation in the range 1-2.8 kJ/m^2 (depending on the wavelength). With the radiant flux of the global solar radiation (defined as the total solar radiation reaching the earth's surface (WHO, 1994) in the mid- or southern European region being of the order of 800-1,000 W/m^2, and with UVA making up approximately 5-6% of solar radiation (i.e. 40-60 W/m^2), a person exposed to natural sunlight can receive 1 kJ/m^2 of UVA within a period of seconds to minutes (1 J = 1 W.sec) (Moseley et al., 1981). Because 25 µg/cm^2 was the only dose applied in the corresponding study, it is concluded that this corresponds to the human LOAEL, although, in view of the findings in guinea pigs, it effects in humans may occur at substantially lower levels. Consequently the animal LOAEL (140 ng/cm^2) and NOAEL (14 ng/cm^2) will also be considered in Risk Characterisation.

4.1.2.6 Repeated dose toxicity

4.1.2.6.1 Studies in animals

Inhalation

In a report in secondary source, which gives no other specific information, it is mentioned that chronic inhalation of anthracene aerosol by albino rats, at concentrations of 0.05 and 0.01 mg/l, is associated with a reduced gain in body weight and blood changes (decrease in hemoglobin, reticulocytosis, leukopenia, and increase in residual blood nitrogen) (Volkova, 1983). The same blood changes were observed after intragastric administration. No conclusions can be drawn from this report in view of its uninformative nature.

Oral

In a study mentioned in a secondary reference, repeated intragastric exposure of rats with anthracene at unspecified doses and duration was said to produce a decrease in hemoglobin, reticulosis, leukopenia and an increase in residual blood nitrogen (Volkova, 1983).

In a study on enzyme induction by polycyclic aromatic hydrocarbons, intragastric administration of anthracene (100 mg/kg/day) to rats for 4 days was found to induce an increase in microsomal carboxylesterase activity in the gastrointestinal mucosa but not in the kidney (Nousiainen et al., 1984), and a slight increase in cytosolic aldehyde dehydrogenase activity in the liver (Torronen et al., 1981). These changes do not have any obvious toxicological significance.

In a detailed study, conducted under GLP conditions for the US EPA (US EPA, 1989), anthracene dissolved in corn oil was administered to groups of 20 male and female CD-1 (ICR)BR mice by oral gavage, at doses of 0, 250, 500, and 1,000 mg/kg/day for 13 weeks. Mortality, clinical signs, body weights, food consumption, ophthalmology findings, hematology and clinical chemistry results, organ weights, organ-to-body weight ratios, gross pathology, and histopathology findings were evaluated. No significant treatment-related effects on any of these parameters were noted. A statistically significant increase in mean ovary weight (absolute and relative to terminal body weight) for the group receiving 500 mg/kg/day (but not any of the other doses), as well as non-dose-related changes in serum globulin and total protein concentrations, and numbers of segmented neutrophils, were considered incidental and of no pathological significance.

In a chronic bioassay, a group of 28 BD I and BD III rats received anthracene in the diet, starting when the rats were approximately 100 days old (Schmahl, 1955). The daily dosage was initially 5 mg/rat, later increased to 15 mg/rat (corresponding to 17-50 mg/kg/day, assuming average animal weight of 300 g), and the experiment was terminated on the 550th experimental day, when a total dose of 4.5 g/rat had been achieved. The rats were observed until they died, with some living more than 1,000 days. No treatment-related effects on lifespan or gross and histological appearance of tissues were observed. No data on a control group were reported, body weights were not mentioned, and hematological parameters were not measured, making the standard of this old study lower than would be desirable.

Diet containing 1 mg/kg anthracene was fed ad libitum to a group of 7 Swiss mice (unspecified sex) for 17 days (Rigdon and Giannukos, 1964). Subsequently, the anthracene content of the diet was increased to 5 mg/kg for days 18-24 and then to 25 mg/kg for days 25-32. The daily intakes of anthracene during these periods were approximately 150 mg/kg/day (day 1-17),

750 mg/kg/day (day 18-24) and 3,750 mg/kg/day (day 25-32). A control group was concurrently fed the same diet without anthracene. The anthracene-treated group had a slightly higher food consumption than in the controls and showed a correspondingly increased gain in weight. Histological examination of the kidneys and the liver did not reveal any significant anthracene-induced changes.

No effect on liver regeneration was observed in partially hepatectomised rats fed anthracene (514 mg/kg bw per day) in the diet for 10 days (Gershbein, 1975).

Other routes

Daily i.p. administration of anthracene (28.5 mg/kg/day; in corn oil) for 14 days to B6C3F1 mice did not significantly affect their immune response as indicated by their antibody-forming cell response to sheep erythrocytes (White et al., 1985).

An old study mentioned in a secondary reference indicates that treatment-related lymphoid effects, including increases in reticulum cells, accumulation of iron, decreased lymphoid cells and dilated lymph sinuses were seen in albino mice receiving weekly subcutaneous injections of a 0.05% colloidal solution of anthracene in gelatine for 40 weeks (Hoch-Ligeti, 1941).

4.1.2.6.2 Studies in humans

No information on the repeated dose toxicity of anthracene in humans is available. It seems likely that the “acute effects” discussed in Section 4.1.2.2.2 refer also to effects seen after repeated human exposure.

4.1.2.6.3 Summary

According to an old and rather poorly described study, administration of anthracene to rats in the diet for up to 550 days, at a daily dose of up to 50 mg/kg, did not reveal any adverse effects. On the other hand, a well-conducted study showed that daily administration of anthracene by gavage to mice for at least 90 days, at doses up to 1,000 mg/kg/day, did not result in any treatment-related effects of toxicological significance (NOEL).

There is lack of information on the effects on animals or humans of anthracene after inhalation is available. However, in view of the absence of any toxic effects after a 90-day oral exposure study, the generally low toxicity of anthracene, and the low levels of human inhalation exposure (see Section 4.1.3, Risk Characterisation), it is not considered that an inhalation study is justified.

4.1.2.7 Genetic toxicity

The genetic toxicity of anthracene has been examined in a large number of studies, too numerous to discuss individually. In the following discussion, only representative negative studies and rare studies reporting positive results are discussed, where appropriate, and the overall conclusions presented.

As is the case with other studies with anthracene, the use of adequate protection from UV radiation is not always described in the reports examined. No reports specifically intended to assess the genotoxicity of photodegradation products of anthracene have been found. However, as has already been mentioned (see Section 4.1.2.5.1), in the presence of UV radiation

anthracene can form DNA adducts *in vitro* as well as in monkey kidney and human skin epithelial cells in culture (Sinha and Chignell, 1983; Blackburn et al., 1973; Blackburn and Tausig, 1975). On the other hand, no results on the induction of anthracene-related DNA damage in the presence of UV *in vivo*, or of the induction of genetic end-points other than DNA adducts (e.g. gene or chromosome mutations) in cell cultures have been reported.

4.1.2.7.1 *In vitro* studies

Studies in bacteria and lower eukaryotes

Anthracene has been tested for the induction of genotoxicity (DNA damage and mutations) in a large number of bacterial systems, including Escherichia coli, Salmonella typhimurium and Bacillus subtilis, with and without metabolic activation, giving negative results in the great majority of cases. Results on its testing for mutagenicity in Salmonella typhimurium has been described in tens of reports, involving use of strains TA1535, TA1536, TA1537, TA1538, TA97, TA98 and TA100, without or with S9 metabolic systems from the liver of rats and guinea pigs subjected to various enzyme-inducing pre-treatments (no reports of genotoxicity testing using metabolic systems other than S9 have been traced). Almost invariably, clearly negative results were reported in there studies. For example, it was tested in Salmonella typhimurium TA1535, TA1538, TA98 and TA100 with S9 extract from Arochlor-induced rat liver, with negative results (Purchase et al., 1976). Rare reports of positive mutagenic activity in Salmonella typhimurium were based on marginal activity observed in TA97 or TA100 with metabolic activation (less than 3fold increase over spontaneous mutation frequency – the use of UV protection was not mentioned) (Sakai et al., 1985; Carver et al., 1986).

Anthracene was also negative in a large number of tests for the induction of gene mutations or cytogenetic damage in Saccharomyces cerevisiae and Saccharomyces pombe.

Studies in mammalian cells

Anthracene did not induce DNA damage in human peripheral blood leukocytes *in vitro*, without metabolic activation. Also, it failed in a large number of studies to induce unscheduled DNA synthesis in primary rat hepatocytes (Williams, 1988), Chinese hamster ovary cells, human HeLa cells with metabolic activation (Martin et al., 1978), while it gave a marginally positive, non-dose-related response in primary human skin epithelial cells (the use of UV protection was not mentioned) (Lake at al., 1978).

It was negative for the induction of gene mutations in a large number of studies, including studies using Chinese hamster ovary cells and human lymphoblast cells. In mutagenesis studies with mouse lymphoma L5178/TK+/-cells it was negative in most cases. However, in one study it gave weak mutagenicity when tested (with protection from UV) in the presence of S9 extract from C57Bl/6J mouse liver (but not with 6 other types of S9 extract) (Amacher and Turner, 1980).

Anthracene has been tested for the induction of sister chromatid exchanges in Chinese hamster ovary cells with metabolic activation, in a rat liver epithelial cell line and in a combined *in vitro*/*in vivo* test using Chinese hamster V79 cells implanted into mice. All studies reported negative results except for one which was marginally positive (Perry and Thomson, 1981).

Anthracene was negative for the induction of chromosomal aberrations in one study with Chinese hamster ovary cells and one with rat liver RLL cells. However, it was reported to give a

positive result using Chinese hamster ovary cells with (but not without) metabolic activation when tested at concentrations up to 0.02 mg/ml (Sofuni et al., 1985).

In tests for the induction of cell transformation *in vitro*, sometimes followed by examination of the *in vivo* growth potential of the transformed foci obtained, anthracene was reported to be negative in over one dozen studies. For example, it was clearly negative for the induction of morphologically transformed foci of cells capable of forming a tumour after s.c. injection into syngeneic animals when tested up to a concentration of 10 µg/ml in mouse BALB/3T3 cells (DiPaolo et al., 1972), and in the induction of transformed foci in Syrian hamster embryo cells after exposure for 7 days at 50 µg/ml (LeBoeuf et al., 1996). However, it was reported positive in a study with Syrian hamster kidney cells BHK 21 C13/HRC 1, with and without metabolic activation (the use of UV protection was not mentioned) (Purchase et al., 1976). It was also reported to be positive in an assay measuring the acquisition of attachment independence of Rauscher leukemia virus-infected rat embryo cells (2FR450) (Traul et al., 1981). Finally, in a test involving treatment with anthracene (at concentration 0.001-50 µg/ml) of Fischer rat embryonal cell line F1706 P88, infected with Rauscher leukemia virus, followed by inoculation of transformed cells into newborn Fischer rats and analysis of tumour induction, an ambiguous result was obtained (marginally positive in one experiment but negative in a subsequent one). The use of UV protection was not mentioned (Freeman et al., 1973)

4.1.2.7.2 *In vivo* studies

Studies in animals

Anthracene did not induce sex-linked recessive lethal mutations in Drosophila melanogaster. However, it tested positive in the wing somatic mutation and recombination test (SMART), causing the appearance of small single spots in a high bioactivation (but not in the normal) strain of insects (no information on the use of light protection is given) (Delgado-Rodriguez et al., 1995).

All *in vivo* tests for genotoxicity of anthracene in mammals have given negative results. It has been tested with negative results for the induction of unscheduled DNA synthesis in various tissues (including liver, kidney and testes) of male C57Bl mice after i.p. doses up to 125 mg/kg (Friedman and Straub, 1976). It was also negative for the induction of mouse micronuclei in bone marrow 96 hours after a single dose of 344 mg/kg (Salamone, 1981) as well in peripheral blood erythrocytes 24 hours after 4 daily administrations of up to 2,500 mg/kg/day (Oshiro et al., 1992). Administration of 2 i.p. doses of 450 mg/kg to mice and Chinese hamsters gave no increase in the frequency of sister-chromatic exchanges and chromosome aberrations in bone marrow cells (Roszinsky-Koecher et al., 1979).

Anthracene was also negative for the induction of transformed cell colonies in an *in vivo*-*in vitro* test system involving i.p. administration (10-30 mg/kg) to pregnant Syrian golden hamsters on days 10-11 of gestation, excision of embryos 2-3 days later and *in vitro* cultivation of embryo cells (DiPaolo et al., 1973). A number of carcinogenic compounds tested in parallel were positive in this system.

No DNA adducts could be detected in mouse skin, using the sensitive ^{32}P-postlabelling assay, after 4 applications (at 0, 6, 30 and 54 hours) of 0.21 mg anthracene (dissolved in acetone) to the skin of Balb/c mice, followed by sacrifice 24 hours later (no information on the use of light protection is given) (Reddy et al., 1984).

It is recalled that one study, discussed in Section 4.1.2.1.1, reported detecting the formation of 9,10-dimethylanthracene, a weakly genotoxic (Fujikawa et al., 1993; Spano et al., 2001) and carcinogenic (LaVoie et al., 1985) compound, in rat skin in contact with anthracene after subcutaneous application, as well as after *in vitro* incubation of anthracene with an extract of rat liver fortified with the methylating agent S-adenosylmethionine (Myers et al., 1988).

Studies in humans

No data on the genotoxicity of anthracene in humans exist.

4.1.2.7.3 Summary

The genotoxicity of anthracene has been examined in a large number of studies. The great majority of these studies, which involved examination of its ability to induce DNA damage, point mutations, chromosome aberrations, sister chromatid exchanges and morphological cell transformation, using systems of varying complexity ranging from bacteria *in vitro*, through host-mediated bacterial studies, lower eukaryotes, and mammalian cells *in vitro*, to *in vivo* rodent studies, have negative results. Many of the genotoxicity tests involving anthracene were conducted in the context of inter-laboratory and assay-comparison studies, based on common, well defined protocols (e.g. Bridges et al., 1981; Brookes and Preston, 1981) and, for this reason, the trend towards negative outcomes must be considered valid. Occasional reports of marginally or inconsistent positive responses do not seem sufficient to overturn the overall conclusion that anthracene is not genotoxic. Furthermore, the consistent absence of any genotoxic activity in a range of *in vivo* tests strongly suggests that the formation of the weakly genotoxic 9,10-dimethylanthracene as a metabolite of anthracene probably does not have any significant biological consequences in terms of genotoxicity.

Limited studies indicate that, in the presence of UV light, anthracene can bind to DNA, but no *in vitro* or *in vivo* biological consequences of such binding have been demonstrated.

4.1.2.8 Carcinogenicity

4.1.2.8.1 Studies in animals

Anthracene has been examined in a large number of studies of varying design and validity. These studies have been assessed in detail by IARC (1983b). The overall conclusion of IARC was that these data provide no evidence that anthracene is carcinogenic to experimental animals and that anthracene cannot be classified as to its carcinogenicity in humans.

Inhalation

No information on the inhalation carcinogenicity of anthracene was found.

