

Annex VI report

PROPOSAL FOR HARMONISED CLASSIFICATIONS AND LABELLING

Substance Name: Pitch, coal tar, high temp.

EC Number: 266-028-2

CAS Number: 65996-93-2

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PROPOSAL FOR HARMONISED CLASSIFICATION AND LABELLING

Substance Name: Pitch, coal tar, high temp.

EC Number: 266-028-2

CAS number: 65996-93-2

Registration number (s):

Purity:

Impurities:

Proposed classification based on Directive 67/548/EEC:

Human Health hazards

Carc. Cat. 1, R45

Muta. Cat. 2, R46

Repr. Cat. 2, R60-61

Environment

N, R50/53

Proposed classification based on Regulation EC 1272/2008:

Human health hazards

Carc. 1A; H350

Muta. 1B; H340

Repr. 1B; H360FD

Environment

Aquatic Acute 1; H400

Aquatic Chronic 1; H410

Proposed labelling based on Directive 67/548/EEC:

Symbol: T, N

Risk phrases:

R45	May cause cancer.
R46	May cause heritable genetic damage.
R60	May impair fertility.
R61	May cause harm to the unborn child.
R50/53	Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment

Safety phrases: 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).

S60/61 This material and its container must be disposed of as hazardous waste. Avoid release to the environment.

Proposed labelling based on Regulation EC 1272/2008:

Signal word: Danger

Symbol: GHS08 Germ cell mutagenicity, hazard category 1B; Carcinogenicity, hazard category 1A; Reproductive toxicity, hazard category 1B.

GHS09 Hazardous to the aquatic environment.

Hazard statement codes: H340 May cause genetic defects.

H350 May cause cancer.

H360 May damage fertility or the unborn child.

H400 Very toxic to aquatic life.

H410 Very toxic to aquatic life with long lasting effects.

As precautionary statements are not included in Annex VI of Regulation EC 1272/2008, no proposal is made.

Proposed specific concentration limits (if any):

-

Proposed notes (if any):

-

JUSTIFICATION

Considerations about the classification and labelling of CTPHT

Coal tar pitch, high temperature (CTPHT) is a UVCB substance and possibly contains thousands of substances, and because of this complexity and variability of CTPHT, great difficulties have been encountered in assessing exposure in the epidemiological studies. Generally, the presence of coal tars and derived products is detected by the presence of their specific constituents, especially coal tar pitch volatiles (CTPV) and Polycyclic Aromatic Hydrocarbons (PAHs) (IARC, 1985).

The database on possible health hazards induced by CTPHT is rather limited, and it is, therefore, hardly possible to perform a full hazard assessment for all the required endpoints. There is, though, quite some information from epidemiological studies on workers in specific industrial processes where CTPHT is produced and/or used, that indicate that carcinogenicity is a hazard associated with CTPHT. This is attributed to the presence of the PAHs in CTPHT. Given the uncertainties with respect to the effects of other chemical constituents of CTPHT (and related substances), it is not completely sure that carcinogenicity is the only relevant effect of CTPHT. However, as it is also noted that the carcinogenic potencies of these PAHs are quite high, limitation of the risks for cancer will automatically reduce the risk for any other possible human health effect, quite possibly even to zero. Therefore, in view of the limited database, it is decided that this classification and labelling report will focus on the CMR properties, using PAHs as guidance substances, in particular benzo[a]pyrene (see for further information Sections 5.7, 5.8, and 5.9).

For the environmental hazard assessment it is well realised that each of the many substances that constitute coal tar pitch may be relevant for the receiving environment. In this report, however, the hazard assessment will be focused on the hazards of polycyclic aromatic hydrocarbons (PAHs) only. Based on the available information, it is only for the 16 homocyclic PAHs that were defined as priority substances by the United States Environmental Protection Agency (US EPA; <http://www.epa.gov/waterscience/methods/pollutants.htm>; further referred to as EPA-PAHs) that sufficient effect and exposure data are available. It is for this reason that the hazard assessment of CTPHT for the environment is restricted to this group of PAHs, accepting that the potential risk of CTPHT might be underestimated (see for further information Section 7).

1 IDENTITY OF THE SUBSTANCE AND PHYSICAL AND CHEMICAL PROPERTIES

1.1 Name and other identifiers of the substance

There are various ‘pitches’, which all exist of mixtures of an enormous range of individual substances. The individual substances - if identified - have usually a CAS registry number. Furthermore, a whole range of CAS numbers occurs in literature for pitches. Many of these CAS numbers can not be found in the official CAS listings. Council Directive 76/769/EEC (EU, 1976) gives a comprehensive listing of “substances”, amongst others “pitches”, consisting of mixtures of individual substances. Another comprehensive list of various mixtures that may contain polycyclic aromatic hydrocarbons (PAHs) is found in Appendix A of (US-EPA, 1999). Some of the coal tar pitches present in these lists are shown in Table 1.1.1.

The feedstock for the production of coal tar pitch high temperature (CTPHT) is tar, coal, high-temperature with CAS number 65996-89-6. This “substance” is defined in Council Directive 76/769/EEC (EU, 1976) as ‘the condensation product obtained by cooling, to approximately ambient temperature, of the gas evolved in the high temperature (greater than 700 °C (1292 °F))

destructive distillation of coal. Coal tar is a black viscous liquid, denser than water and composed primarily of a complex mixture of condensed ring aromatic hydrocarbons. It may contain minor amounts of phenolic compounds and aromatic nitrogen bases'. Coal tars are condensation products obtained during the production of coke and/or natural gas through the destructive distillation of coal, called carbonisation or coking. The composition and properties of a coal tar (and coal tar pitch derived thereof) depend mainly on the temperature of carbonisation and, to a lesser extent, on the nature of the coal used as feedstock.

Table 1.1.1 Different types of pitches as defined in official listings.

CAS number	Product ¹⁾	List
61789-60-4	Pitch	(EU, 1976)
65996-93-2	<i>Pitch, coal tar, high temperature</i>	(EU, 1976; US-EPA, 1999)
92061-94-4	Residues (coal tar), pitch distillation	(EU, 1976; US-EPA, 1999)
94114-13-3	Pitch, coal tar, high temperature, secondary	(EU, 1976; US-EPA, 1999)
121575-60-8	Pitch, coal tar, high temperature, heat treated	(EU, 1976; US-EPA, 1999)

¹⁾ All of these products are classified in Annex VI of Regulation (EC) 1272/2008 as Category 1B Carcinogen (in Table 3.1) and Category 2 Carcinogen (in Table 3.2).

The distillation of high-temperature coal tars results in tar oils (including naphthalene oil, creosote oil, anthracene oil, and creosote) and a solid fraction (CTPHT) (IARC, 1985). When CTPHT is heated, coal tar pitch volatiles (CTPV) are released (ATSDR, 2002). However, the term CTPV is not only used for volatiles released when coal tar pitch (CTP) is heated, but also for volatiles released when coal tar or its products are heated (HSDB, 2004). Figure 1.1.1 shows how CTPHT and the other coal tar products are produced. A definition of the coal tar products can be found in the glossary (see Annex I).

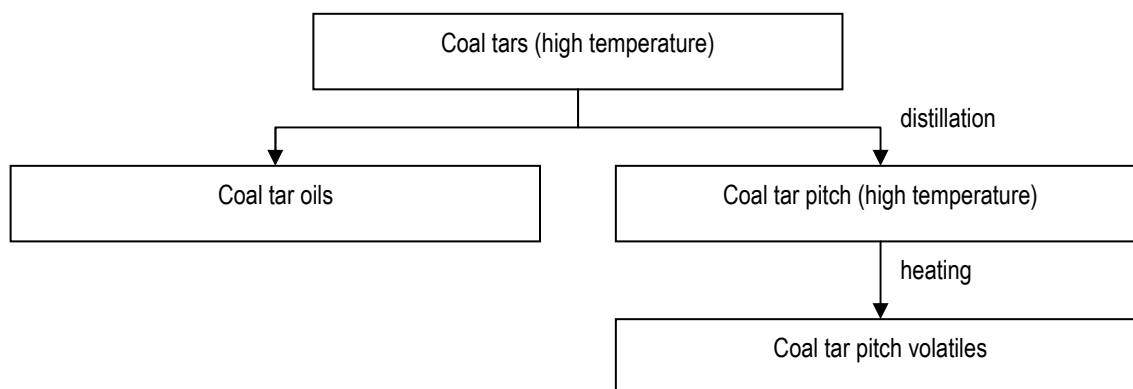


Figure 1.1.1. Origin of high temperature coal tar products.

This proposal concerns only CTPHT with CAS # 65996-93-2.

CAS Number: 65996-93-2¹

EINECS Number: 266-028-2

IUPAC Name: not applicable

Molecular formula: not applicable for UVCB² compounds; coal tar pitch high temperature is a complex hydrocarbon mixture consisting of three- to seven-membered condensed ring aromatic hydrocarbons (90%) and of high molecular weight compounds. Besides these polycyclic aromatic hydrocarbons and their (poly)methylated derivatives, it contains heterocyclic compounds and benzocarbazoles (Steinhauser, 1997).

Structural formula: not applicable

Molecular weight: not applicable

Synonyms: anode pitch, binder pitch, clay pigeon binder, electrode pitch, hard pitch, impregnating pitch, pitch, soft pitch, vacuum pitch

1.2 Composition of the substance

CTP and related substances like CTPV, creosotes and tars are complex and have variable compositions. CTP is a complex hydrocarbon mixture consisting of three- to seven-membered condensed aromatic hydrocarbons and of high molecular weight compounds. It is a shiny, dark brown to black solid produced during the distillation of coal tars. Coal tars are the condensation products obtained by cooling of the gas evolved in the carbonisation of coal. The relative proportions of the components in the mixture of CTP are complex and variable and dependent on whether low temperature or high temperature processes were involved in the production of the tar. Over 400 compounds have been identified in coal tars, and probably as many as 10,000 are actually present (Trosset et al., 1978; McNeil, 1983; both cited in IARC, 1985). The number of compounds present in most coal tar pitches is estimated in the thousands. Because of variation in source materials and manufacturing processes, including different temperatures and times of carbonization, no two coal tars or pitches are chemically identical. In general, however, approximately 80% of the total carbon present in coal tars exists in aromatic form. Volatile fumes, designated CTPV, are released when coal tar, CTP, or their products, are heated (HSDB, 2004).

¹ It should be noted that this CAS registry number may have been applied to records that deal with (coal) tar pitches in a more general sense in files like TOXLINE and NIOSHTIC, whereas relevant records in files like MEDLINE and CA will not be retrieved due to absence of the registry number in indexing. Therefore additional searches on “coal tar pitch” and “coal-tar pitch” were performed in MEDLINE, TOXLINE and CURRENT CONTENTS. However, it is still possible that some relevant data were not found with these searches and therefore not discussed in this C&L Report.

² UVCB: Unknown, of Variable Composition, or of Biological origin.

Purity/Impurities, Additives

The content of the sixteen EPA-PAHs in pitch used for impregnation and binding is presented in Table 1.2.1, along with other aromatic hydrocarbons. Most relevant for the classification and labelling is the composition for binder pitch, as it is the main source for the production of anodes and electrodes (Netherlands, 2005). The structural formulae of the 16 EPA-PAHs are shown in Figure 1.2.1 (those marked with * belong to the Borneff 6). Besides polycyclic aromatic hydrocarbons and their (poly)methylated derivatives, coal tar pitch contains heterocyclic compounds and benzocarbazoles. The amount of benzo[a]pyrene is estimated at 0.1 - 1.5%. Naphthalene and acenaphthylene were not detected in either of the pitches used for impregnation and binding. Yet, the environmental hazards of these compounds are discussed in this report (see section 7) as it cannot be ruled out that their individual toxicity contributes to the overall hazard of CTPHT.

Table 1.2.1. PAH content in CTPHT (16 EPA-PAHs and other aromatic hydrocarbons).

	Impregnation Pitch		Binder Pitch	
	(mg/kg) ^{a)}	(%)	(mg/kg) ^{a)}	(%)
<i>Aromatic hydrocarbons</i>				
Ethylbenzene	n.d.	n.d.	n.d.	n.d.
<i>m-p</i> -Xylene	n.d.	n.d.	n.d.	n.d.
1,2,4-Trimethylbenzene	n.d.	n.d.	n.d.	n.d.
3- Ethyltoluene	n.d.	n.d.	n.d.	n.d.
1,3,5-Trimethylbenzene	n.d.	n.d.	n.d.	n.d.
1,2,3-Trimethylbenzene	n.d.	n.d.	n.d.	n.d.
Indene	n.d.	n.d.	n.d.	n.d.
1,2,4,5-Tetramethylbenzene	n.d.	n.d.	n.d.	n.d.
Naphthalene ^{b)}	n.d.	n.d.	n.d.	n.d.
2,4,6- Trimethylbenzene	n.d.	n.d.	n.d.	n.d.
2- Methylnaphthalene	n.d.	n.d.	n.d.	n.d.
1- Methylnaphthalene	n.d.	n.d.	n.d.	n.d.
Biphenyl	n.d.	n.d.	n.d.	n.d.
Dimethylnaphthalenes	n.d.	n.d.	n.d.	n.d.
Acenaphthylene ^{b)}	n.d.	n.d.	n.d.	n.d.
Acenaphthene ^{b)}	390	0.039	432	0.043
Fluorene ^{b)}	144	0.014	472	0.047
2-Methylfluorene	50	0.005	112	0.011
1-Methylfluorene	n.d.	n.d.	61	0.006
Phenanthrene ^{b)}	3874	0.387	6299	0.630
Anthracene ^{b)}	737	0.074	1311	0.131
3-Methylphenanthrene	n.d.	n.d.	n.d.	n.d.
2-Methylphenanthrene	n.d.	n.d.	n.d.	n.d.
2-Methylanthracene	n.d.	n.d.	n.d.	n.d.
Cyclopenta[def]phenanthrene	918	0.092	821	0.082
4-Methylphenanthrene	n.d.	n.d.	n.d.	n.d.
1-Methylphenanthrene	n.d.	n.d.	n.d.	n.d.
Fluoranthene ^{b)}	17389	1.739	10789	1.079
Acephenanthrylene	828	0.083	386	0.039
Pyrene ^{b)}	14849	1.485	9449	0.945
Benzo[a]fluorene	4509	0.451	1974	0.198
Benzo[b]fluorene	4306	0.431	2456	0.246
Benz[a]anthracene ^{b)}	15008	1.501	7715	0.772

	Impregnation Pitch		Binder Pitch	
	(mg/kg) ^{a)}	(%)	(mg/kg) ^{a)}	(%)
Chrysene ^{b)}	14041	1.404	8053	0.805
Benzo[b]fluoranthene ^{b)}	17408	1.741	12131	1.213
Benzo[k]fluoranthene ^{b)}	8704	0.870	6065	0.607
Benzo[e]pyrene	11891	1.189	8976	0.898
Benzo[a]pyrene ^{b)}	12924	1.292	10021	1.002
Perylene	5014	0.501	3167	0.317
Dibenz[a,h]anthracene ^{b)}	2209	0.221	1749	0.175
Indeno[1,2,3-cd]pyrene ^{b)}	11106	1.111	9061	0.906
Benzo[ghi]perylene ^{b)}	9945	0.994	8664	0.866
Anthantrene	4581	0.458	3464	0.346
<i>Tar acids / phenolics</i>				
Phenol	n.d.	n.d.	n.d.	n.d.
<i>o</i> -Cresol	n.d.	n.d.	n.d.	n.d.
<i>m</i> -/ <i>p</i> -Cresol	n.d.	n.d.	n.d.	n.d.
2,6-Xylenol	n.d.	n.d.	n.d.	n.d.
2,5-Xylenol	n.d.	n.d.	n.d.	n.d.
3,5-Xylenol	n.d.	n.d.	n.d.	n.d.
3,4-Xylenol	n.d.	n.d.	n.d.	n.d.
4-Isopropylphenol	n.d.	n.d.	n.d.	n.d.
2,3,5-Trimethylphenol	n.d.	n.d.	n.d.	n.d.
3,4,5-Trimethylphenol	n.d.	n.d.	n.d.	n.d.
<i>Tar bases / nitrogen-containing heterocycles</i>				
Benzonitrile	n.d.	n.d.	n.d.	n.d.
<i>o</i> -Tolunitrile	n.d.	n.d.	n.d.	n.d.
<i>m</i> -Tolunitrile	n.d.	n.d.	n.d.	n.d.
<i>p</i> -Tolunitrile	n.d.	n.d.	n.d.	n.d.
Quinoline	n.d.	n.d.	n.d.	n.d.
Isoquinoline	n.d.	n.d.	n.d.	n.d.
Indole	n.d.	n.d.	n.d.	n.d.
Chinaldine	n.d.	n.d.	n.d.	n.d.
Acridine	242	0.024	264	0.026
Carbazole	1556	0.156	1664	0.166
<i>Sulphur-containing heterocycles</i>				
Thionaphthene	n.d.	n.d.	n.d.	n.d.
Dibenzothiophene	269	0.027	438	0.044
<i>Oxygen-containing heterocycles / furans</i>				
Dibenzofuran	n.d.	n.d.	215	0.022
Total	162,892	16.289	116,209	11.621

^{a)} n.d.: not detected (detection limit: 50 mg/kg). ^{b)} These are the 16 EPA-PAHs.

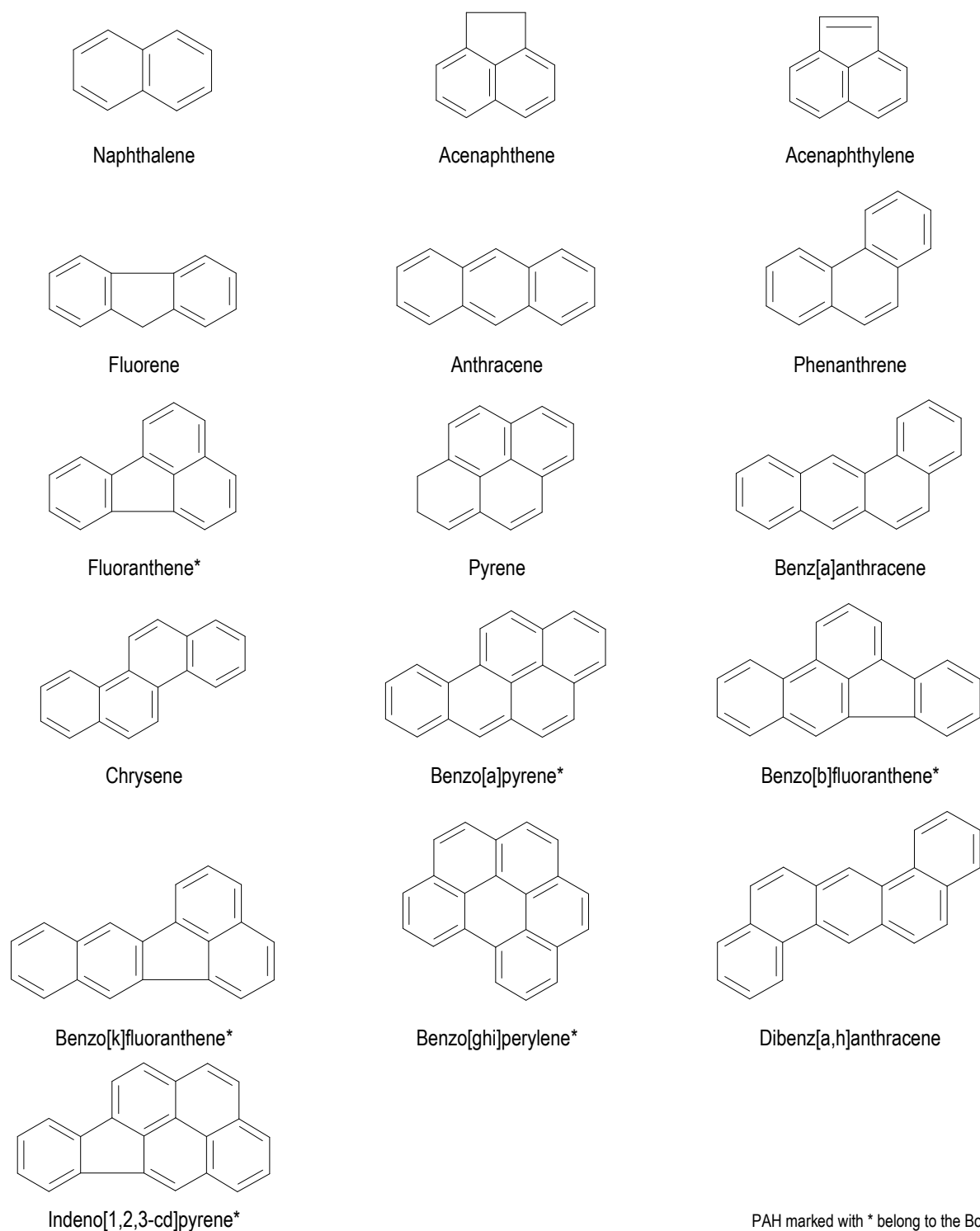


Figure 1.2.1. Structural formulae of polycyclic aromatic hydrocarbons covered in this classification and labelling report.

1.3 Physicochemical properties

The physicochemical characteristics of coal tar pitch high temperature are presented in Table 1.3.1. Because of the importance for the classification and labelling, the water solubility of CTPHT is discussed in detail. In Table 1.3.2 and in Table 1.3.3 the relevant data on water solubility of coal-tar

pitch are summarized. In Table 1.3.4 the physicochemical properties of the individual EPA-PAHs are presented.

Table 1.3.1. Physicochemical properties of CTPHT.

REACH ref Annex, §	Property	IUCLID section	Value	Comment/reference
VII, 7.1	Physical state	4.1	black solid	at 20°C and 101.3 KPa
VII, 7.2	Melting point	4.2	65-150 °C	softening range (CCSG, 2006)
VII, 7.3	Boiling point	4.3	>360 °C	at 1013 hPa
VII, 7.4	Relative density	4.4	1.15-1.40 g/cm ³	at 20 °C (ASTM, 2004; CCSG, 2006)
VII, 7.5	Vapour pressure	4.6	<0.1 hPa <10 hPa	at 20 °C at 200 °C (CCSG, 2006; OECD, 2006)
VII, 7.6	Surface tension	4.10	--	not applicable
VII, 7.7	Water solubility	4.8	~0.040 mg/L	16 EPA-PAHs, at a loading of 10 g/L at 22 °C (Rütgers VFT, 1999a, b)
VII, 7.8	Partition coefficient n-octanol/water (log value)	4.7	--	not applicable
VII, 7.9	Flash point	4.11	>250 °C	(ISO, 2002; CCSG, 2006)
VII, 7.10	Flammability	4.13	non flammable	84/449/EEC (EU, 1984); Coal tar pitch, when heated above its initial boiling point, may generate vapours that may ignite in the presence of air and a source of ignition (CCSG, 2006)
VII, 7.11	Explosive properties	4.14	not explosive	(CCSG, 2006)
VII, 7.12	Self-ignition temperature		>450 °C	at 1013 hPa (DIN, 2003; CCSG, 2006)
VII, 7.13	Oxidising properties	4.15	not oxidizing	(CCSG, 2006)

Water solubility

The lowest values were found after elution from a percolation column (Table 1.3.2, No. 1 and 2) and after settling following mechanical agitation (Table 1.3.2, No. 5). In the first case, the surface of the coal-tar pitch material remained undisturbed, while in the second case the settling time may have favoured removal from the water-phase by adsorption to the glass wall of the test vessel. Repetitive elution in an undisturbed system demonstrated the limited release of PAHs from coal-tar pitch (Table 1.3.2: continuation of test No. 1). On the other hand, under agitated conditions, the permanent mechanical stress on the particle surface prevented a significant decrease in the PAH level in the water phase over time (Table 1.3.2, No. 7). The highest value found was about 140 µg/L at an elevated temperature (Table 1.3.2, No. 4). Overall, the data show high consistency taking into account the different test and analytical conditions.

Multiple elution

Within the scope of a comprehensive analytical programme on the availability of PAH from coal-tar pitch in water (Rütgers VFT, 1999a, b), a column containing 10 g of finely powdered pitch (20-200 µm) was force-percolated by 1.1 L of tap water (water recycling for 1 wk). Each experimental period was terminated by withdrawal of 1 L of the extract and renewal of the volume by fresh-water exchange of 1 l each. This procedure was continued for 39 wk. The total of the EPA-PAHs comprised 9.9 % (after GC) or 9.2 % (after HPLC) in the pitch sample applied. The dissolved EPA-PAHs were determined by HPLC analysis after toluene extraction from the water samples (2 mL/500 mL water, in duplicate) after each water exchange. After the first run, 36.5 µg PAH/L were found; after 15 cycles, the PAH decreased to 11.8 µg/L, and after 39 cycles to 0.9 µg/L. The first water-soluble fraction was dominated by the presence of acenaphthene, phenanthrene, fluoranthene, and pyrene, followed by naphthalene and fluorene (Table 1.3.3). All other PAHs were distinctly

below 1 µg/L). The total cumulative amount of water-extractable EPA-PAHs amounted to approx. 370 µg/10g (= ~0.004 %). The water-soluble fraction of single PAHs remained far below their theoretical water-solubilities, which confirms that freely available PAHs got gradually exhausted. Table 1.3.3 summarises key data and results obtained from this experiment.

The highest water solubility in relation to the loading was 0.0014 % (30 °C) at maximum (Table 1.3.2, No.4). The use of granules resulted in a water-soluble fraction that was a factor of about 10-25 lower than that for the powder form (Table 1.3.2, compare No. 2 with No. 1, No. 6, or No. 7).

Table 1.3.2. Water solubility data of CTPHT.

No.	Form	Loading (g/L)	Concentration in WSF ^{a)} (µg/L)	Temp (°C)	Analytics (HPLC)	Extraction Method	Source
1	Powder	10	37	22	EPA-PAHs	Elution column	(Rütgers VFT, 1999a, b)
2	Granules	100	27	25	EPA-PAHs	Elution column	(Rütgers VFT, 2000)
3	Molten pitch	42	91	22-25	EPA-PAHs	Hot injection into the water phase and 24-h settling	(Rütgers VFT, 1997)
4	Powder	10	137	30	EPA-PAHs	24-h stirring, membrane filtration (0.45 µm)	(UBA, 1997)
5	Powder	10	22	30	EPA-PAHs	24-h stirring, 24-h settling, centrifugation	(UBA, 1997)
6	Powder	10	67	22	EPA-PAHs	8-d stirring, membrane filtration (0.45 µm)	UBA, 1999
7	Powder	10	54-75	22	EPA-PAHs	8-d stirring (6x), 6-fold repetitive water extraction, membrane filtration (0.45 µm)	UBA, 1999

^{a)} WSF = Water-Soluble Fraction based on the 16 EPA-PAHs.

Table 1.3.3. Multiple elution of coal-tar pitch (10 g/L as powder) in an elution column as compared to composition of pitch and water solubility of pitch PAHs.

16 EPA-PAHs	Content in 10 g Pitch (rounded values)		Concentration (µg/L) (rounded values)		
	(µg)	(%)	1 st cycle	15 th cycle	39 th cycle
Naphthalene	100	0.001	1.5	0.8	0.1
Acenaphthylene	n.d. ^{a)}	n.d. ^{a)}	n.d. ^{a)}	n.d. ^{a)}	n.d. ^{a)}
Acenaphthene	10,000	0.1	7.3	2.7	0.02
Fluorene	4,000	0.04	1.2	0.7	0.01
Phenanthrene	20,000	0.2	8.8	0.7	0.03
Anthracene	3,600	0.036	0.6	0.1	0.02
Fluoranthene	100,000	1.0	9.3	3.6	0.05
Pyrene	90,000	0.9	6.7	2.6	0.25
Benz[a]anthracene	85,000	0.85	0.5	0.2	0.08
Chrysene	100,000	1.0	0.4	0.3	0.15
Benzo[b]fluoranthene	130,000	1.3	0.045	0.1	0.09
Benzo[k]fluoranthene	64,000	0.64	0.028	0.01	0.03
Benzo[a]pyrene	110,000	1.1	0.041	0.06	0.03
Dibenz[a,h]anthracene	23,000	0.23	0.01	0.01	0.003
Benzo[ghi]perylene	100,000	1.0	0.02	0.01	0.02
Indeno[1,2,3-cd]pyrene	100,000	1.0	0.01	0.01	0.01
Total	920,000	9.2	36.5	11.8	0.9

Values from Rütgers VFT (1999a, b). ^{a)} n.d.: not detected.

Table 1.3.4. Physicochemical properties of various PAHs.

Substance	CAS nr	Molecular formula	Molecular weight (g/mol)	Melting point (°C)	Boiling point (°C)	Water solubility (µg/L)	Log K_{ow} (-)	Vapour pressure (Pa at 25 °C)	Density (g/cm ³)	Henry's constant (Pa m ³ /mol at 25 °C)
Naphthalene	91-20-3	C ₁₀ H ₈	128.2	81	217.9 ^{d)}	31900 ^{a)}	3.34 ^{d)}	11.2 ^{g)}	1.154	50 ^{m)}
Acenaphthene	83-32-9	C ₁₂ H ₁₀	152.2	96	278	3910 ^{b)}	4.00 ^{f)}	3.3·10 ⁻¹ ⁱ⁾	0.899	14.3 ^{m)}
Acenaphthylene	208-96-8	C ₁₂ H ₈	150.2	92	279	16100 ^{b)}	3.62 ^{g)}	4.8·10 ⁻¹ ^{j)}	1.024	11.5 ^{m)}
Fluorene	86-73-7	C ₁₃ H ₁₀	166.2	115-116	295 ^{f)}	1800 ^{a)}	4.22 ^{f)}	8.3·10 ⁻² ^{j)}	1.203	8.5 ^{m)}
Anthracene	120-12-7	C ₁₄ H ₁₀	178.2	216.4	342 ^{f)}	47 ^{a)}	4.68 ^{e)}	9.4·10 ⁻⁴ ^{j)}	1.283	4.3 ^{m)}
Phenanthrene	85-01-8	C ₁₄ H ₁₀	178.2	100.5	340	974 ^{a)}	4.57 ^{e)}	2.6·10 ⁻² ^{j)}	0.980	3.7 ^{m)}
Fluoranthene	206-44-0	C ₁₆ H ₁₀	202.3	108.8	375	200 ^{a)}	5.20 ^{e)}	1.2·10 ⁻³ ⁱ⁾	1.252	1.1 ^{o)}
Pyrene	129-00-0	C ₁₆ H ₁₀	202.3	156	360	125 ^{a)}	4.98 ^{f)}	1.0·10 ⁻³ ^{j)}	1.271	1.4 ⁿ⁾
Benz[a]anthracene	56-55-3	C ₁₈ H ₁₂	228.3	160.7	435	10.2 ^{a)}	5.9 ^{e)}	7.6·10 ⁻⁶ ^{j)}	1.226	0.81 ^{p)}
Chrysene	218-01-9	C ₁₈ H ₁₂	228.3	253.8	448	1.65 ^{a)}	5.81 ^{e)}	5.7·10 ⁻⁷ ^{k)}	1.274	0.079 ^{q)}
Benzo[a]pyrene	50-32-8	C ₂₀ H ₁₂	252.3	175	496	1.54 ^{a)}	6.13 ^{e)}	7.3·10 ⁻⁷ ^{k)}	1.35	0.034 ^{o,r)}
Benzo[b]fluoranthene	205-99-2	C ₂₀ H ₁₂	252.3	168.3	481	1.28 ^{a)}	6.12 ^{g)}	3.3·10 ⁻⁶ ^{l)}	-	0.051 ^{o,r)}
Benzo[k]fluoranthene	207-08-9	C ₂₀ H ₁₂	252.3	217	480	0.93 ^{a)}	6.11 ^{e)}	1.3·10 ⁻⁷ ^{l)}	-	0.043 ^{o,r)}
Benzo[ghi]perylene	191-24-2	C ₂₂ H ₁₂	276.3	277	545 ^{l)}	0.14 ^{a)}	6.22 ^{e)}	1.4·10 ⁻⁸ ^{k)}	1.329	0.027 ^{o,r)}
Dibenz[a,h]anthracene	53-70-3	C ₂₂ H ₁₄	278.4	266.6	524	0.82 ^{b)}	6.50 ^{f)}	3.7·10 ⁻¹⁰ ^{k)}	1.282	1.3·10 ⁻⁴ ^{q)}
Indeno[1,2,3-cd]pyrene	193-39-5	C ₂₂ H ₁₂	276.3	163.6	536	0.1 ^{c)}	6.58 ^{g)}	1.7·10 ⁻⁸ ^{l)}	-	0.046 ^{q)}

The data presented in the table were taken from Mackay *et al* (1992). ^{a)} The values for water solubility were based on generated column methods using geometric means; ^{b)} The values for water solubility were based on shake-flask using geometric means; ^{c)} For indeno[1,2,3-cd]pyrene no data were available, a default value of 0.1 µg/L was used; ^{d)} The values for log K_{ow} were based on slow-stirring/generator column using average values; ^{e)} The values for log K_{ow} were based on slow-stirring methods using average values; ^{f)} The log K_{ow} values were based on the shake-flask method; ^{g)} The log K_{ow} values were calculated using ClogP model; ^{h)} The values for vapour pressure were based on manometry/gas saturation using geometric means; ⁱ⁾ The values for vapour pressure were based on gas saturation using geometric means; ^{j)} The values for vapour pressure were based on gas saturation/effusion using geometric means; ^{k)} The values for vapour pressure were based on effusion method using geometric means; ^{l)} The values for vapour pressure were estimated using EPIWIN; ^{m)} The selected values for the Henry's constant were based on batch/gas stripping/wetted-wall column using geometric means; ⁿ⁾ The selected values for the Henry's constant were based on batch/gas stripping using geometric means; ^{o)} The selected values for the Henry's constant were based on gas stripping using geometric means; ^{p)} The selected values for the Henry's constant were based on batch column using geometric means; ^{q)} No data were available, so constants were calculated using EUSES 2.0; ^{r)} Measurements were performed at 20 °C.