Oral

A group of 28 BDI or BDIII rats of unspecified sex, 14 weeks old, was administered in its diet initially 5 mg and later 15 mg anthracene “without impurities” per rat on 6 days per week for 78 weeks, so that the total dose received by each rat was 4.5 g (Schmahl, 1955). No control group was used. The animals were observed for life, and had a mean survival time of 700 days. One

animal developed a liver sarcoma after 18 months and another had an adenocarcinoma of the uterus after 25 months. None of these tumours was attributed by the investigators to anthracene. The absence of controls and the relatively low doses used in this old study mean that it is not adequate for the assessment of anthracene carcinogenicity.

Dermal

Anthracene has been tested for carcinogenicity by skin application in a number of studies using different types of protocols aimed at the detection of full carcinogenic potential, tumour initiating activity or photocarcinogenicity. Many of these studies are old, of poor quality and/or poorly reported.

In an early study, 100 mice of unspecified strain, sex or age had a 40% suspension of anthracene in lanolin painted on their skin (Kennaway, 1924a). No details of the purity, dose or number of applications were given. No skin tumours were found among 45 animals which survived more than 6 months.

Skin application (of unspecified number or dose) of anthracene in benzene or sesame oil on the skin of 41 albino mice of unspecified strain, sex or age did not produce any skin tumours (Pollia, 1939). Tumours were produced in a positive control group treated with 1,2,5,6-dibenzanthracene in the same experiment.

Five female Swiss mice of unspecified age received skin applications of a 10% solution of anthracene in acetone (the purity and dose of anthracene were not reported) 3 time per week for life (Wynder and Hoffman, 1959). No skin tumours were observed in any of the animals, all of whom died within 10-20 months of the start of the experiment. Benzo[a]pyrene, employed as a positive control in the same study, produced a high yield of skin papillomas and carcinomas.

The tumour initiating activity of anthracene was examined by painting on the skin of 20 "S" mice, of unspecified sex or age, 0.3 ml 0.5% anthracene (unspecified purity) in acetone twice with an interval of 30 minutes, 3 times per week, so that each animals received a total of 30 mg anthracene (Salaman and Roe, 1956). This was followed by 18 weekly skin applications of the tumour promoter croton oil in acetone, beginning 25 days after the first anthracene application, as follows: one 0.3 ml application of 0.17% croton oil, two 0.3 ml applications of 0.085% croton oil and a further 15 0.3 ml applications of 0.17% croton oil. Control animals received only the croton oil treatments. All surviving animals (17/20 anthracene-treated and 19/20 controls) were killed after the end of the croton oil treatment. No skin tumour induction by anthracene was found, as 3 of anthracene-treated animals exhibited a total of 4 skin papillomas, while among the control animals 4 had a total of 4 skin papillomas.

In another initiation-promotion study, 30 female CD-1 mice, 8 weeks old, received a single skin application of 1,782 µg anthracene (chromatographically purified) in benzene, followed 1 week later by skin application of the tumour promoter TPA (probably 5 µg and not 5 µmol as indicated in the relevant publication) 2 times per week for 34 weeks (Scribner, 1973). A control group received only TPA. At the end of treatment, 4 out of the 28 surviving anthracene-treated animals had one skin papilloma each, as did 1 of the 30 surviving controls. The author concludes that this indicates "borderline initiating activity", but no statistical analysis is included.

Anthracene has also been tested in a number of studies for skin carcinogenicity in combination with UV or visible radiation. In one such study (Miescher, 1942), 2 groups of 44 mice of unspecified strain, sex or age, received skin applications (on the back of the ears) of 5% anthracene (of unspecified purity and dose) in petroleum jelly-olive oil 3 times per week for life. One of the groups also received UV radiation (wavelength > 320 nm) for 40 or 60 min, 2 hours

after each skin application. A third group of 100 mice was treated in a similar way with anthracene but received UV irradiation for 90 min. In all groups most animals died within 7-11 months. While the group receiving the combined anthracene and UV treatment showed "broadness of the epidermis", but no skin papillomas or carcinomas were observed in either group.

In another photocarcinogenesis study (Heller, 1950), white mice were treated on the skin with 10% anthracene (of unspecified purity) in petroleum jelly-olive oil, followed by irradiation for 5 hours with UV light (wavelength 405-320 nm,) alone or in combination with visible light. No information was given on the dose of anthracene and the duration of treatment. A high incidence of skin tumours (including carcinomas) were observed within 5-8 weeks in the group receiving the combined treatment of anthracene plus UV and visible radiation, but none in those receiving either anthracene or UV radiation alone or UV plus visible radiation alone. The unusually short latency time and the poor reporting make it difficult to draw reliable conclusions from this study.

In a relatively recent, well designed and reported photocarcinogenicity study (Forbes, 1976), a group of 24 outbred Skh:hairless-1 mice (mixed males and females), 3 weeks old, were administered skin applications of 4 µg anthracene (of unspecified purity) dissolved in 40 µl methanol once per day, 5 days per week, for 38 weeks, followed after each application by 2 hours of UV irradiation (300 J/m^2, wavelength > 290 nm). Each test animal received a total of 0.76 mg anthracene. Similar groups of negative and positive controls received only methanol or 4 µg 8-methoxypsoralen, respectively, as well as the UV radiation. After 38 weeks, 20, 19 and 16 animals survived from the vehicle control, anthracene-treated and positive control groups, respectively. The times to 50% tumour incidence were 27.2 and 28.2 weeks for the negative controls and the anthracene-treated group, respectively, the difference not being statistically significant. In contrast, the time to 50% tumour incidence for the group of positive controls was significantly shorted at 20.0 weeks. The tumours observed in all groups were mainly squamous-cell carcinomas.

Subcutaneous

In an old study with a small number of animals, 10 rats (unspecified strain, sex, age and body weight) were given weekly s.c injections of 2 ml of a 0.05% suspension of anthracene (unspecified purity) in water for life (Boyland and Burrows, 1935). The maximum total dose administered was 103 mg per animal. Mortality rates were 0/10 after 6 months, 7/10 after 12 months and 8/10 after 18 months. No subcutaneous sarcomas were reported. Groups of positive controls treated in a similar way with 1,2,5,6-dibenzanthracene developed s.c. sarcomas in at least 6/10 and 9/18 rats.

In a small study of short duration, 5 Wistar rats (unspecified sex, age 6-8 weeks) were given s.c injections of 0.5 ml of a solution of anthracene (unspecified purity) in sesame oil (5 mg anthracene/injection) once per week (Pollia, 1941). At 10 months, 4 of the animals were killed. No tumours were reported, in contrast to the positive control groups of animals treated with 1,2,5,6-dibenzanthracene, where s.c tumours were seen.

To a group of 10 BDI and BDII rats (unspecified sex), 14 weeks old, s.c. injections of 1 ml of 2% highly purified anthracene in oil (unspecified type) were administered (total dose 660 mg per animal) once a week for 33 weeks, and the animals observed for life (Schmahl, 1955). Fibrosarcomas (partly with sarcomatous areas) at the site of injection were observed in 5/9 animals, with a mean latency of 26 months. No concurrent vehicle control was used. However, a group of rats similarly treated with naphthalene dissolved in oil (of unspecified type) did not develop any tumours.

Anthracene (8 mg per mouse, dissolved in refined sunflower oil) was given s.c. daily or as a single intragastric dose to BALB/C, C3H/A and C57BlxCBAF1 hybrid strains during the last week of gestation (Shabad et al., 1972). Fragments of embryonic kidney were cultured. In contrast to the control cultures, cultures from the anthracene-treated animals showed an increase in survival and hyperplastic epithelial changes. The changes seen were qualitatively similar but less strong that those produced by treatment with the carcinogen 7,12-dimethylbenz[a]anthracene. While the observed changes were considered by the authors as indicative of pre-malignancy, the lack of a consistent correlation between the effects of known carcinogens and non-carcinogenic analogues and of consistent dose-response relationships makes the significance of these effects difficult to assess.

In the assessment of carcinogenicity of anthracene after subcutaneous administration, it is relevant to recall (see Section 4.1.2.1.1) that one metabolism study demonstrated the formation of 9,10-dimethylanthracene, a weak carcinogen, after subcutaneous administration of anthracene to rats as well as during *in vitro* metabolism with a rat liver extract fortified with S-adenosylmethionine (Myers et al., 1988).

Intraperitoneal

To a group of 10 BDI and BDII rats (unspecified sex), 14 weeks old, i.p. injections of 1 ml of 2% highly purified anthracene in oil were administered (total dose 660 mg per animal) once a week for 33 weeks, and the animals observed for life, the mean survival time being over 2 years. One rat developed a spindle-cell sarcoma of the abdominal cavity. No concurrent vehicle control was used (Schmahl, 1955).

Implantation

Sixty female Osborne-Mendel rats, 3-6 months old, were given 1 pulmonary implant of 0.05 ml of 0.5 mg anthracene (unspecified purity) in 1:1 beeswax:tricaprylin. No lung tumours were observed when "nearly half" of the animals were killed after 1 year. Lung epidermoid carcinomas were observed in groups of rats treated under similar conditions with 3-methylcholanthrene (Stanton et al., 1972).

In a study that was too small to yield useful results, 9 rabbits of various breeds, ages and weights (unspecified strain, sex, age and bodyweight received an implant of a pellet of 4-20 mg anthracene (purity unspecified) into the cerebrum, cerebellum or eye. Animals died or were killed 20-54 weeks after implantation. No gliomas were found (Russel, 1947).

4.1.2.8.2 Studies in humans

Among workers handling crude anthracene (40%, no other information on composition), 3 were reported to have developed epitheliomas of the hand, cheek and wrist, respectively (Kennaway, 1924a,b). Two of these workers had been exposed for 30 and 32 years, respectively. Workers in the same factory who had contact only with purified anthracene did not develop tumours or other skin lesions.

No other information on the carcinogenicity of anthracene in humans was found.

4.1.2.8.3 Summary

Anthracene has been tested for complete carcinogenicity, tumour initiating activity and photocarcinogenicity (in combination with UV light) by various routes of administration (oral, dermal, subcutaneous, intraperitoneal, pulmonary implantation) in a large number of studies using various strains of rats and mice. Many of these studies were carried out many years ago and were of small size or poor quality. This is particularly true of the skin carcinogenicity studies other than those looking for photocarcinogenicity.

All the mouse skin application studies gave negative results for complete carcinogenicity and for tumour-initiating activity. Of 3 studies of photo-carcinogenicity, 2 (including the most recent and best conducted study) did not yield evidence of carcinogenicity, while the third, positive study was poorly reported and exhibited an unusually short tumour induction latency period.

Oral, subcutaneous and intrapulmonary administration to rats gave negative results, except for one subcutaneous injection study which showed induction of fibrosarcomas at the sight of injection. It is noted, in this context, that a metabolism study reported the formation of 9,10-dimethylanthracene, a weak carcinogen, after subcutaneous application of anthracene to rats or after *in vitro* metabolism in the presence of added S-adenosylmethionine. These observations would be compatible with a potential of anthracene to cause local sarcomas after sub-cutaneous administration. However, the consistent absence of any genotoxic activity in a range of *in vivo* tests strongly suggests that the production of this metabolite does not have any significant biological consequences in terms of genotoxicity and carcinogenicity.

Despite the fact that most of the carcinogenicity studies described above were not up to present day standards, overall, the available data do not provide evidence of carcinogenicity for anthracene alone or in combination with light. This conclusion is further supported by the consistent absence of evidence of genotoxic activity of anthracene in *in vitro* and *in vivo* test systems (see section 4.1.2.7 - Genetic Toxicity).

Having in mind the negative outcome of the available carcinogenicity studies, the absence of genotoxicity, and the relatively low levels of human inhalation exposure (see Section 4.1.3, Risk Characterisation), it is not considered that a long-term inhalation study is justified.

4.1.2.9 Reproductive and developmental toxicity

4.1.2.9.1 Studies in animals

Few studies on the reproductive and developmental effects of PAH have been reported. However, studies with PAH other than anthracene demonstrate that PAH are able to cross the placenta, and benzo[a]pyrene is embryotoxic and teratogenic and causes reduced fertility in mice (IPCS, 1998). These effects were partly dependent on the genetically determined modulation (inducibility) by PAH of PAH-metabolising enzymes in the mother and the fetus, mediated by interaction with the Ah receptor. In this context it is useful to note that anthracene does not bind to the Ah receptor (Machala et al., 2001).

No formal tests of the reproductive and developmental effects of anthracene in animals appear to have been conducted.

In the context of the 90-day oral exposure study already described (see Section 4.1.2.6.1), the weights and histology of the testes and ovaries, as well as clinical chemistry and haematology

values, were examined after treatment of CD-1 mice with daily doses of 0, 250, 500 and 1,000 mg/kg/day for 90 days (US EPA, 1989). Although a statistically significant increase in the mean ovary weight and the ovary-to-terminal body weight ratio of animals fed 500 mg/kg/day (but not the other two treated groups) was noted, this was considered incidental and of no toxicological relevance. The clinical chemistry and hematology analyses did not include any parameters which could be of use in the further assessment of this finding, the only changes observed being an increase in total protein in all male treated groups, and decreases in serum globulin and segmented neutrophils in the group of males treated with the low dose. All these changes were also judged to be incidental and of no toxicological relevance. No histological changes in any organs, including the testes and the ovaries, were observed.

In the context of a transplacental carcinogenicity study already described (see Section 4.1.2.8.1), anthracene (8 mg per mouse, dissolved in refined sunflower oil) was given s.c. daily or as a single intragastric dose to BALB/C, C3H/A and C57BlxCBAF1 hybrid strains during the last week of gestation (no information on maternal toxicity is given) (Shabad et al., 1972). Fragments of embryonic kidney from the embryos were cultured. In contrast to the control cultures, cultures from the anthracene-treated animals showed an increase in survival and hyperplastic epithelial changes. The changes seen were qualitatively similar but less strong that those produced by treatment with the carcinogen 7,12-dimethylbenz[a]anthracene. While the observed changes were considered by the authors as indicative of pre-malignancy, the lack of a consistent correlation between the effects of known carcinogens and non-carcinogenic analogues, and of consistent dose-response relationships, makes the significance of these effects difficult to assess. In a similar study, the transplacental effect of anthracene in mice was examined by oral administration of 8 mg of the substance per animal, followed by organ cultures of embryonic kidneys (Sorokina, 1971). Hyperplasia of individual tubules and outgrowths of atypical epithelial structures were observed in 15.6% of the tissues from animals given anthracene, compared to 1.8% seen in tissues from control animals.

In the context of a study of the effects of components of cigarette smoke on the metabolic activity of the placenta, it was shown that oral treatment of 18-day pregnant rats (unspecified strain or age) with 40 mg/kg anthracene resulted 24 hours later in a greater-than-6fold increase in the levels of benzo[a]pyrene hydroxylase activity in the placenta (Welch et al., 1969). However, no change in the levels of the same enzyme, or of 3-methyl-4-monomethylaminoazobenzene demethylase, was observed in the liver of F1 Sprague-Dawley rats after intragastric administration of 60 mg/kg anthracene (dissolved in DMSO) to pregnant animals (age unspecified) on the 19th day of gestation (Welch et al., 1972). These data cannot be utilised for the assessment of the reproductive or developmental toxicity of anthracene.

4.1.2.9.2 Studies in humans

No information on the reproductive and developmental effects of anthracene in humans was found.