2 MANUFACTURE AND USE

2.1 Manufacture and import of a substance

High temperature coal tar pitch is produced by distillation of high temperature coal tar. The latter product with CAS No 65996-89-6 is defined as ‘the condensation product obtained by cooling, to approximately ambient temperature, of the gas evolved in the high temperature destructive distillation of coal. It is a black viscous liquid denser than water, composed primarily of a complex mixture of condensed ring aromatic hydrocarbons. It may contain minor amounts of phenolic compounds and aromatic nitrogen bases’ (EU, 1976). Coal tar is produced at the coke plants of primary steel works or coke plants as such as a by-product. It is supplied to coal tar refineries, which may be part of the coke oven plant or operate independently at another site.

Distillation of coal tar produces several oil fractions and pitch as given in Figure 2.1.1. From 100 tonnes of coal tar; 1 tonne of light oil, 2 tonnes of carbolic oil, about 10 tonnes of respectively naphthalene oil, wash oil and anthracene oil, 12 tonnes of base oil and 50 tonnes of pitch are produced.

Within the European Union, high temperature coal tar pitch is produced by ten companies at eleven sites in nine countries. The total European Union production capacity in 2004 was 1,127,000 tonnes. The actual production output of coal tar pitch in that year was about 817,800 tonnes. Import from outside the EU was reported to be about 91,600 tonnes per year and export was about 355,600 tonnes per year. The total consumption of coal tar pitch in the EU from these figures is estimated to be about 554,000 tonnes per year. Table 2.1.1 presents the market data for coal tar pitch within the European Union.

Figure 2.1.1 also presents an overview of pitch applications (uses). Each application requires different characteristics and therefore there is no ‘standard’ pitch with a chemical composition, which can be characterised within narrow margins, so several ‘types of pitches are distinguished. It should be noted that it might be difficult to distinguish between production and formulation. Some of the pitches can be regarded as blends, which may be formulated at the production site of pitch or at the site of a user. Pitch coke can be regarded as a product made from pitch (industrial application) for which the raw material pitch is processed.

Each type of pitch will have a typical chemical composition or – more specific – a PAH pattern. It is obvious that within one type pitch some variation in the pattern will occur. Especially formulations such as plasticized pitch will have quite a different content and pattern as PAH-containing components are added. In uses where such a type of pitch is used (*e.g.* as a binding agent in a formulation with bitumen for road paving) the PAH pattern and content may also change considerably.

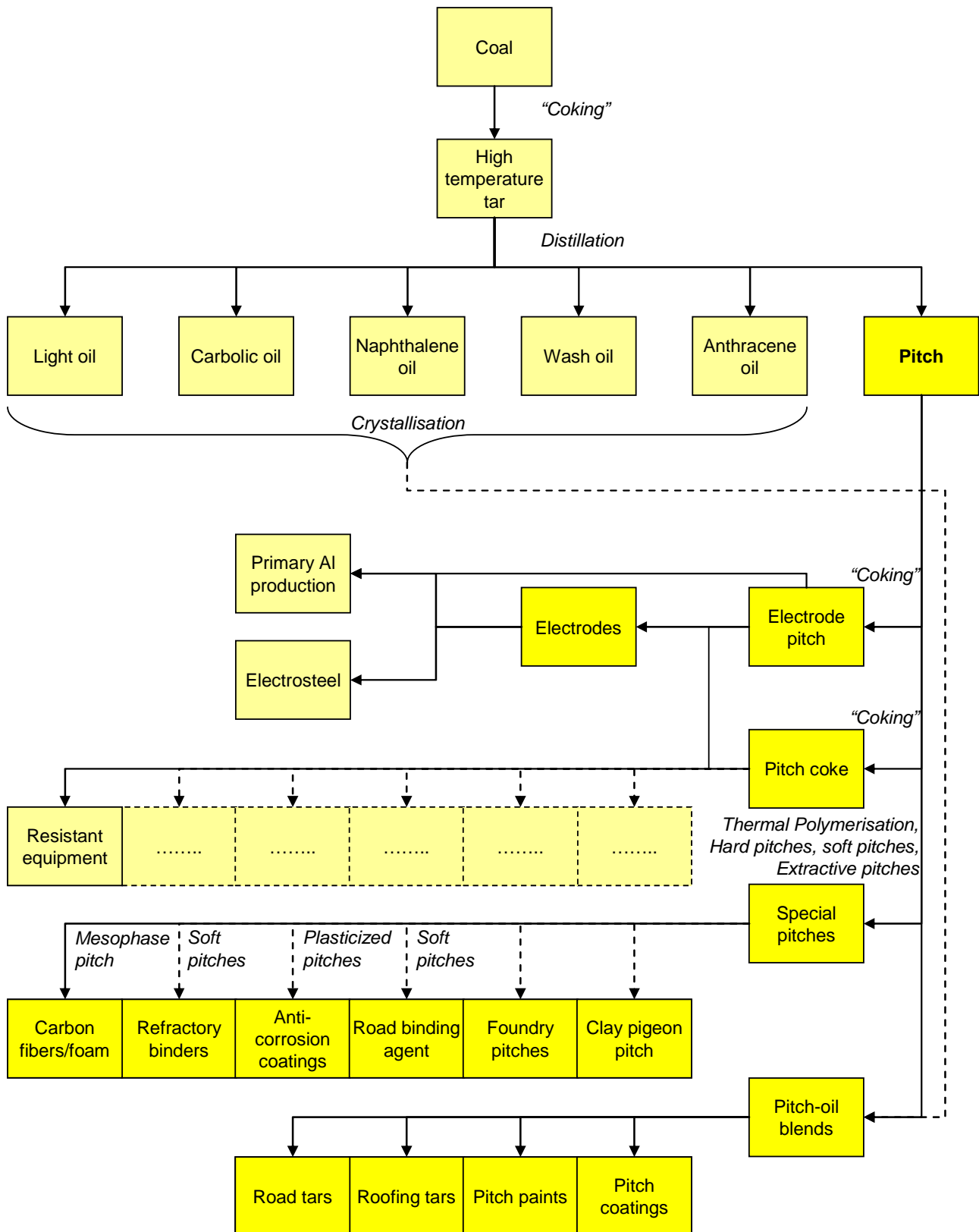


Figure 2.1.1. Schematic overview of the production of CTPHT and the subsequent processing and formulation possibilities.

Table 2.1.1. Market data for coal tar pitch in the European Union, 2004.

Country	Production	Import	Export
Belgium	129,300		107,800
Denmark	138,000		-
France	48,400	41,600	6,800
Germany	208,000	50,000	158,000
Netherlands	56,300		-
Spain	170,000		83,000
United Kingdom	67,800		0
Total	817,800	91,600	355,600

2.2 Uses

2.2.1 Introduction

Coal tar pitch is mainly used as a binding agent in the production of carbon electrodes, anodes and Söderberg electrodes for instance for the aluminium industry. It is also used as a binding agent for refractories, clay pigeons, active carbon, coal briquetting, road construction and roofing. Furthermore small quantities are used for heavy duty corrosion protection, see Table 2.2.1.

A description of the various applications of coal tar pitch is given in the following sections.

Table 2.2.1. Use pattern for coal tar pitch, based on sales in the EU in 2003.

Application	Industry category ^{a)}	Use category ^{b)}	Quantity (tonnes/year)	Percentage of total sales
Anodes	8	2	322,500	71.3
Electrodes	8	2	81,400	18.0
Refractories	0	2	22,500	5.0
Road construction	16	2	800	0.2
Active carbon	0	2	7,900	1.7
Heavy duty corrosion protection	14	2/39	4,700	1.0
Roofing	16	2	3,200	0.7
Clay pigeons	0	2	5,800	1.3
Coal briquetting	9	2	3,700	0.9
Total			452,400	100

^{a)} Industrial category 0 is others, industrial category 8 metal extraction, refining and processing industry, industrial category 9 is mineral oil and fuel industry, industrial category 14 is paints, lacquers and varnishes industry, industrial category 16 is engineering industries: civil and mechanical; ^{b)} Use category 2 is adhesives and binding agents and use category 39 is non-agricultural biocides.

2.2.2 Use as binding agent in the production of electrodes

As indicated before, the largest application of pitch is its use as a binding agent in the production of electrodes (including anodes for the primary aluminium production) with 87 percent of the total amount of pitch used. The electrodes are mainly used in the production of primary metals, ferro-alloys, non-ferrous metals and metal alloys, calcium carbide and silicon carbide.

The production of electrodes starts with the green paste or Söderberg paste production. Green paste is produced from either coal coke or petroleum coke and up to 28% of pitch, which acts as a binder. Cokes is ground and mixed with pitch in heated mixers at a temperature between 100-150 °C and pressed in the desired form which, after cooling, results in so called Söderberg electrodes. These electrodes are commonly used in submerged electric arc furnaces for instance for the production of ferro-alloys and in the electrolysis process for primary aluminium production.

Prebaked anodes are produced from the green paste but especially prebaked anodes might contain residual material from old anodes. The green electrodes are baked in large furnaces at a temperature of about 1100 °C in the absence of air for about 14 days. The equipment used for baking may be open or closed top ring furnaces. Open furnaces use a horizontal duct and closed furnaces use a vertical flue. Open furnaces account for 60% of the capacity. During the baking process the coal tar is converted into coke, making the material electrically conductive. There is a 5% loss in weight during baking as volatiles and filter tar (EC, 2001). Besides the use of coke also used anodes are applied for the production of prebaked anodes.

These prebaked electrodes are mainly applied as anodes in the primary aluminium production. The baking of anodes might be done at a plant at the same site where also aluminium production takes place. However, there are also companies producing anodes and shipping them to their customers. It should be noted that at sites where anodes for the aluminium are baked also manufacturing of graphite electrodes may take place.

Graphite electrodes are obtained after additional impregnation with pitch and consecutive graphitisation at temperatures of 2800 °C for three weeks, usually carried out in Acheson or Castner furnaces. Single chamber furnaces or pit furnaces are used as well as closed ring furnaces for the baking process. Tunnel furnaces are used for small-scale production of speciality carbon (EC, 2001).

These electrodes are used in electric arc furnaces for the production of a variety of products as ferro-alloys, silicon carbide calcium carbide and phosphorous.

Prebake and graphite electrodes are produced from an average of 16% pitch and 84% of petroleum coke. In addition for the manufacture of prebake anodes for the aluminium industry remainders of used anodes are also applied beside cokes.

2.2.3 Use as binding agent in the production of other products

Other carbon and graphite products

Graphite products such as seals, brushes and similar products are produced in a similar way as graphite electrodes. There are differences in the size and complexity of the products and this affects the processes that are used. Other additives such as sulphur of metals can be added to the blend of raw materials to give the desired physical properties to the product. Green shapes are formed by moulding and these may be baked at temperatures up to 1300 °C, re-baked and graphitised by heating the shapes up to 2800 °C. Baking and re-baking of green shapes is done by using a variety of furnaces such as tunnel, single chamber, multiple chamber or annular furnaces depending on size and complexity of the product. Graphitising is done in Acheson, tunnel, Castner or induction furnaces. The shapes are then subjected to a number of finishing processes such as machining and polishing (EC, 2001). Porous graphite is also produced in the basic process of blending sawdust with the raw materials. During baking sawdust is combusted and a porous matrix of carbon of graphite remains, see also the section on Production of active carbon below. High purity graphite is produce in a similar way but the graphitising process is used to remove included impurities such as metals.

Refractory brick

Refractories are materials that provide linings for high-temperature furnaces and other processing units operating at high temperature. Refractories must be able to withstand physical wear, corrosion by chemical agents and high temperatures (above 500 °C). Refractories are produced as formed objects, *i.e.* bricks and shapes and unformed as granulated composites. A typical refractory

application is as lining of basic oxygen furnaces and electric arc furnaces in steelmaking (US-EPA, 1995; Hubble *et al*, 1998).

Several types of refractories are available ranging from pitch-bonded to the advanced refractories that are made with resin bonds, metallics, graphites and sintered or fused magnesia. Different types of pitch-bonded refractories are used like pitch-impregnated fired bricks, pitch-bonded unfired or tempered bricks (high alumina), pitch-bonded unfired or tempered magnesia, magnesia dolomite and dolomite bricks and shapes and pitch-bonded or tempered oxide-graphite products (Routschka & Granitzki, 2002).

Production of active carbon

For those applications where active carbon has to be shaped, the raw material, such as coal, can be pulverised, briquetted by using a binder, and finally carbonised. There are a number of patents describing the production of microporous spheres from pitch. The process involves several stages: melting, dispersing, oxidising with air to render the material insoluble, and finally, activation by steam (Vohler *et al*, 2002).

Binder for road construction and roofing

For road paving various products, consisting of all kinds of mixtures of PAH-containing materials and (in the case of asphalt) minerals, are used. In many of them pitch is one of the components. The following 'road tars' may be distinguished:

- Road tar for low traffic roads based on blends of 60-80% normal pitch with middle oils (boiling range 170-270 °C), heavy oils (270-300 °C), and anthracene oils (boiling range > 300 °C).
- Ageing-resistant road tars have an increased ratio of anthracene oil II (boiling range > 350 °C) and anthracene oil I (boiling range up to 350 °C).
- Bitumen-containing road tar with low oil content containing 15% asphalt basic distillate bitumen.
- Bitumen-rich road tar containing 35-40% bitumen
- Pitch-bitumen contains 70-85% bitumen
- Carbobitumen is a blend of soft pitch and hard bitumen, containing 20-30% of a special pitch.

Because of the varying content of PAHs and the variable amounts of the types of road tars, emissions will show a wide range.

Roofing tars used as impregnating, coating, and adhesive material for tarred felts and tarred sealing webs and are usually blends of pitch and filtered anthracene oil; by using plasticized pitches or by adding extenders the plasticity and temperature stability of roofing tars is improved considerably (Collin & Höke, 2002).

On the whole the amount of pitch used for these two applications decrease as it is replaced by petroleum pitch on account of the lower PAH content because most of the European countries (for instance the Netherlands) have banned the use of coal tar pitch in road construction by law or agreement between trade unions and road building companies. According to industry, only very particular applications such as anti-kerosene coatings for parking lots and fuel stations still use pitch emulsions.

Clay pigeons

Clay pigeon pitch is used as a brittle binding agent with increased softening point for clay pigeons used in sport shooting (Collin & Höke, 2002). Clay pigeons are designed to withstand being thrown

from traps at very high speeds, but at the same time being easily broken when hit by just a very few pellets from a gun. Instead of clay chalk or dolomite limestone and instead of coal tar pitch petroleum pitch may be used. Some manufacturers claim to produce “environmentally” friendly clay pigeons by applying petroleum pitch in order to meet the EEC environmental protection directives, or to apply no binder at all (mixture of several clays) (Lireko, 2002; Shootingworld, 2002; Claypigeon Company Ltd., 2010). Clay pigeons manufacturers, claiming environmental protection, displaced coal tar pitch by petrochemical binders for more than 80% of their production and the former clay pigeons are exported outside the European Union. However, information provided by industry contradicts the assumption that the use of CTPHT in clay pigeons has been reduced significantly and that in the short term this application will be phased out. For this reason, industry is requested to provide information on the release of PAHs from the production and use of clay pigeons in order to assess the environmental risk.

Coal briquetting

Coal briquetting is intended to turn fine-grained coal (< 6 mm) into a lump form. This conversion leads to a better manageable solid fuel especially for domestic heating. Briquetting is also necessary if coal is reacted in a fixed bed subjected to a gas flow (*e.g.* in fixed-bed gasification) and for lump or formed coke production from non-caking coal.

Regarding the temperature range, briquetting processes are subdivided into cold briquetting (< 100 °C) and hot briquetting processes (400-500 °C). The production of high quality briquettes by cold briquetting without binding agent is only possible with soft brown coal. Binding agents such as pitch, tar, and bitumen were formerly used for low volatile coals. Because of their carcinogenic effect (which is particularly pronounced if such binding agents are based on hard coal), they are being replaced by other binding agents, *e.g.* biomass materials (for example molasses). In some Western European countries the use of coal tar pitch is forbidden (Germany and Scandinavia). Hot briquetting is a less common method of compacting. Here, a caking coal in its softening range is used as the binding agent. Low sulphur emission briquettes can be produced by adding sulphur binding components such as milk of lime to reduce the sulphur dioxide emission during low- and medium-temperature combustion (Sauter & Reimert, 2002). Based on information provided by industry, the capacities for briquetting are decreasing from 2000 ktonnes per year in the 1980s to currently 150 ktonnes per year. Together with increasingly use of environmental friendly binders like molasses and starch this eventually leads to a complete phase out of the pitch for this application. However, recent information provided by industry contradicts the assumption that the use of CTPHT in coal briquetting will be phased out. For this reason, industry is requested to provide information on the release of PAHs from the production and use of coal briquetting in order to assess the environmental risk.

Heavy duty corrosion protection

Pitch coatings are used for anticorrosion protection. As the plasticity of normal pitch is too low, hard pitches with a high content of toluene insolubles are adjusted to the desired softening point with high-boiling tar oils (Collin & Höke, 2002). For heavy duty corrosion protection as mentioned in Table 2.2.1 and for application as sealing compounds a further increase in the plasticity range is achieved by hot-mixing these pitches with extenders such as finely ground coal, minerals, diatomaceous earth, or fly ash; to meet especially high anticorrosion requirements, coal tar pitches are combined with polymers (Collin & Höke, 2002). Such pitch-polymer combinations may consist of two-pack systems with epoxy or polyurethane or one-pack systems with other polymers or elastomers.

Physically drying pitch coatings

Special coal tar pitches are used in the production of one-pack physically drying paints; the physical and chemical properties have been modified by special processes, such as polymerisation (Stoye *et al.*, 2002). For waterborne coatings high-boiling coal tar distillates, mineral oil extracts rich in aromatics, or plasticizers normally used in the paint industry (*e.g.* benzylphthalate) may be used as plasticizers for hard pitches (Stoye *et al.*, 2002).

Pitch paints have been used to protect concrete against aggressive water, for corrosion protection of steel constructions in industry, hydraulic steel structures, and underground pipelines.

Pitch combination coatings

In one-pack, physically drying pitch polymer combination paints, pitch paints are mixed with thermoplastic polymers such as PVC, chlorinated rubber, polychloropyrene, polyacrylonitrile, or polystyrene (4-8% wt. % of polymer) (Stoye *et al.*, 2002). They are superior to the conventional pitch paints, and are especially used to protect structures in the sewerage and effluent sector (Stoye *et al.*, 2002).

In two-pack chemically drying combination paints pitch and solvent or pitch and high-boiling tar oils are mixed with reaction-curing resins such as epoxy resins and polyurethanes (Stoye *et al.*, 2002). They are widely used in hydraulic steel structures, ship building (antifouling) and harbour construction, sewerage sector, and pipeline construction (Stoye *et al.*, 2002).

According to industry, corrosion protection with pitch-based products is declining and phasing out of these artefacts is predicted in the next few years. Also there is a European Union wide ban on the use of coal tar (pitch) containing coatings for use on ships and quays etc. However, information provided by industry contradicts the assumption that the use of CTPHT in heavy duty corrosion protection has been reduced significantly and that in the short term this application will be phased out. For this reason, industry is requested to provide information on the release of PAHs from the production and use of heavy duty corrosion protection in order to assess the environmental risk.

2.2.4 Application of carbon and graphite electrodes

Aluminium production

Aluminium is produced in reduction plants by the Hall-Heroult process. The electrolytic reduction of aluminium oxide (alumina) takes place in a molten bath of cryolite (sodium aluminium fluoride) at a temperature of approximately 960 °C, with up to 5% alumina dissolved in this. Aluminium fluoride is added to lower the melting point of the bath. A reduction cell comprises a carbon cathode, insulated by refractory bricks inside a rectangular steel shell, and a carbon anode suspended into the molten charge. The cells are covered with a hood for gas collection and are connected in series to form an electrical reduction line (potline). The oxygen in the alumina reacts with the carbon of the anodes, to form carbon dioxide, hence consuming the carbon anodes. Liquid aluminium deposits at the bottom of the cell and is drawn off.

In the primary aluminium production two different types of anodes are applied, Søderberg anodes and prebaked anodes. The Søderberg process applies continuous anode paste (green paste). The paste is baked *in situ* in the electrolytic cell during the production process. This process does not require changing of anodes. Prebaked anodes are manufactured at a separate anode plant, which sometimes is an integrated part of the primary aluminium production plant.

There are different sub-processes to be distinguished in primary aluminium production depending on the positioning of the current carrying studs in the anodes, a factor which may influence emissions from the electrolytic reduction process: Horizontal Stud Søderberg (HSS) and Vertical

Stud Söderberg (VSS). Also the processes with prebaked anodes differ in the place where the pot working (crust breaking and alumina addition) takes place. The three types are Centre-Worked Prebake (CWPB), Point Feed Prebake (PFPB) and Side-Worked Prebake (SWPB) (US-DOE, 1997; EC, 2001). In case of SWPB cells, alumina is fed into the cells after the crust is broken around the circumference. The gas collection hoods over the length of the cells have to be opened during this operation. CWPB cells are fed with alumina after the crust is broken along the centreline or at selected points on the centre line of the cell (PFPB). CWPB and PFPB systems use an automatic feeding system and can be operated without opening the gas collection hoods (EC, 2001).

Total primary aluminium production in the fifteen EU member states in 2003 was 2,573 ktonnes. In Western Europe, Norway is the largest producer of primary aluminium with 1,190 ktonnes in 2003. Other primary aluminium producing countries in the Western Europe are Iceland and Switzerland producing 266 and 44 ktonnes primary aluminium respectively. Altogether these three countries have a share of 1500 ktonnes primary aluminium in a total amount of 4,073 ktonnes produced in Western Europe (37%). The share of Söderberg technology in the total amount of primary aluminium produced within the European Union is 10 percent, see Table 2.2.2.

The Söderberg technology is applied in Sweden and Spain within the EU 15 and in Norway. About 90 percent of the total aluminium production is produced by prebake technologies.

Table 2.2.2. Primary aluminium production in the European Union, including technology shares and production capacities for the year 2006.

Country	Production ^{a)} (ktonnes/year)	Capacity ^{a)} (ktonnes/year)	Technology share ^{b)}		
			VSS ^{c)}	(SW)PB ^{d)}	CWPB ^{e)}
Austria					
Belgium					
Denmark					
France	444	440		0.11	0.89
Finland					
Germany	516	664			1.00
Greece	163	163			1.00
Iceland ^{f)}	325	391			1.00
Ireland					
Italy	190	190			1.00
Luxembourg					
Netherlands	283	340			1.00
Norway ^{f)}	1379	1388	0.13		0.87
Portugal					
Spain	400	402	0.45		0.55
Sweden	100	100	0.75		0.25
Switzerland ^{f)}	44 12	43			1.00
United Kingdom	362	366			1.00
Slovakia	158	159			1.00
Slovenia	119	132			1.00
Poland	55	55	1.00		
Total EU	2800	3020	0.08	0.02	0.90

^{a)} Production and capacity figures from EAA (2004); ^{b)} There is no Horizontal Stud Söderberg production in Europe; ^{c)} Vertical Stud Söderberg; ^{d)} (Side-Worked) Prebake; ^{e)} Centre-Worked Prebake; ^{f)} Norway, Iceland and Switzerland are not part of the EU, but together are significant and additionally part of the EU Environmental legislation.

Ferro-alloys and non-ferro metals (alloys)

There are principally two primary production processes for the production of ferroalloys, the carbo-thermic and the metallo-thermic reduction of oxidic ores or concentrates. The most important process is the carbo-thermic reduction in which carbon, in the form of coke, coal or charcoal is used as a reducing agent. The metallo-thermic reduction is mainly carried out with either silicon or aluminium as the reducing agent.

There are three types of furnaces, which are primarily used for the production of ferro-alloys:

Electric Arc Furnace (EAF)

An electric ‘submerged’ arc furnace is any type of furnace wherein electrical energy is converted to heat by transmission of a current between electrodes partially submerged in the furnace charge. This can be done by using alternating electric current in a furnace with usually three carbon electrodes or by using a direct current in a furnace where the arc strikes between a number of electrodes and the carbon furnace lining. The furnace can be operated batch wise or continuously with a molten charge. The applied electrodes can be of the Søderberg or the prebake type. In the case of continuous operation with a molten charge the electrodes are submerged and do not strike an arc but operate as an electric resistance furnace or electric submerged arc furnace. The electrodes are consumed in the process and must therefore be replaced continuously at certain intervals requiring shutdown of the furnace. To eliminate this Søderberg electrodes were developed. With this type of electrode it is possible to operate continuously. A carbon paste is baked to a fixed electrode inside the furnace as it approaches gradually the warmer part of the furnace. The carbon of the electrodes is consumed during the reduction process or wears away by the action of the arc. Some installations use hollow electrodes, which allow raw material to be fed into the furnace through the electrode. The furnaces can be open, semi-sealed or totally sealed. The open furnace has a fume collection hood above the top of the furnace shell leaving an open area between the furnace and the hood. Sealed or semi-sealed furnaces have no open area between the hood and the furnace. The cover has feed chutes and sealing valves for charging and holes for electrodes to pass through. Sealed furnaces that partially close the hood openings with charge material are referred to as semi-sealed.

Table 2.2.3. Applied technologies for ferro-alloy and non-ferrous metal production.

Material produced ^{a)}	Furnace type ^{b)}	Electrode	Share	electrode consumption (kg/tonne) ^{c)}	Comments
Ferro-Manganese	EAF	Søderberg	77	(8-20)	
	BF	n.a.	23		
Silico-manganese	EAF	Søderberg	100	20-30	
Silicon-metal	EAF			(100)	
Ferro-chromium	EAF	Søderberg	100	7-25	carbo-thermic + silico-thermic
FeCrSi	EAF				same type as ferro-chromium
Ferro-silicon	EAF	Søderberg	100	40-70 (50)	Norway is biggest EU producer, which uses semi-open systems
Calcium-silicon	EAF	Søderberg	100	90 (120)	carbo-thermic method commonly used. Also alumino-thermal is used
Chromium	EAF	-	-	-	Alumino-thermic, silico-thermic and carbo-thermic
Silicon	EAF	Graphite/Prebake	-	100-140	
Ferro-nickel	EAF	Søderberg	100		
Lead	BF	n.a.			
Secondary lead	BF, EF, EAF	-	-	-	few EU smelters apply BF
Zinc	EL	graphite	-	-	only for electro-thermic distillation

Material produced ^{a)}	Furnace type ^{b)}	Electrode	Share	electrode consumption (kg/tonne) ^{c)}	Comments
Nickel	EAF, RF, FS	Søderberg/Prebake			smelting
Tin	EF	graphite	33	10	also reverberatory furnaces are used
Copper slag cleaning	EF	-	-	-	Slag concentration is an alternative process. No electrodes used.
Steel (electric)	EAF	-	100	1.3-14	
White phosphorous	EF	Søderberg/prebake ^{d)}		50	
Calciumcarbide	EAF	Søderberg	100	12	
Siliconcarbide	EF	Graphite	100	-	

Information taken from Sjardin (2003) and Ullmann (2002). n.a. not applicable; no information available; ^{a)} There is no longer primary magnesium production in the European Union, only magnesium recycling; ^{b)} EL: electric furnace; EAF: electric arc furnace; BF: Blast furnace; RF: reverberatory furnace; FS: flash smelting. ^{c)} Electrode consumption based on Ullmann (2002); ^{d)} Share of Søderberg technology in total production capacity. Søderberg technology is applied in Europe, generally in the United States of America prebake is used (Diskowski, 2002).

Electric resistance furnace

This type of furnace uses a similar arrangement to the electric arc furnace, Depending on the size of the furnace 3 to 6 Søderberg or prebaked electrodes are immersed in the liquid raw material. The temperature is maintained by means of electric resistance heating. These furnaces usually operate with coke or slagging agents depending on the application. The electrodes are consumed as the metal oxides are reduced.

Blast furnace

No carbon or Søderberg electrodes are used with this type of furnace. It uses heated air, which is blow into the furnace at the lower part to burn the coke in the furnace charge, which furthermore exists of metal ore and secondary material. Part of the cokes is burned, which provides the heat to produce the melt. CO gas is formed, which reduces the metal oxides. The consumption of coke in blast furnaces is higher than in submerge arc furnaces since coke is also used as a heating source.

There is a large variety of ferro-alloys and non-ferrous metals (alloys) produced applying electric arc furnaces, electric resistance furnaces or blast furnaces. For each type of material produced the type of furnace generally applied in combination with the type of electrode commonly used is given in Table 2.2.3.

Table 2.2.4 gives the production volumes or production capacities of ferro-alloys and non-ferrous metals other than aluminium in the European Union. Largest commodities are the ferrochromium and ferrosilicon alloys with respectively 390,633 and 238,356 tonnes in 1998. As a non-member state Norway is the largest ferro-alloy and non-ferrous metal producer in Western Europe. The majority of these ferro-alloys are produced in electric arc furnaces (EAF) applying Søderberg electrodes (Table 2.2.4).

Table 2.2.4 Ferro-alloy and non-ferro-metal production (1000 tonne/year) in the European Union and other Western European countries in 2001.

Country	Mg	Cr ^{a)}	Sn	Fe-EAF	FeNi ^{b)}	FeMn ^{b)}	FeCr ^{b)}	FeSi ^{b)}	SiMn ^{b)}	Si-metal ^{b)}	CaC	Si ^{a)}	SiC
Austria	n.a.	n.a.	n.a.	503,000	4,000	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Belgium	n.a.	n.a.	8,000 ^{d)}	2,433,000	n.a.	20,000	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Denmark	n.a.	n.a.	100 ^{d)}	790,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
France	4,000	7,000	n.a.	8,059,000	n.a.	130,000	n.a.	100,000	50,000	75,000		139,000	16,000
Finland	n.a.	n.a.	n.a.	901,000	n.a.	n.a.	236,710	n.a.	n.a.	n.a.		n.a.	n.a.
Germany	n.a.	1,000	- ^{e)}	12,096,000	n.a.	n.a.	19,308	n.a.	n.a.	25,000		n.a.	36,000
Greece	n.a.	n.a.	150 ^{d)}	1,109,000	84,200	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Iceland ^{c)}	n.a.	n.a.	n.a.	-	n.a.	n.a.	n.a.	111,948	n.a.	n.a.		46,000	n.a.
Ireland	n.a.	n.a.	n.a.	358,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Italy	n.a.	n.a.	n.a.	15,272,000	n.a.	40,000	-	n.a.	90,000	6,000		n.a.	n.a.
Luxembourg	n.a.	n.a.	n.a.	2,477,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Netherlands	n.a.	n.a.	n.a.	152,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	65,000
Norway ^{c)}	36,000	n.a.	50 ^{d)}	633,000	n.a.	240,000	82,600	450,000	230,000	10,000	180,000	391,000	80,000
Portugal	n.a.	n.a.	- ^{e)}	475,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Spain	n.a.	n.a.	-/25 ^{d)}	10,537,000	n.a.	10,000	-	40,000	100,000	30,000		n.a.	n.a.
Sweden	n.a.	n.a.	n.a.	1,946,000	n.a.	n.a.	109,198	22,000	n.a.	n.a.	45,000	55,000	n.a.
Switzerland ^{c)}	n.a.	n.a.	n.a.	800,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
United Kingdom	n.a.	7 000	-	3,889,000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.		n.a.	n.a.
Total EU	4,000	15,000	8,325	60,997,000	88,200	200,000	365,216	162,000	240,000	136,000		194,000	117,000

n.a. not applicable: probably no production in this country;-: production is zero/terminated; open space: no information available; company information from the internet (<http://www.waddenzee.nl> or <http://www.esk-sic.de>); ^{a)} Production capacity; ^{b)} Produced with electric furnaces; ^{c)} Norway Iceland and Switzerland are not part of the European Union but are the large ferro-alloy producing countries in Western Europe; ^{d)} Secondary production; ^{e)} Primary and secondary production.

3 CLASSIFICATION AND LABELLING

3.1 Classification in Annex VI of Regulation (EC) 1272/2008

CTPHT is included in Annex VI, part 3 under Index No 648-055-00-5.

Classification in Table 3.1

Classification Carc. 1B

H350

Labelling GHS08 Dgr

H350

Note H

Classification in Table 3.2

Classification Carc. Cat. 2;

R45

Labelling T

R45

S53-45

Note H

3.2 Self classification(s)

Not applicable.

4 ENVIRONMENTAL FATE PROPERTIES

The data presented have been taken from different handbooks (e.g. Mackay *et al*, 1992; Douben, 2003). It should be noted that these data have not been re-evaluated.

4.1 Degradation

4.1.1 Stability

PAHs are chemically stable, with no functional groups that results in hydrolysis. Under environmental conditions, therefore, hydrolysis does not contribute to the degradation of PAHs (Howard *et al*, 1991). The main abiotic transformation is photochemical decomposition, which in natural water takes place only in the upper few centimetres of the aqueous phase. PAHs are photodegraded by two processes, direct photolysis by light with a wavelength < 290 nm and indirect photolysis by least one oxidizing agent (Volkering & Breure, 2003). Singlet oxygen usually plays the main role in this process, however, reactions with nitrite and to a lesser extent with nitrate may take place, to form nitro- and hydroxyl-nitro-aromates (Suzuki *et al*, 1987). Endoperoxides may also form an intermediate stage in the reaction chain. The degradation is related to the content of oxygen dissolved and may be accelerated by humic acid (as energy carriers) and increases exponentially with the temperature (Moore & Ramamoorthy, 1984). When PAHs are absorbed on particles, the accessibility for photochemical reactions may change, depending on the nature of the particles. It was shown by Zepp & Schlotzhauer that for PAHs in true solution in “pure” water or seawater, direct photolysis is considerably more significant than photooxidation by means of singlet oxygen. No molecular oxygen is required for this photolysis. In pure water a photodegradation constant for anthracene of approximately 1 hour was measured (Zepp & Schlotzhauer, 1979). There are great differences in photochemical reactivity between the various PAHs. Zepp & Schlotzhauer (1979) studied the photoreactivity of a series of PAHs in water and included the partitioning of PAHs between water and suspended sediment in the experimental half-lives, giving the following sequence of half-lives: anthracene < benz[a]anthracene < benzo[a]pyrene < chrysene < phenanthrene < fluorene < naphthalene.