4.1.2.9.3 Summary

Anthracene did not cause any detectable toxic effects on the reproductive system of mice during a 90-day feeding study On the other hand, limited studies suggest that, when administered to pregnant mice or rats, anthracene may possibly exert toxic effects on the developing embryo, including the induction of morphological changes and the upregulation of PAH-metabolising

enzymes. However, the poor quality of the data and the absence of any quantitative information do not permit the derivation firm qualitative or dose-response-related conclusions.

In view of the very limited data on reproductive toxicity, the lack of any studies of the ability of anthracene to affect pregnancy outcome or the development of mammalian organisms, the limited evidence of induction in rat embryos of toxic effects in an *in vivo*/*in vitro* study, and the established ability of other members of the PAH family to cross the placenta and cause embryotoxic and teratogenic effects (IPCS, 1998), it is concluded that further data on the reproductive and developmental toxicity of anthracene are needed. In evaluating the importance of the current absence of such data, one should take into account the fact that, as already indicated at the beginning of Section 4.1.2.9.1, the developmental toxicity of PAHs is at least partly dependent on binding to the Ah receptor, and that anthracene does not show such binding to any significant extent.

4.1.3 Risk characterisation

4.1.3.1 General aspects

Human exposure to anthracene can occur during the production and packaging of the pure compound and during the use of anthracene in the manufacture of pyrotechnics. Until recently, exposure could also take place in the context of the use of anthracene for the production of anthracene-9-aldehyde, a process which has now ceased to operate in Europe. Exposure can also occur during additional activities and processes which do not involve the production or use of anthracene, such as the distillation of coal tar for purposes other than the production of anthracene, the production and use of creosote, the production and use of coal tar- or creosote-containing products, the combustion of organic materials and certain industrial processes.

4.1.3.1.1 Exposure

In Europe about 100 workers appear to be involved in the distillation of coal tar to give various products, including light anthracene oil from which anthracene is produced. Anthracene production from light anthracene oil takes place in one plant in Europe, where 12 workers, all males, are involved. Until recently, use of anthracene in chemical synthesis took place at 1 plant in Europe, where 10-15 workers were involved in relevant activities. This production has now ceased.

Information on human exposure associated with the production and uses of anthracene is extremely limited and in most cases does not permit the use of measured data for risk characterisation. Consequently most exposure data employed for this purpose in the present Report have been derived using the EASE modelling programme. Although information for the derivation of specific exposure scenarios for the different occupation- or consumer-related activities is also limited, reasonable worst-case assumptions have been made and used to model the corresponding exposures (see Section 4.1.1 and its sub-sections). The quantitative conclusions of this exposure assessment are summarised in **Table 4.2** and **Table 4.3**.

4.1.3.1.2 Toxicity

Absorption and metabolism

Human exposure to anthracene occurs mainly via the dermal or inhalation routes, and to a limited degree via the oral route (the latter arising mostly through the ingestion of dust cleared from the nasopharyngeal and the tracheobronchial compartments by the mucociliary mechanism). While some animal toxicity data after oral or dermal administration are available, practically no inhalation toxicity data exist. The scarcity of toxicological information is even more severe when it comes to human data.

a) Absorption after inhalation

A short reference in a secondary source to reduced body weight and changes in blood biochemistry after chronic inhalation of rats to an anthracene aerosol, is impossible to assess or utilise further. However, the possibility that anthracene undergoes significant systemic absorption via the lung is supported by the finding that 99.7% of a dose of dissolved anthracene given to rats intratracheally disappears rapidly from the lung. Thus, although it is likely that absorption of particulate anthracene via the lung will be less than 100%, in the absence of other information, complete absorption of inhaled anthracene (vapour, aerosol, or particulate of respirable size) will be assumed.

b) Absorption after dermal exposure

Studies in humans and experimental animals indicate that anthracene is absorbed from the skin and enters systemic circulation, but do not permit the estimation of the rate or degree of systemic absorption in humans. Studies in rodents suggest that dermally applied anthracene (applied in solution form) is absorbed at a rate of the order of 0.3-1% per hour during the first 12-24 hours, the rate thereafter decreasing, thus resulting in an overall absorption of approximately 50% over a period of 5-6 days.

Human occupational exposure via the skin is expected to involve anthracene in dissolved form (e.g. coal tar, anthracene oil) as well as in particulate form (e.g. during packaging or handling of solid anthracene). It is reasonable to expect that systemic absorption via the skin of particulate anthracene will be lower than that of dissolved anthracene. In the absence of other information, the figure of 10% per day (i.e. per 24 hours) will be taken as a conservative estimate of the rate of absorption after dermal exposure to anthracene solution, and 2% per day after dermal exposure to particulate anthracene. When estimating systemic absorption in workers, it is assumed that the time during which the substance remains on the skin is that of the working shift (8 hours), after which the material remaining on the skin surface is removed by washing, However, in view of the lipophilicity of anthracene ($logP_{ow}$ = 4.54), material passing through the epidermis it is likely to accumulate in the stratum corneum and form a reservoir from which it will continue to diffuse into the systemic circulation after the remaining anthracene has been washed from the skin surface. Therefore systemic uptake will continue for longer than the 8 hour exposure period, and a 24 hour period is considered as more appropriate for estimating the dose absorbed as a result of exposure during an 8-hour working shift.

c) Absorption after oral exposure

There are no data on the systemic absorption of anthracene after oral ingestion in humans, and the corresponding studies in animals are limited and of poor quality. A single, old study suggests that systemic absorption in rats following oral intake of anthracene mixed with the diet does not

exceed 50%. In the absence of other information, this figure will adopted for systemic absorption after oral ingestion of anthracene by humans.

The values of systemic absorption which will be adopted for the purpose of estimating the absorbed body burden after the various exposures are summarised in **Table 4.5**.

Table 4.5 Systemic absorption in humans after different routes of anthracene exposure

Exposure route	% absorption
Inhalation (all forms)	100%
Dermal: dissolved anthracene	10% per 24 hours
particulate	2% per 24 hours
Oral	50%

Metabolism

Data are available on the metabolism of anthracene *in vitro* and in rats *in vivo*, suggesting that the major route followed is based on 1,2-epoxidation followed by epoxide hydrolysis and conjugation of the resulting dihydrodiol. There is also some evidence that, in the rat, methylated metabolites (including the weakly tumourigenic 9,10-dimethylanthracene) may also be formed by a different metabolic route. Information on anthracene metabolites other than those found in the urine is almost completely lacking. Given that significant metabolism of PAH occurs in the gastrointestinal tract, the absence of data on bile and feces metabolites is notable.

Information regarding toxicokinetics and metabolism in humans is completely lacking. However, the pattern of anthracene metabolism in the rat broadly follows that of other polycyclic aromatic hydrocarbons, for which human metabolism is known to be qualitatively similar to that of the rat (IPCS, 1998). This suggests that, on a qualitative level, human metabolism of anthracene may also be comparable to that seen in the rat. On the other hand, it is clear that no evaluation of the relative quantitative importance of competing metabolic pathways in humans can be made. In the absence of other information, it will be assumed that the metabolic pathways of anthracene in humans are not different from those observed in the experimental animal systems investigated.

Toxicological end-points and critical values

Systemic toxicity after oral, inhalation and dermal exposure

Anthracene exhibits low acute systemic toxicity in animals after oral exposure, with LD_{50} of 8.12 g/kg in the rat. Repeated dose systemic toxicity data from rats (life-time exposure) and mice (90-day exposure) also suggest low toxic potential by the oral route. No specific targets are revealed by these studies, and a small increase in ovary weight in mice receiving 500 mg/kg/day for 90 days, lacking dose-dependence, is not considered to have toxicological significance. Thus values of oral NOEL's of 50 mg/kg (rat; highest dose tested) and 1,000 mg/kg (mouse) have been derived. Although the latter value is derived from 90-day, rather than life-time exposure, it is considered more reliable as it was based on a more recent and better conducted study and for this reason it will be adopted for risk assessment.

With 50% systemic absorption, an oral NOEL of 1,000 mg/kg corresponds to a daily body burden of 500 mg/kg/day. As no data suitable for estimating limit values for systemic toxicity after inhalation or dermal exposure are available, in subsequent sections this value of the body burden will be employed as a limit value corresponding to a NOEL for systemic exposure, and

compared with the body burdens arising from exposure from other routes in order to derive the corresponding MOS values.

Skin irritation and sensitisation after dermal exposure and eye irritation

Limited references to skin irritating activity of anthracene in humans, usually in the context of occupational medicine reports, are impossible to evaluate or utilise further. On the other hand, in view of the strong phototoxic potential of anthracene under normal daily conditions, any limit values adopted for this endpoint are likely to provide protection from any dermal toxicity in the absence of UV light.

Anthracene was negative in a test for eye irritation that closely resembles that of Annex V of Dir. 92/69/EEC. Therefore it is considered that all exposures to anthracene examined do not raise concern over the induction of eye irritation. **Conclusion (ii)**.

Phototoxicity

In contrast to the situation regarding the ability of anthracene to induce irritation or sensitisation in the absence of light, extensive studies in animals and human volunteers demonstrate its potential to induce, in combination with UV radiation, skin irritation, including immediate and delayed erythema as well as weal-and-flare reaction. Such studies, are in agreement with occupational medicine reports of the occurrence of photodermatitis in workers involved in handling tar and other petroleum-derived products.

No formal *in vivo* test for phototoxicity is foreseen under Directive 67/548/EEC. However, the data of Kaidbey and Nonaka (1984) clearly demonstrate induction of phototoxicity in humans after a single skin application of 25 μg of dissolved anthracene per cm^2 of skin in combination with a minimum of 1 kJ/m^2 of UVA radiation. Although the derivation of a true LOAEL or NOAEL would require extensive data obtained with varying doses of both anthracene and irradiation, the outcome of the abovementioned study, in combination with the ease with which sufficient UVA energy on eliciting phototoxicity can be received by a person exposed to moderate sunlight (a few seconds to minutes), justifies consideration of classification anthracene as an irritant, with a practical LOAEL of 25 $\mu g/cm^2$ (for dissolved anthracene). This value, being derived from a study which utilised only a single anthracene dose level, provides a weak basis from which to extrapolate to a NAEL. Indeed, the possibility that effects may occur at levels substantially lower than 25 $\mu g/cm^2$ finds support in the results of a well reported study in guinea pigs, which indicates that a much lower dose (140 ng/cm^2), in combination with 100 kJ/m^2, can still cause skin irritation, while no effects were observed at 14 ng/cm^2. As no information is available on the relative sensitivities of humans and guinea pigs, the human as well as animal limit values will be considered for the calculation of MOS.

Genotoxicity, carcinogenicity

Although anthracene belongs to the category of polycyclic aromatic hydrocarbons, which includes a large number of well known genotoxic and carcinogenic members, it appears to lack such activity. It has been tested for genotoxicity in a large number of well-validated assays, involving systems ranging from bacteria to whole animals and end-points ranging from DNA damage to gene mutations and clastogenicity. Many of the tests were conducted in the context of multi-laboratory trials, which were based on strictly defined methodologies and must be considered reliable. Although occasional positive or inconclusive results were obtained, the great majority yielded negative results, supporting an overall conclusion that anthracene is not genotoxic. The absence of explicit clarification regarding the use or not of protection against

UV-induced photodegradation in many of the reported genotoxicity studies is not sufficient to overturn this conclusion.

Anthracene has been examined for carcinogenicity in rats, mice and rabbits, using assays designed to test complete carcinogenicity or tumour initiating activity. Many of these studies are old and do not come up to the standards currently considered acceptable. However, when assessed collectively, and taking into account the lack of genotoxic activity, these studies do not provide evidence of anthracene carcinogenicity by any of the exposure routes employed (oral or dermal). Although no inhalation carcinogenicity study has been conducted, the overall balance of available evidence supports the conclusion that anthracene lacks carcinogenic activity by any route. Assays to examine the potential of anthracene to induce skin cancer in combination with UV light (including some relatively recent and well-conducted studies) have also given negative results. Therefore it is considered that all exposures to anthracene examined do not raise concern over the induction of cancer. **Conclusion (ii)**.

Reproductive/developmental toxicity

No Studies in humans on the reproductive or developmental effects of anthracene are available.

Anthracene has not been examined in a formal reproductive toxicity study. However, a 90-day oral toxicity study did not demonstrate any clear toxicological effects on the gonads.

Studies in animals indicate that anthracene can pass the placenta, but no information suitable for the qualitative or quantitative evaluation of reproductive and developmental toxicity of anthracene is available. The limited evidence from an *in vivo* treatment/*in vitro* organ cultures study, while suggesting a possible embryotoxic potential, cannot be utilised to assess the potential of anthracene to exert developmental toxicity. In view of these observations and having in mind the established ability of other members of the PAH family to cross the placenta and cause embryotoxic and teratogenic effects, the lack of adequate information on the developmental toxicity of anthracene constitutes a hurdle in the risk assessment and it is concluded that a developmental toxicity study would ideally be needed. On the other hand, the absence of binding by anthracene to the Ah receptor, which appears to at least partly mediate the developmental toxicity of PAHs, suggests that anthracene may possess weak, if any, developmental toxicity.

4.1.3.1.3 Summary of critical values used in risk characterisation

Table 4.6 summarises the important quantitative data regarding the toxic effects of anthracene in animals and humans. It can be seen that most of them are based on studies in animals, whereas limit values obtained from observations in humans are also available only for skin phototoxicity.

Table 4.6 Limit values and conclusions taken forward to risk assessment

End-point	Value	Type of study	Limit value taken forward to risk characterisation
systemic, oral toxicity	NOEL: 1,000 mg/kg/day	mouse 90-day repeated dose	Body burden: 500 mg/kg/day
skin irritation	no information		None (but see phototoxicity)
eye irritation	Negative		No concern
sensitisation	no information		None (but see phototoxicity)
phototoxicity	NOAEL: 14 ng/cm^2	guinea pig	NOAEL: 14 ng/cm^2
	LOAEL: 140 ng/cm^2	guinea pig	LOAEL: 140 ng/cm^2
	LOAEL: 25 µg/cm^2	Human	LOAEL: 25 µg/cm^2
mutagenicity	Negative	*in vitro* and mouse *in vivo*	No concern
carcinogenicity	Negative	rat, mouse	No concern
reproductive toxicity	no direct information		Information needed
	no effects on gonads	Mouse	
developmental toxicity	no information		Information needed

4.1.3.1.4 Minimal MOS

In the present Section, the toxicological limit values adopted in the previous section will be compared with the maximum exposure levels (or, where appropriate, the corresponding body burdens) adopted for the various exposure scenarios previously examined (summarised in **Table 4.2 and Table 4.3**), and MOS values will be calculated. The latter will be assessed by comparison with the minimal MOS values which are considered acceptable, obtained by using the safety factors described in **Table 4.7**.

The uncertainty factors of 10 adopted for inter- and intraspecies variability and 5 adopted for extrapolation from 90-day to chronic exposures are widely used, standard values. For the extrapolation from LOAEL (human) to NAEL for phototoxicity, an uncertainty factor of 100 is considered necessary in view of the fact that the study from which the LOAEL was derived utilised only a single dose level. The overall minimal MOS is obtained by multiplying the relevant uncertainty factors, depending on the type of extrapolation concerned.