4.1.2 Biodegradation

Aquatic biodegradation

The results from standard test for biodegradation in water show that PAH with up to four aromatic rings are biodegradable under aerobic conditions but that the biodegradation rate of PAH with more aromatic rings is very low (WHO, 1998). Although some evidence for anaerobic transformation of PAHs has been obtained (Thierrin *et al*, 1993; Coates *et al*, 1997), PAHs are usually considered to be persistent under anaerobic conditions (Neff, 1979; Volkering & Breure, 2003). Because marine sediments are often anaerobic, degradation of PAHs in this compartment is expected to be very slow.

The biochemical pathway for the aerobic biodegradation of PAHs has extensively been investigated. It is understood that the initial step in the aerobic catabolism of a PAH molecule by bacteria occurs via oxidation of the PAH to a dihydrodiol by a multi-component enzyme system. These dihydroxylated intermediates may then be processed through either an ortho cleavage type of pathway, in which ring fission occurs between the two hydroxylated carbon atoms, or a meta cleavage type of pathway, which involves cleavage of the bond adjacent to the hydroxyl groups,

leading to central intermediates such as protocatechates and catechols. These compounds are further converted to tricarboxylic acid cycle intermediates (Van der Meer *et al*, 1992). For the lower molecular weight PAHs, the most common route involves the fission into a C3 compound and a hydroxyl aromatic acid compound. The aromatic ring can thereafter either undergo direct fission or can be subjected to decarboxylation, leading to the formation of a dihydroxylated compound. This compound can be dissimilated as described above. When degraded via these pathways, the low molecular weight PAHs can be completely mineralized to CO₂ and H₂O (Volkering & Breure, 2003).

Although the biodegradation pathway of the different PAHs is very similar their biodegradation rates differ considerably. In general, the biodegradation rate decreases with increasing number of aromatic rings. For example, for degradation by bacteria from estuary half lives for anthracene and benzo[a]pyrene of more than 145 and 1750 days, respectively, were found (Gerlach, 1981). For anthracene in pond water, however, a half-life of over 2 days was found (Leslie *et al*, 1987). In static experiments complete decomposition for naphthalene and phenanthrene, partial decomposition for anthracene and chrysene and no decomposition for fluoranthene was found (Richards & Shieh, 1986). According to Volkering & Breure (2003), two factors are considered responsible for the difference in degradation rate. First, the bacterial uptake rates of the compounds with higher molecular weight have been shown to be lower than the uptake rates of the low molecular weight PAHs. The second and most important factor is the bioavailability of PAHs, due to sorption on suspended organic matter and sediment. Since the K_{OW} and the K_{OC} are strongly correlated, high molecular weight PAHs will degrade slower than low molecular weight PAHs. This is illustrated by Durant *et al* (1995) who found that the half-life of PAHs in estuarine sediment was reversely related to the K_{OW} . Biodegradation rates also are extremely dependent on the (a)biotic conditions both in the laboratory and in the field. Important influencing factors are (1) the substrate concentration; with low PAH concentrations leading to longer half-lives; (2) temperature, which reversely relates to the half-live and (3) the presence or absence of a lag-phase (De Maagd, 1996). In addition, the desorption rate of PAH appears to decrease with increase of the residence time of PAHs due to slow sorption into micropores and organic matter, and polymerization or covalent binding to the organic fraction. The consequence of this aging process is a decreased biodegradability and a decreased toxicity (Volkering & Breure, 2003).

Obviously, due to the large variations it is difficult to predict half-lives of PAHs. For the classification and labelling it is decided to use the suggested mean half-lives by Mackay *et al* (1992).

Biodegradation in soil

Biodegradation is the major mechanism for removal of PAH from soil, although PAHs with fewer than four aromatic rings may also be removed by volatilization and photolysis (WHO, 1998). Many different species of bacteria (both Gram-negative and Gram-positive), fungi, yeasts and algae are known to degrade PAHs (Cerniglia, 1992; Cerniglia *et al*, 1992; Juhasz & Naidu, 2000; Kanaly & Harayama, 2000), of which bacteria are generally assumed to be the most important group of soil micro-organisms contributing in the biodegradation of PAHs in soils (Kästner *et al*, 1994; McGillivray & Shiaris, 1994). Fungi may play a significant role in PAH degradation in the top soil (Cerniglia *et al*, 1992).

Although the toxicity of the metabolites is often lower than the toxicity of the parent compound, their bioavailability may be higher because of their higher water solubility. Comparing the toxicity before and after bioremediation however reveals a decrease in toxicity along with PAH biodegradation (Wang *et al*, 1990; Baud-Grasset *et al*, 1993). According to Volkering & Breure (2003), a possible explanation for this decrease in toxicity is that the intermediates, which are more

reactive than saturated PAHs, undergo polymerization reactions or chemical reactions with the soil organic matter. Recent investigations (Burgos *et al*, 1999; Kästner *et al*, 1999; Nieman *et al*, 1999) have shown that under natural conditions, intermediates of PAH degradation is irreversibly incorporated in the humic soil fraction.

Like for the aquatic environment, there is a relationship between PAH environmental persistence and increasing number of benzene rings which is consistent with the results of various studies correlating environmental biodegradation rates and PAH molecule size (Bossert & Bartha, 1986; Banerjee *et al*, 1995), probably due to changes in the aqueous solubility, bioavailability and structural stability of PAHs through the compound group. Studies on the microbial ecology of PAH-contaminated soils have shown that the number of PAH-degrading micro-organisms, as well as the degrading capacity, are much higher in PAH-contaminated soils than in pristine soils, which implies that an adapted microbial population has been developed (Herbes & Schwall, 1978; Carmichael & Pfaender, 1997). The rate of biodegradation in soil also depends on other external factors, like temperature, the characteristics of the soil (soil type, pH, moisture content, oxygen content and nutrients) and its microbial population (Sims & Overcash, 1983). Some of these factors may also explain why the half-lives observed under laboratory conditions are much shorter than those obtained from long-term field-based experiments. In a study with sandy loams, forest soil, and roadside soil partially loaded with sewage sludge from a municipal treatment plant (Wild & Jones, 1993), the half lives were in the range of weeks, while in a study with soils enriched with PAH-contaminated sewage sludge under field conditions (Wild *et al*, 1991) the half-lives were in the range of years (see Table 4.1.1). Wild & Jones (1993) argued that in the laboratory, soils were kept under conditions that tends to optimize biodegradation potential, that is stable temperatures (20-30 °C), stable moisture content, good lighting and aeration and often added nutrients, while in the field low temperature suspend degradation, oxygen supply may be limited and water saturation may make the soil anaerobic.

Table 4.1.1. The biodegradation rate of PAHs in soil found under laboratory and field conditions.

PAH (number of rings)	Half lives obtained from soil microcosms (days)	Half lives obtained from long term field experiment (years)
Naphthalene (2)	14-48	< 2.0
Acenaphthene and Fluorene (3)	44-74	> 3.2
Phenanthrene (3)	83-193	5.7
Anthracene (3)	48-120	7.9
Fluoranthene (4)	110-184	7.8
Pyrene (4)	127-320	8.5
Benz[a]anthracene and chrysene (4)	106-313	8.1
Benzo[b]fluoranthene (5)	113-282	9.0
Benzo[k]fluoranthene (5)	143-359	8.7
Benzo[a]pyrene (5)	120-258	8.2
Benzo[ghi]perylene (6)	365-535	9.1
Coronene	603-2030	16.5

Data taken from Wild *et al* (1991) and Wild & Jones (1993).

For the classification and labelling it is decided to use the suggested half-lives by Mackay *et al* (1992), which based on a literature search, ranked the PAHs in different classes (see Section 4.1.3).

4.1.3 Summary and discussion of persistence

On the basis of model calculations, Mackay *et al* (1992) ranked the 16 EPA-PAHs according to their persistence in, water, soil and sediment in different classes (Table 4.1.2) which correspond to a specific half-live in these compartments (Table 4.1.3). For the classification these values are used.

Table 4.1.2. Ranking of PAH in different classes.

Compound	Water	Soil	Sediment
Naphthalene	3	5	6
Acenaphthene ^{a)}	3	5	6
Acenaphthylene ^{a)}	3	5	6
Fluorene	4	6	7
Anthracene	4	6	7
Phenanthrene	4	6	7
Fluoranthene	4	7	8
Pyrene	5	7	8
Benz[a]anthracene	5	7	8
Chrysene	5	7	8
Benzo[a]pyrene	5	7	8
Benzo[b]fluoranthene ^{a)}	5	7	8
Benzo[k]fluoranthene	5	7	8
Benzo[ghi]perylene ^{a)}	5	7	8
Dibenz[a,h]anthracene	5	7	8
Indeno[1,2,3-cd]pyrene	5	7	8

^{a)} Classified based on information from literature by the rapporteur.

Table 4.1.3. Suggested half-life classes of PAHs in various environmental compartments.

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	

Classification as suggested by Mackay *et al* (1992).

4.2 Environmental distribution

4.2.1 Adsorption/desorption

Many studies have been performed to determine the organic carbon-water partition coefficient (K_{OC}) of aromatic hydrocarbons, both mono-aromatic and polycyclic compounds. A well known relationship between K_{OC} and K_{OW} is the following equation of Karickhoff *et al* (1979) based on experiments with 10 compounds of which 8 are non-halogenated aromatic compounds, mostly PAHs, in three sediments:

$$\log K_{OC} = \log K_{OW} - 0.21$$

Data for mono-aromatic 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*, 1998b).

Poerschmann & Kopinke (2001) measured the partition coefficient of PAHs and *n*-alkanes to dissolved humic organic matter (HOM). When these partition coefficients are corrected for the

percentage organic carbon in organic matter (by the standard factor of 1.7), the resulting $\log K_{OC}$ values for PAHs are in accordance with the other data for PAHs (Figure 4.2.1).

The last years, more evidence becomes available that sorption of organic chemicals into soils and sediments can be better described by a two-phase model. This model assumes that two main types of organic carbon exist: amorphous organic carbon, with a linear sorption, and black carbon (or carbonaceous geosorbents) with non-linear (Freundlich) sorption (Cornelissen *et al*, 2005). A model to describe a two-phase system is that of Bucheli & Gustafsson (2000) and Accardi-Dey & Gschwend (2003):

$$K_{PM} = K_{POC} f_{POC} + K_{BC} f_{BC} [PAH]_{Dissolved}^{(n-1)}$$

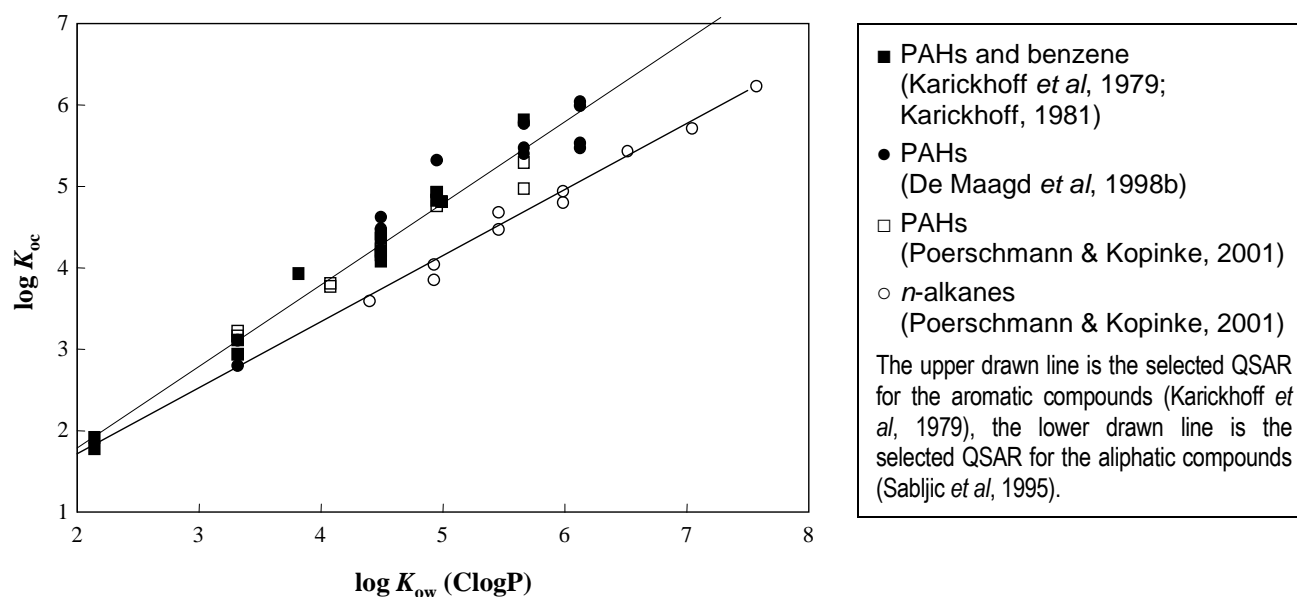


Figure 4.2.1. Organic carbon-water partition coefficients as a function of $\log K_{ow}$.

From several studies (Jonker & Koelmans, 2001; Burgess *et al*, 2004; Lohmann *et al*, 2004; Vinturella *et al*, 2004) it appears that the partition coefficients to soot-like particles (black carbon; K_{BC}) are much higher than the partition coefficients normalised to the total of organic carbon in the sediment or soil (K_{OC}). These values for K_{BC} are a factor of 10 to 59 higher than the values used in the risk assessment, except from the data by Jonker & Koelmans (2001), which are 59 to 950 times higher than the values used in the risk assessment, but only 3.5 to 22 times as high as the K_{OC} values for amorphous organic carbon determined in the same way. Overall, the partitioning to carbonaceous materials can be up to 60 times higher than the partitioning to the commonly used organic carbon.

The relative importance of the non-linear sorption depends on both the concentration of black carbon and the concentration of the PAH (see Figure 4.2.2). For more information the reader is referred to Koelmans *et al* (2006). Cornelissen *et al* (2005) state that at 10-40 % of the aqueous solubility of a compound, black carbon has the potential to dominate sorption. De Maagd (1996) has used concentrations of 7.5-75% of their aqueous solubilities, with aqueous solubilities ranging from 0.137 $\mu\text{g/L}$ (benzo[ghi]perylene) to 34800 $\mu\text{g/L}$ (naphthalene). For these concentrations, Koelmans *et al* (2006) showed that both sorption to black carbon and sorption to amorphous organic matter play a role. Which of these two sorption sites is dominant depends entirely on the concentration used and the compound used.

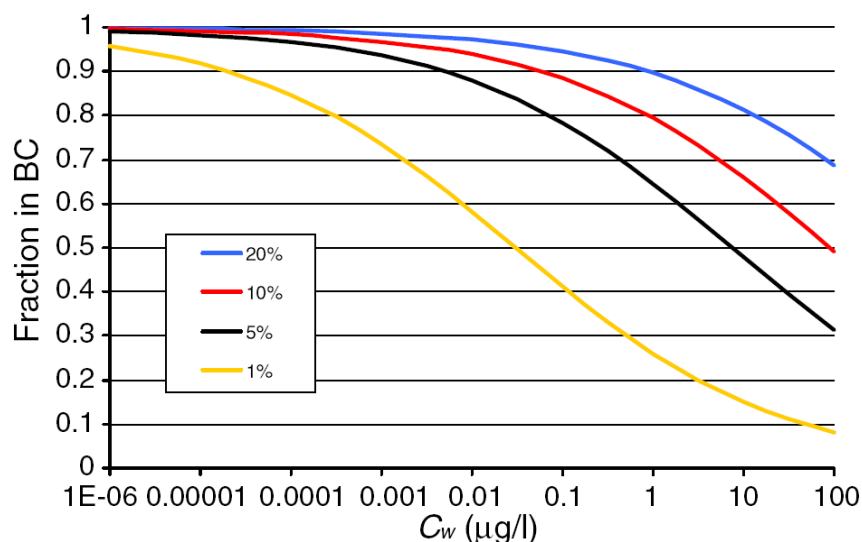
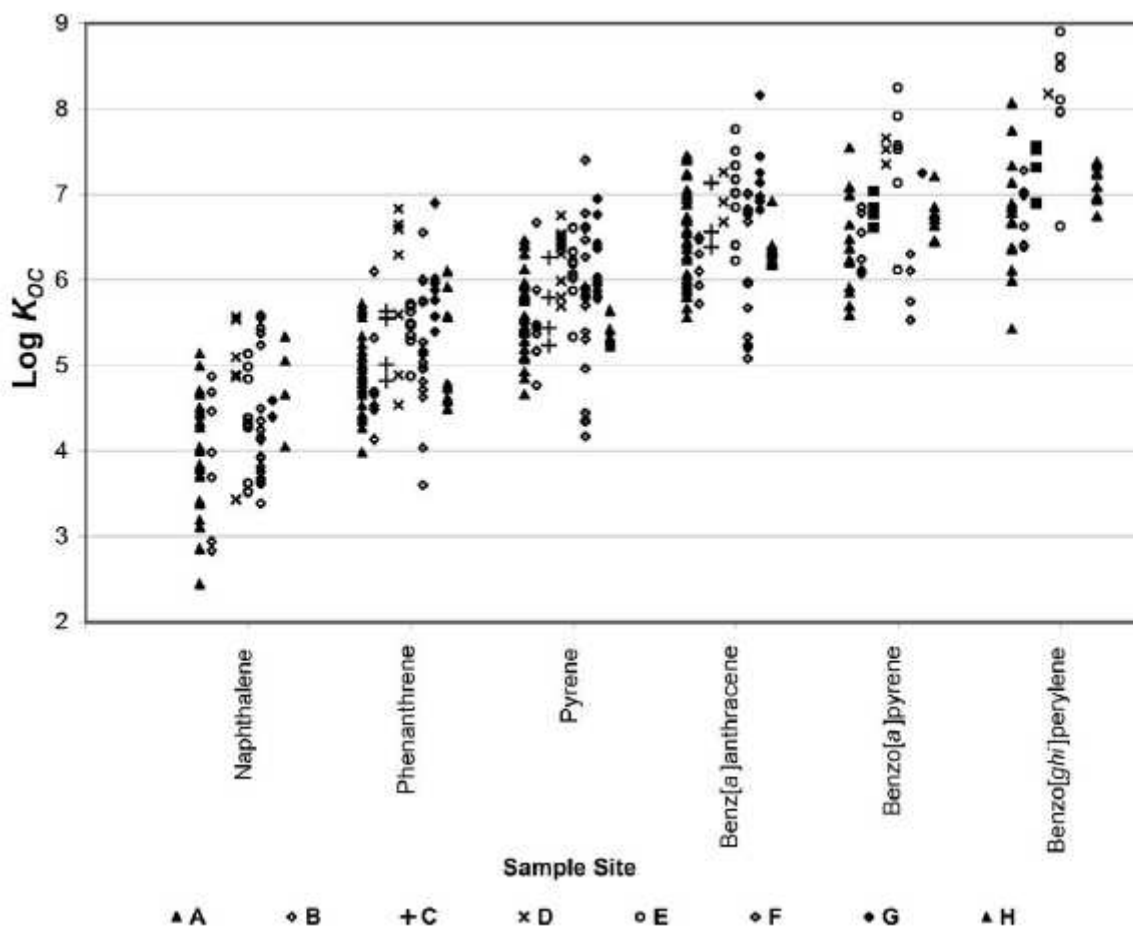


Figure reproduced from Koelmans *et al* (2006).

Figure 4.2.2. Fraction of organic pollutant bound to black carbon as a function of aqueous carbon concentration (C_w), for 1%, 5%, 10% and 20% black carbon (of total organic carbon).



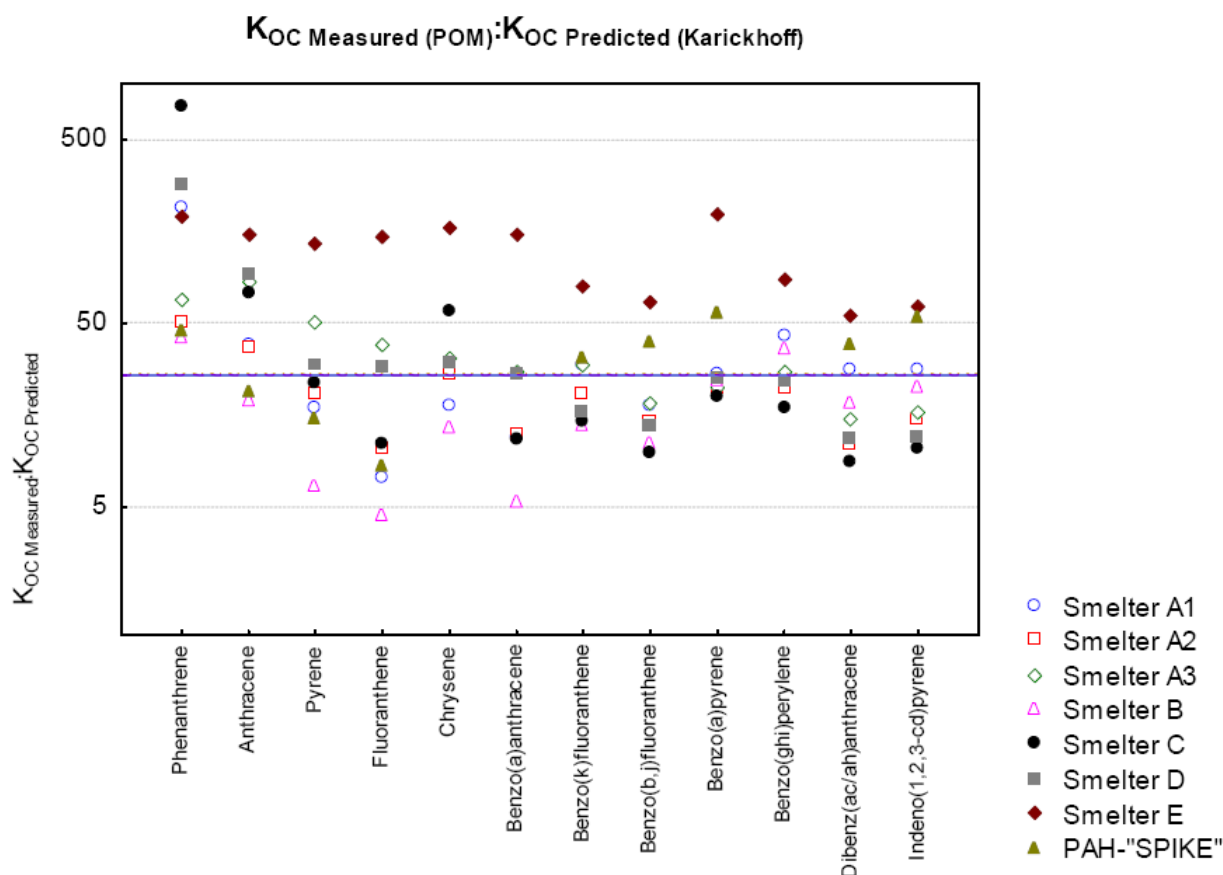
Determined by Hawthorne *et al* (2006). Sites A up to F are rural or urban (light) industrial sites with manufactured gas plants as likely PAH sources. Site G and H are rural industrial sites with aluminium smelters as likely PAH sources.

Figure 4.2.3. Log sediment organic carbon-water partitioning coefficients (K_{OC}) values for several polycyclic aromatic hydrocarbons at different industrial sites.

At field-realistic amounts of 1% black carbon (of total organic carbon), at concentrations of 1 µg/L 80% is sorbed to amorphous organic matter and 20% is sorbed to black carbon. Thus, for the study

of De Maagd (1996) the sorption of chrysene, benzo[k]fluoranthene, benzo[a]pyrene, and benzo[ghi]perylene, was not dominated by black carbon.

In a study of Hawthorne *et al* (2006) it was demonstrated that at different historically contaminated sites, the K_{OC} value of nearly all PAHs show a high degree of variation up to three orders of magnitude (see Figure 4.2.3), likely as a result of a range of carbon types having different sorption characteristics. The lowest K_{OC} values measured were close to the ones proposed by Karickhoff *et al* (1979), whereas the median value was an order of magnitude higher. It was concluded that the dependence of K_{OC} values on the site location was significant for some sites; however it did not appear to be related to the likely source of PAHs. This was illustrated by the difference between the two sites contaminated by the aluminium smelters, where one site had high K_{OC} values while the other site had among the lowest K_{OC} values (see Figure 4.2.3). In this absence of information on the black carbon content no relationship between the K_{OC} values and the black carbon content can be made.



Free-energy relationship calculations are following Karickhoff *et al* (1979). The ratio is presented for all sediments and all PAHs (from phenanthrene) presented from left to right with increasing K_{OW} s. The median (26) is presented by a blue line. Note logarithmic scale (Ruus *et al*, 2007)

Figure 4.2.4. The ratio between the organic carbon:water-partitioning coefficient (K_{OC}) deduced by POM-solid phase extraction and the predicted K_{OC} derived from K_{OW} , using free-energy relationship.

In order to investigate the particle affinity of PAHs associated with coal tar pitch, freely dissolved PAH-fractions in sediments outside several Nordic aluminium smelters were measured by the use of polyoxymethylene-solid phase extraction (POM-SPE), and sediment-water partitioning coefficients (K_d) were determined (Ruus *et al*, 2007). The results showed that measured K_d s were much higher than predicted from free-energy relationships, following Karickhoff *et al* (1979) (see Figure 4.2.4). However, there was no clear relationship between the black carbon content in sediment (ranging from 0.11-5.7%) and the K_{OC} values measured. Moreover, the K_{OC} values

measured in spiked sediment from a reference site with a relatively low black carbon content (< 0.1%) were one of the highest. It is therefore difficult to interpret these data in the current classification and labelling. The PAH concentrations in the sediments with a high black carbon content (*i.e.* at the vicinity of smelter B and C) were also significantly higher than in the other sediments. In view of the concentration dependency, sorption at these sites might be less dominated by black carbon.

In conclusion, the two-phase model may have better predictive powers than a one-phase model (e.g. Moermond *et al*, 2005; Koelmans *et al*, 2006). Research in this field is still on-going. To be able to use this two-phase sorption model, it is important to know the fraction of black carbon and the fraction of amorphous organic carbon. It should also be noted that the quantification of carbonaceous materials still suffers from operational shortcomings (Cornelissen *et al*, 2005). Thus, although the two-phase model seems to be an improvement over the one-phase model, in practice it can only be used when black carbon is measured. This is very site-specific. Moreover, care should be given to the fact that when partition coefficients for the ‘pure’ organic carbon phases are combined, this exceeds the actual, experimentally measured sorption. Thus, K_{BC} values for pure black carbon are not necessarily valid under *in situ* conditions, probably due to attenuation effects by dissolved organic matter molecules (Koelmans *et al*, 2006).

For the purpose of the CLH-report the one-phase model as proposed by Karickhoff *et al* (1979), which incorporates field-derived sediments with mixtures of all types of organic carbon (including both black carbon and amorphous organic carbon), is used to derive ‘general’ K_{OC} values for the different PAHs (see Table 4.2.1).

Table 4.2.1. The log K_{OC} for the 16 EPA-PAHs.

Compound	Log K_{ow}	Log K_{OC} ^{a)}
Naphthalene	3.34	3.13
Acenaphthene	4.00	3.79
Acenaphthylene	3.62	3.41
Fluorene	4.22	4.01
Anthracene	4.68	4.47
Phenanthrene	4.57	4.36
Fluoranthene	5.20	4.99
Pyrene	4.98	4.77
Benz[a]anthracene	5.91	5.70
Chrysene	5.81	5.60
Benzo[a]pyrene	6.13	5.92
Benzo[b]fluoranthene	6.12	5.92
Benzo[k]fluoranthene	6.11	5.90
Benzo[ghi]perylene	6.22	6.01
Dibenz[a,h]anthracene	6.50	6.29
Indeno[1,2,3-cd]pyrene	6.58	6.37

^{a)} Values are based on the equation of Karickhoff *et al* (1979).

Factors influencing the sorption and bioavailability of PAHs

Aging

The residence time of PAHs in soil and sediment, also referred to as aging, will alter the sorption and in concomitance with the bioavailability (Alexander, 1995; Belfroid *et al*, 1996; White *et al*, 1997; Chung & Alexander, 1998; Nam *et al*, 1998; Tang & Alexander, 1999). Studies with sediment showed that when freshly spiked PAHs are more readily desorbed and thus more bioavailable than PAHs from aged sediments (Kukkonen & Landrum, 1995; Chung & Alexander, 1998). Landrum *et al* (1992) observed that sorption of pyrene and phenanthrene increased with

increasing residence time (in order of months). Belfroid *et al* (1996) hypothesized that sediment and also soil can be considered as more-compartment systems, in which the organic contaminant partitions between interstitial water and several compartments in the sediment/soil particles. The molecules bound to the particles will become available only after diffusion into the interstitial water, which is biphasic process with a rapid initial desorption phase followed by a slow phase. The impact of this slow desorption process seems to increase with increasing residence time of the contaminant in soil and sediment. Several studies indicate that bioavailability decreases with increasing residence time, resulting in a reduction of bioaccumulation in benthic organisms (Landrum *et al*, 1992; Harkey *et al*, 1994; Harkey *et al*, 1995). However, the extent of aging differs between soils at which the soil organic carbon content appears to be a major determinant, as been demonstrated by Nam *et al* (1998) who found no aging effects in soils with an organic carbon content less than 2%. The low carbon content in combination with high test concentrations could also explain the absence of aging effects on the toxicity of pyrene and phenanthrene to *Folsomia fimetaria* (Sverdrup *et al*, 2002). Since the standard soil according to the EU TGD has a organic carbon content of 2% and the fact that aging is insufficiently quantifiable, aging is as yet not considered in the classification and labelling.

Relationship between sorption and the bioavailability of PAHs

As mentioned above, the origin of the organic carbon to which the PAHs are associated may have its influence on the partition coefficients and the kinetic rate of desorption. In this way, strong sorbing carbonaceous materials may limit the bioavailability of PAHs to soil and sediment species more than amorphous organic carbon on average does. Especially the role of carbonaceous materials such as black carbon, coals and kerogen is subject of discussion. This has been reviewed extensively by Cornelissen *et al* (2005) and Koelmans *et al* (2006).

The higher partition coefficients to black carbon indicate that soot-like materials may have a major influence on the bioavailability to soil and sediment species. However, the implication for classification and labelling of coal tar pitch is difficult to interpret. With respect to the emission from the aluminium production (including paste preparation and anode baking), all of the 2-, 3- and part of the 4-ring PAHs (*i.e.* fluoranthene and pyrene) are mainly emitted in the gaseous or dissolved form, whereas the 5-rings and some 4-ring PAHs (*i.e.* benz[a]anthracene and chrysene) are mainly bound to particles. The effect of the sorption on carbonaceous materials on uptake of PAHs by biota is still unclear. Where some studies show that uptake of PAHs is significantly decreased in the presence of carbonaceous materials, others show that this effect is not present or negligible (see below).

Ghosh *et al* (2003) determined for two field sediments desorption and biodegradation of PAHs. Various types of carbonaceous materials were picked out of these sediments, and for these individual types of black carbon also desorption and biodegradation of PAHs was determined. They conclude that PAHs present in coal tar pitch were more bioavailable than PAHs sorbed to carbonaceous materials such as coal, coke and charcoal. PAHs associated with the black carbon fraction were not available for biodegradation, while PAHs associated with coal tar pitch were available for biodegradation. Differences in biodegradation between the two sediments were explained by a difference in PAH desorption rates from organic carbon fractions. Finally they show that even in the presence of black carbon, PAHs may remain primarily associated with original source materials such as coal tar pitch and may be available for microorganisms for biodegradation.

In the effect of activated carbon on the bioavailability of organic compounds was investigated by Zimmerman *et al* (2004) and Millward *et al* (2005), which measured accumulation of PCBs from sediment for polychaetes (*Neanthes arenaceodentata*) and amphipods (*Leptocheirus plumulosus*). In 29 day accumulation experiments it was shown that 6 months after the addition of 3.4% activated carbon to sediment, bioaccumulation is reduced with 87% for *N. arenaceodentata* and with 75% for *L. plumulosus*. Water concentrations and accumulation in semi-permeable membrane devices

(SPMDs) were reduced in the same order of magnitude. However, while the reduction of bioaccumulation was very clear for activated carbon, the addition of the same amount of coke did not have any effect on bioaccumulation, although water concentrations were reduced with 38-64%.

Zimmerman *et al* (2005) tested the effect of the addition of different amounts of activated carbon on field sediments on water concentrations and accumulation in SPMDs. After one month of equilibration, water concentrations were reduced by 81% in the sediments treated with 1.7% activated carbon. More activated carbon did not reduce the water concentrations further. However, uptake of PAHs by SPMDs was further reduced with increasing activated carbon content: at 3.4% activated carbon SPMD accumulation was reduced with 90%. No uptake in biota was measured for PAHs, but for PCBs in the same study bioaccumulation by *N. arenaceodentata* and *L. plumulosus* was reduced with 93% and 90% respectively, at 3.4% activated carbon addition.

McLeod *et al* (2004) reported the effect of sorption of benzo[a]pyrene on various types of organic carbon on bioavailability through food uptake for the clam *Macoma balthica*. The absorption efficiency was 41% when diatoms were added as an organic carbon source, and decreased in the order of diatoms > wood > char > anthracite > peat > coke > activated carbon. For coke and activated carbon, the absorption efficiency was only 11% and 2%, respectively.

Several experiments show that despite the effect of carbonaceous materials on sorption to sediments, bioavailability does not seem to decrease significantly with increasing black carbon content. From experiments with eight marine benthic invertebrates (*Cirriformia grandis*, *Clymenella torquata*, *Macoma balthica*, *Mulinia lateralis*, *Mya arenaria*, *Nereis virens*, *Pectinaria gouldii*, and *Yoldia limatula*) it appeared that, normalised to organic carbon, bioaccumulation factors of PAHs (1-methylfluorene, phenanthrene, fluoranthene, benz[a]anthracene, chrysene, benzo[a]pyrene, and 7-methylbenzo[a]pyrene, 1-methylphenanthrene, and 3,6-dimethylphenanthrene) were not significantly influenced by the amendment of the sediment with soot, from diesel exhaust. Median ratios between accumulation with and without soot were only 1.3. The authors consider it as surprising that significant bioaccumulation occurs of PAHs from sediments amended with high levels of soot (1.9% by weight), but they state that this might be explained by digestive exposure to soot-bound PAHs. It is concluded that it cannot be assumed that soot-bound PAHs are not available to benthic species or that there is a uniform reduction in bioavailability for all benthic species. Other sources of soot than diesel exhaust were not investigated (Rust *et al*, 2004a).

In a second study by Rust *et al* (2004b) the effect of coal dust, tire rubber, diesel soot, creosote, crude oil, and fuel oil on the bioaccumulation of PAHs to three marine benthic invertebrates (*Cirriformia grandis*, *Clymenella torquata*, and *Macoma balthica*) was studied. Except from coal dust, the normalised bioaccumulation factors (BAFs) were within a factor of two for the studied PAHs (1-methylfluorene, phenanthrene, 1-methylphenanthrene, 3,6-dimethylphenanthrene, fluoranthene, pyrene, chrysene, and benzo[a]pyrene) in almost all cases. Log BAF values ranged from about -1 to +0.5.

Also for the saltwater deposit feeder *Nereis succinea* it was concluded that the presence of soot had less influence on the bioavailability of benzo[a]pyrene than on the desorption from sediment (Lamoureux & Brownawell, 2004). In another study bioavailability seems to be strongly reduced but concentrations in organisms were not directly compared to sediment concentrations (Vinturella *et al*, 2004). Further, the effect of black carbon from field sediments on the bioaccumulation by the amphipod *Monoporeia affinis* was studied (Sundelin *et al*, 2004). Although it is mentioned that the content of black carbon (ranging 0.13 to 0.45%) has an influence on the bioaccumulation, a closer look at the data reveals that for the six spiked PAHs (phenanthrene, pyrene, fluoranthene, benz[a]anthracene, benzo[a]pyrene, and benzo[ghi]perylene) this is only the case for phenanthrene. For the six compounds log lipid normalised BAF values range from -2 to 0.67.

In order to investigate bio-availability of PAHs associated with coal tar pitch, Ruus *et al* (2007) evaluated in the same study as mentioned above the bioaccumulation of PAHs from sediments outside several Nordic aluminium smelters in a mesocosm experiment using the polychaete *Nereis diversicolor*, the gastropod *Hinia reticulata* and the bivalve *Nuculoma tenuis*.

The biota-sediment accumulation factors (BSAFs) measured for *Nereis diversicolor* and *Hinia reticulata* were very similar to BSAFs expected based on the POM-deduced sediment-water partitioning coefficients (see Figure 4.2.5). In contrast, the expected biota concentrations calculated from the Karickhoff *et al* (1979) free-energy relationship, sediment concentrations and BCFs corresponded not as good with the concentrations actually measured in these species.

The good correspondence with the POM-deduced partitioning coefficients was not observed for *Nuculoma tenuis*, in which higher PAH concentrations were measured. Here the PAH profiles (relative concentrations of PAH compounds) showed stronger resemblance to the sediments (relatively higher concentrations of higher molecular weight compounds).

It was argued that logistical intractabilities connected to this species biology and size rendered it probable that particulate sedimentary matter contaminated the *N. tenuis* tissues samples. According to the authors, it would seem less likely that the difference between the other species is due to differences in metabolism and elimination rates. However, in order to draw any conclusion more information is needed on the exposure routes and metabolic transformation capacities of the species. For *Nereis sp.* it is known that they are able to metabolize PAHs.

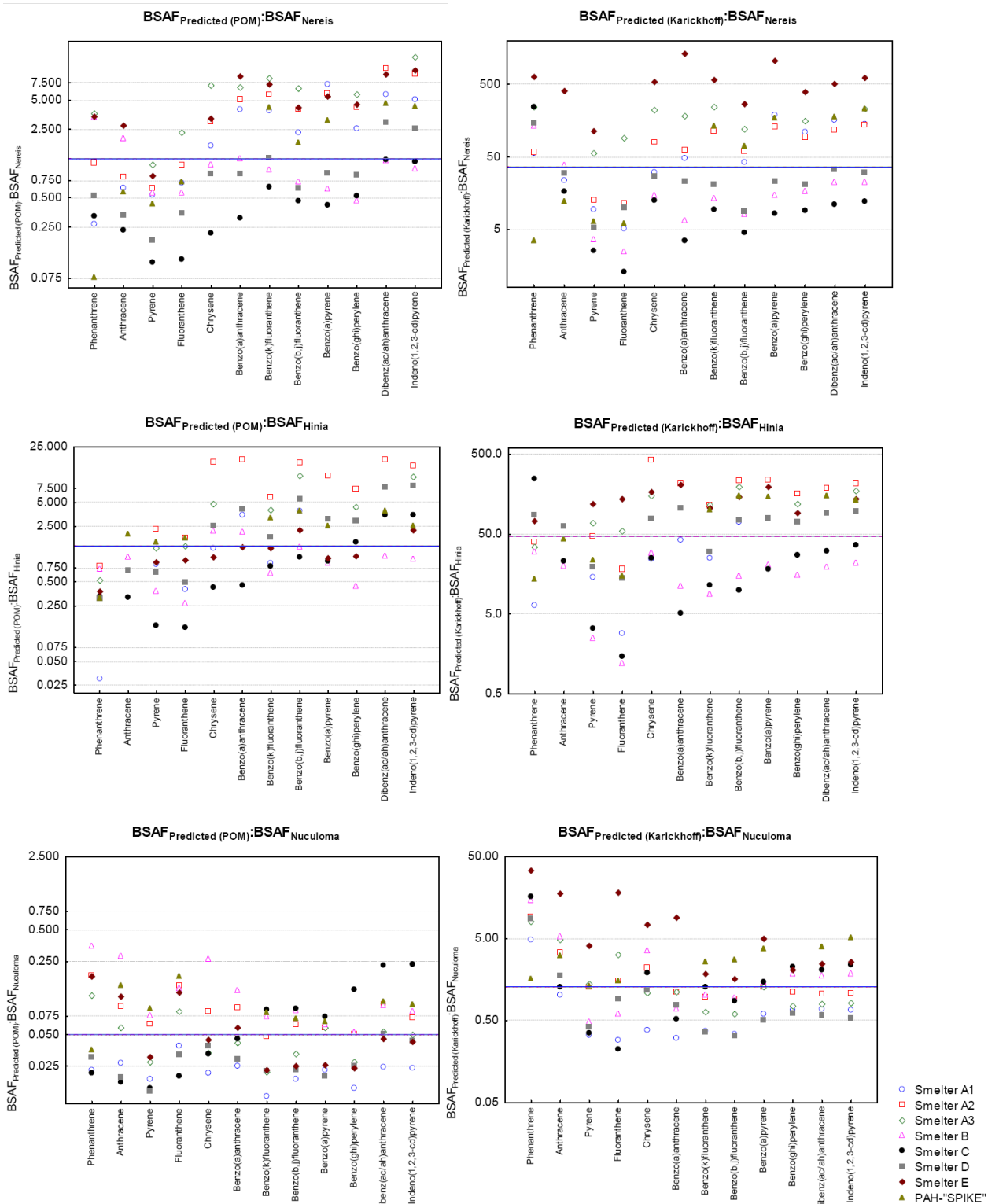
It should also be noted that the degree of variation did not significantly decline when the “POM-predicted” biota concentrations were compared to the actual measured concentrations in the species. If the “POM method” would be a measure for the real bioavailability, we would expect the variation among sediments to be lower for the POM-based estimations than when Karickhoff-based estimations are used. Furthermore, the measured concentration in biota is even lower than what would be expected with POM-based estimations. This could indicate that other processes than bioavailability (like metabolic transformation!) also play a role in these low BSAFs.

Summary

Several studies indicate that bioavailability decreases with increasing residence time. The extent of aging seems to be dependent on the organic carbon content. As no ageing effect were found at an organic carbon content of standard soil (2%) and the fact that this phenomenon is insufficiently quantifiable, aging is not considered in the classification and labelling.

The adsorption and desorption of PAHs to carbonaceous materials can show a high degree of variation, likely as a result of the origin of the organic carbon to which the PAHs are associated. Consequently, strong sorbing carbonaceous materials may limit the bioavailability of PAHs to soil and sediment species. However, the implication for classification and labelling of coal tar pitch is as yet difficult to interpret. In addition, the effect of the sorption on carbonaceous materials on uptake of PAHs by biota is still unclear. Where some studies show that uptake of PAHs is significantly decreased in the presence of carbonaceous materials, others show that this effect is not present or negligible.

Based on these considerations and the uncertainties on this topic the eventual binding to soot-like materials is not taken into account in the classification of CTPHT.



From top to bottom: *Nereis diversicolor*, *Hinia reticulata* and *Nuculoma tenuis*. Medians are presented by blue lines. Note different scales (logarithmic) on the figures (Ruus *et al.* 2007).

Biota to sediment accumulation factors (BSAFs) calculated from the POM-SPE deduced sediment-water partitioning coefficients, K_{OWs} and the organic carbon normalized sediment PAH-concentrations. The actual measured BSAFs are calculated from the lipid normalized concentrations in the organism and the organic carbon normalized concentrations in the sediments; The “Karickhoff-predicted” BSAF=1.62.

Figure 4.2.5. The ratio between the “POM-predicted” biota to sediment accumulation factors and the actual measured BSAFs (left), and the ratio between the “Karickhoff-predicted” biota to sediment accumulation factor, and the actual measured BSAFs (right).

4.2.2 Volatilisation

The Henry law constants for PAHs rank from 49 Pa m³/mol for naphthalene to 0.007 Pa m³/mol for dibenz[a,h]anthracene (see Table 1.3.4). Volatilization plays a significant role in surface water and it depends on temperature, water movement, wind and the molecular size of the PAHs. Especially for naphthalene and the 3-ring PAHs volatilization is significant (Southworth, 1979).

Under field conditions the following observations were made: For the Dutch big rivers the Rhine, Meuse and IJssel the half-lives of PAH have been calculated in two extreme cases: (a) a slow-moving river (0.14 m/s) at a wind velocity of 2 m/s (approximately Force 2) and (b) a fast-moving river (1.7 m/s) at a wind velocity of 20 m/s (approximately Force 9). The average depth of water was put at 5 m and the water temperature at 11 °C. For naphthalene and benzo[a]pyrene the half-lives for volatilization are in situation (b) 0.4 and 420 hours, respectively. At comparatively low temperature the Henry coefficient decreases whereas the half-lives increase (Slooff *et al*, 1989). Naphthalene was volatilized from soil at a rate of 30% after 48 hours, with negligible loss of PAH with three or more rings (Park *et al*, 1990).

For the classification of CTPHT the Henry's law constants shown in Table 1.3.4 were used.

4.2.3 Distribution modelling

The distribution of the 16 EPA-PAHs in sewage treatment plants has been calculated using the model SIMPLETREAT integrated to EUSES (EU, 2008a) based on the K_{OC} values and the Henry's law constants presented in Table 4.2.1 and Table 1.3.4, respectively. They are presented as an example in Table 4.2.2.

Table 4.2.2. Estimation of removal of the 16 EPA in a sewage treatment plant.

PAH compound	% to air	% to water	% to sludge	% degraded	% removal
Naphthalene	38.7	47.2	12.6	1.5	52.8
Acenaphthene	11.0	47.4	40.3	1.3	52.6
Acenaphthylene	12.4	62.8	22.9	1.8	37.2
Fluorene	5.7	41.6	52	0.3	58.4
Anthracene	1.5	25.2	73.1	0.2	74.8
Phenanthrene	1.6	29	69.2	0.2	71.0
Fluoranthene	0.1	14.3	85.5	0.1	85.7
Pyrene	0.3	18	81.7	0.0	82.0
Benz[a]anthracene	0.0	9.3	90.7	0.0	90.7
Chrysene	0.0	9.6	90.3	0.0	90.4
Benzo[a]pyrene	0.0	8.8	91.2	0.0	91.2
Benzo[b]fluoranthene	0.0	8.8	91.2	0.0	91.2
Benzo[k]fluoranthene	0.0	8.8	91.2	0.0	91.2
Benzo[ghi]perylene	0.0	8.7	91.3	0.0	91.3
Dibenz[a,h]anthracene	0.0	8.3	91.7	0.0	91.7
Indeno[1,2,3-cd]pyrene	0.0	8.3	91.7	0.0	91.7

Values according to EUSES 2.0.

4.3 Bioaccumulation

4.3.1 Aquatic bioaccumulation

Accumulation of PAHs from water is strongly dependent on the physicochemical properties of the compound and the species exposed: the rates of accumulation and elimination generally decrease

with increasing molecular weight (corresponding to a lower solubility). Different factors linked to animal behaviour and characteristics influence uptake and accumulation of PAHs, such as biotransformation, size of the organism, avoidance of highly contaminated sites, burrowing behaviour, density of the organism population and bioturbation. Metabolism may be very important in explaining PAH accumulation patterns. It is suspected that high molecular weight PAHs (HPAHs) are more rapidly metabolized than low molecular weight PAHs (LPAHs) due to differences in enzyme affinity (Schnell *et al*, 1980). PAHs are metabolized by the phase I enzymes of the mixed function oxygenase system (MFO) to more hydrophilic products like phenols, dihydrodiols, quinines and epoxides (Lech & Vodcnik, 1985; Sijm & Opperhuizen, 1989). Some of the PAHs can be excreted directly as unconjugated polar metabolites in bile (via the gallbladder), but most PAH will be excreted after conjugation by phase II enzymes (Vermeulen *et al*, 1992). Molluscs have a very limited ability to metabolize PAHs, while in algae and oligochaete worms no evidence of PAHs metabolism has been found. Although polar metabolites of PAHs are excreted rapidly to water, some metabolites are released more slowly than the unmetabolized parent compounds (Slooff *et al*, 1989).

Other important behavioural aspects that influence bioavailability and uptake of PAHs are life history and the feeding strategy: *i.e.* whether an organism is a suspension feeder, a deposit feeder, a herbivore browsing on particle surfaces, or a predator (Leppänen, 1995; Kaag *et al*, 1997). Owing to their greater ingestion rates, deposit feeders tend to accumulate more PAHs than suspension feeders. Parkerton (1993) concluded from an extensive literature study that organic carbon- and lipid-normalised BSAF levels in filter feeders, deposit feeders, and omnivores were not similar. Although the scatter within the groups was considerable, the general pattern revealed that BSAFs for deposit feeders comprising oligochaete and polychaete worms were slightly higher. Also a study with two types of bivalves showed that the deposit feeding tellinid clams (*Macomona liliana*) accumulated higher PAH levels than the suspension-feeding cockles (*Austrovenus stutchburyi*) and oysters (*Crassostrea gigas*). BSAFs deviated by one to three orders of magnitude from the predicted equilibrium BSAF value of 1.7 (Hickey *et al*, 1995). Another study with the infaunal amphipod *Rhepoxynius abronius* that does not ingest sediment, and the infaunal deposit feeding polychaete *Armandia brevis* found similar accumulations of the LPAHs by the two species but substantially more accumulation of HPAHs by the polychaete (Meador *et al*, 1995). The results of this study suggest that deposit and non-deposit-feeding infaunal invertebrates will acquire most of their body burden of LPAHs through pore water, regardless of feeding strategy. Experimental and fitted BCFs provided evidence that the uptake for *Lumbriculus rubellus* of PAHs with $\log K_{OW} < 5$ is mediated through direct contact with the soluble phase. For PAHs of higher $\log K_{OW}$, the dietary uptake may provide an additional route of exposure, but will not exceed 10% of the total uptake in earthworms (Belfroid *et al*, 1994).

Recently, bioaccumulation studies of the 16 EPA-PAHs (see Table 1.2.1) in aquatic organisms were extensively evaluated (Bleeker & Verbruggen, 2009). The key studies from this evaluation are summarized in Table 4.3.1. For naphthalene, acenaphthene, acenaphthylene and fluorene invertebrates showed lower BCF values than fish, so in Table 4.3.1 only fish data are included. For the other PAHs also data on molluscs and crustaceans is included (if available), because considerable differences were found for the different test species due to differences in biotransformation capabilities.

Table 4.3.1 Summary of highest reliable BCF values for the 16 EPA-PAHs.

Substance	BCF value (WW)	BCF value (5% lipid norm.) ^{a)}	Species	Type ^{b)}	Reference ^{c)}
Naphthalene ¹⁾	999	515	<i>Cyprinodon variegatus</i> (Fish)	Kin.	(Jonsson <i>et al.</i> , 2004)
Acenaphthene ¹⁾	760	1000	<i>Cyprinus carpio</i> (Fish)	Equi.	(RIITI, 1990a)
Acenaphthylene ¹⁾	387	509	<i>Cyprinus carpio</i> (Fish)	Equi.	(RIITI, 1990b)
9H-fluorene ¹⁾	1459	1658	<i>Pimephales promelas</i> (Fish)	Kin.	(Carlson <i>et al.</i> , 1979)
Anthracene	4973	– ²⁾	<i>Pimephales promelas</i> (Fish)	Equi.	(Hall & Oris, 1991)
	– ³⁾	19000	<i>Perna viridis</i> (Mollusc)	Equi.	(Richardson <i>et al.</i> , 2005)
	39727	21098 ⁴⁾	<i>Pontoporeia hoyi</i> (Crustacean)	Kin.	(Landrum, 1988)
Phenanthrene	3611	4751	<i>Pimephales promelas</i> (Fish)	Kin.	(Carlson <i>et al.</i> , 1979)
	28043	14893 ⁴⁾	<i>Pontoporeia hoyi</i> (Crustacean)	Kin.	(Landrum, 1988)
Fluoranthene	2439	2772	<i>Pimephales promelas</i> (Fish)	Kin.	(Carlson <i>et al.</i> , 1979)
	– ³⁾	12250	<i>Perna viridis</i> (Mollusc)	Equi.	(Richardson <i>et al.</i> , 2005)
	58884	– ⁵⁾	<i>Diporeia spec.</i> (Crustacean)	Kin.	(Schuler <i>et al.</i> , 2004)
Pyrene	1297	1474	<i>Pimephales promelas</i> (Fish)	Kin.	(Carlson <i>et al.</i> , 1979)
	– ³⁾	12250	<i>Perna viridis</i> (Mollusc)	Equi.	(Richardson <i>et al.</i> , 2005)
	166000	88157 ⁴⁾	<i>Pontoporeia hoyi</i> (Crustacean)	Kin.	(Landrum, 1988)
Benz[a]anthracene	260	– ⁶⁾	<i>Pimephales promelas</i> (Fish)	Equi.	(De Maagd <i>et al.</i> , 1998a)
	63000	33457 ⁴⁾	<i>Pontoporeia hoyi</i> (Crustacean)	Kin.	(Landrum, 1988)
Chrysene	6088	– ⁷⁾	<i>Daphnia magna</i> (Crustacean)	Equi.	(Newsted & Giesy, 1987)
Benzo[a]pyrene	608	– ⁸⁾	<i>Lepomis macrochirus</i> (Fish)	Kin.	(Jimenez <i>et al.</i> , 1987)
	191000 ⁹⁾	119375	<i>Dreissena polymorpha</i> (Mollusc)	Kin.	(Gossiaux <i>et al.</i> , 1996)
	73000	38768 ⁴⁾	<i>Pontoporeia hoyi</i> (Crustacean)	Kin.	(Landrum, 1988)
Benzo[b]fluoranthene	–	–	No experimental data available	–	–
Benzo[k]fluoranthene	13225	– ¹⁰⁾	<i>Daphnia magna</i> (Crustacean)	Equi.	(Newsted & Giesy, 1987)
Benzo[ghi]perylene	28288	– ¹¹⁾	<i>Daphnia magna</i> (Crustacean)	Equi.	(Newsted & Giesy, 1987)
Dibenz[a,h]anthracene	50119	– ¹²⁾	<i>Daphnia magna</i> (Crustacean)	Equi.	(Newsted & Giesy, 1987)
Indeno[1,2,3-cd]pyrene	–	–	No experimental data available	–	–

^{a)} BCF values are normalized to organisms with a lipid content of 5%. ^{b)} Kin.: kinetic BCF value (k_1/k_2); Equi. BCF value at (assumed) equilibrium ($C_{organism}/C_{water}$).

¹⁾ For these compounds invertebrates showed lower BCF values than fish, show only fish data are given. ²⁾ In this study no lipid content was given, but based on lipid contents in fathead minnows reported by Carlson *et al.* (1979) lipid content is expected to be 5 – 6 %, which would result in lipid normalized values ranging from 4100 – 5000. ³⁾ In this study only lipid-based BCF values were given, but lipid content itself was not reported ⁴⁾ In this study lipid content was expressed only as percentage of dry weight (35%). In addition the ratio between total wet weight and dry weight was given (0.269). For lipid normalization it was assumed that the same ratio holds for lipids, resulting in a lipid content of 9.4% based on wet weight. ⁵⁾ In this study no lipid content was given, but for a lipid normalized value to fall below the trigger value of 500 the lipid content needs to be 590%, which is impossible. ⁶⁾ In this study no lipid content was given, but for a lipid normalized value to exceed the trigger value of 500 the lipid content needs to be 2.6% or lower, which seems to be unrealistically low To fall below the trigger value of 100 the lipid content needs to be 13% or higher, which is also not likely. ⁷⁾ In this study no lipid content was given, but Liu *et al.* (1996) report lipid contents in *Daphnia magna* ranging from 4 – 6%, suggesting that a lipid normalized BCF value will be similar to the wet weight value. If the lipid content is higher than 61%, the normalized BCF value will be below 500, but such a lipid content is highly unlikely. ⁸⁾ In this study no lipid content was given, but for a lipid normalized value to exceed the trigger value of 500 the lipid content needs to be 6.1% or lower. ⁹⁾ Higher BCF values were reported, but this is the highest value for which lipid normalization could be applied. ¹⁰⁾ In this study no lipid content was given, but for a lipid normalized value to fall below the trigger value of 500 the lipid content needs to be 130%, which is impossible. ¹¹⁾ In this study no lipid content was given, but for a lipid normalized value to fall below the trigger value of 500 the lipid content needs to be 280%, which is impossible. ¹²⁾ In this study no lipid content was given, but for a lipid normalized value to fall below the trigger value of 500 the lipid content needs to be 500%, which is impossible.

4.3.2 Terrestrial bioaccumulation

According to the Technical Guidance Document (EU, 2003), the bioconcentration in earthworm can be described as a hydrophobic partitioning between the pore water and the phases inside the organism. This equilibrium partitioning approach can be modelled according to the following equation as described by Jager (1998):

$$BCF_{\text{earthworm}} = (0.84 + 0.012K_{\text{OW}}) / \rho_{\text{earthworm}}$$

where for $\rho_{\text{earthworm}}$ by default a value of 1 ($\text{kg}_{\text{wwt}}/\text{L}$) can be assumed.

The feasibility of this QSAR for bioconcentration of PAHs in earthworm is evaluated by Jager *et al* (2000). *Eisenia andrei* were exposed to artificial soil spiked with series of phenanthrene, pyrene, fluoranthene and benzo[a]pyrene concentrations. Because the concentration in the organisms did not reach a steady state, the BCF were expressed dynamically as ratio of the uptake and elimination rate constants. The BCF were slightly higher than expected from the QSAR which might have been a feature of PAHs in particular but was considered more likely caused by experimental errors (*e.g.* a slightly overestimated sorption). The BCF based on the elimination rate constant (k_e) from the accumulation phase agree much better with the expected values, thereby indicating that k_e from the depuration experiment was erroneously low. Jager *et al* (2003) also determined the BCF values of PAHs in *E. andrei* exposed to field contaminated soils. Both the BCF and BSAF values were generally lower than the equilibrium partitioning estimate (on average a factor of 11) and also lower than the maxima observed in spiked artificial soil medium. Ma *et al* (1998) even found on average a factor of four lower values for PAHs in *Lumbricus rubellus*. However, the actual concentration that the field-collected earthworms had been exposed to is not easily reconstructed. It was postulated that the kinetics of depletion of pore water PAHs, and the kinetics of their replenishment, have influenced the accumulation patterns and the steady-state body residues. Jager *et al* (2003) concluded that the equilibrium partitioning approach can be considered to estimate the maximum amount that can be taken up, but the total variation in body residues and uptake kinetics may be driven by differences in assimilation efficiencies between soils, as well as differences in desorption kinetics of PAHs from soils. As a reasonable worst case subsequent BCF values for the selected PAHs are used for the classification and labelling, which are given in Table 4.3.2.

Table 4.3.2. Calculated BCF values in earthworm.

Compound	Log K_{OW}	BCF ^{a)}
Naphthalene	3.34	27
Acenaphthene	4.00	120
Acenaphthylene	3.62	51
Fluorene	4.22	200
Anthracene	4.68	580
Phenanthrene	4.57	450
Fluoranthene	5.20	1900
Pyrene	4.98	1200
Benz[a]anthracene	5.91	9800
Chrysene	5.81	7800
Benzo[a]pyrene	6.13	16000
Benzo[b]fluoranthene	6.12	16000
Benzo[k]fluoranthene	6.11	15000
Benzo[ghi]perylene	6.22	20000
Dibenz[a,h]anthracene	6.50	38000
Indeno[1,2,3-cd]pyrene	6.58	46000

^{a)} Calculations based on the equation described in the Technical Guidance Document (EU, 2003).

While the equilibrium partitioning model is applicable for the earthworm-soil system, in which the BSAF is independent of the log K_{OW} , the BSAFs for the isopods *Porcellio scaber* and *Philoscia muscorum* are clearly negatively correlated with the log K_{OW} . It appears that earthworms accumulate higher concentrations of PAH than isopods. Field sampling of three species of isopods and an earthworm species from PAH-contaminated sites showed that earthworms accumulated one to two order of magnitude more PAHs than the isopods (Van Brummelen *et al.*, 1996). The deviation from the equilibrium partitioning model can be explained via non-equilibrium conditions: (a) metabolism of PAHs, (b) limited contact among the compartments *e.g.* through a lack of pore water abundance or through physical boundaries (exoskeleton in isopods) or (c) restricted contact with soil (Van Brummelen *et al.*, 1996).

4.3.3 Summary and discussion of bioaccumulation

Almost all EPA-PAHs show BCF values for fish above the criterion for bioaccumulating potential of 500 for Regulation EC 1272/2008 (EU, 2008b), when values are corrected to a standard fish with a lipid content of 5%, although values for acenaphthylene and benzo[a]pyrene are close to this criterion. Due to uncertainties in lipid content in fish, lipid normalized values for benz[a]anthracene and benzo[a]pyrene are not certain, but are likely to fall between 100 and 500. Consequently BCF values for all EPA-PAHs are above the criterion from EU Directive 67/548/EEC (EU, 1967).

4.4 Secondary poisoning

There are several indications that biomagnification of PAHs does not occur in both the aquatic and terrestrial environment, partly being the result of the relatively high rates of metabolism and excretion of PAHs in vertebrates and some invertebrates (Neff, 1979; Broman *et al.*, 1990; Clements *et al.*, 1994; Suedel *et al.*, 1994). Although some primary consumers and detritivores may accumulate high levels of PAHs, predators usually contain low levels (Niimi & Dookhran, 1989; Hellou *et al.*, 1991; Lemaire *et al.*, 1993; Clements *et al.*, 1994). This phenomena of biominification (process of decreasing concentrations with rising trophic levels) is also observed in extensive biomonitoring studies on aquatic organisms in the Rhine-Meuse estuary, with the highest PAH concentrations found in aquatic plants, oligochaetes, isopods and freshwater clams, lower concentrations in other molluscs and chironomids and concentrations below the detection limits in roach and liver of 7 week-old cormorant chickens feeding on roach and other cyprinids (Van Hattum *et al.*, 1993; Den Besten *et al.*, 1995; Van Hattum *et al.*, 1996; Van Hattum *et al.*, 1998).

Although biomagnification of PAHs is not expected for food webs involving fish, species from the lower trophic levels that are not able to effectively metabolize these compounds may exhibit food web transfer. Predatory molluscs and polychaetes that prey on the other polychaetes and molluscs would likely have higher PAH tissue residues than other similar species that only ingest sediment (Meador, 2003).

Food web transfer of PAHs metabolites is another area that has received little attention. Even though parent PAHs may not be biomagnified, prey species may contain high levels of metabolites that could be accumulated by predators. This was examined by McElroy & Sisson (1989), who fed polychaetes (*Nereis virens*) containing benzo[a]pyrene and accompanying metabolites to winter flounder (*Pseudopleuronectes americanus*) and found that fish had accumulated the metabolites.

5 HUMAN HEALTH HAZARD ASSESSMENT

The database on human health hazards induced by CTPHT is rather limited, and it is, therefore, hardly possible to perform a full effect assessment for all the required endpoints. There is, though, quite some information from epidemiological studies on workers in specific industrial processes where CTPHT is produced and/or used, that indicate that carcinogenicity is a hazard associated with CTPHT (see Section 5.8.4). This is attributed to the presence of PAHs in CTPHT, for which benzo[a]pyrene has been chosen as exposure indicator (see Section 1 on general substance information). For the human health effects assessment, as well as for risk characterisation, these worker population studies, therefore, are very important. Because of the rather different scenario-specific CTPHT-PAH profiles, these data were originally grouped per scenario, using benzo[a]pyrene as a scenario-specific effect indicator. However, as is indicated in Section 5.8.4, such a scenario-specific approach appeared only possible for the aluminium smelter industry.

In addition, the scarce data from experimental studies using CTPHT and related substances, and additional relevant literature were evaluated for the below section.

In October 2006 CTPHT was discussed in the TC C&L (see Annex II). In this discussion the classification of CTPHT for several non-CMR hazard classes has been agreed. In addition it was agreed to classify CTPHT as a category 1 carcinogen and as a category 2 mutagen and reprotoxic compound. In this light, additional classifications for non-CMR hazard classes have limited added value. Therefore, no classification is proposed for non-CMR health classes and these endpoints are not further discussed in this document.

5.1 Toxicokinetics (absorption, metabolism, distribution and elimination)

No data were available which allow a quantitative estimation of absorption of CTPHT from inhalation, dermal, and oral exposure. The absorption of those components that are considered relevant with regard to the critical toxicological effects is, of course, of utmost importance. The absorption is different for the different toxicologically relevant components of CTPHT, as illustrated by different absorption rates for different non-particle-bound PAHs. Due to the variable physical form and composition of CTPHT and CTPVHT the predictive value of absorption studies conducted with non-particle-bound PAHs is limited. Absorption after inhalation of particle-bound PAHs depend on particle size, the smaller the particles, the more extensive the PAH elute from the particles. Oral and dermal absorption of PAHs from solid CTPHT is probably low compared to the absorption of PAHs from CTPVHT and fine dust, due to the binding of PAHs in the pitch matrix of solid CTPHT. Based on the calculated dermal absorption of ten different PAHs from dermally applied coal tar to pig-ears (ranging from 1 % to > 30%; Van Rooij *et al*, 1995) a dermal absorption of PAHs from CTPHT of 30% is proposed as worst case estimate. Based on these data a dermal absorption of 30% is taken forward to classification and labelling.

Since quantitative data on the absorption of PAHs from CTPHT and CTPVHT after inhalation and oral exposure are lacking, default values for absorption can be used (EC, 2003): for CTPHT default values of 100% (in this case) may be used for absorption of critical components via inhalation and oral exposure. Although these default values are probably too high, especially for the absorption of PAHs from solid CTPHT, it is not possible to quantify the extent of this likely overestimation of the inhalation and oral absorption rates.

5.2 Acute toxicity

Not relevant for this CLH-report.

5.3 Irritation

Not relevant for this CLH-report.

5.4 Corrosivity

Not relevant for this CLH-report.

5.5 Sensitisation

Not relevant for this CLH-report.

5.6 Repeated dose toxicity

Not relevant for this CLH-report.

5.7 Mutagenicity

In vitro and *in vivo* genotoxicity tests with CTP or CTPV are summarised in Table 5.7.1 and Table 5.7.2, respectively.

5.7.1 In vitro data

In vitro: bacteria, yeast and mammalian cells

CTP was mutagenic in *Salmonella typhimurium* strain TA98 when tested in the presence of a metabolic activating system. Negative results were obtained when tested without S9 or in strain TA100 both with and without S9. The doses tested were 0, 0.05, 0.25, 2.5, and 5 mg/plate. The results were confirmed by a second trial (Solorzano *et al*, 1993).

Referring to papers presented at congresses, Schimberg *et al* (1980) stated that cyclohexane extracts of dust samples collected from different work phases and foundries-amongst others those using CTP-showed mutagenic activity in *S. typhimurium* strains TA98 and TA100 in the presence of an S9 liver metabolic activating system. The mutagenic activity did not directly correlate with the benzo[a]pyrene concentration in the samples (no more details presented; Schimberg *et al*, 1980).

Condensates of fumes generated from CTP by heating 10 kg samples to 232 or 316°C were strongly mutagenic when tested in *S. typhimurium* strain TA98 in the presence of an induced hamster liver S9 mix. The condensate generated at 316°C had a mutagenic index 2 to 3 times higher than that generated at 232°C, and contained significantly higher concentrations of PAH. Both condensates contained considerably lower concentrations of PAHs than the CTP from which they were generated (Machado *et al*, 1993).

A DMSO extract of (unspecified) CTP was found positive in *S. typhimurium* strains TA1537, TA98, and TA100 in the presence of an S9 mix, while negative results were obtained when tested without metabolic activation (IARC, 1985).

Results from testing dichloromethane extracts of roofing-tar pot emissions in *S. typhimurium* TA1537, TA1538, TA98, and TA100 were positive in the presence and negative in the absence of a metabolic activation system. No mutagenic activity was seen in strain TA1535. This material-which was characterised in the several underlying papers as a pitch-based tar, an asphalt tar or derived from a CTP-was examined in a number of other test systems as well. Negative results were obtained in *S. cerevisiae* D3 (endpoint: mitotic recombination; only one concentration tested) and in Syrian hamster embryo cells (endpoint: DNA fragmentation); both tests were performed without metabolic

activation only. Results were positive in BALB/c3 3T3 cells (ouabain resistance) and in mouse lymphoma L5178Y cells (TK^{+/+} mutation), both with and without metabolic activation. In Chinese hamster ovary cells, the material induced a significant increase in the frequency of sister chromatid exchanges (tested both with and without S9), but no mutations (tested without S9 only). Finally, increases in morphologically-transformed foci in BALB/c 3T3 cells (both with and without metabolic activation; not statistically significant) and in viral transformation in Syrian hamster embryo cells (statistically significant) were found (IARC, 1985).