Table 4.7 Uncertainty factors employed in the estimation of minimal MOS

Source of uncertainty	Uncertainty factor
Interspecies variability	10
Intraspecies variability	10
Exposure duration (90-day to chronic exposure)	5
LOAEL to NAEL (phototoxicity)	100

4.1.3.2 Workers

Occupational exposure to anthracene is expected to occur during the purification of anthracene oil for the manufacture of anthracene and during anthracene packaging.

4.1.3.2.1 Manufacture of anthracene from anthracene oil

In the context of the assessment of exposure during the manufacture of anthracene from anthracene oil, two stages at which worker exposure can occur are recognised, the first referring to the preparation of crude anthracene and the second referring to the refining of the latter.

Exposure to anthracene during both stages can occur by inhalation and by skin contact during loading and unloading as well as during cleaning and maintenance operations.

Systemic toxicity

Inhalation exposure may involve exposure to anthracene vapour or in particulate form. Assuming that particulate anthracene is in respirable form, an inhalation rate of 10 m^3/shift and 100% systemic absorption, the body burden for workers exposed for 8 h per shift to the airborne concentrations derived from measurement and from modelling shown in **Table 4.3** have been calculated (**Table 4.8**). In the calculations it has been assumed that a worker spends a full shift on only one of the two stages of the process. However, the broadly similar levels of exposure (measured or modelled) expected at the two stages, in combination with the large MOS estimated, indicate that the overall conclusion of the risk characterisation is not dependent on the proportion of the shift time spent on each job.

Dermal exposure may involve anthracene in a dissolved state as well as solid anthracene (during removal of solidified anthracene from pipelines and vessels), and is likely to take place via contact with the hands and forearms (total area = 1,980 cm^2). It is not possible to estimate the proportion (duration or amount) of skin contact with dissolved or solid anthracene. Using the levels of dermal contact predicted by EASE, the resulting body burdens have been calculated assuming 10% absorption per 24 hours for dissolved anthracene. If contact is with particulate anthracene (2% absorption), the body burden and MOS would be 5 fold lower (not shown in **Table 4.8**).

Based on the fact that the limit value of the body burden (corresponding to the NOEL) is derived from the sub-chronic mouse feeding study, a minimal MOS of 500 is adopted. As seen in **Table 4.8**, the estimated MOS values exceed the minimal MOS for inhalation, dermal and combined exposures not only for the measured air concentrations but also for the much higher (and probably substantially exaggerated) ones predicted by EASE. **Conclusion (ii)**.

Skin photoirritation

The dermal exposure levels estimated by EASE for the two stages of the process considered are similar to or approximately 4 times lower than the human LOAEL of 25 $\mu g/cm^2$. They are also substantially higher than the animal LOAEL and NOAEL. This suggests that **Conclusion (iii)** applies.

Table 4.8 Risk characterisation for anthracene production

Systemic toxicity							
Inhalation	**Air conc. (µg/m³)**	**Body burden[1] (µg/kg/day)**	**Mean life-time body burden[3] (µg/kg/day)**		**MOS[4]**	**Min. MOS[5]**	**Concl.**
Preparation of crude anthracene	Estimated from Measurement: 5	0.71	0.33		$1.5 \cdot 10^{6}$	500	ii
	Modelled: 44	6.3	2.9		$1.7 \cdot 10^{5}$		
Purification	Estimated from Measurement: 7.8	1.1	0.51		$9.8 \cdot 10^{5}$		
	Modelled: 60	8.6	3.9		$1.3 \cdot 10^{5}$		
Dermal	**Dermal exposure (µg/cm²/day)**	**body burden[2] (µg/kg/day)**					
Preparation of crude anthracene	Modelled: 6	17.0	7.8		$6.4 \cdot 10^{4}$	500	ii
Purification	Modelled: 30	84.9	39.0		$1.3 \cdot 10^{4}$		
Combined							
Preparation of crude anthracene	Inhal^{n} from measurement	17.7	8.1		$6.2 \cdot 10^{4}$	500	ii
	Inh. Modelled	23.3	10.7		$4.7 \cdot 10^{4}$		
Purification	Inhal^{n} measured	85.6	39.6		$1.3 \cdot 10^{4}$		
	Inhal^{n} modelled	93.5	42.9		$1.2 \cdot 10^{4}$		
Dermal phototoxicity	**Dermal exposure (µg/cm²/day)**			**Limit value**	**MOS**	**Min MOS[6]**	
Preparation of crude anthracene	Modelled: 6	N/A	N/A	Human LOAEL	4	100	iii
				Animal LOAEL	$2 \cdot 10^{-2}$		
				Animal NOAEL	$2 \cdot 10^{-3}$		
Purification	Modelled: 30	N/A	N/A	Human LOAEL	0.8	100	iii
				Animal LOAEL	$5 \cdot 10^{-3}$		
				Animal NOAEL	$5 \cdot 10^{-4}$		

1 Exposure · (inhalation rate 10 m³/8-hour shift) · (100% systemic absorption) / (70 kg bd.wt.)
2 Exposure · (1,980 cm²) · (10% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL body burden (500 mg/kg/day)
5 Interspecies variability · intraspecies variability x subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

4.1.3.2.2 Anthracene packaging

Exposure during the packaging of pure anthracene is expected to occur primarily by skin contact or oral ingestion of airborne, non-respirable particles cleared by the mucociliary system and subsequently ingested. To a small extent it may also occur by inhalation of anthracene vapours, although it is recognised that the value of vapour concentration brought forward from the modelled estimation of exposure (concentration corresponding to saturated vapour pressure at room temperature) is likely to represent a serious overestimation.

Systemic toxicity

The body burdens resulting from the modelled exposures have been calculated using the absorption rates adopted and assuming that the all of the inhaled dust is cleared by the mucociliary system and is eventually ingested. Dermal exposure is expected to involve solid anthracene and is likely to take place via contact with the hands and forearms (total area = 1,980 cm^2). As can be seen in **Table 4.9**, the MOS values calculated for systemic toxicity exceed the minimal MOS for all three routes considered individually or combined. Taking into account that the modelling procedure employed probably leads to significant overestimation of the exposure, it is concluded that **Conclusion (ii)** applies.

Skin photoirritation

The maximal dermal exposure level estimated by EASE (1 mg/cm^2) is significantly higher than the human LOAEL of 25 $\mu g/cm^2$ (MOS = $2.5 \cdot 10^{-2}$), as well as the animal LOAEL and NOAEL, indicating that **Conclusion (iii)** applies.

Table 4.9 Risk characterisation for anthracene packaging

Systemic toxicity						
	Air conc. ($\mu g/m^3$)	**Body burden[1] (μg/kg/day)**	**Mean life-time body burden[3] (μg/kg/day)**	**MOS[4]**	**Min. MOS[5]**	**Concl.**
Ingestion of non-respirable dust	5,000	357	164	$3.0 \cdot 10^3$	500	ii
Inhalation of vapour	60	8.6	3.9	$1.3 \cdot 10^5$	500	ii
Dermal	**Dermal exposure ($\mu g/cm^2$/day)**	**Body burden[2] (μg/kg/day)**				
	1,000	566	260	$1.9 \cdot 10^3$	500	ii
Combined exposure			428	$1.2 \cdot 10^3$	500	ii

Table 4.9 continued overleaf

Table 4.9 continued Risk characterisation for anthracene packaging

dermal phototoxicity	**Dermal exposure ($\mu g/cm^2/day$)**			**Limit value**	**MOS**	**Min. MOS**[6]	
Dermal	1,000	N/A	N/A	Human LOAEL	$2.5 \cdot 10^{-2}$	100	iii
				Animal LOAEL	$1.4 \cdot 10^{-4}$	-	
				Animal NOAEL	$1.4 \cdot 10^{-5}$		

1 Exposure · (inhalation rate 10 m^3/8 hour shift) · (systemic absorption) / (70 kg bd.wt.); absorption: 50% for ingested dust, 100% for inhaled vapour
2 Exposure · (1,980 cm^2) · (2% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL body burden (500 mg/kg/day)
5 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

4.1.3.2.3 Use of anthracene in the manufacture of pyrotechnics

Exposure during the manufacture of pyrotechnics is expected to occur primarily by skin contact or oral ingestion of airborne, non-respirable particles cleared by the mucociliary system and subsequently ingested. To a small extent it may also occur by inhalation of anthracene vapours, although it is recognised that the value of vapour concentration brought forward from the modelled estimation of exposure (concentration corresponding to saturated vapour pressure at room temperature) is likely to represent a serious overestimation.

Systemic toxicity

The body burdens resulting from the modelled exposures have been calculated using the absorption rates adopted and assuming that all of the inhaled dust is cleared by the mucociliary system and is eventually ingested. Dermal exposure is expected to involve solid anthracene and is likely to take place via contact with the hands and forearms (total area = 1,980 cm^2). As can be seen in **Table 4.10**, the MOS values calculated for systemic toxicity exceeds the minimal MOS for all three routes considered individually or combined. Taking into account that the modelling procedure employed probably leads to significant overestimation of the exposure, it is concluded that **Conclusion (ii)** applies.

Skin photoirritation

The maximal dermal exposure level estimated by EASE (1 mg/cm^2) is significantly higher than the human LOAEL of 25 $\mu g/cm^2$ (MOS = $2.5 \cdot 10^{-2}$), as well as the animal LOAEL and NOAEL, indicating that **Conclusion (iii)** applies.

Table 4.10 Risk characterisation for manufacture of pyrotechnics

Systemic toxicity							
	Air conc. (μg/m³)	**Body burden[1] (μg/kg/day)**	**Mean life-time body burden[3] (μg/kg/day)**	**MOS[4]**		**Min. MOS[5]**	**Concl.**
ingestion of non-respirable dust	5,000	357	164	$3.0 \cdot 10^{3}$		500	ii
inhalation of vapour	60	8.6	3.9	$1.3 \cdot 10^{5}$		500	ii
Dermal	**Dermal exposure (μg/cm²/day)**	**Body burden[2] (μg/kg/day)**					
	1,000	566	260	$1.9 \cdot 10^{3}$		500	ii
Combined exposure			428	$1.2 \cdot 10^{3}$		500	ii
Dermal phototoxicity	**Dermal exposure (μg/cm²/day)**			**Limit value**	**MOS**	**Min. MOS[6]**	
Dermal	1,000	N/A	N/A	human LOAEL	$2.5 \cdot 10^{-2}$	100	iii
				animal LOAEL	$1.4 \cdot 10^{-4}$	-	
				animal NOAEL	$1.4 \cdot 10^{-5}$		

1 Exposure x (inhalation rate 10 m³/8 hour shift) x (systemic absorption) / (70 kg bd.wt.); absorption: 50% for ingested dust, 100% for inhaled vapour
2 Exposure x (1,980 cm²) x (2% systemic absorption) / (70 kg bd.wt.)
3 Body burden x (5/7 days/week) x (45/70 years)
4 Compared to mouse NOEL body burden (500 mg/kg/day)
5 Interspecies variability x intraspecies variability x subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

4.1.3.3 Consumers

There is no consumer exposure to anthracene produced as an industrial product or to products containing added anthracene.

4.1.3.4 Exposure of the general population via the environment

Exposure of the general population via the environment is likely to result in a maximal oral intake (via the diet and drinking water) of 143 ng/kg/day and a maximal likely airborne exposure of 9.7 ng/kg/day. These intakes would correspond a total body burden of 81.2 ng/kg/day, leading to a MOS = $6.2 \cdot 10^{6}$ (compared with a minimal MOS of 500) (**conclusion (ii)**) (**Table 4.11**). The large MOS indicates that this conclusion will not be affected even if the level of exposure is significantly higher.

Table 4.11 Risk characterisation for the general population exposed via the environment

Systemic toxicity				
	Body burden[1] (ng/kg/day)	**MOS[2]**	**Min. MOS[3]**	**Concl.**
Oral intake (ng/kg/day): 143	71.5	$7.0 \cdot 10^{6}$	500	ii
Inhalation intake (ng/kg/day): 9.7	9.7	$5.2 \cdot 10^{7}$		ii
Combined exposure	81.2	$6.2 \cdot 10^{6}$		ii

1 Intake · (50% absorption rate)
2 Intake · (100% absorption rate)
3 Compared to mouse NOEL (500 mg/kg/day)
4 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation

4.1.3.5 Combined exposure

Given the extremely low levels of exposure of the general population via the environment, relative to occupational exposures, and absence of consumer exposure, it was not considered useful to produce a combined exposure assessment.

4.1.3.6 Exposures to anthracene not related to the production and current uses of anthracene

The following discussion on exposures arising from sources NOT related to the production and current use of anthracene does not directly come under the terms of reference of the present Report, and is presented for illustrative purposes. For this reason, formal conclusions are not included in this Section.

4.1.3.6.1 Occupational exposure during use of anthracene during chemical synthesis

Even though the use of anthracene for chemical synthesis appears to have now ceased, it is also discussed for illustrative purposes given its use until very recently.

Exposure during chemical synthesis could occur by inhalation of airborne anthracene vapours as well as by skin contact with dissolved anthracene during loading, sampling and routine cleaning and maintenance operations. It is recognised that the value of the vapour concentration brought forward from the estimation of exposure (concentration corresponding to saturated vapour pressure at room temperature) is likely to represent a serious overestimation.

Systemic toxicity

As can be seen in **Table 4.12**, the body burden resulting from the modelled exposure leads to MOS values greater than 10^4 for systemic toxicity from inhalation and dermal exposure individually and combined.

Skin photoirritation

The maximal dermal exposure level estimated by EASE (100 µg/cm^2) is 4 times higher than the human LOAEL, and also substantially higher than the animal LOAEL and NOEL, indicating concern.

Table 4.12 Risk characterisation for use of anthracene in chemical synthesis

Systemic toxicity					
	Air conc. (µg/m^3)	**Body burden[1] (µg/kg/day)**	**Mean life-time body burden[3] (µg/kg/day)**	**MOS[4]**	**Min. MOS[5]**
Vapour	60	8.6	3.9	$1.3 \cdot 10^5$	500
Dermal	**Dermal exposure (µg/cm^2/day)**	**Body burden[2] (µg/kg/day)**			
	100	283	130	$3.8 \cdot 10^3$	500
Combined exposure		292	134	$3.7 \cdot 10^3$	500
Dermal phototoxicity	**Dermal exposure (µg/cm^2/day)**			**Limit value**	**Min. MOS[6]**
Dermal	100	N/A	N/A	Human LOAEL	0.25
				Animal LOAEL	$1.4 \cdot 10^{-3}$
				Animal NOEL	$1.4 \cdot 10^{-4}$

1 Exposure · (inhalation rate 10 m^3/8 hour shift) · (systemic absorption) / (70 kg bd.wt.); absorption: 50% for ingested dust, 100% for inhaled vapour
2 Exposure · (1,980 cm^2) · (10% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL (500 mg/kg/day)
5 Interspecies variability · intraspecies variability x subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

4.1.3.6.2 Coal tar distillation

Exposure during coal tar distillation is expected to occur by inhalation of anthracene vapours as well as by skin contact with dissolved anthracene during loading, sampling and routine cleaning and maintenance operations.

Systemic toxicity

As can be seen in **Table 4.13**, the body burden resulting from the modelled exposure leads to MOS values greater than 10^4 for systemic toxicity from inhalation and dermal exposure individually and combined.