In vitro: human body fluids

Heussner *et al* (1985) monitored genotoxicity in 27 smoking and 23 non-smoking workers exposed to CTPV in an aluminium reduction plant and in 28 smoking and 22 non-smoking non-exposed workers from various other sites of this plant. Exposure was not determined at the time of the investigations, but measurements performed approximately 10 years before showed levels of CTPV of 0.5 to 3.42 mg/m³ in the anode production area. Extracts of 86 urine samples were analysed for the presence of mutagenic substances by performing two separate assays at 2 concentrations in *S. typhimurium* strains TA98 and TA100 both with and without induced rat liver homogenates. 43 samples were from the exposed workers and an equal number were from non-exposed workers. Fourteen out of 43 exposed workers and 7/43 non-exposed workers had mutagenic compounds in their urine. Among the non-mutagenic samples, toxicity was observed in 15 exposed and 7 non-exposed samples. Negative results (no mutagenic or toxic response) were observed in 14/43 exposed and 29/43 non-exposed workers samples. The presence of toxic urines complicates the interpretation of the results. The difference in mutagenic compounds in urine between exposed and non-exposed workers was statistically significant if toxic urines were not included ($p < 0.01$). If the toxic urines are pooled with the non-mutagenic urines the significance level drops ($p < 0.08$). However, if the toxic urines were combined with the mutagenic urines, the observed difference is highly significant ($p < 0.002$). Cigarette smoking was related to urine mutagenicity in similar ways in both the exposed and non-exposed workers. Among smokers, incidences of mutagenic urine were 10/23 and 6/19 in exposed and non-exposed workers ($p < 0.15$), respectively; among non-smokers, 4/20 and 1/24, respectively ($p < 0.05$). Overall chromosome aberration rates in lymphocytes were similar in both exposed and non-exposed workers. Among exposed workers a significant inverse correlation ($p < 0.05$) between age and chromatid aberration rate was observed. Results of semen analysis failed to detect differences between exposed and non-exposed workers (Heussner *et al*, 1985).

In its review on selected non-heterocyclic polycyclic aromatic hydrocarbons, WHO (1998) summarised studies in which the mutagenicity of urine of persons exposed to polycyclic aromatic hydrocarbons has been tested in *S. typhimurium* strains TA98 or TA100 both with and without metabolic activation. Frequently, several urine samples from both exposed and control persons appeared to be too toxic to allow evaluation of the mutagenic potential. In most of the studies of workers exposed during activities such as coking, coal tar distillation, work in aluminium (Søderberg potrooms), anode, and graphite electrode plants, the results were negative. Only in cases of heavy exposure (patients with psoriasis to coal tar applications; workers at coke ovens or in a carbon plant) positive results were obtained. In addition, expectorate from workers in a coke and UK in a aluminium (Søderberg potrooms) plant were reported to be positive when tested in the presence of a metabolic activating system in *S. typhimurium* strains TA98 and TA100 (WHO, 1998).

Table 5.7.1. Genotoxicity of CTP or CTPV *in vitro*.

Assay	Compound	Species	Result	Reference
<i>Bacteria</i>				

Assay	Compound	Species	Result	Reference
Bacterial gene mutation test	CTP	<i>S. typhimurium</i> (TA 98, 100)	Positive +S9 in TA98, negative -S9 in TA98 and +S9 and -S9 in TA100	(Solorzano <i>et al</i> , 1993)
Bacterial gene mutation test	Cyclohexane extracts of dust samples in iron foundries (using CTP)	<i>S. typhimurium</i> (TA 98 100)	Positive +S9	(Schimberg <i>et al</i> , 1980)
Bacterial gene mutation test	Condensates of fumes generated from CTP by heating to 232 or 316 °C	<i>S. typhimurium</i> (TA 98)	Positive +S9	(Machado <i>et al</i> , 1993)
Bacterial gene mutation test	DMSO extract of (unspecified) CTP	<i>S. typhimurium</i> (TA 98, 100, 1537)	Positive +S9, negative -S9	(IARC, 1985)
Bacterial gene mutation test	Dichloromethane extract of roofing-tar pot emissions	<i>S. typhimurium</i> (TA 98, 100, 1535, 1537, 1538)	Positive +S9 in TA 98, 100, 1537, 1538, negative +S9 in TA 1535 and -S9 in TA 98, 100, 1535, 1537, 1538	(IARC, 1985)
<i>Yeast</i>				
Mitotoc recombination	Dichloromethane extract of roofing-tar pot emissions	<i>S. cerevisiae</i> D3	Negative without metabolic activation	(IARC, 1985)
<i>Mammalian cells</i>				
DNA fragmentation	Dichloromethane extract of roofing-tar pot emissions	Syrian hamster embryo cells	Negative without metabolic activation	(IARC, 1985)
Gene Mutation	Dichloromethane extract of roofing-tar pot emissions	BALB/c3 3T3 cells (ouabain resistance)	Positive with and without metabolic activation	(IARC, 1985)
Gene Mutation	Dichloromethane extract of roofing-tar pot emissions	Mouse lymphoma L5178Y cells (TK ^{+/-})	Positive with and without metabolic activation	(IARC, 1985)
Sister Chromatid Exchange	Dichloromethane extract of roofing-tar pot emissions	Chinese hamster ovary cells	Positive with and without metabolic activation	(IARC, 1985)
Gene mutation	Dichloromethane extract of roofing-tar pot emissions	Chinese hamster ovary cells	Negative without metabolic activation	(IARC, 1985)
Morphological transformation	Dichloromethane extract of roofing-tar pot emissions	BALB/c3 3T3 cells	Increase in transformed foci (not statistically significant) with and without metabolic activation	(IARC, 1985)
Viral transformation	Dichloromethane extract of roofing-tar pot emissions	Chinese hamster ovary cells	Increase in transformed foci (statistically significant) with and without metabolic activation	(IARC, 1985)
<i>Human body fluids</i>				
Bacterial gene mutation test	Human urine sample, occupational exposure in an aluminium reduction plant to a.o. CTPV	<i>S. typhimurium</i> (TA 98 100)	Positive with and without metabolic activation	(Heussner <i>et al</i> , 1985)
Bacterial gene mutation test	Human urine sample, occupational exposed during coking, coal-tar distillation, work in Søderberg potrooms of aluminium plants, anode plants, and graphite electrode plants	<i>S. typhimurium</i> (TA 98 100)	Negative with and without metabolic activation	(WHO, 1998)
Bacterial gene mutation test	Human urine sample, heavy exposure of psoriasis patients to coal-tar applications, and coke oven, and carbon plant workers	<i>S. typhimurium</i> (TA 98 100)	Positive with and without metabolic activation	(WHO, 1998)

Assay	Compound	Species	Result	Reference
Bacterial gene mutation test	Human expectorate sample, occupational exposed workers of coke plant and aluminium (Søderberg potrooms) plant	<i>S. typhimurium</i> (TA 98 100)	Positive with metabolic activation	(WHO, 1998)

In addition to these tests with CTP(V) or human body fluids of workers exposed to CTP(V), various *in vitro* genotoxicity studies with coal tar, coal tar products and several individual PAHs demonstrated the genotoxicity of these substances (studies not summarised) (WHO, 1998; ATSDR, 2002).

5.7.2 In vivo data

There were no data on the results of testing the potential genotoxicity of CTPHT in experimental animals.

Oral administration of coal tar or coal tar waste resulted in increased DNA adduct formation in several *in vivo* studies (ATSDR, 2002). Dose-related increases in DNA adduct levels were observed in B6C3F₁ mice fed up to 2 g coal tar/100 g food for 28 days (Culp & Beland, 1994; Culp *et al.*, 1996a, b). DNA adducts were detected in liver, lung, and forestomach by ³²P-postlabeling.

In addition to this study, several other *in vivo* genotoxicity studies in experimental animals with coal tar, coal tar waste, coal tar products, and individual PAHs demonstrated the genotoxicity of these substances (studies not summarised) (WHO, 1998; ATSDR, 2002).

5.7.3 Human data

Several studies have been carried out on mutagenic/genotoxic effects (*e.g.* micronuclei, chromosomal aberrations, SCEs in lymphocytes, DNA adducts) in individuals and populations occupationally exposed to mixtures of PAH among which CTP. However, basically, these investigations did not address the potential *in vivo* mutagenicity/genotoxicity due to exposure to CTP or other complex mixtures containing PAH but aimed at finding sensitive methods for measuring exposure to CTP or similar PAH-containing mixtures.

Heussner *et al.* (1985) monitored genotoxicity in workers exposed to CTPV in an aluminium reduction plant (study is described in more detail under *in vitro* studies). Blood was sampled for cytogenetic analysis. No statistically significant differences in chromosomal aberrations were found between exposed and non-exposed groups (Heussner *et al.*, 1985).

Buchet *et al.* (1995) investigated cytogenetic endpoints (sister chromatic exchanges, high frequency cells, and micronuclei) in peripheral lymphocytes of 56 male workers of 2 coke oven/steel foundry plants and 93 workers of one graphite electrode plant. A control group consisting of 137 workers mainly from the steel foundry (rolling mills) plants was included. PAH exposure was assessed by means of personal air sampling of 13 selected PAH and measurement of 1-hydroxypyrene concentrations in post-shift urine. The groups did not differ with respect to education level and smoking habits but the mean age of the graphite electrode workers was slightly lower than that of the coke oven workers and the controls. Although there was no statistically significant difference in mean total PAH exposure levels between the coke oven and graphite electrode workers-15.96 (range: 0.540-1106.4 mg/m³) and 20.5 mg/m³ (range: 0.13-1212 mg/m³, respectively, *vs.* 0.700 mg/m³ (0.120-3.830 mg/m³) in controls -, some differences in individual PAH among which benzo[a]pyrene (0.068 and 0.219 mg/m³, respectively) were seen. In addition, graphite electrode workers had significantly higher mean urinary 1-hydroxypyrene levels although pyrene air levels were similar. When compared to controls, (arithmetic) mean values for the percentage of micronuclei found in lymphocytes were lower in the exposed groups. There were increases in the

(arithmetic) mean number of SCEs per cell in the non-smoking graphite electrode workers (4.8, n=4 observations vs. 3.9 in non-smoking controls, n=29) as well as in the smoking coke-oven workers (4.9, n=16 vs. 4.0, n=30), but decreases in SCEs in smoking graphite electrode workers (3.7, n=1 vs. 4.0, n=30) and non-smoking coke-oven workers (4.0, n=16 vs. 2.9, n=71) were observed. The (square-root) mean percentage of high-frequency cells was increased in the non-smoking and smoking coke-oven workers (5.3, n=16 vs. 2.3, n=29, and 7.0, n=16 vs. 3.1, n=30, respectively) and decreased in smoking and non-smoking graphite workers (0.9, n=4 vs. 2.3, n=29, and 2.6, n=1 vs. 3.1, n=30). Based on logistic regression, high frequency cells were associated with the intensity of current exposure to PAHs, but not with duration of exposure. No consistent associations between other cytogenic effects and PAH exposure were found (Buchet *et al*, 1995).

Van Delft *et al* (1998) examined PAH-DNA adduct levels in peripheral blood lymphocytes of workers of a carbon-electrode manufacturing plant by a ^{32}P -postlabelling method. On basis of job conditions, workers were divided into three groups with presumed low, intermediate, and high exposure, based on historic data from air sample analysis. The low-exposure group consisted of 5 smoking and 14 non-smoking laboratory and office workers and served as a control group, the high-exposure group of 9 smoking and 8 non-smoking workers from the carbon-anode factory while 19 (7 smokers, 12 non-smokers) workers basically not stationed in this factory-mainly maintenance technicians active across the entire plant -formed the intermediate-exposure group. Groups were comparable as to age although within the high-exposure group, there was a considerable difference in average age between smokers and non-smokers (47 vs. 35 y, respectively). Personal air sampling resulted in median total PAH levels of 8.4 (range: 1.8-80 mg/m^3 ; n=12 air samples) for the intermediate exposure group and 32 mg/m^3 (range: 2.3-185 mg/m^3 ; n=18; p=0.099) for high exposure group, while those of benzo[a]pyrene were 0.37 (range 0.09-5.0 mg/m^3) and 1.20 mg/m^3 (range: 0.43-3.2 mg/m^3 ; p=0.024) for the intermediate and high exposure group, respectively. Urinary 1-hydroxypyrene levels were significantly higher in the intermediate-exposure (3.6-fold) and high-exposure (8.2-fold) groups when compared with the control group. There were no statistically significant differences for any of the PAH-DNA adduct clusters (*i.e.* combining all adduct areas or taking various zones and spots separately) between groups. The levels of total adducts and of adducts in some zones and spots were higher in lymphocytes of smokers than in those of non-smokers, being statistically significant for the latter only (Van Delft *et al*, 1998).

Arnould *et al* (1999) monitored benzo[a]pyrene-DNA adducts in leucocytes from 17 (12 smoking, 5 non-smoking) workers of a carbon-electrode-producing plant by a ^{32}P -postlabelling method and a competitive immunoassay using polyclonal antibodies obtained from rabbits immunised with DNA modified by benzo[a]pyrene-*trans*-7,8-dihydrodiol-9,10-epoxide. The control group consisted of 10 (5 smoking, 5 non-smoking) administrative workers. The exposed workers were older than the controls (age ranges: 27-53 and 18-35 y, respectively). Benzo[a]pyrene exposure levels determined by sampling at different fixed workstations in the plant ranged from 0 mg/m^3 for the control group to 575 to 1149 ng/m^3 for the exposed group. Levels of adducts (expressed as fmol/50 μg of DNA) obtained by the immunoassays were significantly higher than those obtained by postlabelling. Adduct levels in smokers were higher than those in non-smokers and those in exposed higher than those in non-exposed. No statistical analysis was presented (Arnould *et al*, 1999).

Carstensen *et al* (1999b) have analysed aromatic adduct formation to DNA in peripheral lymphocytes from 98 male potroom workers (median age: 35 y; range: 22-60 y) in an aluminium reduction plant and 55 male blue-collar workers (mail carriers and city council employees; median age: 41 y; range: 22-61) from the same town as a control group, using a ^{32}P - postlabelling method. Thirty-one percent of the exposed group were smokers as compared with 22% of the control group. Personal air sampling of both particulate and gas phase PAHs performed during a full workday for the workers and for 5 randomly selected controls resulted in median levels of the sum of 22 selected particulate PAHs of 13.2 (range: 0.01-270 $\mu\text{g}/\text{m}^3$) and 0.11 $\mu\text{g}/\text{m}^3$ (range: 0.01-0.37 $\mu\text{g}/\text{m}^3$) for

workers and 3/5 controls (no detectable levels in 2 other ones), respectively. Median Benzo[a]pyrene levels were $1 \mu\text{g}/\text{m}^3$ (range: $0.02\text{-}24 \mu\text{g}/\text{m}^3$) for the workers and 0.004 and $0.02 \mu\text{g}/\text{m}^3$ for the 2 controls with measurable levels. Levels of the sum of 7 gas phase congeners ranged from 0.01 to $131 \mu\text{g}/\text{m}^3$ (median: $16.3 \mu\text{g}/\text{m}^3$) in workers to 0.008 to $0.41 \mu\text{g}/\text{m}^3$ (median: $0.20 \mu\text{g}/\text{m}^3$) in controls. No difference in the frequency in DNA adducts was found between the potroom and blue-collar workers. Smoking habits did not affect results (Carstensen *et al*, 1999b).

In a study on the influence of genetic polymorphisms of biotransformation enzymes on genotoxic events, micronuclei in peripheral CD4^+ and CD8^+ lymphocytes, DNA single-strand breaks, HPRT mutation frequency, and urinary 8-hydroxydeoxyguanosine were investigated in the aforementioned potroom and blue-collar workers. No differences in these endpoints were found between the 2 groups (Carstensen *et al*, 1999a).

Concerning other genotoxic endpoints, it was stated in the WHO review (1998) that no increases in the rates of micronuclei, chromosomal aberrations, or sister chromatid exchanges were reported in coke-oven, carbon-plant, aluminium-plant, or graphite-electrode plant workers, or in chimney-sweeps; in most cases, significant effects of smoking could be detected. In one study in coke-oven workers in which an increase in chromatid aberrations and sister-chromatid exchanges was observed, no difference was found between smokers and non-smokers. Elevated DNA adduct levels have been reported in studies on, among others, workers in coke-oven plants, aluminium manufacturing, and foundries (WHO, 1998).

In a review on the validity of the biomarkers mentioned above for estimating individual exposure to PAH, Dor *et al* only discussed DNA-adducts because the other markers were stated to have poor specificity for PAH (Dor *et al*, 1999).

In addition to these studies, several other *in vivo* genotoxicity studies of workers exposed to coal tar, coal tar products, and individual PAHs demonstrated the genotoxicity of these substances (studies not summarised) (WHO, 1998; ATSDR, 2002).

Table 5.7.2 Genotoxicity of CTP or CTPV *in vivo*.

Endpoint	Compound	Species	Result	Reference
<i>Human blood cells</i>				
Chromosomal aberrations	Occupational exposure in (smoking and non-smoking) aluminium reduction plant workers	Human blood	No statistically significant differences between exposed and non-exposed	(Heussner <i>et al</i> , 1985)
Sister Chromatid Exchange (SCE)	Occupational exposure in (smoking) coke oven and (non-smoking) graphite electrode workers	Human peripheral blood lymphocytes	No consistent associations between SCEs and PAH exposure were found	(Buchet <i>et al</i> , 1995)
High frequency Cells (HFCs)	Occupational exposure in (smoking and non-smoking) coke oven workers	Human peripheral blood lymphocytes	HFCs were associated with the intensity of current exposure to PAHs, but not with duration of exposure.	(Buchet <i>et al</i> , 1995)
Micronuclei	Occupational exposure in coke oven and graphite electrode workers	Human peripheral blood lymphocytes	No consistent associations between micronuclei and PAH exposure were found	(Buchet <i>et al</i> , 1995)
PAH-DNA adducts (by ^{32}P -postlabelling)	Occupational exposure in carbon-electrode manufacturing workers	Human peripheral blood lymphocytes	No statistically significant differences	(Van Delft <i>et al</i> , 1998)

Endpoint	Compound	Species	Result	Reference
Benzo[a]pyrene-DNA adducts (by ³² P-postlabelling and immunoassay)	Occupational exposure in carbon-electrode manufacturing workers	Human leucocytes	Increase in exposed compared to non-exposed (and smokers compared to non-smokers)	(Arnould <i>et al</i> , 1999)
Aromatic DNA adducts (by ³² P-postlabelling)	Occupational exposure in potroom workers of an aluminium reduction plant	Human peripheral blood lymphocytes	No statistically significant differences between exposed and non-exposed	(Carstensen <i>et al</i> , 1999b)
Micronuclei	Occupational exposure in potroom workers of an aluminium reduction plant	Human peripheral CD4 ⁺ and CD8 ⁺ lymphocytes	No statistically significant differences between exposed and non-exposed	(Carstensen <i>et al</i> , 1999a)
DNA single-strand breaks	Occupational exposure in potroom workers of an aluminium reduction plant	Human peripheral CD4 ⁺ and CD8 ⁺ lymphocytes	No statistically significant differences between exposed and non-exposed	(Carstensen <i>et al</i> , 1999a)
HPRT mutation frequency	Occupational exposure in potroom workers of an aluminium reduction plant	Human peripheral CD4 ⁺ and CD8 ⁺ lymphocytes	No statistically significant differences between exposed and non-exposed	(Carstensen <i>et al</i> , 1999a)
Micronuclei	Occupational exposure in coke-oven, carbon-plant, aluminium-plant, or graphite-electrode plant workers, or in chimney sweeps	Human lymphocytes	No increase between exposed and non-exposed (in most studies differences between smokers and non-smokers were observed)	(WHO, 1998)
Chromosomal aberrations	Occupational exposure in coke-oven, carbon-plant, aluminium-plant, or graphite-electrode plant workers, or in chimney sweeps	Human lymphocytes	No increase between exposed and non-exposed (in most studies differences between smokers and non-smokers were observed)	(WHO, 1998)
Sister Chromatid Exchange	Occupational exposure in coke-oven, carbon-plant, aluminium-plant, or graphite-electrode plant workers, or in chimney sweeps	Human lymphocytes	No increase between exposed and non-exposed (in most studies differences between smokers and non-smokers were observed)	(WHO, 1998)
Chromosomal aberrations	Occupational exposure in coke-oven workers	Human blood cells	Increased in exposed compared to non-exposed (however, no difference between smokers and non-smokers was observed)	Bender (1988 cited in WHO, 1998)
DNA-adducts	Occupational exposure in coke-oven, aluminium-plant, and foundry workers	Human lymphocytes	Increased in exposed compared to non-exposed	(WHO, 1998)

5.7.4 Summary and discussion of mutagenicity

From mutagenicity testing in *S. typhimurium* conducted according to EC guidelines, it is concluded that CTP is a bacterial mutagen. Results from *in vitro* genotoxicity testing in mammalian cells are somewhat inconsistent, but mostly positive. Human body fluids are generally not mutagenic in bacterial gene mutation tests, except for urine samples of heavily exposed psoriasis patients (to coal-tar applications), and coke oven, and carbon plant workers.

There were no data on *in vivo* genotoxicity testing of CTPHT in experimental animals. Results on genotoxic endpoints in human blood cells after occupational exposure to CTP(V) are inconsistent, but in heavily PAH-exposed people increased DNA-adduct levels have been reported.

The data set available on the mutagenicity/genotoxicity of CTPHT does not meet the basic requirements as specified in Annex VIIA of Directive 67/548/EEC (completed by Directive 2001/59/EC; EU, 2001). However, numerous genotoxicity studies with coal tar, coal tar waste, coal tar products, and individual PAHs demonstrated the genotoxicity of these substances (WHO, 1998; ATSDR, 2002).

In its criteria document, WHO (1998) has discussed the mutagenicity/genotoxicity testing of 33 individual PAHs. Based on the evaluations of IARC (1983) and the results of genotoxicity studies reported after 1983, it was concluded that the only compounds with negative results in all tests were anthracene, fluorene, and naphthalene. Of the other compounds, 16 (including benzo[a]pyrene) were considered to be genotoxic, 8 probably genotoxic, while data concerning the remaining compounds were inadequate or inconsistent (WHO, 1998).

According to EU Regulation (EC) 1272/2008 (EU, 2008b), substances containing more than 0.1% of a mutagen category 1A or 1B (in Table 3.1 of Annex VI) or a mutagen category 1 or 2 (in Table 3.2 of Annex VI) need to be classified as a category 1A or 1B (or 1 or 2) mutagen. CTPHT contains a variable amount of mutagenic PAHs, whose individual mutagenic effects are considered to be at least additive in nature. This includes benzo[a]pyrene (CAS 50-32-8) which is included in Annex VI of EU Regulation (EC) 1272/2008 (entry 601-032-00-3) with amongst others the classification Muta 1B; H340.

Therefore, based on the available genotoxicity data on CTPHT, CTPVHT, coal tar, coal tar waste, coal tar products, and individual PAHs, and the fact that the amount of category 1B mutagenic PAHs in CTPHT is estimated to be more than 0.1% (on a weight/weight basis) in almost all circumstances, classification of CTPHT as a category 1B mutagen is proposed (H340) according to Regulation (EC) 1272/2008 and a category 2 mutagen is proposed (T; R46) according to 67/548/EEC (EU, 1967).

Note:

The classification of CTPHT for mutagenicity according to 67/548/EEC (Muta. Cat. 2; R46) has been discussed and agreed by the TC-C&L in October 2006 (see Annex II).

5.8 Carcinogenicity

5.8.1 Carcinogenicity: oral

There were no data available on the potential carcinogenicity of CTPHT after oral exposure in experimental animals. In 1989 the RIVM judged the available bioassay data in PAH mixtures all to be of insufficient quality for human cancer risk assessment. However, in 2001, they investigated the implications of two more recently conducted studies on the carcinogenic effects of oral administration of coal tar mixtures and/or benzo[a]pyrene in rats and mice, on human cancer risk assessment after exposure to PAHs (Kroese *et al.*, 2001). These two studies are described underneath.

In a study conducted by Culp *et al.* (1998), the tumorigenicity of two coal tar mixtures was compared to that of benzo[a]pyrene in female B6C3F1 mice (48 mice per group) after 2 years of feeding. Coal tar mixture 1 (CT1), a composite of coal tar from seven manufacture gas plant waste sites, was fed to female B6C3F1 mice at doses of 0, 100, 300, 1000, 3000, 6000, and 10000 ppm (calculated by the rapporteur member state as equivalent to 0, 12, 36, 120, 360, 720, and 1200 mg/kg_{bw}); coal tar

mixture 2 (CT2), which was composed of coal tar from two of the seven waste sites and another site having a high benzo[a]pyrene content, was fed at doses of 0, 300, 1000, and 3000 ppm (calculated by the rapporteur member state as equivalent to 0, 36, 120, and 360 mg/kg_{bw}). benzo[a]pyrene was fed at doses of 0, 5, 25, and 100 ppm (calculated by the rapporteur member state as equivalent to 0, 0.6, 3, and 12 mg/kg_{bw}). Two additional groups of 48 mice served as controls, one group was fed the standard diet, while the other group was fed the standard diet treated with acetone in a manner identical to the benzo[a]pyrene diets.

A significantly lower survival rate was observed in mice exposed to both coal tar mixtures at doses of 360 mg/kg_{bw} and higher and in mice exposed to benzo[a]pyrene doses of 3 mg/kg_{bw} and higher. Food consumption and body weight was significantly decreased in mice fed 720 and 1200 mg/kg_{bw} CT1 and 360 mg/kg_{bw} CT2. Liver weights of mice fed 360 mg/kg_{bw} CT1 or CT2 were significantly increased (approximately 40%; corresponding benzo[a]pyrene doses were 0.8 and 1.1 mg/kg_{bw}, corrected for reduced food consumption) compared to the control group, whereas treatment with 3 mg/kg_{bw} benzo[a]pyrene did not result in increased liver weights (liver weights of higher exposed animals were not determined due to tumour development accompanied by decreases in body weights) (Culp *et al*, 1998).

Table 5.8.1. Incidence of neoplasms in female B6C3F1 mice fed coal tar mixtures 1 and 2.

Site	Mixture	Coal tar concentration (ppm)							p-value for dose related trend
		0	100	300	1000	3000	6000	10000	
Incidence									
Liver (hepatocellular adenomas and/or carcinomas)	1	0/47	4/48	2/46	3/48	14/45 ^{a)}	1/42	5/43	0.007
	2	0/47		7/47	4/47	10/45 ^{a)}			0.0004
Lung (alveolar/bronchiolar adenomas and/or carcinomas)	1	2/47	3/48	4/48	4/48	27/47 ^{a)}	25/47 ^{a)}	21/45 ^{a)}	<0.00001
	2	2/47		4/48	10/48 ^{a)}	23/47 ¹⁾			<0.00001
Forestomach (papillomas and/or carcinomas)	1	0/47	2/47	6/45	3/47	14/46 ^{a)}	15/45 ^{a)}	6/41	<0.00001
	2	0/47		3/47	2/47	13/44 ^{a)}			<0.00001
Small intestine (adenocarcinomas)	1	0/47	0/46	0/45	0/47	0/42	22/36 ^{a1)}	36/41 ^{a)}	<0.00001
	2	0/47		0/47	0/47	1/37			Not significant
Hemangiosarcomas ^{b)}	1	1/48	0/48	1/48	1/48	11/48 ^{a)}	17/48 ^{a)}	1/45	<0.00001
	2	1/48		1/48	4/48	17/48 ^{a)}			<0.00001
Histiocytic sarcomas	1	1/48	0/48	0/48	1/48	7/48	5/48	0/45	<0.00001
	2	1/48		3/48	2/48	11/48 ^{a)}			0.00003
Sarcomas ^{c)}	1	1/48	4/48	3/48	2/48	7/48	1/48	2/45	0.006
	2	1/48		0/48	4/48	5/48			0.003

^{a)} Significantly different ($p < 0.05$) from control group; ^{b)} Organs involved include skin, mesentery, mesenteric lymph nodes, heart, spleen, urinary bladder, liver, uterus, thoracic cavity, ovary and skeletal muscle; ^{c)} Organs involved include mesentery, forestomach, skin and kidney.

The coal tar diets induced a dose related increase in hepatocellular adenomas and/or carcinomas, alveolar/bronchiolar adenomas and/or carcinomas, forestomach squamous epithelial papillomas and/or carcinomas, small intestine adenocarcinomas, histiocytic sarcomas, hemangiosarcomas in multiple organs and sarcomas (see Table 5.8.1). The incidence of the liver, lung, and forestomach neoplasms and hemangiosarcomas was statistically significant greater than the control group at dose levels of 360 mg/kg_{bw} and higher. Benzo[a]pyrene treatment resulted in a dose related increase in papillomas and/or carcinomas of the forestomach, oesophagus, tongue and larynx (see Table 5.8.2). The incidence of the forestomach neoplasms was statistically significant greater than the control group at dose levels of 3 mg/kg_{bw} and higher, while the incidence of oesophagus and tongue neoplasms was statistically significant increased at dose levels of 12 mg/kg_{bw}.

Table 5.8.2. Incidence of neoplasms in female B6C3F1 mice fed benzo[a]pyrene.

Site	Benzo[a]pyrene concentration (ppm)				p-value for dose related trend
	0	5	25	100	
	Incidence				
Liver (hepatocellular adenomas)	2/48	7/48	5/47	0/45	Not significant
Lung (alveolar/bronchiolar adenomas and/or carcinomas)	5/48	0/48	4/45	0/48	Not significant
Forestomach (papillomas and/or carcinomas)	1/48	3/47	36/46 ^{a)}	46/47 ^{a)}	<0.00001
Esophagus (papillomas and/or carcinomas)	0/48	0/48	2/45	24/46 ^{a)}	0.0014
Tongue (papillomas and/or carcinomas)	0/48	0/48	2/46	23/48 ^{a)}	0.0003
Larynx (papillomas and/or carcinomas)	0/35	0/35	3/34	5/38	0.014
Hemangiosarcomas ^{b)}	1/48	2/48	3/47	0/48	Not significant
Histiocytic sarcomas	2/48	2/48	1/47	0/48	Not significant
Sarcomas ^{c)}	1/48	2/47	7/47	0/48	Not significant

^{a)} Significantly different ($p < 0.05$) from control group; ^{b)} Organs involved include liver, mesentery and spleen; ^{c)} Organs involved include forestomach, glandular stomach, skin and skeletal muscle.

A comparison of the results indicated that the benzo[a]pyrene in the coal tar diets could be responsible for the forestomach tumours. In contrast, the lung and liver tumours appeared to be due to other genotoxic components contained within the coal tar mixture, while small intestine tumours appeared to result from chemically-induced cell proliferation (determined by no. of S-phase cells) that occurred at high doses of coal tar in addition to DNA adduct formation (by ³²P-postlabeling) (Culp *et al*, 1998; Goldstein *et al*, 1998).

In a study conducted by the RIVM (Kroese *et al*, 2001), Riv:TOX rats of the Wistar strain (52 per dose, per sex) were administered 0, 3, 10, or 30 mg benzo[a]pyrene/kg_{bw} dissolved in soy-oil by gavage 5 days a week for 104 weeks.

Table 5.8.3. Incidences of some major treatment-related neoplasms in rats treated with benzo[a]pyrene.

Site	Dose (mg/kg _{bw}) females				Dose (mg/kg _{bw}) males			
	0	3	10	30 ^{a)}	0	3	10	30 ^{a)}
	Incidence females				Incidence males			
Forestomach								
Squamous cell papilloma	1/52	3/51	20/51***	25/52***	0/52	7/52*	18/52***	17/52***
Squamous cell carcinoma	0/52	3/51	10/51**	25/52***	0/52	1/52	25/52***	35/52***
Liver								
Hepatocellular adenoma	0/52	2/52	7/52*	1/52	0/52	3/52	15/52***	4/52
Hepatocellular carcinoma	0/52	0/52	32/52***	50/52***	0/52	1/52	23/52***	45/52***
Auditory canal ^{b)}								
Squamous cell papilloma	0/0	0/1	0/0	1/20	0/1	0/0	0/7	4/33
Carcinoma ^{c)}	0/0	0/1	0/0	13/20**	0/1	0/0	2/7	19/33***

The most advanced stage of lesions is scored. ^{a)} Note that this group had a significantly shorter lifetime; ^{b)} These tissues were examined only when abnormalities were observed upon macroscopic examination; ^{c)} Composite tumours of squamous and sebaceous cells apparently arisen from the pilosebaceous units/ "Zymbal glands"; * Significantly different ($p < 0.01$) from control group; ** Significantly different ($p < 0.001$) from control group; *** Significantly different ($p < 0.0001$) from control group.

A dose related decrease in survival was observed in both males and females. In males of the highest dose group (30 mg/kg_{bw}), body weights were decreased from week 10 onwards, food consumption was statistically significantly reduced (with less than 10%) from week 36 onwards. Water consumption was statistically significantly and dose related increased in males from week 13 onwards. Benzo[a]pyrene treatment had no major effect on body weight, food consumption and water consumption in female rats. Dose dependent increases in tumour incidence in a variety of organs and/or tissues were observed in both sexes (see Table 5.8.3). The most prominent

carcinogenic effects were observed in the liver, forestomach, and epidermal structures (amongst others auditory canal, lip, and skin), of which the liver is considered the most relevant for human risk assessment in terms of pathogenesis and sensitivity. A statistically significant increase in incidence of liver neoplasms was observed in males and females exposed to benzo[a]pyrene doses of 10 mg/kg_{bw} and higher (Kroese *et al*, 2001).