Skin photoirritation

The maximal dermal exposure level estimated by EASE (0.1 mg/cm^2) is 4 times higher than the human LOAEL and substantially higher than the animal LOAEL and NOEL, indicating concern.

Table 4.13 Risk characterisation for coal tar distillation

Systemic toxicity						
	Air conc. (µg/m³)	**Body burden[1] (µg/kg/day)**	**Mean life-time body burden[3] (µg/kg/day)**	**MOS[4]**		**Min. MOS[5]**
Vapour	11	1.6	0.7	$6.9 \cdot 10^5$		500
Dermal	**Dermal exposure (µg/cm²/day)**	**Body burden[2] (µg/kg/day)**				
	1.5	4.2	1.9	$2.6 \cdot 10^5$		500
Combined exposure		5.8	2.7	$1.9 \cdot 10^5$		500
Dermal phototoxicity	**Dermal exposure (µg/cm²/day)**			**Limit value**	**MOS**	**Min. MOS[6]**
Dermal	1.5	N/A	N/A	Human LOAEL	17	100
				Animal LOAEL	$9 \cdot 10^{-2}$	-
				Animal NOEL	$9 \cdot 10^{-3}$	

1 Exposure · (inhalation rate 10 m³/8 hour shift) · (systemic absorption) / (70 kg bd.wt.); absorption: 50% for ingested dust, 100% for inhaled vapour
2 Exposure · (1,980 cm²) · (10% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL (500 mg/kg/day)
5 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

4.1.3.6.3 Occupational exposures via creosote

Creosote blending

Exposure during creosote blending is expected to occur by inhalation of anthracene vapours as well as by skin contact with dissolved anthracene during loading, sampling and routine cleaning and maintenance operations.

Systemic toxicity

As can be seen in **Table 4.14**, the body burden resulting from the modelled exposure leads to MOS values greater than 10^4 for systemic toxicity from inhalation and dermal exposure individually and combined.

Skin photoirritation

The maximal dermal exposure level predicted by EASE (15 µg/cm²) is comparable with the human LOAEL, leading to a MOS of 1.7 as compared to the minimal MOS of 100. It is also substantially higher than the animal LOAEL and NOEL, indicating concern.

Table 4.14 Risk characterisation for creosote blending

Systemic toxicity						
	Air conc. ($\mu g/m^3$)	**Body burden[1] (µg/kg/day)**	**Mean life-time body burden[3] (µg/kg/day)**	**MOS[4]**		**Min. MOS[5]**
Vapour	11	1.6	0.73	$6.9 \cdot 10^5$		500
Dermal	**Dermal exposure ($\mu g/cm^2/day$)**	**Body burden[2] (µg/kg/day)**				
	15	42.5	19.5	$2.6 \cdot 10^4$		500
Combined exposure		44.1	20.2	$2.5 \cdot 10^4$		500
Dermal phototoxicity	**Dermal exposure ($\mu g/cm^2/day$)**			**Limit value**	**MOS**	**Min. MOS[6]**
Dermal	15	N/A	N/A	Human LOAEL	1.7	100
				Animal LOAEL	$9 \cdot 10^{-3}$	-
				Animal NOEL	$9 \cdot 10^{-4}$	

1 Exposure · (inhalation rate 10 m^3/8 hour shift) · (systemic absorption) / (70 kg bd.wt.); absorption: 50% for ingested dust, 100% for inhaled vapour
2 Exposure · (1,980 cm^2) · (10% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL (500 mg/kg/day)
5 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

Creosote packaging

Exposure during creosote packaging is expected to occur by inhalation of anthracene vapours as well as by skin contact with dissolved anthracene during loading, sampling and routine cleaning and maintenance operations.

Systemic toxicity

As can be seen in **Table 4.15**, the body burden resulting from the modelled exposure leads to MOS values greater than 10^4 for systemic toxicity from inhalation and dermal exposure individually and combined.

Skin photoirritation

The maximal dermal exposure level estimated by EASE (15 $\mu g/cm^2$) is comparable with the human LOAEL, leading to a MOS of 1.7 as compared to the animal MOS of 100. It is also substantially higher than the animal LOAEL and NOEL, indicating concern.

Table 4.15 Risk characterisation for creosote packaging

Systemic toxicity						
	Air conc. (µg/m³)	**Body burden[1] (µg/kg/day)**	**Mean life-time body burden[3] (µg/kg/day)**	**MOS[4]**		**Min. MOS[5]**
Vapour	11	1.6	0.73	$6.9 \cdot 10^4$		500
Dermal	dermal exposure (µg/cm²/day)	body burden[2] (µg/kg/day)				
	15	42	19.5	$2.6 \cdot 10^4$		500
Combined exposure		43.6	20.2	$2.5 \cdot 10^4$		500
Dermal phototoxicity	**Dermal exposure (µg/cm²/day)**			**Limit value**	**MOS**	**MOS[6]**
Dermal	15	N/A	N/A	human LOAEL	1.7	100
				animal LOAEL	$9 \cdot 10^{-3}$	-
				animal NOEL	$9 \cdot 10^{-4}$	

1 Exposure · (inhalation rate 10 m³/8 hour shift) · (100% systemic absorption) / (70 kg bd.wt.); absorption: 50% for ingested dust, 100% for inhaled vapour
2 Exposure · (1,980 cm²) · (2% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL (500 mg/kg/day)
5 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

Timber impregnation

Exposure during timber impregnation is expected to occur by inhalation of anthracene in particulate form as well as by skin contact with dissolved anthracene during loading, sampling and routine cleaning and maintenance operations. No information is available on the size of the anthracene-containing particles, and they are assumed to be respirable, leading to 100% absorption.

Systemic toxicity

As can be seen in **Table 4.16**, the body burden resulting from the exposures estimated from measured values leads to MOS values greater than 10^3 for systemic toxicity from inhalation and dermal exposure individually and combined.

Skin photoirritation

The maximal dermal exposure level leads to a MOS of $7 \cdot 10^{-2}$ compared to the human LOAEL. It is also substantially higher than the animal LOAEL and NOEL, indicating concern.

Table 4.16 Risk characterisation for timber impregnation

Systemic toxicity						
	Air conc. (µg/m³)	**Body burden[1] (µg/kg/day)**	**Mean life-time body burden[3] (µg/kg/day)**		**MOS[4]**	**Min. MOS[5]**
Particulate	19	2.7	1.2		$4.2 \cdot 10^{5}$	500
Dermal	Dermal exposure (µg/cm²/day)	Body burden[2] (µg/kg/day)				
	334	945	435		$1.1 \cdot 10^{3}$	500
Combined exposure		948	436		$1.1 \cdot 10^{3}$	500
Dermal phototoxicity	**Dermal exposure (µg/cm²/day)**			**Limit value**	**MOS**	**Min. MOS[5]**
Dermal	334	N/A	N/A	Human LOAEL	$7 \cdot 10^{-2}$	100
				Animal LOAEL	$4 \cdot 10^{-4}$	-
				Animal NOEL	$4 \cdot 10^{-5}$	

1 Exposure · (inhalation rate 10 m³/8 hour shift) · (100% systemic absorption) / (70 kg bd.wt.)
2 Exposure · (1,980 cm²) · (10% systemic absorption) / (70 kg bd.wt.)
3 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL (500 mg/kg/day)
5 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
6 LOAEL-to-NAEL extrapolation

Creosote brushing

Exposure of professionals (e.g. gardeners) during creosote brushing is expected to occur by dermal contact with dissolved anthracene.

Systemic toxicity

In **Table 4.17** the body burden has been calculated based on the dermal exposures to the hands (taken to have an area of 840 cm^2) and the trunk (area 5,690 cm^2) estimated from an experimental study on volunteers, as well as based on the predictions of the EASE model (in this case taking the area exposed as the sum of the hands and trunk, i.e. 6,530 cm^2). The body burden figures indicated, and on which the calculations of MOS for systemic toxicity are based, is that corresponding to continuous lifetime exposure. It can be seen that the MOS values thus obtained are greater than 10^2. Actual exposure for consumers is not expected to exceed a few days per year, at most, leading to a substantially greater MOS value.

Skin photoirritation

It has been assumed that risk of photoirritation exists for the part of the body most likely to be exposed to the light, i.e. the hands. The dermal exposure of the hands leads to a MOS of 3.1 relative to the human LOAEL, while it is substantially lower than the animal LOAEL and NOEL. Furthermore, the exposure predicted by EASE is comparable to the human LOAEL (MOS = 0.3) and substantially higher than the animal LOAEL and NOEL. These observations indicate concern.

Table 4.17 Risk characterisation for creosote brushing

	Dermal exposure ($\mu g/cm^2/day$)	**Body burden[1] ($\mu g/kg/day$)**	**MOS[2]**			**Min. MOS[3]**
Systemic toxicity	Estimated from measurements Through the hands: 8 Through the trunk: 54	449	$1.1 \cdot 10^3$			500
	Modelled by EASE 75	700	715			
Dermal phototoxicity	Estimated from measurements Through the hands: 8	N/A	Limit value	Exposure	MOS	Min. MOS[4]
			Human LOAEL	Measure-ments	3.1	100
				EASE	0.3	
	Modelled by EASE 75		Animal LOAEL	Measure-ments	$1.7 \cdot 10^{-2}$	-
				EASE	$1.8 \cdot 10^{-3}$	
			Animal NOEL	Measure-ments	$1.7 \cdot 10^{-3}$	
				EASE	$1.8 \cdot 10^{-4}$	

1 [hand exposure · (840 cm^2) + (trunk exposure x (5,690 cm^2)] · (10% systemic absorption)/ (70 kg bd.wt.); for EASE-derived exposure, body burden = exposure · (6,530 cm2) · (10% systemic absorption)/(70 kg bd.wt.)
2 Compared to mouse NOEL (500 mg/kg/day)
3 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
4 LOAEL-to-NAEL extrapolation

4.1.3.6.4 Occupational exposure from consumer products

Use of coal tar paints and related products

Exposure during the use of coal tar paints and related products is expected to occur by dermal contact with dissolved anthracene.

Systemic toxicity

As can be seen in **Table 4.18**, the body burden has been calculated based on the dermal exposures to the hands (taken to have an area of 840 cm^2) and the trunk (area 5,690 cm^2) estimated from an experimental study on volunteers. It has also been estimated using the modelled dermal exposure obtained using the EASE programme (in this case taking the area exposed as the sum of the hands and trunk, i.e. 6,530 cm^2). The body burden figures indicated, and on which the calculations of MOS for systemic toxicity are based, is that corresponding to continuous lifetime exposure. It can be seen that the MOS values thus obtained are greater than 10^4. Actual exposure for consumers is not expected exceed a few days per year, at most, leading to a substantially greater MOS value.

Skin photoirritation

It has been assumed that risk of photoirritation exists for the part of the body most likely to be exposed to the light, i.e. the hands. The dermal exposure of the hands, estimated from the experimental data, lead to a MOS of 1.4 relative to the human LOAEL, while it is substantially lower than the animal LOAEL and NOEL. On the other hand, the exposure level modelled by EASE is equal lead to the human LOAEL (MOS = 1), and substantially lower than the animal LOAEL and NOEL. These observations indicate concern.

Table 4.18 Risk characterisation for use of coal tar paints and related products

	Dermal exposure ($\mu g/cm^2/day$)	**Body burden[1] ($\mu g/kg/day$)**	**MOS[2]**			**Min. MOS[3]**
Systemic toxicity	Estimated from measurements Through the hands: 18.3 Through the trunk: 2.7	43.9	$1.1 \cdot 10^{4}$			500
	Modelled by EASE: 25	233	$2.1 \cdot 10^{3}$			
Dermal phototoxicity	Estimated from measurements	N/A	limit value	expo-sure	MOS	min. MOS[4]
	Through the hands: 18.3		Human LOAEL	measure-ments	1.4	100
				EASE	1	
	Modelled by EASE: 25		Animal LOAEL	measure-ments	$7.7 \cdot 10^{-3}$	
				EASE	$5.6 \cdot 10^{-3}$	
			Animal NOEL	measure-ments	$7.7 \cdot 10^{-4}$	
				EASE	$5.6 \cdot 10^{-4}$	

1 [hand exposure · (840 cm^2) + (trunk exposure · (5,690 cm^2)] · (10% systemic absorption) / (70 kg bd.wt.); for EASE-derived exposure, body burden = exposure · (6,530 cm2) · (10% systemic absorption) / (70 kg bd.wt.)
2 Compared to mouse NOEL (500 mg/kg/day)
3 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
4 LOAEL-to-NAEL extrapolation

4.1.3.6.5 Occupational exposure from other industrial sources

Inhalation exposure to anthracene can occur in the context of industrial sources not related to the production of use of anthracene. **Table 4.19** presents the body burdens estimated for various occupational activities, based on the maximal exposures estimated from the limited data available. In all cases, the calculated MOS exceeds the minimal MOS for systemic toxicity by many orders of magnitude, indicating no concern.

Table 4.19 Risk characterisation for occupational exposure from other industrial sources

activity	Air conc. (µg/m³)	body burden[1] (µg/kg/day)	Mean life-time body burden[3] (µg/kg/day)	MOS[4]	Min. MOS[5]
Coal processing and related activities	52	7.4	3.4	$1.5 \cdot 10^5$	500
Carbon anode/graphite	5.51	0.8	0.4	$1.3 \cdot 10^6$	
Silicon carbide	139	19.9	9.1	$5.5 \cdot 10^4$	
Aluminium and other metals	256	36.6	16.8	$3 \cdot 10^4$	
Iron and steel	22	3.1	1.4	$3.6 \cdot 10^5$	

1 Exposure · (inhalation rate 10 m³/8 hour shift) · (100% systemic absorption) / (70 kg bd.wt.)
2 Body burden · (5/7 days/week) · (45/70 years)
4 Compared to mouse NOEL (500 mg/kg/day)
5 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation

4.1.3.6.6 Consumer exposure during use of coal tar paints and related products

Consumer exposure to anthracene can only occur in the context of using coal tar paints and related products, and is expected to occur via skin contamination.

Systemic toxicity

In **Table 4.20**, the body burden has been calculated using the maximal levels which have been estimated indirectly from measured data, and assuming exposures to the hands to involve an area of 840 cm^2 and those to the trunk an area of 5,690 cm^2.

Skin photoirritation

It has been assumed that risk of photoirritation exists for the part of the body most likely to be exposed to the light i.e. the hands. The dermal exposure of the hands leads to a MOS of 0.4 relative to the human LOAEL, while it is substantially lower than the animal LOAEL and NOEL.

Table 4.20 Risk characterisation for consumer use of coal tar paints and related products

<table>
<tr><th></th><th>Dermal exposure ($\mu g/cm^2$/day)</th><th>Body burden[1] (µg/kg/day)</th><th colspan="2">MOS[2]</th><th>Min. MOS[3]</th></tr>
<tr><td>Systemic toxicity</td><td rowspan="5">Through the hands: 100
Through the trunk: 0.8</td><td>127</td><td colspan="2">$4.0 \cdot 10^4$</td><td>500</td></tr>
<tr><td rowspan="4">Dermal phototoxicity</td><td rowspan="4">N/A</td><td>Limit value</td><td>MOS</td><td>Min. MOS[4]</td></tr>
<tr><td>Human LOAEL</td><td>0.25</td><td>100</td></tr>
<tr><td>Animal LOAEL</td><td>$1.4 \cdot 10^{-3}$</td><td rowspan="2">-</td></tr>
<tr><td>Animal NOEL</td><td>$1.4 \cdot 10^{-4}$</td></tr>
</table>

1 [hand exposure · (840 cm^2) + (trunk exposure · (5,690 cm^2)] · (10% systemic absorption) / (70 kg bd.wt.)
2 Compared to mouse NOEL (500 mg/kg/day)
3 Interspecies variability · intraspecies variability · subchronic-to-chronic extrapolation
4 LOAEL-to-NAEL extrapolation

4.2 HUMAN HEALTH (PHYSICOCHEMICAL PROPERTIES)

No need for further information or for classification according to Directive 67/548/EEC arises in connection with the physicochemical properties of anthracene.

5 RESULTS

5.1 EXPOSURES RELATED TO THE PRODUCTION AND USE OF ANTHRACENE

Table 5.1 summarises the outcome of the risk assessment as regards exposures related to the production and use of anthracene. These conclusions are the following:

a) **Conclusion (ii)** has been reached for systemic toxicity via all routes of exposure considered with regard to occupational exposure during production and packaging of anthracene, during the use of anthracene for manufacture of pyrotechnics and with regard to exposure of the general population via the environment. This conclusion was reached on the basis of MOS values which in most cases exceeded the minimal MOS substantially. Furthermore, in those cases (the majority of scenarios) where modelled, rather than measured, maximum exposure levels were utilised, these levels are likely to have been highly conservative. Consequently adoption of **Conclusion (ii)** is based on a high confidence level.

b) **Conclusion (iii)** has been reached for skin phototoxicity via dermal exposure with regard to occupational exposure during anthracene production and packaging. In all cases, the maximal exposures estimated are higher than the human LOAEL as well as the animal LOAEL and NOEL, and the calculated MOS values are substantially smaller than the minimal MOS. Although the studies from which these limit values were derived were not based on a formally approved protocol, they appear to have been well conducted and well reported, providing clear evidence of induction of skin irritation after a single application of anthracene in combination with a dose of UVA radiation which can be readily received by a person exposed for a short time to natural sunlight.

Even if estimated exposures may exceed the true ones due to the conservative nature of the assumptions made during modelling, the degree by which the estimated MOS is exceeded by the minimal MOS is such as to suggest that there is significant concern for adverse effects and a need for limiting risks.

c) A significant lack of measured exposure data has been identified for workers involved in anthracene production from anthracene oil, anthracene packaging and anthracene use in the manufacture of pyrotechnics.

d) There is need for further information regarding the reproductive and developmental toxicity of anthracene, and studies (e.g. a test based on OECD TG 414 (Prenatal Developmental Toxicity Study)) would be required to fill this information gap.

On the other hand, it is noted that, as far as the situation in the EU in 2003 is concerned, only one production site exists in the EU, with 99% of production volume exported outside the EU and only a very minor use in pyrotechnics. There are no consumer exposures to the commercially-produced substance and human environmental exposures are very low. The potential for worker exposure using modelled estimates is low, and limited measured data and control measures, known to be applied at the production site, indicate that the model predictions are probably over-estimates. Finally, mechanistic considerations (absence of Ah receptor binding by anthracene) make it likely that any reproductive toxicity of anthracene would probably be weak. On this basis, the execution of the abovementioned reproductive toxicity study may not be required as long as the exposure situation does not change.

Further measured exposure data for workers involved in anthracene production from anthracene oil, anthracene packaging and anthracene use in the manufacture of pyrotechnics, would of course increase the level of confidence in this decision.

The situation should be closely monitored and if there are any indications that production and use patterns are changing the potential for increasing exposure should be reconsidered and the need to request a developmental toxicity study revisited.

e) Although anthracene has not been formally tested for the induction of skin irritation or sensitisation in the absence of UVA light, no convincing data have been found to support the suggestion that it has such activity. On the other hand, as stated above, in combination with UVA light it has clear photoirritating activity for the skin. Classification of substances as "phototoxic" is not currently foreseen by Directive 67/548/EEC or any of its derivative legislation. Because the levels of irradiant energy of a magnitude sufficient to elicit anthracene photoirritation can be received easily through short exposures to sunlight, it is proposed that anthracene must be considered in practice as a skin irritant and be classified as "irritating to the skin". In this case there would not be a need for further tests on its skin irritating or sensitising activity in the absence of UVA light.

5.2 EXPOSURES NOT DIRECTLY RELATED TO THE PRODUCTION OR USE OF ANTHRACENE

Table 5.2 summarises the outcome of risk assessment as regards exposures which are not related to the production or use of anthracene. This assessment has been included for illustrative purposes, and covers occupational activities such as use of anthracene for chemical synthesis (which was conducted in a European plant until recently), coal tar distillation, occupational exposure during creosote production, packaging and use in timber impregnation and wood brushing, occupational exposure during use of coal tar paints and related products and occupational exposure associated with industrial sources involving combustion of organic materials. It also includes consumer exposure during the use of coal tar paints and related products. In all cases, there was no concern for systemic toxicity, with a high degree of confidence, arising from exposure via inhalation or dermal contact. However, there were concerns for skin phototoxicity in all cases where dermal exposure was considered.

5.3 PHYSICOCHEMICAL PROPERTIES (HUMAN HEALTH)

No need for further information or for classification according to Directive 67/548/EEC arises in connection with the physicochemical properties of anthracene.

Table 5.1 Summary of risk assessment conclusions

Exposure/toxicity end-point	Conclusion
Occupational exposures	
Manufacture of anthracene from anthracene oil	
Systemic toxicity by inhalation	ii
Systemic toxicity by dermal exposure	ii
Systemic toxicity by combined inhalation and dermal exposure	ii
Dermal phototoxicity	iii
Anthracene packaging	
Systemic toxicity by ingestion of airborne dust	ii
Systemic toxicity by inhalation	ii
Systemic toxicity by dermal exposure	ii
Systemic toxicity by combined inhalation, oral and dermal exposure	ii
Dermal phototoxicity	iii
Use of anthracene in the manufacture of pyrotechnics	
Systemic toxicity by ingestion of airborne dust	ii
Systemic toxicity by inhalation	ii
Systemic toxicity by dermal exposure	ii
Systemic toxicity by combined inhalation, oral and dermal exposure	ii
Dermal phototoxicity	iii
Exposure of the general population via the environment	ii

Table 5.2 Summary of scenarios for which information can be found in this report regarding exposures not related to production and current uses of anthracene

Occupational exposure during use of anthracene for chemical synthesis (not in operation in Europe)
Systemic toxicity by inhalation
Systemic toxicity by dermal exposure
Systemic toxicity by combined inhalation and dermal exposure
Dermal phototoxicity
Coal tar distillation
Systemic toxicity by inhalation
Systemic toxicity by dermal exposure
Systemic toxicity by combined inhalation and dermal exposure
Dermal phototoxicity

Table 5.2 continued overleaf

Table 5.2 continued Summary of scenarios for which information can be found in this report regarding exposures not related to production and current uses of anthracene

Occupational exposure via creosote	
Creosote blending	
Systemic toxicity by inhalation	
Systemic toxicity by dermal exposure	
Systemic toxicity by combined inhalation and dermal exposure	
Dermal phototoxicity	
Creosote packaging	
Systemic toxicity by inhalation	
Systemic toxicity by dermal exposure	
Systemic toxicity by combined inhalation and dermal exposure	
Dermal phototoxicity	
Timber impregnation	
Systemic toxicity by inhalation	
Systemic toxicity by dermal exposure	
Systemic toxicity by combined inhalation and dermal exposure	
Dermal phototoxicity	
Creosote brushing	
Systemic toxicity by dermal exposure	
Dermal phototoxicity	
Occupational exposure from other industrial sources	
Coal processing and related activities	Systemic toxicity by inhalation
Carbon anode/graphite	Systemic toxicity by inhalation
Silicon carbide	Systemic toxicity by inhalation
Aluminium and other metals	Systemic toxicity by inhalation
Iron and steel	Systemic toxicity by inhalation
Occupational exposure from consumer products	
Exposure during creosote brushing	
Systemic toxicity by dermal exposure	
Dermal phototoxicity	
Exposure during the use of coal tar paints and related products	
Systemic toxicity by dermal exposure	
Dermal phototoxicity	
Consumer exposure during use of coal tar paints and related products	
Systemic toxicity by dermal exposure	
Dermal phototoxicity	

6 REFERENCES

Akhtar MN, Hamilton JG, Boyd DR, Braunstein A, Seifried HE and Jarina DM (1979) Anthracen 1,2-oxide: Synthesis and role in the metabolism of anthracene by mammals. J. Chem. Soc. Perkin Trans. **1**, 1442-1446.

Alison AC, Magnus IA and Yound MR (1966) Role of lysosomes and of cell membranes in photosensitization. Nature **209**, 874.

Amacher DE and Turner GN (1980) Promutagen activation by rodent-liver postmitochondrial fractions in the L5178YT/K cell mutation assay. Mutat. Res. **74**, 485-501.

Andersson K, Levin J-O and Nilsson C-A (1983) Sampling and analysis of particulate and gaseous PAH's from coal-tar sources in the working environment. Chemosphere **12**, 197-204.

Argenbright LW, Forbes PD and Stewart GJ (1980a) Quantitation of phototoxic hyperemia and permeability to protein: II. Inhibition by histamine (H1 and H2) receptor antagonists in mouse skin. J. Invest. Dermatol. **75**, 417-420.

Argenbright IW, Forbes PD and Stewart GJ (1980b) Quantitation of phototoxic hyperemia and permeability to protein: I. Inhibition by histamine (H1 and H2) and serotonin receptor antagonists in pig skin. Exp. Molec. Pathol. **32**, 154-161.

Becher G and Bjorseth A (1985) A novel method for the determination of occupational exposure to polycyclic aromatic hydrocarbons by analysis of body fluids. In: Cooke M, Dennis AJ, eds. Polynuclear aromatic hydrocarbons: Mechanisms, methods and metabolism. Columbus, OH: Battelle Press, 145-155.

Betts (2002) Personal communication with Betts.

Bjorseth A, Bjorseth O and Fjeldstad P (1978a) Polycyclic aromatic hydrocarbons in the work atmosphere: I. Determination of area specific concentrations and job specific exposure in a vertical pin Sonderberg aluminium plant. Scand. J. Work Environ. Health **4**, 212-223.

Bjorseth A, Bjorseth O and Fjeldstad P (1978b) Polycyclic aromatic hydrocarbons in the work atmosphere: II. Determination in a coke plant. Scand. J. Work Environ. Health **4**, 224-236.

Blackburn GM, Buckingham J, Fenwick RG, Taussig P and Thompson MH (1973) Photochemical binding of anthracene and other aromatic hydrocarbons to deoxyribonucleic acid with attendant loss of tritium. J. Chem. Soc. Perkin I, 2809-2813.

Blackburn GM and Taussig PE (1975) The photocarcinogenicity of anthracene: photochemical binding to deoxyribonucleic acid in tissue culture. Biochem. J. **149**, 289-291.

Bock FG and Burnham M (1961) The effect of experimental conditions upon the concentration of hydrocarbons in mouse skin after cutaneous application. Cancer Res. **21**, 510-515.

Bock FG and Mund R (1958) A survey of compounds for activity in the suppression of mouse sebaceous glands. Cancer Res. **18**, 887-892.

Bond JA, Baker SM and Bechtold WE (1985) Correlation of the octanol/water partition coefficient with clearance half-times of intratracheally instilled aromatic hydrocarbons in rats. Toxicology **36**, 285-95.

Boogaard PJ and van Sittert NJ (1995) Urinary 1-hydroxypyrene as biomarker of exposure to polycyclic aromatic hydrocarbons in workers in petrochemical industries: baseline values and dermal uptake. Sci. Total Environ. **163**, 203-9.

Boyland, E and Burrows H (1935) The experimental production of sarcoma in rats and mice by a colloidal aqueous solution of 1:2:5:6-dibenzanthracene. J. Pathol. Bacteriol. **41**: 231-238.

Boyland E and Levi AA (1935) Metabolism of polycyclic compounds. I. Production of dihydroxydihydroanthracene from anthracene. Biochem. J. **29**, 2679-2683.

Boyland E and Levi AA (1936a) Metabolism of polycyclic compounds. II. Production of dihydroxydihydroanthracene glycuronic acid from anthracene. Biochem. J. **30**, 728-731.

Boyland E and Levi AA (1936b) Metabolism of polycyclic compounds. III. Anthrylmercapturic acid. Biochem. J. **30**, 1225-1227.

Bridges BA, McGregor DB and Zeiger E. et al. (1981) Summary report on the performance of bacterial mutation assays. **In**: Evaluation of Short-term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, Vol. 1, F.J. de Serres and J. Ashby, Ed. Amsterdam, Elsevier, North Holland. 49-67.

Broman D, Naef C and Zebuehr Y (1991) Long-term high and low-volume air sampling of polychlorinated dibenzo-p-dioxins and dibenzofurans and polycyclic aromatic hydrocarbons along a transect from urban to remote areas on the Swedish Baltic coast. Env. Sci. Technol. **25**, 1841-1850.

Brookes P and Preston RJ (1981) Summary report on the performance of in vitro mammalian assays. **In**: Evaluation of Short-term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, Vol. 1, de Serres FJ and Ashby J (eds), Amsterdam, Elsevier, North Holland. 77-85.

Brune K, Kalin H, Schmidt R and Hecker E (1978) Inflammatory, tumor initiating and promoting activities of polycyclic aromatic hydrocarbons and diterpene esters in mouse skin as compared with their prostaglandin releasing protency in vitro. Cancer Lett. **4** (1978), 333-342.

Burnham K and Rahman M (1992) Effects of petrochemicals and ultraviolet radiation on epidermal IA expression in vitro. J. Toxicol. Environ. Health **35**, 175-85.

Carver JH, Machado ML and MacGregor JA (1986) Application of modified Salmonella/microsome prescreen to petroleum-derived complex mixtures and polynuclear aromatic hydrocarbons (PAH). Mutat Res, **174**, 247-253.

Cautreels W and van Cauwenberghe K (1978) Experiments on the distribution of organic pollutants between airborne particulate matter and the corresponding gas phase. Atmos. Environ. **12**, 1133-1141.

Chaloupka K, Santosstefano M, Goldfarb IS, Liu G, Myers MJ, Tsyrolv IB, Gelboin HV, Krishnan V and Safe S (1994) Aryl hydrocarbon (ah) receptor-independent induction of Cyp1a2 gene expression by acenaphthylene and related compounds in B6C3F1 mice. Carcinogenesis **15**, 2835-2840.

Chang LH (1943) The fecal excretion of polycyclic hydrocarbons following their administration to rats. J. Biol. Chem. **151**, 93-99.

Clar E (1964) Polycyclic Hydrocargbons, Academic Press **1**, 288-307.

Clayton GD and Clayton FE (1981) Patty's Industrial Hygiene and Toxicology, Volume 2A, 2B, 2C, Toxicology. 3rd ed. New York, John Wiley Sons.

Collin G and Zauder M (1982) Tar and Pitch. **In**: Ullmann's Encyclopedia of Technical Chemistry.

Collin G and Höke H (1985) Anthracene. In: Ullman's Encyclopedia of Industrial Chemistry, 5th Ed., Vol. 2.