5.8.2 Carcinogenicity: inhalation

Female rats (Wistar; n=72 group) were exposed to 0, 1.1, or 2.6 mg/m³ of a CTPHT aerosol, 17 hours/day, 5 days/week, for 43 or 86 weeks followed by an exposure-free period of up to 86 or 43 weeks, respectively. The aerosol was generated by heating CTP to 750°C under nitrogen atmosphere and diluting the high temperature tar/pitch vapour with 12°C clean air, resulting in a PAH-rich condensation aerosol with a mass median aerodynamic diameter (MMAD) of 0.5 µm. The 1.1- and 2.6-mg/m³ aerosols contained among others 20 and 46 µg/m³ benzo[a]pyrene, respectively, resulting in cumulative doses of inhaled benzo[a]pyrene of 71 (43-wk exposure), 142 (86-wk exposure), 158 (43-wk exposure), and 321 (86-wk exposure) mg benzo[a]pyrene/m³/h, respectively. Exposure to 2.6 mg/m³ for 43 or 86 weeks caused an increased mortality rate when compared to those of controls. Especially the animals exposed for 86 weeks had to be sacrificed because of the development of large, multiple lung tumours. No exposure-related tumours were observed in organs other than the lung. Most of the lung tumours were benign and malignant keratinizing squamous-cell tumours while some broncho-alveolar adenomas and adenocarcinomas were found. Tumour rates were 4.2 and 33.3% for the animals exposed to 1.1 mg/m³ for 43 and 86 weeks, respectively, and 38.9 and 97.2% for the animals exposed to 2.6 mg/m³ for 43 and 86 weeks, respectively (Heinrich *et al*, 1986; Heinrich *et al*, 1994a; Heinrich *et al*, 1994b).

Intratracheal instillation was used to study the carcinogenic effects of CTPHT in male and female Wistar rats. A total of 190 animals were divided into four groups receiving 10 weekly instillations of charcoal powder suspension and of about 0.65, 13.7, and 20.0 mg CTPHT (particle size distribution: 90% <10 µm; 75% <5 µm) suspended in physiological saline per animal per treatment (group sizes not given). Thirty-six of these animals received one treatment only (see also Section 5.2); the remaining animals were killed one, three, 12, and 18 months after the last instillation, respectively. Treatment did not affect survival rates or average body weights when compared to controls. Histological changes found were mainly located in the bronchiolo-alveolar areas, and dose-dependent as to severity. They ranged from hyperplastic, metaplastic, and dysplastic changes to extensive cancers. No tumours were found in the rats treated with a total dose of about 6.5 mg while incidences were 4/32 and 10/40 in animals given total amounts of about 137 and 200 mg, respectively. Most of these tumours were squamous-cell carcinomas (10/14) (Chang *et al*, 1992).

Eight-week old female mice (Iva:NMRI; n=28-31) were exposed to combustion product from a coal stove, containing 0.3 µg/m³ benzo[a]pyrene, 16 hours/day, 5 days/week, for 8 months, and subsequently to a PAH-rich effluent gas generated by heating CTP to 750°C under nitrogen atmosphere, containing about 60 µg/m³ benzo[a]pyrene, 16 hours/day, 5 days/week, for 15 months. The particle mass concentrations and the MMAD were 1.1 mg/m³ and 0.1 µm, respectively, for the coal stove combustion product and 4.6 (±5.1) mg/m³ and 0.8 µm, respectively, for the CTP product. This treatment induced statistically significant increases in lung tumour incidence (79% vs. 32% in control animals) and in multiplicity, *i.e.* the average number of tumours per lung (7.0 ± 7.9 vs. 0.7 ± 1.7 in controls). From previous similar experiments, the authors suggested most of the tumours to be benign adenomas, but results of histological examinations were not available. The total duration of the experiment was 25 months, *i.e.* the lifespan of the mouse (Heinrich *et al*, 1986).

Newborn female mice (NMRI/BR; n=40/group) were exposed to 0, 0.5 (± 0.85), or 2.44 (± 0.40) mg/m³ of an aerosol generated by pyrolyzing preheated CTP in nitrogen atmosphere at 750-800°C

and diluted with fresh air, 16 hours/day, 5 days/week, for 44 weeks from postnatal day 1 onwards. The MMAD of the aerosols was $0.55 \pm 0.03 \mu\text{m}$. They contained 0, 50, and $90 \mu\text{g}/\text{m}^3$ benzo[a]pyrene, respectively. At the end of the exposure period of 44 weeks, survival rates were 38/40 and 35/40 in the low- and high-concentration group, respectively, vs. 39/40 in the control group. Treatment induced multiple foci of bronchiolo-alveolar hyperplasia in almost all mice (low concentration: 38/40, high concentration: 39/40, controls: 0/40) and squamous metaplasia in 6/40 animals of the high-concentration group, and caused statistically significant increases in the incidence of lung adenomas (low: 40/40, high: 40/40, control: 5/40), of lung adenocarcinomas (10/40, 33/40 vs. 6/40), and of lung squamous cell carcinomas (0/40, 6/40 vs. 0/40). In addition, one adenosquamous carcinoma was found in an animal of the high-concentration group (Schulte *et al*, 1994).

5.8.3 Carcinogenicity: dermal

When 40% solutions of CTP (not further specified) in benzene were painted on the hairy skin of white mice (n=49; strain and sex not reported), once weekly, for 19 months, painted skin lost its hair after the first application. The first tumour appeared three months after the first application, and, by the end of month 12, there were skin tumours in 37 of the 43 mice alive at that time, 29 being keratinizing squamous-cell carcinomas. Other tumours observed were pulmonary adenomas in eight animals and a squamous cell carcinoma of the stomach. Animals of the control group were painted with pure benzene. They developed epidermic atrophy, focal hyperplasia, atrophy of the hair follicles and sebaceous glands only (characteristic effects of this substance). There were no skin tumours among the control group, although one mouse developed pulmonary adenomas (Kireeva, 1968). There were no data on control animals (untreated or solvent-treated) (Kireeva, 1968).

Application of about 1.7 mg each of two different samples of CTP (from coke-oven production and of the grade commonly used in roofing, no further information of its origin was given) dissolved in benzene to the shaved back skin of mice (Swiss albino; n=15/sex/group), twice a week, resulted in a decreased mean survival time (31 wk vs. 82 wk for benzene-treated controls). Treatment with CTPHT caused increases in the number of tumour-bearing animals (53/58 vs. 1/26 in benzene-treated controls), in the incidence of skin carcinomas (31/58 vs. 0/26) and papillomas (53/58 vs. 1/26). Some other tumours were found as well, but it was stated that the numbers observed were not significantly greater than would be expected in a control group (Wallcave *et al*, 1971).

When male mice (C3H/HeJ; n=50) were dermally treated with 50 mg of a toluene solution of a “traditional CTP”, twice weekly, 45/49 and 3/49 mice had developed malignant and benign skin tumours, respectively, by the end of the experiment after 32 weeks. The average time of appearance of papillomas was 18.0 weeks. Both incidence and latency period differed statistically significantly from those found in the toluene- and benzo[a]pyrene control groups: no tumours were found in the toluene-exposed group, while the benzo[a]pyrene group showed an average latency period of 31.8 weeks and incidences of 24/39 and 7/39 for malignant and benign tumours, respectively (Emmett *et al*, 1981).

IARC (1985) refers to an experiment from the 1920s in which dermal treatment with a benzene extract of a hard residue from a coke-oven tar induced lung tumours, but no skin tumours in mice (strain and sex not reported), while no lung or skin tumours were found in control animals.

In a Polish study, it was reported that application of several pitches to the skin of mice, twice weekly, for 22 weeks, induced skin tumour incidences of 27-50% (Gorski, 1959).

5.8.4 Carcinogenicity: human data

Already in the 19th century, reports on the induction of cancer in persons occupationally exposed to combustion products containing PAHs have been published. Studies on possible carcinogenic effects due to exposure to CTPV have been reviewed by several working groups of the International Agency for Research on Cancer (IARC, 1984, 1985, 1987) and by the UK Health and Safety Executive (HSE) (HSE, 1993; Armstrong *et al*, 2003). The IARC concluded that there is sufficient evidence that coal-tar pitches are carcinogenic in humans (IARC, 1985, 1987, 2010). Several additional studies have been published (Ronneberg & Langmark, 1992; Armstrong *et al*, 1994; Partanen & Boffetta, 1994; Ronneberg & Andersen, 1995; Tremblay *et al*, 1995; Cullen *et al*, 1996; Stern *et al*, 2000; Armstrong *et al*, 2004). Quantitative cancer risk estimates have been calculated by Armstrong *et al* (Armstrong *et al*, 1986; 1994), and Tremblay *et al* (1995) attempted to quantify the relationship between exposure to CTPV in Söderberg potrooms and the risk of bladder and lung cancer (based on a Canadian cohort of aluminium production workers). More recently, Armstrong *et al* (2003; 2004) performed a meta-analysis on lung and bladder cancer risk after exposure to PAHs.

The epidemiological data relevant for each exposure scenario are summarised in the next section. In addition a summarizing table is included in Annex III. The summary is based on the publications by IARC and HSE. These publications were not consulted individually. Below the next section, the meta-analysis of Armstrong *et al* (2003; 2004), which combined studies conducted in industries that share (almost exclusive) exposure to PAHs, is described.

Meta-analysis on lung and bladder cancer risk after exposure to PAHs

The meta-analyses by Armstrong *et al* (2003; 2004) combined studies conducted in the industries that share (almost exclusive) exposure to PAHs. By combining a much larger body of data, the risk estimates become statistically much more stable.

The meta-analyses included 39 occupational cohorts exposed to PAHs for which risk estimates for lung cancer could be estimated and 27 cohorts for which risk estimates were published for bladder cancer. Only epidemiological studies on occupational exposure by inhalation were included. Biomarker studies, studies only reporting proportional cancer analyses, non-English publications and non-primary research papers (*e.g.* reviews) were excluded. Studies in which PAH was considered unlikely to be the predominant lung or bladder carcinogen (because of the presence of other known, possibly confounding tissue specific carcinogenic substances, *e.g.* in workplaces including those in the rubber industry and foundries and those involving exposure to diesel exhaust) were excluded as well. Also studies for which assessment of exposure was not possible (*e.g.* case-control and registry studies) were excluded. To avoid double counting of information from the same workforce reported in several papers, only the last reported results were included.

The cohorts included in the meta-analyses were occupationally exposed to CTPVHT in several industries (aluminium smelting, carbon anode plants, asphalt, and tar distillation), but also cohorts from other industries exposed to PAHs were included, such as coke ovens, coal gas production and carbon black production, where the main cause of cancer induction is their exposure to PAHs (*i.e.* irrespective of whether they are scenarios in this Annex VI report on CTPHT). Although it is likely that the composition (PAH profile) and therefore the carcinogenic potential of the exposures is not exactly similar across industries, deriving a statistically stable risk estimate based on all PAH-exposed cohorts is still considered superior to deriving industry-specific but very uncertain estimates. In a meta-analysis, exposures have to be defined as the same metric on the same scale. The underlying studies, however, showed a substantial variation in exposure definition, ranging from no explicit definition to quantitative assessment of exposure to benzo[a]pyrene. Exposures were measured as benzo[a]pyrene, as a proxy (benzene-soluble matter, total PAHs, carbon black) that could be converted to benzo[a]pyrene, or no measure of exposure. For the studies lacking

information on exposure, the authors defined supplementary estimates for exposure to benzo[a]pyrene for each industry/workgroup combination, based on available published exposure estimates in the same industries. Furthermore, the exposure variables were converted to cumulative exposure (duration \times time-weighted mean concentration), if necessary. Where risk by cumulative exposure was not published, it was derived as the product of mean estimated concentration of exposure in each group for which risk was reported and the mean duration of exposure in that group. In absence of information on duration of exposure, 20 years was assumed, representing the average found in studies for which the duration was reported.

In the meta-analyses, relative risks (RRs) were estimated for each study for a benchmark exposure level of 100 $\mu\text{g}/\text{m}^3\cdot\text{year}$ cumulative benzo[a]pyrene. The authors had chosen this benchmark level such that it was comprised within the exposure ranges of the studies included in the meta-analyses. These Unit Relative Risks (URRs) were estimated by fitting an exposure-risk model to the data with Poisson regression. The fitted model was a log-linear (exponential) model as normally used in epidemiological studies and meta-analyses thereof:

$$\ln(\text{RR}) = bx \text{ (equivalent to } \text{RR} = e^{bx} \text{)}$$

where x is the cumulative exposure and b is the slope of the exposure-risk relationship. Meta-regression was applied to assess the impact of study characteristics on the final risk estimate.

Lung cancer

An overall relative risk estimate (URR) of 1.20 (95% confidence interval: 1.11-1.29) per *unit* of 100 $\mu\text{g}/\text{m}^3\cdot\text{year}$ cumulative benzo[a]pyrene exposure was calculated for lung cancer. This implies that the risk for lung cancer was 20% higher in workers exposed to 100 $\mu\text{g}/\text{m}^3\cdot\text{year}$ cumulative benzo[a]pyrene (\sim 40 years exposure to an average concentration of 2.5 $\mu\text{g}/\text{m}^3$ benzo[a]pyrene). In a meta-analysis, it is common practice to investigate whether the data from the studies included are sufficiently in agreement with each other by testing for heterogeneity. In the current meta-analysis, a statistically significant heterogeneity in URRs between the individual studies was observed, indicating that some studies (mainly the smallest, *i.e.* least precise studies) had deviating estimates. Nevertheless, statistical significant heterogeneity was observed between industry groups, but not between and within the major contributing groups, *i.e.* coke ovens, gas works and aluminium smelters. The combined URR estimate in aluminium smelters, the only industry exposed to CTPVHT for which a statistically stable industry-specific estimate could be established, was 1.16 (95% confidence interval: 1.05-1.28) per unit of 100 $\mu\text{g}/\text{m}^3\cdot\text{year}$ cumulative benzo[a]pyrene exposure.

For other characteristics (such as study design, region or type of exposure measurement) no statistically significant heterogeneity was detected.

Although limited, information on total dust exposure did not suggest that dust exposure was an important confounder or effect modifier.

A requirement for establishing and quantifying an association between PAH exposure and lung cancer is that confounding due to other risk factors of lung cancer, such as smoking, are unlikely to explain the results. Confounding can arise from smoking habits that differ between the exposed and unexposed groups. In general, in occupational epidemiological studies the effect is limited, but unpredictable, as there is no systematic and consistent association between exposure and smoking (unlike studies on *e.g.* lifestyle and cancer, where smoking is always prevalent in persons with the least healthy lifestyle habits). Regarding the meta-analysis of Armstrong *et al* (2003; 2004), only in four out of 39 studies (mainly nested case-control studies from cokes ovens and aluminium smelters) in the meta-analysis for lung cancer, adjustment of risk estimates for confounding due to smoking was performed; the meta-analysis observed borderline statistically significant higher estimates for the studies adjusted for smoking than for those that had not (URR = 1.31, 95%

confidence interval: 1.16-1.48 versus 1.16, 95% confidence interval: 1.11-1.21, respectively). Failure to adjust for smoking in the majority of the studies is, if anything, therefore more likely to underestimate than to overestimate the true risk estimate. This higher risk estimated from studies that did control for smoking prove at least that the risk of cancer is not always overestimated when no adjustment is made.

Bladder cancer

An overall relative risk estimate (URR) of 1.33 (95% confidence interval: 1.17-1.51) per unit of 100 $\mu\text{g}/\text{m}^3$ ·year cumulative benzo[a]pyrene exposure was calculated for bladder cancer (Armstrong *et al*, 2003). Although the results support a PAH-bladder cancer association, this finding was less robust than that for lung cancer, as it appeared to be largely dependent on two studies of aluminium production workers (Tremblay *et al*, 1995; Romundstad *et al*, 2000a; Romundstad *et al*, 2000b). For the aluminium production industry the evidence for an association was strong. Although the URRs from other industries were statistically compatible with those for aluminium, there was little independent evidence for an association of bladder cancer with PAH in coke ovens or in other industries.

Armstrong *et al* (2003) concluded:

“Previous reviews have similarly concluded that there is a much stronger weight of evidence that PAH causes lung than that it causes bladder cancer. One recent review (Negri & La Vecchia, 2001) noted specifically that the evidence for bladder was confined to the aluminium production industry. Other co-exposures, in particular aromatic amines and nitro-PAH (Tremblay *et al*, 1995) known to be present in small concentrations in aluminium potrooms, have been suggested as alternative causal agents. However, it is unclear why these would not also be present in other PAH-exposed workplaces.”

The combined URR estimate in aluminium smelters, the only industry exposed to CTPVHT for which a rather precise industry-specific estimate could be established, was 1.42 (95% confidence interval: 1.23-1.65) per unit of 100 $\mu\text{g}/\text{m}^3$ ·year cumulative benzo[a]pyrene exposure.

5.8.5 Other relevant information

Epidemiological data relevant for the different exposure scenarios

Scenario 1: Production of CTPHT in coal tar distillation plants

In a review by the HSE (Armstrong *et al*, 2003) three cohort studies were identified, none of which contained data on exposure. The study by Hansen (1989) was not solely related to tar distillation but also to asphalt and roof felt processing. Statistically non-significant increased lung cancer risks were observed in all three studies and non-significant increased bladder cancer in two of the three studies.

Scenario 2: Use as a binder for electrodes

Sub-scenario 2i: in the aluminium industry (studies on aluminium production workers)

Several studies among aluminium production workers in Canadian, French, Italian, Norwegian, US and Russian industries have been published. Most are taken from a review of Ronneberg & Langmark (1992), complemented with information from the IARC (1984, 1987, 2010) and HSE (HSE, 1993; Armstrong *et al*, 2003). Five more recent publications not included in the reviews (Armstrong *et al*, 1994; Ronneberg & Andersen, 1995; Tremblay *et al*, 1995; Cullen *et al*, 1996; Ronneberg *et al*, 1999; Romundstad *et al*, 2000a; Romundstad *et al*, 2000b; Armstrong *et al*, 2003; Armstrong *et al*, 2004) were also consulted.

The lung and bladder have been the most commonly identified sites for excess cancer in populations of aluminium production workers. In Canadian studies dose-response relations were

found for bladder and lung cancer. The Norwegian studies have shown inconsistent results. Excess risk of stomach, kidney, prostate, pancreas, lymphatic and haemopoietic cancer and leukaemia were noted in several studies among aluminium production workers.

The IARC concluded that there is sufficient evidence that certain exposures occurring during aluminium production cause cancer and that pitch volatiles have fairly consistently been suggested in epidemiological studies as being possible causative agents (IARC, 1987).

Sub-scenario 2ii: Use as a binder and impregnation of electrodes

In a review by Armstrong *et al* (2003) three papers (reporting 3 cohort studies and one case control study) were identified on carbon workers. One paper from China includes workers of six carbon plants (not further specified) and one aluminium reduction plant (working potroom and carbon department). Although part of this cohort falls under sub-scenario 2i, the study is described under this scenario, assuming most workers were involved in the use of CTPHT as a binder and impregnation of electrodes. The other two papers describe workers in carbon (graphite) electrode plants in Italy and France.

In one of the available studies on the use of CTPHT as a binder and impregnation of electrodes, a statistically significant increased lung cancer risk was observed (Liu *et al*, 1997). In the other studies non-significant increases in lung and bladder cancer risks were observed (Moulin *et al*, 1989; Donato *et al*, 2000).

Scenario 3: Use as a binder in Asphalt industry and in Roofing

Several studies among asphalt workers have been published. This review of Partanen & Boffetta (1994), who examined and combined the results of 20 epidemiologic studies conducted on asphalt workers and roofers, complemented with information from the IARC (1985) and more recent publications by Stern (2000), Boffetta *et al* (2003), Randem *et al* (2004) and Armstrong *et al* (2003) were consulted. Assuming that the review of Partanen & Boffetta, the IARC document and the meta-analysis of Armstrong *et al* (2003; 2004) contain the most important issues with respect to the evaluated epidemiological studies, original data of these studies were not consulted.

Most of the studies evaluated by Partanen & Boffetta (1994), the IARC (1985) and Armstrong *et al* (2003) have limitations, with respect to power, lack of exposure data, or failure to control for confounding. In roofers, some studies with smoking-adjusted results suggest an excess lung cancer risk unexplained by tobacco smoking. Since roofers work with hot pitch they have probably been exposed to great amounts of carcinogenic PAHs. However, the data were insufficient to specifically address the carcinogenicity of the different exposures encountered in roofing (and other asphalt workers), including coal tar derived exposures.

Scenarios 4 through 8: Use in heavy-duty corrosion protection or as a binder for refractories, active carbon, coal briquetting, and clay pigeons

There are very few epidemiological studies available on these occupational scenarios. In the IARC evaluation (1985) only one study was described on coal briquetting in which an increased mortality due to bladder and prostatic cancer was observed.

5.8.6 Summary of carcinogenicity

The IARC concluded that coal-tar pitches are carcinogenic to humans (Group 1), based on sufficient evidence for carcinogenicity in experimental animals and humans (IARC, 1985, 1987, 2010).

There were no data available on the potential carcinogenicity of CTPHT after oral exposure in experimental animals. However, oral studies with coal tar in mice resulted in increased tumour incidences in various organs, including the liver, lung, and forestomach. Oral studies with

benzo[a]pyrene resulted in increased tumour incidences in a.o. the liver, forestomach, and epidermal structures in rats and the forestomach and the upper digestive tract in mice.

Inhalation of CTPHT caused broncho-alveolar lesions and lung tumours in rats and mice, while dermal exposure to CTP (not further specified) and CTPHT caused skin tumours in mice. Although the available experimental animal studies were not conducted according to EU or OECD guidelines, they clearly indicate that CTPHT is carcinogenic following inhalation and dermal exposure.

With respect to human data, statistically non-significant increased lung cancer risks were observed in all three available cohort studies on coal tar distillation. In two of these three studies non-significant increased bladder cancer risks were observed. In the other study a non-significant reduced SMR for bladder cancer was observed. None of the studies contained data on exposure and one of the studies was not solely related to tar distillation but also to asphalt and roof felt processing.

Among populations of aluminium production workers (scenario 2i), the lung and bladder have also been the most common sites identified for excess cancer. In several studies among aluminium production workers excess risks of stomach, kidney, prostate, pancreas, lymphatic and haemopoietic cancer and leukaemia were also noted.

In one of the available studies on the use of CTPHT as a binder and impregnation of electrodes (scenario 2ii), a statistically significant increased lung cancer risk was observed. In the other studies non-significant increases in lung and bladder cancer risks were observed.

Among roofers and asphalt workers (scenario 3) excess lung and skin cancer risks were observed, however, the data were insufficient to specifically address the carcinogenicity of the different exposures, including coal tar derived exposures.

On the other exposure scenarios (scenarios 4 through 8), no or very few epidemiological studies were available.

Based on experimental and epidemiological data on the carcinogenicity of CTPHT and CTPVsHT and the evaluation of these data by the IARC, classification of CTPHT and CTPVsHT as a category 1A carcinogen (H350) is proposed according to 67/548/EEC and a category 1 carcinogen (T; R45) is proposed according to 67/548/EEC (EU, 1967).

Note:

The classification of CTPHT for carcinogenicity according to 67/548/EEC with Carc. Cat. 1; R45 has been discussed and agreed by the TC-C&L in October 2006 (see Annex II).

5.9 Toxicity for reproduction

No experimental data on the potential reproduction toxicity of CTPHT were available. High-boiling coal liquid, coal tar derived products, and creosote have been shown to produce reproductive toxicity in animals by the inhalation, oral, and dermal routes.

5.9.1 Effects on fertility

In its criteria document, the WHO discussed the reproductive toxicity of several individual PAHs, among which benzo[a]pyrene. It was concluded that this PAH had adverse effects on female fertility and reproduction (WHO, 1998).

Inhalation

In a repeated dose inhalation toxicity study by Springer *et al* (1986a; 1987) fertility toxicity was also evaluated by examination of the reproductive organs. No change in relative weights of ovary or

testis were recorded for Fischer rats exposed to 30, 140, and 690 mg/m³ of a high-boiling coal liquid aerosol (heavy distillate, the highest-boiling material derived from the solvent refined coal-II process) for 6 hours/day, 5 days/week for 5 weeks. However, after exposure of Fischer rats and CD-1 mice to the same concentrations of high-boiling coal liquid aerosol for 13 weeks, statistically significantly reduced relative ovary weights were observed at the highest dose level. Testis weights were statistically significantly increased in rats exposed to 140 mg/m³ and higher. Examination of ovarian section showed a significant decrease in the amount of luteal tissue in rats exposed to 690 mg/m³ high-boiling coal liquid aerosol for 5 or 13 weeks.

Oral

In a repeated dose oral toxicity study (Weyand et al., 1994, cited in ATSDR, 2002) fertility toxicity was also evaluated by examination of the reproductive organs. B6C3F1 mice fed a control gel diet or adulterated diets containing 0, 51, 251, or 462 mg/kg/day (males) and 0, 42, 196, or 344 mg/kg/day (females) Manufactured Gas Plant residue (a by-product of coal gasification, coal-tar like material), exhibited no adverse effects on the epididymides, preputial gland, ovaries, uterus, or clitoral gland after treatment for 94 or 185 days.

The summary of a multi-generation reproduction toxicity study with creosotes indicated that oral exposure to creosote produced reproductive toxicity (male and female fertility and pregnancy indices) at a dose level (25 mg/kg_{bw}/day) below maternal toxic dose levels (75 mg/kg_{bw}/day) in rats (CCE, 2004).

Coal tar creosote (fractions or blends of coal tar oils, sometimes including coal tar pitch, which are used for timber preservation) was tested for estrogenic activity using an assay in ovariectomised (OVX) ICR and DBA/2 mice. OVX mice were gavaged with 0, 10, 50, or 100 mg/kg creosote in sesame oil or 0.1 mg/kg 17 α -ethynylestradiol (positive control) once a day for 4 days. Treatment with 17 α -ethynylestradiol produced a significant increase in uterine weight and vaginal cell cornification compared with animals receiving only sesame oil, but no fertility effects (significant increase in uterine weight or vaginal cell cornification) were observed in animals treated with this creosote (Fielden et al., 2000 in ATSDR, 2002).

Dermal

No studies with regard to effects on fertility after dermal exposure are available.

5.9.2 Developmental toxicity

In its criteria document, the WHO discussed the reproductive toxicity of several individual PAHs. According to the WHO, reproductive toxicity studies have been reported on anthracene, benz[a]anthracene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and naphthalene. Embryo-toxicity was reported in response to benz[a]anthracene, benzo[a]pyrene, dibenz[a,h]anthracene, and naphthalene. Benzo[a]pyrene also had adverse effects on postnatal development (WHO, 1998).

Inhalation

In a study by Springer *et al* (1982) mated female rats were exposed to 0, 17, 84, or 660 mg/m³ of a high-boiling coal liquid aerosol (heavy distillate, the highest-boiling material derived from the solvent refined coal-II process) for 6 hours/day on gestational days 12-16. Developmental effects, including a significant increase in the incidence of mid- and late-gestational resorptions, significantly reduced crown-rump length, foetal weight, foetal lung weight, and placental weights and significantly increased incidence of reduced ossification, were observed in the highest dose group, and a significant trend for reduced ossification with increasing coal tar concentrations. Cleft palates were also observed in this group, but the increased incidence was not significant. Animals exposed to the highest dose groups showed some signs of maternal toxicity (statistically significantly reduced thymus and increased lung and spleen weights) (Springer *et al*, 1982).

Oral

Heavy distillate, the highest-boiling coal liquid from the solvent-refined coal-II process, was administered by intragastric intubation to pregnant rats. Five dose levels of heavy distillate (90, 140, 180, 370 and 740 mg/kg/day), were given daily from 12 to 16 days of gestation and the rats were killed at 20 days of gestation (Hackett *et al*, 1984). Maternal body weights and weights of the liver, kidneys, spleen, adrenals, thymus, ovaries and the gravid uterus were obtained. Gravid uteri were evaluated for prenatal mortality. Live foetuses were examined for malformations and weighed; foetal lungs were excised and weighed. Maternal body weight gain (excluding extragestational body weight) was significantly reduced in all dose groups. Placental weight was depressed from the dose level of 140 mg/kg/day. Adrenal weights were increased in all treated animals, except for those in the lowest-dose group. However, the weights of the spleen, liver, kidneys, and ovaries of all dosed groups were similar to controls. There was significant maternal mortality at 740 mg/kg/day. These findings suggest that maternal toxicity may have played a role in the elicitation of the developmental toxicity observed in this study.

Regarding developmental toxicity, a significant decrease in the number of live foetuses/litter and a significant increase in the number of resorptions, were observed at 370 mg/kg/day. A statistically significant increase in resorptions was also observed in rats treated with 180 mg/kg/day. A significant decrease in relative foetal lung weight and a significant increase in anomalous foetuses were observed in offspring of females treated with 140 mg/kg/day. In the offspring of females treated with 370 mg/kg/day, a significant increase in the incidence of cleft palate, syndactyly, ectrodactyly and missing toenails on hind feet, were observed. In addition, increased intrauterine mortality at doses of 370 and 740 g/kg was reported.

Several developmental effects were observed in female Sprague-Dawley rats gavaged with 740 mg/kg/day high-boiling coal liquid (heavy distillate, the highest-boiling material derived from the solvent refined coal-II process) on gestational days 12-14. Early mortality was significantly increased in treated pups, within the first 3 days after birth, 54% of the treated pups died compared with 9% of the untreated pups. Body and lung weights of treated pups that died or were sacrificed at 1 or 3 days postdelivery were significantly reduced compared with controls. Body weight gain was significantly reduced (15%) for treated pups compared with the controls at all time points. Treated pups that died showed signs of severe dehydration. Thymus and lung weights in treated animals were significantly lower than in the corresponding control animals. In treated pups that died, the incidence of small lungs (size more than two standard deviations below the mean of the control group) was 27% (in 90% of litters). 10% (in 80% of litters) had cleft palates, and 33% of the pups (in 80% litters) had both small lungs and cleft plates. No controls had small lungs or cleft palates. No malformations were detected in 30% of the treated pups that died and microscopic examination of foetal lung tissue revealed no overt histological differences between treated and control animals. The data from this study suggest that high-boiling coal liquid is a teratogen in Sprague-Dawley rats, however, given the moderate, yet statistically significant maternal toxicity, the possibility of foetal effects secondary to maternal toxicity cannot be excluded (Springer *et al.*, 1986a in ATSDR, 2002).

Summaries of two teratogenicity studies with creosotes indicate that oral exposure to creosote produced reproductive toxicity (increased post-implantation loss, foetus length and weight, impairment of viability, and morphological malformations) at or above maternal toxic dose levels (NOAEL of 75 mg/kg_{bw}/day and LOAEL of 175 mg/kg_{bw}/day) in rats (CCE, 2004).

In a summary of a multi-generation reproduction toxicity study, however, it was reported that oral exposure to creosote produced reproductive toxicity (male and female fertility and pregnancy indices) at a dose level (25 mg/kg_{bw}/day) below maternal toxic dose levels (75 mg/kg_{bw}/day) in rats (CCE, 2004).

Dermal

In a developmental study Sprague-Dawley rats and CD-1 mice, were dermally exposed to 500 or 1500 mg/kg high-boiling coal liquid on gestational days 11-15. A significant decrease in gravid uterine weight compared with controls in both rats and mice at both dose levels exposed to coal tar was reported. In mice, no difference in extragestational body weight gain was observed, next to significantly increased weights of the liver, kidney, and spleen at both dose levels. In rats, extragestational body weights were decreased compared to the controls and the relative weights of maternal liver and kidney were significantly increased while those of the thymus were significantly decreased at both dose levels.

Developmental effects, including a dose dependent decrease in foetal and placental weights and crown-rump lengths, and decreased foetal lung absolute and relative weights were observed in both low and high dose rat groups. Resorptions were significantly increased for all exposed rats and high dose mice compared to controls. There was no significant difference between foetal weight, placental weight and foetal lung weights and crown-rump lengths in control and exposed mouse foetuses. A significantly increased incidence of small lungs, cleft palate, oedema, midcranial lesion, and reduced cranial ossification was observed in exposed rat foetuses and a significantly increased incidence of cleft palate, dilated ureter, and retal pelvic cavitation was observed in exposed mouse foetuses (Zangar et al., 1989 cited in ATSDR, 2002).

Another animal study reported that dermal contact with coal tar creosote-treated wood produced fetotoxic effect in pregnant sows. Four sows were confined to wooden farrowing crates for 2-10 days before delivery. The platforms of the crates were coated with three brush applications of a commercial wood preservative containing 98.5% coal tar creosote. Following contact with creosote, 24 of the 41 pigs delivered were dead at birth, and 11 pigs died by day 3 postfarrowing. The surviving pigs had rough skin and suffered from dehydration and severe diarrhoea. The pigs failed to gain weight until they were 5-6 weeks old. No toxic effects on the sows were reported. Four sows confined to untreated lumber crates at least 24 hours before farrowing delivered 36 pigs, 1 died within 24 hours and 3 died postfarrowing. No toxic effects were noted in mother or baby pigs (Schipper 1961 cited in ATSDR, 2002).

5.9.3 Human data

No differences in sperm count, sperm morphology, and the frequency of sperm-carrying fluorescent bodies-“1-F” and “2-F”, the latter thought to represent nondisjunction of the Y chromosome in meiosis-were found between 20 workers exposed to CTPV in an aluminium reduction plant and 20 unexposed controls matched as to age, smoking and alcohol-drinking habits from the same facility. Exposure data were not given (Heussner *et al*, 1985). These findings were confirmed by Ward (1988 cited in ATSDR, 2002), who also found no adverse effects on sperm characteristics, including sperm count and morphology, in workers exposed to CTPV in an aluminium reduction plant (Ward 1988 cited in ATSDR, 2002).

No other human data on the reproductive toxicity of CTP(V) were available. Dermal exposure to coal tar was studied in a retrospective human study including 64 women who had been treated with coal tar for psoriasis or dermatitis. Fifty-six of the women returned the questionnaire. In total the women had been pregnant 103 times. In 59 of these pregnancies, no coal tar had been used, in 21 pregnancies it was unclear whether coal tar had been used or not, and in the remainder, coal tar had been used at some point during pregnancy. Untreated pregnancies resulted in 19% spontaneous abortion while treated pregnancies resulted in 26% spontaneous abortion. The authors did not consider this to be a significant increase in spontaneous abortion compared with the general population, but pointed out that their sample size was small and this study probably did not have sufficient resolution to detect a modest increase in risk ((Franssen et al., 1999 cited in IARC, 2002).

Studies on developmental effects in humans are not available.