CRC Handbook of Chemistry and Physics, (1987) CRC Press.

Crow KD, Alexander E, Buck WHL, Johnson BE, Magnus IA and Porter AD (1961) Photosensitivity due to pitch. Brit. J Dermatol, **73**, 220-232.

Danish EPA (1995) Human health and environmental concerns of creosote. Danish EPA, Copenhagen.

Dankovich DA, Wright CW, Zangar RC and Springer DL (1989) Complex mixture effects on the dermal absorption of benzoa]pyrene and other polycyclic aromatic hydrocarbons from mouse skin. J. Appl Toxicol. **9**, 239-244.

Dayhaw-Barker P, Sambuco CP, Forbes PD and Barker FM (1985) Development of an animal model to study the phototoxic effects of anthracene and UV in skin and ocular tissues. Photochem. Photobiol. **41**, 122S.

Dean J (1979) Lange's Handbook of Chemistry, 12th ed., Dean J (ed.).

Delgado-Rodriguez A, Ortiz-Marttelo R, Graf U, Villalobos-Pietrini R and Gomez-Arroyo S (1995) Genotoxic activity of environmentally important polycyclic aromatic hydrocarbons and their nitro derivatives in the wing spot test of Drosophila melanogaster. Mutat. Res. **341**, 235-47.

DiPaolo JA, Takano K and Popuscu NC (1972) Quantitation of chemically induced neoplastic transformation of BALB/3T3 cloned cell lines, Cancer Res. **32**, 2686-2695.

DiPaolo JA, Nelson RL, Donovan PJ and Evans CH (1973) Host-mediated in vivo-in vitro assay for chemical carcinogenesis, Arch. Pathol. **95**, 380-385.

Dufresne A, Lesage J and Perrault G (1987) Evaluation of occupational exposure to mixed dusts and polycyclic aromatic hydrocarbons in silicon carbide plants. Am. Ind. Hyg. Assoc. J. **48**, 160-6.

Elson LA et al. (1945) Biochem. J. **39**, 301-308.

Emmett EA (1986) Cutaneous and ocular hazards of roofers. Occup. Med. **1**, 307-22.

Fazio T and Howard JW (1983) **In**: Handbook of Polycyclic Aromatic Hydrocarbons, Bjoresth A (ed.), Marcel Dekker, Inc., New York.

Forbes PD, Davies RE and Urbach F (1976) Phototoxicity and photocarcinogenesis: comparative effects of anthracene and 8-methoxypsoralen in the skin of mice. Fd. Cosmet. Toxicol. **14**, 303-306.

Franck H-G and Stadlhofer JW (1987) Industrial Aromatic Chemistry, Raw Materials, Processes and Products.

Freeman AE, Weisburger EK, Weisburger JH, Wolford RG, Maryak JM and Huebner RJ (1973) Transformation of cell cultures as an indication of the carcinogenic potential of chemicals. J. Natl. Cancer Inst. **51**, 799-808.

Friedman MA and Staub J (1976) Inhibition of mouse testicular DNA synthesis by mutagens and carcinogens as a potential sample mammalian assay for mutagenesis. Mutat. Res. **37**, 67-76.

Fujikawa K, Fort FL, Samejima K and Sakamoto Y (1993) Genotoxic potency in Drosophila melanogaster of selected aromatic amines and polycyclic aromatic hydrocarbons as assayed in the DNA repair test. Mutat. Res. **290**, 175-82.

Garrod AN, Guiver R and Rimmer DA (2000) Potential exposure of amateurs (consumers) through painting wood preservative and antifoulant preparations. Ann. Occup. Hyg. **44**, 421-6.

Gershbein LL (1975) Liver regeneration as influenced by the structure of aromatic and heterocyclic compounds. Res. Commun. Chem. Pathol. Pharmacol. **11**, 445-466.

Geyer H, Politzki G and Freitag D (1984) Prediction of ecotoxicological behaviour of chemicals: relationship between n-octanol/water partition coefficient and bioaccumulation of organic chemicals by *alga chlorella*. Chemosphere **13**, 269-284.

Gloxhuber H (1970) Phototoxicity testing of cosmetic materials, J. Soc. Cosmetic Chemists **21**, 825-833.

Grote D (1979a) Űberprüfung des toxischen Wirkungsbildes nach einmaliner, oraler Applikation von 16,0 g/kg Kg2. Auftragsnummer 335a, S. 1-3, Huntingdon Research Centre, Deutschland, Münster, Upublished Report.

Grote D (1979b), Prüfung der Hautverträglichkeit nach einmaliger Applikation auf die intakte oder skarifizierte Haut beim Kaninchen, Auftragsunummer 335b, Huntington Research Centre, Deutschland, Münster, Upublished Report.

Grote D (1979c), Verträglichkeitspruefung am Auge nach einmaliger Applikation beim Kaninchen, Auftragsnummer 335c, Huntington Research Centre, Deutschland, Münster, Upublished Report.

Hall M and Grover PL (1987) Differential stereoselectivity in the metabolism of benzo[a]pyrene and anthracene by rabbit epidermal and hepatic microsomes. Cancer Letters **38**, 57-64.

Harper KH (1959) The intermediary metabolism of polycyclic hydrocarbons. Brit. J. Cancer **13**, 718-731.

Hashimoto Y, Tokura K, Kishi H and Strachan WMJ (1984) Prediction of seawater solubility of aromatic compounds. Chemosphere **13**, 881-888.

Hausigk D and Koelling G (1968) Űber Reaktionen von partiell hydrierten mehrkernigen aromatischen Verbindungen mit 1,4-dichlor-butan. Chem. Ber. **101**, 469-472.

Heikkila PR, Hameilae M, Pyy L and Raunu P (1987) Exposure to creosote in the impregnation and handling of impregnated wood. Scan. J. Work Environ. Health **13**, 431-437.

Heller W (1950) Experimental study on tumours produced by light. 2. Tumours produced by light through photosensitisation (Ger.). Strahlentherapie **81**, 529-548. Cited in IARC (1983b).

Hoch-Ligeti C (1941) Studies on the changes in the lymphoid tissue of mice treated with carcinogenic and non-carcinogenic hydrocarbons. Cancer Res. **1**, 484-488. Described in: Faust RA (1991) Toxicity summary for antrhacene. Report prepared for the Oak Ridge Reservation Environmental Restoration Program.

Höke (2000) Personal communication with Höke.

Höke (2002) Personal communication with Höke.

Höke (2003) Personal communication with Höke.

Hopia A, Pyysalo H and Wickström K (1986) Margarines, butter and vegetable oils as sources of polycyclic aromatic hydrocarbons. J. Am. Oil Chem. Soc. **63**, 889-93.

HSE (2002) Personal communication from the Health and Safety Executive (UK).

IARC (1983a) International Agency for Research on Cancer Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Humans. Vol. 32, Bitumens, coal-tars and derived products, shale-oils and soots, Part 1.

IARC (1983b) International Agency for Research on Cancer Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Humans, Vol. 32, Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. Anthracene, 105.

Ingram LL Jr, McGinnis GD, Gjovik KR et al. (1982) Migration of creosote and its components from treated piling sections in marine environments. Proceedings of the American Wood Preservation Association, **78**, 120-128.

IPCS (International Programme of Chemical Safety) (1998) Environmental Health Criteria. 202. Selected non-heterocyclic polycyclic aromatic hydrocarbons. World Health Organisation, Geneva.

Jandris LJ and Forcė RK (1983) Determination of polycyclear aromatic hydrocarbons in vapor phases by laser-induced molecular fluorescence. Anal. Chim. Acta **151**, 19-27.

Joshi PC and Pathak MA (1984) The role of active oxygen ($^1O_2$ and $O_2^{.-}$) induced by crude coal tar and its ingredients used in photochemotherapy of skin diseases. J. Invest. Dermatol. **82**, 67-73.

Kaidbey KH and Nonaka S (1984) Action spectrum for anthracene-induced photosensitization of human skin, Photochem. Photobiol. **39**, 375-378.

Karcher W et al. (eds.) (1985) .Spectral Atlas of Polycyclic Aromatic Compounds, Karcher W. et al. (eds.).

Karickhoff SW et al. (1979), Sorption of hydrophobic pollutants on natural sediments. Water Res. **13**, 241-248.

Kennaway FL (1924a) On cancer-producing factor in tar. Brit. Med. J. **1**, 564-567. Cited in IARC (1983b).

Kennaway FL (1924b) On cancer-producing tars and tar-fractions. J. Ind. Hyg. **5**, 462-488. Cited in IARC (1983b).

Kleffner HW, Talbiersky J and Zander M (1981), Simple scheme for the estimation of concentrations of polycyclic aromatic hydrocarbons in tars. Fuel **60**, 361-362. Cited in IARC (1983a).

Kochevar IE, Armstrong RB, Einbinder J, Walther RR and Harber LC (1982) Coal tar photoxocitiy: active compounds and action spectra. Photochem. Photobiol. **36**, 65-69.

Kveseth K and Sortland B (1982) Polycyclic aromatic hydrocarbons in sewage, mussels and tap water. Chemosphere **11**, 623-39.

Lake RS, Kropko ML, Pezzutti MR, Shoemaker RH and Igel HJ (1978) Chemical induction of unscheduled DNA synthesis in human skin epithelial cell cultures. Cancer Res. **38**, 2091-2098.

Lang KF and Eigen I (1967), Forstschr. Chem. Forsch. **8**, 91-141.

LaVoie JE, Coleman DT, Rice JE, Geddie NG and Hoffmann D (1985) Tumor-initiating activity, mutagenicity and metabolism of methylated anthracenes. Carcinogenesis **10**, 1483-1488.

Lax E and Synowietz C (1964) Taschenbuch für Chem. und Physicker. Lax E and Synowietz C (eds.).

LeBoeuf RA, Kerckaert GA, Aardema MJ, Gibson DP, Brauninger R and Isfort RJ (1996) The pH 6.7 Syrian hamster embryo cell transformation assay for assessing the carcinogenic potential of chemicals. Mutat. Res. **21**, 85-127.

Lehman E, Auffarth J and Hager J (1984) Determination of selected polycyclic aromatic hydrocarbons in coal-tar, coal-tar pitch and tar products. Staub-Reinhalt. Luft **44**, 452-455.

Lesage J, Perrault G and Durand P (1987) Evaluation of worker exposure to polycyclic aromatic hydrocarbons. Am. Ind. Hyg. Assoc. J. **48**, 753-9.

Lide DR (1991) CRC Handbook of Chemistry and Physics, 72nd ed., Lide DR (ed.), CRC Press, Boca Raton.

Lo M and Sandi E (1978) Polycyclic aromatic hydrocarbons in foods. **In**: Gunter FA (ed.) Residue Review. Springer-Verlag, New York, **69**, 35-86.

Lorenz LJ and Gjovik LR (1972) Analysing creosote by gas chromatography. **In**: Proceedings of the Annual Meeting of the American Wood-Preservers' Association, 32-42.

Lovell WW and Sanders DJ (1992) Phototoxicity testing in guinea-pigs. Food Chem. Toxic. **30**, 155-160.

Machala M, Vondracek J, Blaha L, Ciganek M and Neca JV (2001) Aryl hydrocarbon receptor-mediated activity of mutagenic polycyclic aromatic hydrocarbons determined using in vitro reporter gene assay. Mutat. Res. **497**, 49-62.

Mackay, D. and Shiu, W. Y., "Aqueous Solubility of Polynuclear Aromatic Hydrocarbons", J. Chem. Eng. Data, **22(4)**, 399-402 (1977).

Maga JA (1986) Polycyclic aromatic hydrocarbon (PAH) composition of mesquite (Prosopis fuliflora) smoke and grilled beef. J. Agric. Food Chem. **34**, 249-51.

Marlich LI and Leukevitch ZK (1973) Capillary chromatograph as a method for the rapid determination of the main components in coal tar and coal tar fractions. Zh. ana. Khim. **28**, 1193-1198. Cited in IARC (1983a).

Martin CN, McDermid AC and Garner RC (1978) Testing of known carcinogens and noncarcinogens for their ability to induce unscheduled DNA synthesis in HeLa cells, Cancer Res. **38**, 2621-2627.

Masclet P, Pistikopoulos P, Beyne S and Mouvier G (1988) Long range transport and gas-particle distribution of polycyclic aromatic hydrocarbons at a remote site in the Mediterranean sea, Atmos. Environ. **22**, 639-50.

May W, Walik SP, Miller MM, Tewari UB, Brown-Thomas JM and Goldgerg RN (1983) Solution thermodynamics of some slightly soluble hydrocarbons in water. J. Chem. Eng. Data **28**, 197-200.

McNeil D (1983) Tar and Pitch. **In**: Encyclopedia of Chemical Technology. Kirk RE and Othmer DF (eds.), 3rd edn.

Mellon Institute (1977) Range Finding Toxicity Tests on Rabbits and Rats, EPA Doc. No. 86-870001568, Fiche No. OTS0516149.

Menichini E, Bocca A, Merli F, Ianni D and Monfredini F (1991) Polycyclic aromatic hydrocarbons in olive oils on the Italian market. Food Addit. Contam. **8**, 363-69.

Miescher G (1942) Experimental studies on the formation of tumours by photosensitisation (Ger.) Schweiz. med. Wochenschr. **72**, 1082-1084. Cited in IARC (1983b).

Mitchell CE (1982) Distribution and retention of benzo[a]pyrene in rats after inhalation. Toxicol. Lett. **11**, 35-42.

Montizaan GK, Kramers PGH, Janus JA and Posthumus R (1989) Integrated criteria document PAH: Effects of 10 selected compounds. Appendix to Report No. 758474011. Bilthoven, National Institute of Public Health and Environmental Protection.

Moseley H, Diffey BL, Marks JM and Mackie RM (1981) Personal solar UV-A doses received by patients undergoing oral psoralen photochemotherapy for psoriasis. Brit. J. Dermatol. **105**, 573-7.

Myers SR, Blake JW and Flesher JW (1988) Bioalkylation and biooxidation of anthracene, in vitro and in vivo. Biochem. Biophys. Res. Comm. **151**, 1441-1445.

Nabert K and Schön G (1963) Sickerheitstechnische Kennzahlen brennbarer Gase und Dämpfe, 2nd ed.

Nagornyi PA and Rodionov GA (1969) Data on the toxicology of anthracenetoluolphenol-formaldehyde resin. Gig. Tr. Prof. Zabol. **13**, 59-61 (Russ.).

Neff JM (1979) Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fates and biological effects. Neff JM (ed.), Applied Science Publisheres Ltd., London.

Nestler FHM (1974) The characterization of wood-reserving creosote by physical and chemical methods of analysis (USDE Forest Service Research Paper FPL 195), U.S. Department of Agriculture.

Nousiainen U, Torronen R and Hanninen O (1984) Differential induction of various carboxylesterases by certain polycyclic aromatic hydrocarbons in ther at. Toxicology **32**, 243-251.

Novotny M, Strand JW, Smith SL, Wieslder D and Schwende FJ (1981) Compositional studies of coal tar by capillary gas chromatograph/mass spectrometry. Fuel 60, 213-220. Cited in IARC (1983a).

Old LJ, Benacerraf B and Carswell E (1963) Contact reactivity to carcinogenic polycyclic hydrocarbons. Nature, **198**, 1215-1216.

Oshiro Y, Balwierz PS, Soelter SG, Guzzie PJ and Rohrbacher E (1992) Evaluation of mouse peripheral blood micronucleus assay. Environm. Molec. Mutagen. **19**, 42.

Perry PE and Thomson EJ (1981) Evaluation of the sister chromatid exchange method in mammalian cells as a screening system for carcinogens, in: evaluation of short-term tests for carcinogens: Report of the International Collaborative Program. Prog. Mutat. Res. 1, (F.J. de Serres, J. Ashby, eds.), p. 560-569, Elsevier North-Holland, New York, Amsterdam, Oxford.

Petry T, Schmid P and Schlatter C (1996a) The use of toxic equivalency factors in assessing occupational and environmental health risk associated with exposure to airborne mixtures of polycyclic aromatic hydrocarbons (PAHs). Chemosphere **32**, 639-48.

Petry T, Schmid P and Schlatter C (1996b) Airborne exposure to polycyclic aromatic hydrocarbons (PAHs) and urinary excretion of 1-hydroxypyrene of carbon anode plant workers. Ann. Occup. Hyg. **40**, 345-57.

Piet GJ and Morra CF (1983) **In**: Artificial Groundwater Recharge. Huisman L and Olsthorn TN (eds.) Pitman Publ, 29-42.

Pollia JA (1939) Investigations on the possible carcinogenic effect of anthracene and chrysene and some of their compounds. J. Indust. Hyg. **21**, 219-220. Cited in IARC (1983b).

Pollia JA (1941) Investigations on the possible carcinogenic effect of anthracene and chrysene and some of their compounds. II. The effect of subcutaneous injection in rats. J. Ind. Hyg. Toxicol. **23**, 449-451.

Purchase IFH, Longstaff E, Ashby J, Styles JA, Anderson D, Lefevre PA and Westwood FR (1976) Evaluation of six short term tests for detecting organinc chemical carcinogens and recommendations for their use. Nature **264**, 624-627.

Rahman A, Barrowman JA and Rahimtula A (1986) The influence of bile on the bioavailability of polynuclear aromatic hydrocarbons from the rat intestine. Can. J. Physiol. Pharmacol. **64**,1214-8.

Reddy MV, Gupta RC, Randerath E and Randerath K (1984) ^{32}P-Postlabeling test for covalent DNA binding of chemicals in vivo: application to a variety of aromatic carcinogens and methylating agents. Carcinogenesis **5**, 231-43.

Riala R, Heikkila P and Kanerva L (1998) A questionnaire study of road pavers' and roofers' work-related skin symptoms and bitumen exposure. Int. J. Dermatol. **37**, 27-30.

Rigdon RH and Giannukos NJ (1964) Effect of carcinogenic hydrocarbons on growth of mice. Arch. Path. **77**, 112-118.

Rispler J and Urbanek RW (1978) Photodynamic induction of remission in psoriasis with anthracene and long ultraviolet irradiation. Photochem. Photobiol. **41**, 103A.

Roff MW (1997) Dermal exposure of amateur or non-occupational users to wood-preservative fluids applied by brushing outdoors. Ann. Occup. Hyg. **41**, 297-343.

Roszinksy-Koecher G, Basler A and Roehrborn G (1979) Mutagenicity of polycyclic aromatic hydrocarbons. 5. Induction of sister-chromatid exchanges in vivo. Mutat. Res. **66**, 65-67.

Russel H (1947) An unsuccelful attempt to induce gliomata in rabbits with chloanthrene. J. Pahtol. Bacteriol. 59, 481-483. Cited in IARC (1983b).

Sakai M, Yoshida D and Mizusaki S (1985) Mutagenicity of polycyclic aromatic hydrocarbons and quinones on Salmonella typhimurium TA97. Mutat. Res. **156**, 61-67.

Salaman MH and Roe FJC (1956) Further tests for tumour-initiating activity: N,N-Di-(2-chloroethyl)-p-aminophenyl-butyric acid (CD1348) as an initiatior of skin tumour formation in the mouse. Br. J. Cancer **10**, 363-378.

Salamone MF (1981) Toxicity of 41 carcinogens and noncarcinogenic analogs. **In**: Evaluation of short-term tests for carcinogens, Progress in mutation research, de Serres FJ and Ashby J (eds.), Elsevier North-Holland, New York, Amsterdam, Oxford. **1**, 682-685.

Sangster J (1989) Octanol-water partition coefficients of simple organic compounds. J. Phys. Chem. Reference Data **18**, 111-1129.

Santodonato J et al. (1981) Hazard Profiles on PAH. Syracuse, NY: Syracuse Res Corp p. 53 TR 81-633.

Sartorelli P, Cenni A, Matteucci, Montomoli L, Novelli MT and Palmi S (1999) Dermal exposure assessment of polycyclic aromatic hydrocarbons: in vitro percutaneous penetration from lubricating oil. Int. Arch. Occup. Environ. Health **72**, 528-232.

Sax NI (1975) Dangerous properties of industrial materials, van Nostrand Reinhold Company, New York; 8th edition.

Sax NI (1992) Dangerous properties of industrial materials, van Nostrand Reinhold Company, New York; 8th edition.

Schmahl D (1955) Examination of the carcinogenic action of naphthalene and anthracene in rats. Z. Krebsforsch. **60**, 697-710.

Schothorst AA, Suurmond D and de Lijster A (1979) A biochemical screening test for the photosensitizing potential of drugs and disinfectants. Photochem Photobiol. **29**, 531-7.

Schwartz FP (1977) Determination of temperature dependence of solubilities of polycyclic aromatic hydrocarbons in aqueous solutions by a fluorescence method. J. Chem. Eng. Data **22**, 273-277.

Scribner JD (1973) Tumor initiation by apparently noncarcinogenic polycyclic aromatic hydrocarbons, J. Natl. Cancer Inst. **50**, 1717-1719.

Shabad LM, Sorikina JD, Golub NI and Bogovski SP (1972) Transplacental effect of some chemical compounds on organ cultures of embryonic kidney tissue. Cancer Res. **32**, 617-627.

Shubik P and Della Porta G (1957) Carcinogenesis and acute intoxication with large doses of polycyclic hydrocarbons. Am. Med. Ass. Arch. Path. **64**, 691-703.

Sims P (1964) Metabolism of polycyclic compounds. 25. The metabolism of anthracene and some related compounds in rats. Biochem. J. **92**, 621-631.

Sinha and Chignell (1983) Binding of anthracene to cellular macromolecules in the presence of light. Photochem. Photobiol. **37**, 33-37.

Smith DJT and Harrison RM (1996) Concentrations, trends and vehicle source profile of polynuclear aromatic hydrocarbons in the U.K. atmosphere. Atmosph. Environ. **30**, 2513-25.

Sofuni T, Hayashi M, Matsuoka A, Sawada M, Hatanaka M and Ishidate M (1985) Mutagenicity tests on organic chemical contaminants in city water and related compounds. II. Chromosome aberration tests in cultured mammalian cells. Bull. Natl. Inst. Hyg. Sci. (Tokyo), 64-75.

Sonnefeld WJ, Zoller WH and May WE (1983) Dynamic coupled-column liquid chromatographic determination of ambient temperature vapor pressures of polynuclear aromatic hydrocarbons. Anal. Chem. **55**, 275-280.

Sorgo PM (1996) Report of the European Commission (DG III) on the justification of the Danish position concerning the Directive on creosote.

Sorokina ID (1971) Transplacental effect of 7,12-dimethylbenz(a)anthracene and its non-carcinogenic analog-anthracene in organ cultures of mouse embryonal kidneys. Bull. Eksp. Biol. Med. **72**, 86-9.

Spano MA, Frei H, Wurgler FE and Graf U (2001) Recombinagenic activity of four compounds in the standard and high bioactivation crosses of Drosophila melanogaster in the wing spot test. Mutagenesis **16**, 385-94.

Speer K, Steeg E, Horstmann P, Kühn Th and Montag A (1990) Determination and distribution of polycyclic aromatic hydrocarbons in native vegetable oils, smoked fish products, mussels and oysters, and bream from the River Elbe. J. High Resol. Chromatogr. **13**, 104-11.

Stanton MF, Miller E, Wrench C and Blackwell R (1972) Experimental induction of epidermoid carcinoma in the lungs of rats by cigarette smoke condensate. J. Natl. Cancer Inst. **49**, 867-877.

Staesse HL (1954) Fractional distillation of creosote and composition of preservatives used in the cooprative creosote programme. Proc. American Wood Preservers' Association **50**, 13-40.

Storer JS, DeLeon I, Millikan LE, Laseter JL and Griffing C (1984) Human absorption of crude coal tar products. Arch. Dermatol. **120**, 874-7.

Thrane KE and Mikalsen A (1981) High-volume sampling of airborne polycyclic aromatic hydrocarbons using glass fiber filters and polyurethane foam. Atmos. Environ. **15**, 909-918.

Thyssen J (1979) Bayer AG, Untersuchung zur Haut- und Schleimhautverträglichkeit, Institut für Toxikologie, Wuppertal-Elberfeld.

Torronen R, Nousiainen U and Hanninen O (1981) Induction of aldehyde dehydrogenase by polycyclic aromatic hydrocarbons in rats. Chem.- Biol. Interact. **36**, 33-44.

Traul KA, Takayama K, Kachevsky V, Hink RJ and Wolff JS (1981) A rapid in vitro assay for carcinogenicity of chemical substances in mammalian cells utilizing an attachment-independence endpoint. 2 - Assay validation. J. Appl. Toxicol. **1**, 190-5.

US EPA (1987) Health and Environmental Effects Profile for Anthracene, US Environmental Protection Agency, Washington, DC.

US EPA (1989) Subchronic toxicity study in mice with anthracene. HLA Study No. 2399-131. Final Report. Prepared by Hazelton Laboratories America, Inc., Rockville, MD, for the Office of solid Waste, US Environmental Protection Agency, Washington, DC.

van Bladeren PJ, Sayer JM, Ryan DE, Thomas PE, Levin W and Jerina DM (1985) Differential stereoselectivity of cytochromes P-450b and P-450c in the formation of naphthalene and anthracene 1,2-oxides. The role of epoxide hydrolase in determining the enantiomer composition of the 1,2-dihydrodiols formed. J. Biol. Chem. **260**, 10226-35.

van Rooij JG, van Lieshout MA, Bodelier-Bade MM and Jongeneelen FJ (1993) Effect of the reduction of skin contamination on the internal dose of creosote workers exposed to polycyclic aromatic hydrocarbons. Scand. J. Work Environ. Health **19**, 200-207.

van Rooij JG, Vinke E, De Lange J, Bruijnzeel PL, Bodelier-Bade MM, Noordhoek J and Jongeneelen FJ (1995) Dermal absorption of polycyclic aromatic hydrocarbons in the blood-perfused pig ear. J. Appl. Toxicol. **15**, 193-200.

Volkova NI (1983) Anthracene and derivatives. **In**: Encyclopaedia of Occupational Health and Safety. Parmeggiani L (ed.), International Labour Office, Geneva, **1**, 162-163.

WEI (2000) Personal communication from the West European Institute for Wood Preservation.

Welch RM, Harrison YE, Gommi BW, Poppers PJ, Finster M and Conney AH (1969) Stimulatory effect of cigarette smoking on the hydroxylation of 3,4-benzpyrene and the N-demethylation of 3-methyl-4-monomethylaminoazobenzene by enzymes in human placenta, Clin. Pharm. Therapeut. **10**, 100-109.

Welch RM, Gommi B, Alvares AP and Conney AH (1972) Effect of enzyme-induction on the metabolsim of benzo(a)pyrene and 3-methyl-4-monomethylaminoazobenzene in the pregnant and fetal rat. Cancer Res, **32**, 973-978.

Weyand EH and Bevan DR (1986) Benzo(a)pyrene disposition and metabolism in rats following intratracheal instillation. Cancer Res. **46**, 5655-61.

White KL, Lysy HH and Holsapple MP (1985) Immunosuppression by polycyclic aromatic hydrocarbons. A structure-activity relationship in B6CeF1 mice and DBA/2 mice. Immunopharmacol. **9**, 155-164.

Wickström K, Pyysalo H, Plaami-Heikkilä S and Tuominen J (1986) Polycyclic aromatic compounds (PAC) in leaf lettuce. Z. Lebensm. Unters Forsch. **183**, 182-5.

Williams GM (1988) Detection of chemical carcinogens by unscheduled DNA synthesis in rat liver primary cell cultures, Cancer Res. **37**, 1845-1851.

Whitehouse BG (1984) The effects of temperature and salinity on the aqueous solubility of polynuclear aromatic hydrocarbons. Marine Chem. **14**, 319-332.

WHO (1994) Ultraviolet Radiation. Environmental Health Criteria (2nd edition), vol. 160, World Health Organisation, Geneva.

Worstmann W (1981) Akute Toxizität von Produkt-Nr. 110.147 (Anthracen) an männlichen und weiblichen Ratten nach dermaler Applikation, Auftragsnummer 874/4/81, S. 1-8, Huntingdon Research Centre Deutschland, Münster, Unpublished Report.

Wynder EL and Hoffman D (1959) A study of tobacco carcinogenesis. VII. The role of higher polycyclic hydrocarbons. Cancer **12**, 1079-1086.

Yamazaki H, Imamura E, Kamei S, Yamauchi A, Kakiuchi Y and Tai HH (1990) Polycyclic aromatic hydrocarbons affect the calcium ionophore induced activation of rabbit platelet. Chemosphere **21**. 21-28.

Yang JJ, Roy TA and Mackerer CR et al. (1986) Percutaneous absorption of anthracene in the rat: comparison of in vivo and in vitro results.Tox. Ind. Health **2**, 79-84.

Yoshida K, Shigeoka T and Yamauchi F (1983), Relationship between molar refraction and n-octanol/water partition coefficient. Ecotox. Environ. Safety **7**, 558-565.

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 II 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 II 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
ECDIN	Environmental Chemicals Data and Information Network
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 II 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 tonnes/annum)
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 II 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/oo	Parts per thousand
O	Oxidising (Symbols and indications of danger for dangerous substances and preparations according to Annex II 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

PAH	Polycyclic aromatic hydrocarbons
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 IV of Directive 67/548/EEC
SAR	Structure-Activity Relationships
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange
SCHER	Scientific Committee on Health and Environmental Risks
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 II 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 Organisation
WWTP	Waste Water Treatment Plant
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)

European Commission

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anthracene – part II – human health, **Volume 78**

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The report provides the comprehensive risk assessment of the substance Anthracene. It has been prepared by Greece 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.

Part I - Environment

This part of the evaluation has not been finalised yet.

Part II – Human Health

This part of the evaluation considers the emissions and the resulting exposure to human populations in all life cycle steps. The scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.

The human health risk assessment concludes that there is concern for workers with regard to skin phototoxicity as a consequence of dermal exposure. There is a need for further information and for testing (on hold) on reproductive and developmental toxicity.

For consumers, humans exposed via the environment and for human health (physico-chemical properties) there is no concern.

The conclusions of this report will lead to risk reduction measures to be proposed by the Commission's committee on risk reduction strategies set up in support of Council Regulation (EEC) N. 793/93.

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Part II – human health

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