5.9.4 Summary and discussion of reproductive toxicity

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTPHT. Animal data was available on high-boiling coal liquid, coal tar derived products and creosote (inhalation, oral and dermal route).

High-boiling coal liquid had *effects on fertility* in a repeated dose inhalation toxicity study (13 weeks): statistically significant increased testis weights were observed in rats from a concentration of 140 mg/m³ (NOAEC 30 mg/m³). At the highest tested concentration (690 mg/m³) also decreased ovary weights and loss of luteal tissue were observed.

Coal tar derived products and coal tar creosote had no effects on fertility in mouse studies (with NOAELs of 344 mg/kg_{bw}/day and 100 mg/kg, respectively). In a summary of a multigeneration study it is reported that creosote had effects on fertility in rats (at a dose level of 25 mg/kg_{bw}/day) below maternal toxic doses (75 mg/kg_{bw}/day) (Springer *et al*, 1982; Hackett *et al*, 1984; Springer *et al*, 1986b; Springer *et al*, 1987; Zangar *et al*, 1989; CCE, 2004).

Table 5.9.1 Summary of reproductive toxicity studies with high-boiling coal liquid, coal tar derived products, and creosote.

Type of study	Species	Exposure	Results	Reference
Repeated dose toxicity study- Inhalation	Mouse	0, 30, 140, or 690 mg/m ³ coal tar aerosol; 6 hours/day; 5 days/week; 13 weeks	Significantly reduced relative ovary weights at 690 mg/ m ³ . Significant decrease in luteal tissue at 690 mg/ m ³	(Springer <i>et al</i> , 1987)
Repeated dose toxicity study- Inhalation	Fischer rat	0, 30, 140, or 690 mg/m ³ coal tar aerosol; 6 hours/day; 5 days/week; 5 weeks	No effects on reproductive organ weights. Significant decrease in luteal tissue at 690 mg/ m ³	(Springer <i>et al</i> , 1986b)
Repeated dose toxicity study- Inhalation	Fischer rat	0, 30, 140, or 690 mg/m ³ coal tar aerosol; 6 hours/day; 5 days/week; 13 weeks	Significantly reduced relative ovary weights at 690 mg/ m ³ . Significant decrease in luteal tissue at 690 mg/ m ³	(Springer <i>et al</i> , 1986b)
Repeated dose toxicity study-Oral	Mouse	0, 51, 251, 462 mg/kg/day (males) 0, 42, 196, 344 mg/kg/day (females) MGP residue ^{a)} for 94 and 185 days	No effects on reproductive organs	(Weyand <i>et al</i> , 1994)

^{a)} Manufactured Gas Plant residue is a coal-tar like material.

Although *developmental effects* were observed in the available studies, it is not clear whether they were directly induced by high-boiling coal liquid, coal tar derived products, and creosote. In most of the studies, the observed foetal deformities appeared to be related to maternal toxicity except for the study by Schipper (1961), which showed an increase in foetal mortality in pigs without apparent maternal toxicity.

In humans no adverse effects on sperm characteristics, including sperm count and morphology, were observed in workers exposed to CTPV in an aluminium reduction plant (Heussner *et al*, 1985; Ward 1988 cited in ATSDR, 2002). In a retrospective study among psoriasis or dermatitis patients, dermal exposure to coal tar did not induce a significant increase in spontaneous abortion compared with the general population (Franssen *et al*, 1999). However, the sample size was probably too small to detect a modest increase in risk.

CTPHT may contain up to 1.5% benzo[a]pyrene, which is classified as toxic for reproduction (Repr.1B, H360-). According to the Regulation (EC) No. 1272/2008 (EU, 2008b), substances containing more than 0.3% of a substance (impurity) classified as toxic for reproduction in category 1B should be classified as a toxic for reproduction. The same applies to 67/548/EEC above 0.5%.

For these reasons it is proposed to classify CTPHT as toxic to reproduction (Repr.1B, H360FD) according to EC 1272/2008 and as category 2 reprotoxic (T, R60/61), according to 67/548/EEC (EU, 1967).

Note:

The classification of CTP for Reproductive toxicity according to 67/548/EEC with Repro Cat 2; R60/61 has been discussed and agreed by the TC-C&L in October 2006 (see Annex II).

5.10 Other effects

Other effects cannot be excluded, but have not been identified.

5.11 Derivation of DNEL(s) or other quantitative or qualitative measure for dose response

Not relevant for this type of report.

6 HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICOCHEMICAL PROPERTIES

6.1 Explosivity

CTPHT is not explosive.

6.2 Flammability

CTPHT is not flammable.

6.3 Oxidising potential

CTPHT is not oxidising.

7 ENVIRONMENTAL HAZARD ASSESSMENT

In the effect assessment below the ecotoxicity data has been evaluated for the 16 EPA-PAHs separately. The data from both literature and other EU RARs are used. In the different sections the most decisive toxicity studies are described. This is based on the data presented in the transition dossier for CTPHT (Netherlands, 2008), in which the studies were summarized in an Annex.

PAHs can be toxic via different mode of actions, such as non-polar narcosis and phototoxicity. The last is caused by the ability of PAHs to absorb ultraviolet A (UVA) radiation (320–400 nm), ultraviolet B (UVB) radiation (290–320 nm), and in some instances, visible light (400–700 nm). This toxicity may occur through two mechanisms: photosensitization, and photomodification. Photosensitization generally leads to the production of singlet oxygen, a reactive oxygen species that is highly damaging to biological material. Photomodification of PAHs, usually via oxidation, results in the formation of new compounds and can occur under environmentally relevant levels of actinic radiation (Lampi *et al*, 2006). The phototoxic effects can be observed after a short period of exposure, which explains why for PAHs like anthracene, fluoranthene and pyrene, where phototoxicity is most evident, the acute toxicity values are even lower than the chronic toxicity values.

According to Weinstein & Oris (1999) there is a growing body of evidence which suggests that phototoxic PAHs may be degrading aquatic habitats, particularly those in highly contaminated areas with shallow or clear water. For example, the photoinduced chronic effects of anthracene have been reported at those UV intensities occurring at depths of 10 to 12 m in Lake Michigan (Holst & Giesy, 1989). In addition to direct uptake of PAHs from the water column, another potential route of exposure for aquatic organisms is their accumulation from sediments (see e.g. Clements *et al*, 1994; Kukkonen & Landrum, 1994), followed by subsequent solar ultraviolet radiation exposures closer to the surface.

Ankley *et al* (2003) also concluded in their peer review that PAHs are present at concentrations in aquatic systems such that animals can achieve tissue concentrations sufficient to cause photoactivated toxicity. Although UV penetration can vary dramatically among PAH-contaminated sites, in their view it is likely that at least some portion of the aquatic community will be exposed to UV radiation at levels sufficient to initiate photoactivated toxicity. They do recognize that at present time, the ability to conduct PAH-photoactivated risk assessment of acceptable uncertainty is limited by comprehensive information on species exposure to PAH and UV radiation during all life stages. PAH exposure and uptake, as well as UV exposure, are likely to vary considerably among species and life stages as they migrate into and out of contaminated locations and areas of high and low UV penetration. For all but sessile species, these patterns of movements are the greatest determinant of the risk for photoactivated toxicity.

Despite these uncertainties, it is thought that the phototoxic effects can not be ignored in the present risk assessment. It should be noted that the UV exposure levels of the selected studies did not exceed the UV levels under natural sun light conditions.

7.1 Aquatic compartment (including sediment)

7.1.1 Toxicity test results

A Water-Accommodated Fraction (WAF) method to determine the toxicity of coal tar pitch was developed by Tadokoro *et al* (1991) by studying different test solution preparation methods in absence of UV irradiation: direct addition to media without filtration, direct addition with supernatant after the solid material was siphoned out of solution and diluting the stock solution of

the saturated concentration. Killifish (*Oryzias latipes*), read sea bream (*Pagrus major*) and daphnia (*Daphnia magna*) were used for testing. In the direct addition method an extraction time of 24 hr was used followed by a settle time of 2 hr. In the dilution method, coal tar pitch was spread over a glass plate at a rate of 50 mg/cm², after which the plate was dipped into the water of an aquarium in order to obtain a wider surface area for the extraction. The possible number of glass plates that could be dipped into 1 litre of water corresponded to 1000 mg/L as an added amount. The total detected amount of major components (not specified) in the prepared test solutions was 0.3 % and 0.13 % (relative to total nominal loading with CTPHT) with the direct addition and dilution method, respectively. Using direct addition (with and without filtration) the LC₅₀ value was between 100 and 1000 mg/L for all species. With the dilution method the LC₅₀ was > 1000 mg/L for *O. latipes* (other species were not tested).

Additional information is available concerning the solubility of pulverized CTPHT (see Section 1.3). At 100 and 10,000 mg/L, stirred (rate unknown) at room temperature for 24 hr and filtered (0.2 µm) afterwards, the concentration in solution, expressed in DOC, is 0.3 mg/L at both loading rates, corresponding to 0.3 and 0.003 % relative to the nominal loading rates. It should however be noted that the authors of this study specified the results as preliminary and not fully reliable. A blank control was not presented for this examination at low loadings. In a second test (pH dependence), the control value was stated to be 0.9 mg/L TOC.

In another experiment a column containing 10 g of finely powered CTPHT (20-200 µm) was force-percolated by 1.1 L of tap water (water recycling for 1 wk). Each experimental period was terminated by withdrawal of 1 L of the extract and subsequent replacement of this volume by 1 L fresh tap water. This procedure was continued for 39 weeks. The total of the EPA-PAHs in the pitch sample applied comprised of 9.9% (after GC) or 9.2% (after HPLC). After the first run, 36.5 µg PAH/L was found, after 15 cycles the total PAH concentration decreased to 11.8 µg/L and after 39 cycles to 0.9 µg/L. The first water-soluble fraction was dominated by the presence of acenaphthene (7.3 µg/L), phenanthrene (8.8 µg/L), fluoranthene (9.3 µg/L) and pyrene (6.7 µg/L), followed by naphthalene (1.5 µg/L), fluorene (1.2 µg/L) and anthracene (0.6 µg/L). The total cumulative amount of water-extractable EPA-PAHs amounted to approximately 370 µg/10 g (= ~0.004%).

Apart from these data, toxicity data for the individual PAHs were analyzed, which are summarized in the next paragraphs.

Fish

Naphthalene

In the EU-RAR for naphthalene (United Kingdom, 2003) a study with fry of the pink salmon (*Oncorhynchus gorbuscha*) tested in a flow-through system for 5 weeks was chosen as the key study, resulting in a NOEC of 120 µg/L (Moles & Rice, 1983). This species was tested in seawater with a salinity of 28‰. The tested life-stage of this species lives in seawater and not in fresh water. The lowest NOEC for naphthalene for fresh water species is 370 µg/L in a similar test with fry of Coho salmon (*Oncorhynchus kisutch*) exposed for 40 days by a continuous flow system (Moles *et al*, 1981). However, Black *et al* (1983) and Milleman *et al* (1984) reported an LC₅₀ of 110 or 120 µg/L for an early life stage study (ELS) with rainbow trout exposed from 20 minutes after fertilization of the eggs until 4 days after hatching of the fry (after 23 d, total exposure 27 d). The presented data by Black *et al* (1983) show a clear dose-response relationship. The LC₅₀ value of 117 µg/L derived from a dose-response relationship with a log-logistic equation ($r^2=0.96$) is similar to the values mentioned above. The EC₁₀ for survival after 4 days post-hatching is 20 µg/L. Clearly, this is the lowest usable effect concentration for naphthalene in fresh water species.

In the RAR of naphthalene the study of Black *et al* (1983), was disregarded because the method could not be repeated with toluene and it generally gives much lower results than standard studies.

After reconsideration, it is thought that there are some differences with naphthalene. For toluene the difference with the other toxicity data is several orders of magnitude. For naphthalene, there are several studies which show the onset of chronic effects or effects on sensitive life stages around the value of 20 µg/L. The EC₁₀ for toluene is also an order of magnitude lower than that for naphthalene, a compound with a log K_{OW} that is 0.6 unit higher. Both EC₁₀s do further not originate from the same publication, or at least toluene has been omitted from the publication. If a read-across is performed with the data for phenanthrene from the same study instead of toluene, the data are very well in line with another study with the same species and with data for other species.

Acenaphthylene

In a 96-h acute toxicity study with the Japanese Medaka (*Oryzias latipes*), the LC₅₀ for acenaphthylene was 6400 µg/L (Yoshioka & Ose, 1993).

Acenaphthene

The lowest EC₅₀s for acenaphthene are for the fish species *Salmo trutta* and *Oncorhynchus mykiss* from studies with continuous flow-system and measured concentrations (Holcombe *et al*, 1983). The 96-h LC₅₀s are 580 and 670 µg/L, respectively.

Two independent ELS tests with *Pimephales promelas* were carried out, one with dimethylformamide as solvent and one without carrier (Cairns & Nebeker, 1982). The fish were exposed by a flow-through system and concentrations were measured. No significant effects on fork length and wet weight were observed at concentrations lower than 330-350 µg/L. In a flow-through ELS study with the marine fish *Cyprinodon variegatus*, the EC₁₀ derived from the presented data for hatching was 760 µg/L (NOEC = 970 µg/L) and for mortality after hatching 610 µg/L (NOEC = 520 µg/L) (Ward *et al*, 1981).

Fluorene

In a continuous flow system fingerlings of *Lepomis macrochirus* were exposed for 30 d. Growth and mortality were scored, resulting in a NOEC for growth of 125 µg/L (based on nominal concentrations; Finger *et al*, 1985).

Anthracene

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; Borovsky *et al*, 1987). The UV-intensity of sunlight on a clear day was measured to be 4245 µW/cm² (Allred & Giesy, 1985). With the assumption that sunlight has a ratio of 100:10:1 visible light:UV-A:UV-B (this is the simulated solar radiation used in most experiments) this corresponds to an UV-intensity of 420 µW/cm².

In the 6-w continuous flow toxicity study with *Pimephales promelas* a light regime during hatching was used of 16:8 light:dark by fluorescent light with UV-A 67.94 ± 9.02 µW/cm² (365 ± 36 nm) and UV-B 6.71 ± 0.81 µW/cm² (310 ± 34 nm). The NOEC for hatching was 6.7 µg/L.

Phenanthrene

The EC₁₀ for the largemouth bass *Micropterus salmoides* was derived from an ELS test without effect concentration and the EC₅₀ with a log-logistic relationship and hence the uncertainty in this value is rather high. Unlike the results for *Oncorhynchus mykiss*, the effect data for the concentration series are not reported for *Micropterus salmoides* (Black *et al*, 1983).

Pyrene

Pimephales promelas were irradiated with UV-radiation at 750 $\mu\text{W}/\text{cm}^2$ for 30 minutes after 30 minutes of incubation and mortality was recorded the next day (Kagan *et al*, 1985). The LC_{50} was 220 $\mu\text{g}/\text{L}$.

Chrysene

In an ELS study with *Brachydanio rerio* no effects were observed up to concentrations of 0.91 $\mu\text{g}/\text{L}$ (Hooftman & Evers-De Ruiter, 1992).

Benz[a]anthracene

For fish, no standard acute toxicity data are available. In a study with larvae of *Pimephales promelas* the median lethal time was determined (Oris & Giesy, 1987). 7-d old larvae were exposed to a measured concentrations of 1.8 $\mu\text{g}/\text{L}$ benz[a]anthracene for an incubation period of 24 hour in the absence of UV-radiation and thereafter exposed to UV-light with an intensity of 20 $\mu\text{W}/\text{cm}^2$ UV-B (290-336 nm), 95 $\mu\text{W}/\text{cm}^2$ UV-A (336-400 nm). After the incubation time of 24 hours, the medium was renewed every 12 hours. The median lethal time after UV-radiation started was 65 hours. Thus, after 89 hours, of which the last 65 hours were with UV radiation, 50% mortality of the fish larvae occurred at 1.8 $\mu\text{g}/\text{L}$.

Fluoranthene

From an ELS study with *Brachydanio rerio* EC_{10} values for length (18 $\mu\text{g}/\text{L}$) and weight effects (21 $\mu\text{g}/\text{L}$) were determined (Hooftman & Evers-De Ruiter, 1992). In an ELS study with *Pimephales promelas* the no effects on growth were determined below 10.4 $\mu\text{g}/\text{L}$, unless UV-light was used, which resulted in a NOEC of 1.4 $\mu\text{g}/\text{L}$ (Spehar *et al*, 1999).

Benzo[a]pyrene

Two ELS studies for fish in fresh water were found. In a 28-d ELS study with *Brachydanio rerio* no significant effects were observed for mortality, hatchability, length, and weight up to measured concentrations of 4.0 $\mu\text{g}/\text{L}$ (Hooftman & Evers-De Ruiter, 1992).

In a 36-d ELS study with *Oncorhynchus mykiss* solutions were renewed every 7 to 10 days and water concentrations were measured every five days. Aqueous concentrations appeared to be rather constant. It appeared that mortality and hatching were not dose-response related in a range of measured concentrations ranging from 0.08 to 3.0 $\mu\text{g}/\text{L}$ (Hannah *et al*, 1982). Only at 2.4 $\mu\text{g}/\text{L}$ a significant difference in mortality was observed. The length of alevins was significantly reduced at all benzo[a]pyrene concentrations. However, a dose-response relationship was completely lacking and the effect percentage did not exceed 8% at all concentrations. At 0.21, 2.4, and 3.0 $\mu\text{g}/\text{L}$ significantly more abnormalities were observed. However, at intermediate concentrations of 0.37 $\mu\text{g}/\text{L}$ and 1.5 $\mu\text{g}/\text{L}$ no significant effects were observed. Therefore, the NOEC for abnormalities is 1.5 $\mu\text{g}/\text{L}$. If the presented data are evaluated with a log-logistic relationship, an EC_{10} of 2.9 $\mu\text{g}/\text{L}$ is derived. Due to the absence of dose-response relationships for mortality, hatching, and length, this EC_{10} for abnormalities is considered as most critical endpoint for *Oncorhynchus mykiss*.

In a 7-d ELS study with the marine fish *Fundulus heteroclitus* mild deformities were observed in the benzo[a]pyrene treatment groups ranging from 0.25 to 10 $\mu\text{g}/\text{L}$, while these effects were not observed in the controls (Wassenberg *et al*, 2002). The percentage effect ranged from 0 to 43% but a dose-response relationship was completely missing. In the second lowest concentration of 0.5 $\mu\text{g}/\text{L}$ 0% deformities were observed. Therefore, no useable endpoint can be derived from this study.

In 6-d ELS study with the marine flatfish *Psettichtys melanostichus* the only tested concentration of 0.1 $\mu\text{g}/\text{L}$ resulted in significantly reduced hatching success (on the fifth day of the study) and in 5% of the embryos deformities were found (Hose *et al*, 1982). However, in the control group only 57.0% hatched on average, with a range from 21.6 to 89.6%. In the treated group the average

hatching success was 28.1% with a range of 7% to 67.6%. The meaning of these results can therefore be questioned, especially because after 120 hours the percentage hatching was almost equal.

Benzo[b]fluoranthene

No toxicity data for fish are available.

Benzo[k]fluoranthene

In two studies, the effects of benzo[k]fluoranthene in an ELS test with *Brachydanio rerio* was examined. In the first 28-d study one concentration of 0.58 µg/L was tested. At this concentration 52% mortality occurred (Hooftman & Evers-De Ruiter, 1992). In a second 42-d study a dose-response relationship was examined. The mentioned concentrations here are based on measured concentrations per concentration and not on average recovery times the nominal concentration as given in the report. The LC₅₀ estimated from the presented data with a log-logistic relationship was 0.65 µg/L. From the data for weight and length EC₁₀ are derived of 0.31 and 0.17 µg/L. Due to the good fit of the log-logistic equation, these estimates have a low uncertainty.

Dibenz[a,h]anthracene

For fish, no standard acute toxicity data are available for dibenz[a,h]anthracene. A study with larvae of *Pimephales promelas* was performed to determine the median lethal time (Oris & Giesy, 1987). 7-d old larvae were exposed to a measured concentrations of 0.15 µg/L dibenz[a,h]anthracene for an incubation period of 24 hour in the absence of UV-radiation and thereafter exposed for 96 hours to UV-light with an intensity of 20 µW/cm² UV-B (290-336 nm), 95 µW/cm² UV-A (336-400 nm). After the incubation time of 24 hours, the medium was renewed every 12 hours. After 120 hours, of which the last 96 hours were with UV radiation, no mortality of the fish larvae occurred at 0.15 µg/L.

Benzo[ghi]perylene

In a study with *Pimephales promelas* 7-d old larvae were exposed to benzo[ghi]perylene concentrations of 0.15 µg/L benzo[ghi]perylene for an incubation period of 24 hour in the absence of UV-radiation and thereafter exposed to UV-light with an intensity of 20 µW/cm² UV-B (290-336 nm), 95 µW/cm² UV-A (336-400 nm). After the incubation time of 24 hours, the medium was renewed every 12 hours and exposure in combination with UV-radiation lasted for 96 hours. After 120 hours, of which the last 96 hours were with UV radiation, less than 20% mortality of the fish larvae occurred at 0.15 µg/L.

In an ELS study with *Brachydanio rerio* no effects were observed up to concentrations of 0.16 µg/L (Hooftman & Evers-De Ruiter, 1992).

Indeno[1,2,3-cd]pyrene

No toxicity data for fish are available.

Aquatic invertebrates

Naphthalene

For the marine environment, some studies are available that show low effect concentrations. Caldwell *et al* (1977) found that naphthalene at 130 µg/L (measured concentration) significantly prolonged the development of larvae of the Dungeness crab (*Cancer magister*) in a 40-d toxicity study with continuous flow. At the lower concentration of 21 µg/L this effect was not observed. However, this effect was only observed with crabs from Alaska and not with crabs from Oregon in a duplicate experiment for 60 d. Still, the results are significant (P<0.01) and were not only

observed for naphthalene but also for the higher concentration of the water soluble fraction of crude oil in the same experiment.

At the only concentration tested of 14 µg/L Ott *et al* (1978) found significant adverse effects on the lifetime (15 days) of adult marine copepods (*Eurytemora affinis*) ($P < 0.01$) and their brood size ($P < 0.01$) and number of eggs ($P < 0.05$). The test was performed in closed bottles and solutions were renewed daily. Concentrations were measured at the start and from preliminary measurements it was concluded that the loss of naphthalene is less than 8% in 24 h.

Sanborn & Malins (1977) tested newly hatched zoea of the Dungeness crab (*Cancer magister*) and stage I and IV larvae of the spot shrimp (*Pandalus platyceros*) with radiolabelled naphthalene (5.10 Ci/mol: regular activity) exposed by continuous flow at only one concentration. The measured concentrations varied from 8-12 µg/L. Within 36 h 100% mortality occurred. In the controls less than 1% mortality occurred.

From the three studies above only the one from Caldwell *et al* (1977) is considered useful as from the two latter studies, no NOEC could be derived. However, these results suggest that marine crustaceans are a sensitive group of species. From the results with *Cancer magister* a NOEC of 21 µg/L can be derived.

NOECs are available for algae, crustaceans, fish and echinodermata. For the latter taxonomic group the NOEC results from a toxicity test with only 4 days of exposure (Falk-Petersen & Lønning, 1982; Saethre *et al*, 1984). However, these studies with the green sea urchin *Strongylocentrotus droebachiensis* are ELS studies with eggs and because it is a vulnerable life-stage it can also be regarded as a chronic NOEC.

Acenaphthylene

In a acute toxicity study 48-h with *Daphnia magna*, acenaphthylene concentrations were measured and the EC₅₀ for immobility was 1800 µg/L (Bisson *et al*, 2000). In a 7-d renewal reproduction study with *Ceriodaphnia dubia* the EC₁₀ was 64 µg/L (Bisson *et al*, 2000).

Acenaphthene

The 7-d EC₁₀ for reproduction of *Ceriodaphnia dubia* exposed to acenaphthene is 42 µg/L (Bisson *et al*, 2000).

Fluorene

The lowest effect value for fluorene is an EC₁₀ of 25 µg/L from the 7-d reproduction study with *Ceriodaphnia dubia* (Bisson *et al*, 2000). The second lowest value is the NOEC of 62.5 µg/L from the 21-d reproduction study with *Daphnia magna* (Finger *et al*, 1985). In the first study the medium was renewed daily, in the second study the exposure system was intermittent flow. The EC₁₀ for *Ceriodaphnia dubia* is similar to the EC₁₀ presented in a figure for the immobility of *Daphnia pulex* from an acute 48-h study (Smith *et al*, 1988).

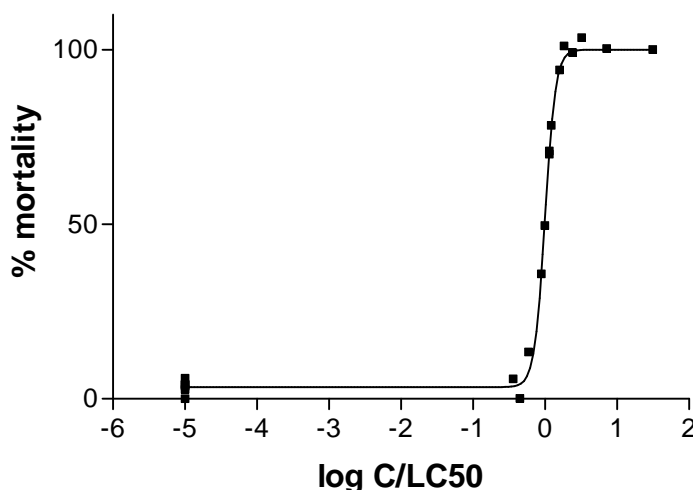
Anthracene

Anthracene is very phototoxic and toxic effects (LC₅₀s) are observed at concentrations lower or equal to the lowest chronic effect concentrations (see also Section 0).

Ultraviolet radiation in the most sensitive chronic toxicity studies was less harsh than that of (simulated) solar radiation. The 21-d static renewal toxicity studies with *Daphnia magna* (Holst & Giesy, 1989; Foran *et al*, 1991) had a light regime of 16:8 light:dark and a total UV-radiation of 117 µW/cm² with a ratio of UV-A:UV-B of 8:1. This resulted in NOECs or EC₁₀s for reproduction of 1.5-2.0 µg/L. In a 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 (see Figure 7.1.1). The LC₅₀ is estimated to be 1 µg/L. From the figures it can be

concluded that all treatments with different UV-intensities result in very steep dose-response relationships. If first the LC₅₀ is estimated and exposure concentrations are expressed as a ration of this LC₅₀ for each light intensity, then a clear dose response relationship can be derived. For exposure to full sunlight, the LC₅₀ is estimated to be 1.0 µg/L.

***Daphnia pulex* exposed to anthracene and natural light**



Reproduced from Allred & Giesy (1985). Anthracene exposure lasted for 24 hrs, followed by ½ hour of full sunlight.

Figure 7.1.1. Dose response curve for *Daphnia pulex* exposed successively to anthracene and full sunlight.

For the marine environment the lowest value is for brine shrimp *Artesia salina*, exposed to anthracene for 10 hours with irradiation by sunlight for the last eight hours (Peachey & Crosby, 1996). Similar to fresh water, this is the lowest effect concentration available. From the figures, the EC₁₀ is estimated to be 1.7 µg/L. This is slightly higher than the EC₅₀ for *Daphnia pulex*. For the brine shrimp, however, the exposure to sunlight is much longer, 8 hours instead of half an hour.

Phenanthrene

For insects only 50% effect concentrations for phenanthrene were reported (Landrum *et al*, 2003). In the study by Bleeker *et al* (2003) exposure is via sediment, which makes it less useful for the aquatic risk assessment. The lowest EC₁₀ (13 µg/L) is for reproduction of *Ceriodaphnia dubia* in a 7-d toxicity test.

At 20 µg/L reproduction effects were observed in comparison with the solvent (acetone) control for the marine polychaete worm *Neanthes arenaceodentata* exposed to phenanthrene for 8 weeks (Emery & Dillon, 1996). However, only one sublethal concentration was tested and effects were seawater without solvent showed the same effects. The validity of these results is therefore limited. For the same species also a 96-h LC₅₀ for emergent juveniles of 51 µg/L was reported. This test was used to determine the sublethal concentration mentioned above (Emery & Dillon, 1996).

Pyrene

The lowest value for pyrene for *Daphnia magna* was observed after exposure of neonates for 24 h with 16:8 hour light:dark, then at an UV-intensity of 370±20 µW/cm² (295-365 nm; peak 340 nm) for 2 hours and 1 hour of recovery in the test medium. The EC₅₀ for immobility was 1.38 µg/L (Wernersson, 2003). In a similar treatment (2 hours of recovery instead of 1, the EC₅₀ for 4-d old daphnids was 5.7 µg/L (Wernersson & Dave, 1997). After one hour of exposure in the dark followed by one hour UV-irradiation, at 1300 µW/cm² (320-400 nm; peak 350 nm), the LC₅₀ was 4 µg/L (Kagan *et al*, 1985; Kagan *et al*, 1987). When exposed to UV-B radiation only (intensity 64

$\mu\text{W}/\text{cm}^2$) for four times two hours during 48 hours, the EC_{50} for immobility of neonates ranges from 2.7 to 20 $\mu\text{g}/\text{L}$ at different hardness of the artificial test media and different concentrations of dissolved organic matter of natural waters (Nikkilä *et al*, 1999). For the fresh water mollusc *Utterbackia imbecilis* the 24-h LC_{50} was 2.63 $\mu\text{g}/\text{L}$ with UV-A radiation (320-400 nm) at an intensity of 70 $\mu\text{W}/\text{cm}^2$ (Weinstein & Polk, 2001). The reported concentrations were not analytically verified.

After 7 days of exposure Bisson *et al* (2000) found an EC_{10} of 2.1 $\mu\text{g}/\text{L}$ for reproduction of *Ceriodaphnia dubia*.

When exposed for 2 hours in the dark followed by one hour with UV-radiation (320-400 nm; peak 350 nm) at an intensity of 1300 $\mu\text{W}/\text{cm}^2$, the LC_{50} for nauplii of *Artemia salina* was 8 $\mu\text{g}/\text{L}$ (Kagan *et al*, 1985; Kagan *et al*, 1987). When exposed for 2 hours in the dark followed by eight hours with UV-radiation (peak 312 nm) at an intensity of 975-1000 $\mu\text{W}/\text{cm}^2$, the LC_{50} for nauplii of *Artemia salina* was estimated from the presented figure to be 36 $\mu\text{g}/\text{L}$ (Peachey & Crosby, 1996). The same treatment with sunlight ($\lambda > 290$ nm) at an intensity of 407-1429 $\mu\text{W}/\text{cm}^2$ resulted in an EC_{50} of 3.4 $\mu\text{g}/\text{L}$ (Peachey & Crosby, 1996). From these results it may be concluded that the maximum intensity of the radiation is more important than the time of irradiation. Of the crustaceans *Mysidopsis bahia* was the most sensitive species. Under ultraviolet light with an intensity of 397 ± 35.1 $\mu\text{W}/\text{cm}^2$ UV-A (365 ± 36 nm) and 134 ± 22.8 $\mu\text{W}/\text{cm}^2$ UV-B (310 ± 34 nm) with a photoperiod of 16:8 hour light:dark the LC_{50} was 0.89 $\mu\text{g}/\text{L}$. Under the same conditions, the LC_{50} for embryos/larvae of *Mulina lateralis* was 0.23 $\mu\text{g}/\text{L}$, while the LC_{50} for juveniles of 1 to 1.5 mm of the same species was 1.68 $\mu\text{g}/\text{L}$ (Pelletier *et al*, 1997). For embryos/larvae of the mollusc *Crassostrea gigas* the shell development was monitored after an exposure of 48 hours under UV-light with an intensity of 456.2 ± 55 $\mu\text{W}/\text{cm}^2$ UV-A and 6.3 ± 0.1 $\mu\text{W}/\text{cm}^2$ UV-B with a photoperiod of 12:12 hour light:dark. The NOEC was 0.5 $\mu\text{g}/\text{L}$ (Lyons *et al*, 2002). Although the exposure time of this study is rather short (48 hours), the endpoint is a chronic one (shell development/malformation). Except from the study by Pelletier *et al* (1997) concentrations were not analytically verified.

Chrysene

The only study, that showed a considerable effect of chrysene, was a determination of the median lethal time to neonates of *Daphnia magna* (Newsted & Giesy, 1987). In this experiment, the daphnids were exposed to one concentration of chrysene (measured concentration of 0.7 $\mu\text{g}/\text{L}$). The test was performed as a static-renewal acute toxicity test. After 24 hours of exposure with a 16:8 light:dark photoperiod, the animals were exposed to UV-light with an intensity of 25 ± 3 $\mu\text{W}/\text{cm}^2$ UV-B (310 ± 36 nm), 120 ± 5 $\mu\text{W}/\text{cm}^2$ UV-A (365 ± 36 nm), and 680 ± 10 $\mu\text{W}/\text{cm}^2$ visible light (400 to 700 nm). The median lethal time after UV-radiation started was 24 hours. Thus, after 48 hours, of which the last 24 hours were with UV radiation, 50% mortality of the daphnids occurred at 0.7 $\mu\text{g}/\text{L}$.

Benz[a]anthracene

The 96-h LC_{50} of *Daphnia pulex* exposed to benz[a]anthracene under a 12:12 h photoperiod to mixed fluorescent and natural light was 10 $\mu\text{g}/\text{L}$ (Trucco *et al*, 1983). The 48-h LC_{50} of *Daphnia magna* from a test in the dark was higher than 9.1 $\mu\text{g}/\text{L}$ (Bisson *et al*, 2000). Also under artificial light with a photoperiod of 16:8 h light:dark 50% mortality was not reached in the highest concentration when *Daphnia magna* was exposed for 24 hour. The same test followed by irradiation with UV (295-365 nm; peak 340 nm) with an intensity of 370 ± 20 $\mu\text{W}/\text{cm}^2$ for 2 hours and 1 hour of recovery in the test medium lead to an LC_{50} of 3.4 $\mu\text{g}/\text{L}$. UV-radiation thus increases the toxicity of benz[a]anthracene.

For *Ceriodaphnia dubia* no effects were observed in a 7-d study at concentrations up to 8.7 $\mu\text{g}/\text{L}$ (Bisson *et al*, 2000).

Fluoranthene

For the annelid *Stylaria lacustris* the reported data are from a study with sediment exposure (Suedel *et al*, 1996) and are also less suitable for the risk assessment for the aquatic compartment. The number of taxonomic groups is thus reduced to six.

The lowest chronic NOECs or EC₁₀ are in between 1.0 and 1.5 µg/L. Bisson *et al* (2000) found an EC₁₀ of 1.2 µg/L for the reproduction of *Ceriodaphnia dubia* exposed to fluoranthene for 7 days under laboratory light with an intensity less than 500 lux. Oris *et al* (1991) found for the same endpoint NOECs of 57 and 32 µg/L. Wilcoxon *et al* (2003) reported a 10-d LC₁₀ for the amphipod *Hyalella azteca* of 1.1 µg/L. This test was performed under UV-enhanced light with a photoperiod of 16:8 hours light:dark and an intensity of 7.54 µW/cm² UV-B, 102.08 µW/cm² UV-A, and 289.24 µW/cm² visible. The LC₁₀ decreased strongly with UV-intensity. Under gold light (intensity of 0.17 µW/cm² UV-B, 0.09 µW/cm² UV-A, 167.72 µW/cm² visible) and fluorescent light (intensity of 1.32 µW/cm² UV-B, 13.65 µW/cm² UV-A, 424.69 µW/cm² visible) the LC₁₀s were 56 and 8.0 µg/L, respectively. However, these values are comparable with the reported EC₅₀s.

When exposed under laboratory ultraviolet light with 283 µW/cm² UV-A and 47 µW/cm² UV-B and a photoperiod of 12:12 h light dark, Spehar *et al* (1999) found a NOEC of 1.4 µg/L for growth of *Daphnia magna*, exposed for 21 days. With UV-enhanced light with an intensity of 102 µW/cm² UV-A, 7.5 µW/cm² UV-B, and 289 µW/cm² visible light and a photoperiod of 16:8 h light:dark, a 10-d LC₁₀ for *Hyalella azteca* was found of 1.1 µg/L (Wilcoxon *et al*, 2003). In all these experiments concentrations were experimentally determined. For the fresh water mollusc *Utterbackia imbecilis* the 24-h LC₅₀ was 2.45 µg/L with UV-A radiation (320-400 nm) at an intensity of 70 µW/cm² (Weinstein & Polk, 2001).

However, the same effect that was observed for anthracene is also observed for fluoranthene. Fluoranthene appears to be extremely phototoxic when some organisms are exposed in combination with ultraviolet radiation, such as sunlight. The acute LC₅₀s of fluoranthene for fresh water species exposed under laboratory lighting with UV are comparable or even lower than the chronic NOEC. The 96-h LC₅₀s for the freshwater oligochaete *Lumbriculus variegatus* and *Hydra americana* were 1.2 µg/L and 2.2 µg/L, respectively, with ultraviolet light with 359-587 µW/cm² UV-A and 63-80 µW/cm² UV-B and a photoperiod of 12:12 h light dark. The 48-h LC₅₀ for *Daphnia magna* was 1.6 µg/L, with ultraviolet light with 783-850 µW/cm² UV-A and 104 µW/cm² UV-B and a photoperiod of 12:12 h light dark (Spehar *et al*, 1999).

In the marine environment studies by Boese *et al* (1999) and Swartz *et al* (1990) are sediment studies in which the overlying water was measured. In the study by Spehar *et al* (1999) a 31-d chronic NOEC for the reproduction of the Mysid shrimp *Mysidopsis bahia* are reported. With a photoperiod of 16:8 hours light:dark in fluorescent light the NOEC was reported to be 11.1 µg/L. If instead UV-radiation was applied (465-724 µW/cm² UV-A and 68-109 µW/cm² UV-B), the NOEC dropped to 0.6 µg/L.

Under the same UV-conditions conditions, also some LC₅₀ values were found. The 48-h LC₅₀ for the marine mollusc *Mulinia lateralis* was 2.8 µg/L, the 96-h LC₅₀ for *Mysidopsis bahia* was 1.4 µg/L, the 48-h LC₅₀ for the urchin *Arbacia punctulata* was 3.9 µg/L and the 96-h LC₅₀ for *Pleuronectes americanus* was 0.1 µg/L (Spehar *et al*, 1999).

Benzo[a]pyrene

Only acute toxicity data with exposure to UV-light result in effects at concentrations near the aqueous solubility (1.2-1.8 µg/L; Mackay *et al*, 2000). The lowest acute value is the EC₅₀ of 1.2 µg/L for immobility of *Daphnia magna* after exposure for 24 h with a 16:8 light:dark photoperiod, then 2 hours exposure to UV (295-365 nm; peak 340 nm) with an intensity of 370 ± 20 µW/cm² for 2 hours, followed by 1 hour of recovery in the test medium (Wernersson, 2003). In an earlier study

with similar exposure except from the fact the recovery period was 1 hour instead of 2 hours, the EC₅₀ was 8.6 µg/L (Wernersson & Dave, 1997).

The 7-d EC₁₀ for reproduction of *Ceriodaphnia dubia* is 0.5 µg/L (Bisson *et al*, 2000). Also in this case concentrations were measured. The EC₁₀, estimated from the presented data, and NOEC for reproduction of *Daphnia magna* in 14-d study were 12.5 µg/L (Atienzar *et al*, 1999), a value which is above the aqueous solubility of benzo[a]pyrene. The actual concentrations were not measured in this study.

A 48-h study with eggs and sperm of the echinoderm *Strongylocentrotus purpuratus* (Hose *et al*, 1983) might be considered as chronic as well, because a sensitive part of the life-cycle of this organism is incorporated. No significant effects were observed on fertilisation success of eggs. After 48 hours however, the embryos exposed to a nominal concentration of 1.0 µg/L benzo[a]pyrene and higher showed a significantly higher percentage abnormalities of the gastrulae. Only the nominal concentration of 0.5 µg/L was not significantly different from the solvent (ethanol) control. All treatments, including the solvent control were significantly different from the sea water control. The percentage effect shows a dose-response relationship in the nominal concentrations of 0.5, 1, and 5 µg/L. At higher concentrations, *i.e.* above the aqueous solubility, the effect percentage remains rather constant. The concentrations were measured and initial concentrations were within 10% of the nominal values. After 48 hours all concentrations had declined to about 0.5 µg/L except from the highest concentrations of 50 µg/L, which had declined to 2 µg/L.

The shell development of embryos of the mollusc *Crassostrea gigas* was investigated in a 48-h study (Lyons *et al*, 2002). Under UV lacking fluorescent laboratory lighting with a photoperiod of 12:12 h light:dark, the NOEC for abnormal shells is 1 µg/L. With a log-logistic relationship, the derived EC₁₀ from the presented data is 1.1 µg/L. When UV irradiation with an intensity of $456.2 \pm 55 \mu\text{W}/\text{cm}^2$ UV-A and $6.3 \pm 0.1 \mu\text{W}/\text{cm}^2$ UV-B with a photoperiod of 12:12 h light:dark was used, the NOEC reduced to 0.5 µg/L. The presented data show a clear dose-response relationship and the EC₁₀ derived from these data with a log-logistic equation is 0.22 µg/L.

A method for evaluating pollutant genotoxicity, embryotoxicity and teratogenicity using sea urchin (*Strongylocentrotus purpuratus*) embryos was developed by Hose (1985) and tested using benzo[a]pyrene. No effects were observed on the fertility up to 50 µg/L. However, significant fewer embryos treated with at least 1 µg/L had completed gastrulation than the control. Genotoxic effect, as evidenced by increased anaphase aberration rates, were even significant at the lowest dose tested, 0.5 µg/L. Chromosomes or acentric fragments outside the spindle apparatus and translocation bridges.

The effect of benzo[a]pyrene and other compounds has been investigated on survival, development, and reproduction of the estuarine copepod *Eurytemora affinis*. For survival of the adult stage a NOEC of 12 µg/L was observed (96-h LC₅₀ was 58 µg/L). Larvae (nauplii stage) exposed to this NOEC never reached the copepodid stage and subsequently died (Forget-Leray *et al*, 2005). The NOEC for the complete life cycle should therefore be < 12 µg/L.

Benzo[b]fluoranthene

Some acute toxicity studies for benzo[b]fluoranthene have been performed with *Daphnia magna*. In a standard 48-h study performed in the dark, no toxicity was found up to 1.1 µg/L (Bisson *et al*, 2000). In a 24-h study with a photoperiod 16:8 h light: dark no toxicity was found either. In the same treatment but extended with 2 hours of irradiation with UV light (295-365 nm; peak 340 nm) with an intensity of $370 \pm 20 \mu\text{W}/\text{cm}^2$ and a recovery period of 2 hours, the EC₅₀ for immobility was 4.2 µg/L (Wernersson & Dave, 1997). This is still above the aqueous solubility of 1.1-1.5 µg/L (Mackay *et al*, 2000). No toxic effects were observed as well in two chronic toxicity studies with

the algae *Pseudokirchneriella subcapitata* and the crustacean *Ceriodaphnia dubia* (Bisson *et al*, 2000).

Benzo[k]fluoranthene

Acute toxicity data for benzo[k]fluoranthene are only available for *Daphnia magna*. However, in the two available studies (Bisson *et al*, 2000; Verriest *et al*, 2001) no effects were observed. However, due to the low solubility of benzo[k]fluoranthene of about 1 µg/L (Mackay *et al*, 2000), acute effects are not anticipated. However, in the 7-d reproduction study with *Ceriodaphnia dubia* no effects were observed either (Bisson *et al*, 2000).

Dibenz[a,h]anthracene

The lowest acute value for immobility of *Daphnia magna* exposed to dibenz[a,h]anthracene is the EC₅₀ of 1.8 µg/L after exposure for 24 h with a 16:8 light:dark photoperiod, then 2 hours exposure to UV (295-365 nm; peak 340 nm) with an intensity of 370 ± 20 µW/cm² for 2 hours, followed by 1 hour of recovery in the test medium (Wernersson, 2003). In an earlier study with similar exposure except from the fact the recovery period was 1 hour instead of 2 hours, the EC₅₀ was 4.6 µg/L (Wernersson & Dave, 1997).

No chronic effect was observed at concentrations up to 0.032 µg/L in a 7-d study with *Ceriodaphnia dubia*.

In studies with the annelid *Neanthes arenaceodentata* (Rossi & Neff, 1978) no effects at concentrations near the aqueous solubility of dibenz[a,h]anthracene were observed.

Benzo[ghi]perylene

The 48-h LC₅₀ for *Daphnia magna* exposed to benzo[ghi]perylene was higher than 0.2 µg/L, which is in itself higher than the aqueous solubility of 0.14 µg/L (Mackay *et al*, 2000). The EC₁₀ of 0.082 µg/L from a 7-d reproduction study with the crustacean *Ceriodaphnia dubia* (Bisson *et al*, 2000) is below the aqueous solubility.

Indeno[1,2,3-cd]pyrene

Indeno[1,2,3-cd]pyrene was not acutely toxic to *Daphnia magna* at concentrations up to 357 µg/L. The 7-d EC₁₀ for reproduction of *Ceriodaphnia dubia* was 0.27 µg/L. In all three cases concentrations were measured (Bisson *et al*, 2000).

Algae and aquatic plants

Naphthalene

In a study with the duckweed *Lemna gibba* the lowest EC₁₀ for growth was 280 µg/L (Ren *et al*, 1994), but this was after photomodification of naphthalene. Otherwise no effects were observed below 2000 µg/L (Ren *et al*, 1994).

Acenaphthylene

In a 72-h static long term study with *Pseudokirchneriella subcapitata* exposed to acenaphthylene the EC₁₀ was 82 µg/L (Bisson *et al*, 2000). An EC₅₀ value was not reported, but should be higher than the reported EC₁₀ value.

Acenaphthene

For acenaphthene no EC₅₀ for algae is reported. However, a good toxicity study with *Pseudokirchneriella subcapitata* is available (Bisson *et al*, 2000) for which only the EC₁₀ of 38 µg/L is reported. The EC₅₀ must therefore be higher than this value.

Fluorene

In a 72-h static long term study with *Pseudokirchneriella subcapitata* exposed to fluorene the EC₁₀ for growth was 820 µg/L (Bisson *et al*, 2000), but in a 7d test with the same species, the EC₁₀ for cell number was 1400 µg/L (Finger *et al*, 1985). In a study with the duckweed *Lemna gibba* the lowest EC₁₀ for growth was 280 µg/L (Ren *et al*, 1994), but this was after photomodification of naphthalene. Otherwise no effects were observed below 2000 µg/L (Ren *et al*, 1994).

Anthracene

Anthracene is very phototoxic and toxic effects (LC_{50s}) are observed at concentrations lower or equal to the lowest chronic effect concentrations (see also Section 0).

In a short-term study (Gala & Giesy, 1992) algae were exposed to anthracene in a static renewal set-up for 34-36 hours with 765 µW/cm² UV-A during the last 22-24 hours. UV-B was filtered out. The NOECs and EC_{10s} for growth rate and primary production ranged from 1.4 to 1.5 µg/L. In these three chronic studies experimental concentrations were measured.

Short-term (24-h) experiments were performed to examine the effect of anthracene on green alga *Scenedesmus armatus* grown in a batch culture system at irradiances of 12, 33, 48, and 64 W/m² of the photosynthetically active radiation range. Cultures were aerated (0.1 or 2% CO₂) or nonaerated. As a result of aeration (evaporation) the concentration of anthracene dropped from 0.45 mg/L at the beginning of the experiment to an undetectable value after 10 h. At nominal concentrations exceeding 0.05 mg/L inhibited the growth of the algae in a concentration- and irradiance-dependent manner. The effect observed at 64 and 48 W/m² was independent of the CO₂ level, whereas the growth inhibition at 33 and 12 W/m² was much greater in cultures aerated with 2% than with 0.1% CO₂.

Phenanthrene

For phenanthrene the lowest EC₁₀ (10 µg/L) is for growth rate of the algae *Pseudokirchneriella subcapitata* (Halling-Sørensen *et al*, 1996), but this is not the only value for this algae species. The authors tested several different experimental set-ups varying in exposure time and enrichment with bicarbonate to control the pH and whether or not the system was closed. In another recent study the EC₁₀ for growth rate of *Pseudokirchneriella subcapitata* was also higher (23 µg/L: Bisson *et al*, 2000).

Pyrene

The lowest chronic value for algae is the 72-h EC₁₀ of 1.2 µg/L for growth of *Pseudokirchneriella subcapitata* (Bisson *et al*, 2000). For the macrophyte *Lemna gibba* (Ren *et al*, 1994; Huang *et al*, 1995; Huang *et al*, 1997a; Huang *et al*, 1997b) and the cyanophyte *Anabaena flos-aqua* (Bastian & Toetz, 1982) no toxic effect were observed at concentrations up to the aqueous solubility. All concentrations except data from Huang *et al* (1997a; 1997b) were analytically verified.

Chrysene

In a 72-h static long term study with *Pseudokirchneriella subcapitata* exposed to chrysene no effects were observed below 1 µg/L (Bisson *et al*, 2000). In a study with the duckweed *Lemna gibba* no effects were observed below 2000 µg/L (Ren *et al*, 1994).

Benz[a]anthracene

The growth of *Pseudokirchneriella subcapitata*, exposed to benz[a]anthracene concentrations far above the aqueous solubility, was not inhibited by 50% when illuminated with a 16:8 h light:dark photoperiod with cool white fluorescent light (Cody *et al*, 1984).

The 72-h EC₁₀ for inhibition of growth of *Pseudokirchneriella subcapitata* from a study with measured concentrations of benz[a]anthracene is 1.2 µg/L (Bisson *et al*, 2000). For the same algae species Cody *et al* (1984) presented a dose-effect relationship. From the data in the figure, a 96-h

EC₁₀ of 18 µg/L can be estimated with a log-logistic relationship. However, the uncertainty in this estimate is substantial due to the flatness of the dose-response curve, probably as a result of solubility limitations: the aqueous solubility of benz(a)anthracene is around 10 µg/L (Mackay *et al*, 2000). Further, this value is based on nominal concentrations. Probably most important, from the presented spectra it is estimated that the total light intensity is less than 50 µW/cm², although the light intensities are given at single wavelengths (Cody *et al*, 1984). The light intensity may play an important role in the lower EC₁₀ from the study by Bisson *et al* (2000). Therefore, the aforementioned values of 1.2 µg/L is considered to be more realistic than this value of 18 µg/L, due to the low light intensity. For the cyanophyte *Anabaena flos-aqua* the NOEC for after two weeks of exposure was 8.3 µg/L although at 5 µg/L also significant effects were observed (Bastian & Toetz, 1982). Therefore, no clear dose-response relationship was observed. The light regime was continuous light at 951-1903 µW/cm² (200-400 foot candles). The concentration of benz[a]anthracene declined by 85% in 14 days. The real effect concentration is therefore overestimated. Not the growth rate was determined in this study, but the biomass after 14 days.

In a study with the duckweed *Lemna gibba* only concentrations far above the aqueous solubility are reported (Huang *et al*, 1997a; Huang *et al*, 1997b).

Fluoranthene

The EC₁₀ derived from the graphs for the cyanobacterium *Anabaena flos-aqua* (Bastian & Toetz, 1982) is rather uncertain. The lowest concentration gave a relatively high effect percentage in relation to both the control and the second concentration. Therefore, a NOEC could not be derived (<0.38 µg/L) (Bastian & Toetz, 1982). The EC₁₀ for the algae *Pseudokirchneriella subcapitata* is 8.6 µg/L (Bisson *et al*, 2000).

Benzo[a]pyrene

The lowest EC₁₀ for algae *Pseudokirchneriella subcapitata* exposed to benzo[a]pyrene is 0.78 µg/L (Bisson *et al*, 2000). This test was performed with a light intensity of 6000 to 8000 lux (~ 2000 µW/cm² with cool white fluorescent lamps). Concentrations were measured. For the same species, the EC₁₀ can be estimated from the data presented by Cody *et al* (1984). The EC₁₀ under cool white fluorescent light was 10 µg/L, under black light 0.96 µg/L. Here, reported concentrations are nominal. Although the light intensities are given at single wavelengths, from the presented spectra it is estimated that the total light intensity is less than 50 µW/cm² in all cases. Therefore, the light intensity may play an important role in the lower EC₁₀ from the study by Bisson *et al* (2000).

Benzo[b]fluoranthene

For algae no EC₅₀ is presented. However, in the 72-h study with *Pseudokirchneriella subcapitata* the EC₁₀ for growth is larger than 1 µg/L (Bisson *et al*, 2000) and hence the EC₅₀ must also be higher than this value.

Benzo[k]fluoranthene

For algae no EC₅₀ is presented. However, in the 72-h study with *Pseudokirchneriella subcapitata* by Bisson *et al* (2000) the EC₁₀ for growth is larger than 1 µg/L and hence the EC₅₀ must also be higher than this value.

Dibenz[a,h]anthracene

For algae no EC₅₀ for dibenz[a,h]anthracene is presented. However, in the 72-h study with *Pseudokirchneriella subcapitata* the EC₁₀ for growth is 0.14 µg/L (Bisson *et al*, 2000) and hence the EC₅₀ must be higher than this value.

For *Lemna gibba* no chronic effects at concentrations near the aqueous solubility were observed (Huang *et al*, 1997a; Huang *et al*, 1997b).

Benzo[ghi]perylene

For algae no EC₅₀ is available. However, in the 72-h study with *Pseudokirchneriella subcapitata* the EC₁₀ for growth is larger than 0.16 µg/L (Bisson *et al*, 2000) and hence the EC₅₀ must also be higher than this value.

For *Lemna gibba* the reported effect concentrations are far above the aqueous solubility (Huang *et al*, 1997a; Huang *et al*, 1997b).

Indeno[1,2,3-cd]pyrene

The 72-h EC₁₀ for the growth rate of *Pseudokirchneriella subcapitata* was 1.5 µg/L.

Sediment organisms

Not relevant for this type of report.

Other aquatic organisms

Not relevant for this type of report.

7.1.2 Calculation of Predicted No Effect Concentration (PNEC)

Not relevant for this type of report.

7.2 Terrestrial compartment

Not relevant for this type of report.

7.3 Atmospheric compartment

Not relevant for this type of report.

7.4 Microbiological activity in sewage treatment systems

Not relevant for this type of report.

7.5 Calculation of Predicted No Effect Concentration for secondary poisoning (PNEC_{oral})

Not relevant for this type of report.

7.6 Conclusion on the environmental classification and labelling

Because CTPHT is a UVCB substance, it is very difficult to classify CTPHT on the basis of the individual components. In addition, not all the components can be analyzed when diluted in water. Furthermore, the different CTPHT components influence each other's solubility in the water phase and consequently the composition in the water phase will not be the same at different loadings.

Therefore, the water-accommodated fraction (WAF) approach is considered most appropriate to classify CTPHT, as recommended for oil products and products such as creosote in the OECD *Guidance Document on Aquatic Toxicity Testing of Difficult Substances and Mixtures* (OECD, 2000). With this approach the toxicity of complex multi-component substances, which are only

partially soluble in water, can be determined by preparing water-accommodated fractions (WAFs) at different loadings (e.g. 1, 10 and 100 mg/L). The term water-accommodated fraction is applied to aqueous media containing only the fraction of multi-component substances that is dissolved and/or present as a stable dispersion or emulsion. Test data obtained with WAFs apply to multi-component substances as an entity. The classification criteria are applied to the loading rate.

A WAF method to determine the toxicity of coal tar pitch was developed by Tadokoro *et al* (1991) by studying different test solution preparation methods in absence of UV irradiation: direct addition to media without filtration, direct addition with supernatant after the solid material was siphoned out of solution and diluting the stock solution of the saturated concentration. Killifish (*Oryzias latipes*), red sea bream (*Pagrus major*) and daphnia (*Daphnia magna*) were used for testing. In the direct addition method an extraction time of 24 hr was used followed by a settle time of 2 hr. In the dilution method, coal tar pitch was spread over a glass plate at a rate of 50 mg/cm², after which the plate was dipped into the water of an aquarium in order to obtain a wider surface area for the extraction. The possible number of glass plates that could be dipped into 1 litre of water corresponded to 1000 mg/L as an added amount. The total detected amount of major components (not specified) in the prepared test solutions was 0.3 % and 0.13 % (relative to total nominal loading with CTPHT) with the direct addition and dilution method, respectively. Using direct addition (with and without filtration) the LC₅₀ value was between 100 and 1000 mg/L for all species. With the dilution method the LC₅₀ was > 1000 mg/L for *O. latipes* (other species were not tested).

Additional information is available concerning the solubility of pulverized CTPHT (see Section 1.3). At 100 and 10,000 mg/L, stirred (rate unknown) at room temperature for 24 hr and filtered (0.2 µm) afterwards, the concentration in solution, expressed in DOC, is 0.3 mg/L at both loading rates, corresponding to 0.3 and 0.003 % relative to the nominal loading rates. It should however be noted that the authors of this study specified the results as preliminary and not fully reliable. A blank control was not presented for this examination at low loadings. In a second test (pH dependence), the control value was stated to be 0.9 mg/L TOC.

In another experiment a column containing 10 g of finely powered CTPHT (20-200 µm) was force-percolated by 1.1 L of tap water (water recycling for 1 wk). Each experimental period was terminated by withdrawal of 1 L of the extract and subsequent replacement of this volume by 1 L fresh tap water. This procedure was continued for 39 weeks. The total of the EPA-PAHs in the pitch sample applied comprised of 9.9% (after GC) or 9.2% (after HPLC). After the first run, 36.5 µg PAH/L was found, after 15 cycles the total PAH concentration decreased to 11.8 µg/L and after 39 cycles to 0.9 µg/L. The first water-soluble fraction was dominated by the presence of acenaphthene (7.3 µg/L), phenanthrene (8.8 µg/L), fluoranthene (9.3 µg/L) and pyrene (6.7 µg/L), followed by naphthalene (1.5 µg/L), fluorene (1.2 µg/L) and anthracene (0.6 µg/L). The total cumulative amount of water-extractable EPA PAHs amounted to approximately 370 µg/10 g (= ~0.004%).

The solubility results obtained with CTPHT in the maximum water soluble form (powder) by Tadokoro *et al* (1991) are compared with the information on the acute aquatic toxicity of the most acutely toxic PAH fluoranthene. Since the DOC concentration (*i.e.* 0.3 mg/L) of pulverized CTPHT in the experiments by Rütgers VFT (1999a, b) was equal at loadings of 100 and 10,000 mg/L, it is assumed that at both loadings the concentrations of available PAHs is also equal. It is considered likely that the same applies to the forced-percolation experiment, in which also an excess amount of 10,000 mg/L of pitch was extracted. The concentration of fluoranthene reported in the first extract (9.3 µg/L) can consequently be linked to a nominal loading of 100 mg/L finely powered pitch. Since the concentration in the force percolate exceeds the LC₅₀ for fluoranthene (*i.e.* 0.1 µg/L) by almost a factor of 100, it is plausible that at loading rates around 1 mg/L or lower CTPHT exerts toxic response and should be classified.

The most important short-coming of these tests, however, is that they were not performed in the presence of UV irradiation in order to take into account possible phototoxic effects. As a

consequence it was concluded that insufficient data is available on aquatic toxicity testing of WAFs of CTPHT. Hence, it proved to be impossible to draw any definitive conclusions on the aquatic classification of CTPHT based on the preferred WAF approach. This analysis includes several uncertainties and is at this stage used as supporting evidence that CTPHT is classifiable. The classification could be subject to revision if sufficiently reliable effects data (in the presence of UV irradiation) on WAFs of CTPHT become available.

Given the considerations mentioned above, it was decided that an alternative for the environmental classification of CTPHT is necessary. The alternative used is based upon the rules laid down in Annex I, section 1 of Regulation (EC) 1272/2008 (EU, 2008b). CTPHT is considered as a 'mixture' in this perspective. For the classification of mixtures two approaches are described in this Regulation, a classification based on summation of classified components and one based on toxicity test data.

For CTPHT the classification based on summation is preferred, because apart from toxicity test data, this method also takes into account the persistence and bioaccumulation potential of the mixture. For this classification each individual component is classified (if possible), and then the (weighed) content of classified components (in the same category) is summed. If this sum exceeds 25% then the whole mixture is classified the same (see Annex I, section 4.1.3.5 of Regulation (EC) 1272/2008). To add more weight to highly toxic components, multiplying factors (M-factors) are assigned to the individual components, which depend on the L(E)C₅₀ value of the component (see §4.1.3.5.5 in Annex I of Regulation (EC) 1272/2008)³.

The 16 individual EPA-PAHs were analysed with respect to their acute aquatic effects data and the lowest available EC₅₀ or LC₅₀ was chosen as a point of departure for aquatic hazard classification. The effects data are described in more detail in Section 7.1. These lowest acute toxicity data were combined with degradability and bioaccumulation data for each of the individual PAHs to come to a classification for each individual PAH (based upon the criteria from Regulation (EC) 1272/2008 and those from Council Directive 67/548/EEC). These data and classifications are summarized in Table 7.6.1. For twelve of the 16 EPA-PAHs this classification is N;R50-53 and Aquatic Acute 1; Aquatic Chronic 1, for one PAH (*i.e.* acenaphthylene) this classification is N;R51/53 and Aquatic Chronic 2. For the other three PAHs the conclusion is 'not classified', either due to non-occurrence of effects up to the limit of water solubility (*i.e.* benzo[b]fluoranthene and benzo[ghi]perylene) or due to relatively low toxicity (*i.e.* naphthalene).

In the next step in classification of CTPHT based on summation, M-factors were assigned to the 16 EPA-PAHs (see Table 7.6.1). For four of the PAHs no M-factors could be assigned, either due to non-occurrence of effects up to the limit of water solubility (*i.e.* benzo[b]fluoranthene and benzo[ghi]perylene) or due to relatively low toxicity (*i.e.* naphthalene and acenaphthylene). For the other PAHs these M-factors were then used to calculate the weight that each of the PAHs contributes to the toxicity of CTPHT by multiplying the weight based percentage of each specific PAH in CTPHT (data for binder pitch, which is considered as most relevant for the classification of CTPHT, taken from Table 1.2.1) with the corresponding M-factor. Since all the PAHs to which an M-factor could be assigned are classified as Aquatic Acute 1; Aquatic Chronic 1 substances, all their contributions to the toxicity of CTPHT were then summed to come to an overall contribution (in %) to the toxicity of CTPHT, *i.e.* 4504% (see Table 7.6.1). Since this value is (far) above the 25% limit from Regulation (EC) 1272/2008, it is proposed to classify CTPHT as a Chronic Category 1

³ In Directive 1999/45/EC (EU, 1999) for classification of preparations (and its adaptations and amendments), weight of individual components was added by setting concentration limits. In Regulation (EC) 1272/2008 (EU, 2008b) these concentration limits are converted to M-factors. For readability only M-factors are mentioned here.

substance, *i.e.* Aquatic Acute 1; Aquatic Chronic 1 according to Table 3.1 of Annex VI of this regulation.

In Directive 1999/45/EC (EU, 1999) the summation method is based on concentration limits instead of M-factors. For N;R50-53 substances a concentration limit of $\geq 25\%$ equals an M-factor of 1, a concentration limit of $\geq 2.5\%$ equals an M-factor of 10, etc. The contribution of each constituent in the overall toxicity of the mixture is then calculated by dividing the weight based percentage of the constituent (in %) by the concentration limit (in %) (see Table 7.6.1). Since all the PAHs to which concentration limits could be assigned are classified as N;R50/53 substances, all their contributions to the toxicity of CTPHT were then summed to come to an overall contribution (in %) to the toxicity of CTPHT, *i.e.* 180% (see Table 7.6.1). Since this value is (far) above the 25% limit from Directive 1999/45/EC, it is proposed to classify CTPHT as a N;R50/53 substance according to this directive (and Table 3.2 of Annex VI of Regulation (EC) 1272/2008).

It should be noted that for this classification it is assumed that all PAH present in CTPHT will dissolve into the water phase and thus be available to (aquatic) organisms. This is likely to give an overestimation of the toxicity of CTPHT. However, since the composition of the WAF is uncertain this toxicity estimate can be seen as a worst case. In addition, it cannot be ruled out that other components of CTPHT will contribute to the overall toxicity of the substance.

Furthermore, it should be noted that in contrast with our classification, naphthalene and benzo[b]fluoranthene are included in Annex VI of Regulation (EC) 1272/2008 with the classification Aquatic Acute 1; Aquatic Chronic 1 / N;R50-53 (as well as chrysene, benz[a]-anthracene, benzo[a]pyrene, benzo[k]fluoranthene, and dibenz[a,h]anthracene). Since CTPHT would be classified as Aquatic Acute 1; Aquatic Chronic 1 based on the other PAHs already, this classification would gain more weight if naphthalene and especially benzo[b]fluoranthene would have been included in the summation.

When CTPHT is used in a mixture, the classification as Aquatic Acute 1; Aquatic Chronic 1 asks for the assignment of an M-factor. For UVCB substances such as CTPHT, however, setting an M-factor is not considered appropriate, since by definition the composition of the substance may vary. Therefore, no M-factor for CTPHT is proposed. For CTPHT-containing mixtures it appears more appropriate to classify these by the summation method by using the classification of the individual toxic components (*i.e.* the PAHs in CTPHT).

If, notwithstanding the arguments above, there is a need for an M-factor for CTPHT, (an indication of) the overall toxicity of CTPHT should be known. For an indication of the overall toxicity the classification method based on test data may become useful (see §4.2.3.5.2 in Annex I of Regulation (EC) 1272/2008). For this classification an overall $L(E)C_{50}$ for CTPHT is calculated using the following equation:

$$\frac{\sum C_i}{L(E)C_{50m}} = \sum_{\eta} \frac{C_i}{L(E)C_{50i}}$$

where:

C_i = concentration of component i (weight percentage)

$L(E)C_{50i}$ = LC_{50} or EC_{50} for component i (in mg/L)

η = number of components

$L(E)C_{50m}$ = $L(E)C_{50}$ of the part of the mixture with test data

The weight based percentage of each specific PAH in CTPHT (data for binder pitch, which is considered as most relevant for the classification of CTPHT, taken from Table 1.2.1) divided by its corresponding lowest acute toxicity value is given in Table 7.6.1. For three of the PAHs this calculation was not possible, either due to non-occurrence of effects up to the limit of water

solubility (*i.e.* benzo[b]fluoranthene and benzo[ghi]perylene) or due to relatively low toxicity (*i.e.* naphthalene). Acenaphthylene was not detected in CTPHT, so toxicity of this compound was not used in calculating the overall toxicity of CTPHT, although it cannot be ruled out completely that the compound is present and thus contributes to the toxicity of CTPHT.

Using these data in the given equation, results in the following equation:

$$\frac{9.2}{L(E)C_{50m}} = 21280, \text{ resulting in } L(E)C_{50m} \text{ being } 0.00043 \text{ mg/L.}$$

This value would result in an M-factor of 1000. It should be noted, however, that for such an exercise the concentration (weight percentage) of the individual toxic components should be known. For CTPHT containing mixtures, when measurements are needed to determine these concentrations, it appears to be more appropriate to determine these in the mixture rather than in CTPHT. Subsequently these concentrations can then be used in the classification based on summation.

Table 7.6.1. Aquatic hazard classification of CTPHT.

16 EPA-PAHs	Degradability ^{a)}	Bioaccumulation ^{b)}	Lowest acute aquatic toxicity value (E/LC ₅₀) in mg/L	Content in CTPHT (% w/w) ^{c)}	Proposed classification of PAH ^{d)}		Multiplying factor (M) ^{e)}	M × content in CTPHT (% w/w) ^{f)}	Concentration limit (%)	Content in CTPHT / Concentration limit ^{g)}	Content in CTPHT (% w/w) / E/LC ₅₀ ^{h)}
				According to Annex IV Table 3.1	According to Annex IV Table 3.2						
Naphthalene	NR	>500	> 4.3	0	not classified ⁱ⁾	not classified ⁱ⁾	– ^{j)}	–	– ^{j)}	–	– ^{l)}
Acenaphthylene	NR	>500	1.8	0	H411	N; R51/53	– ^{j)}	–	– ^{j)}	–	0
Acenaphthene	NR	>500	0.58	0.043	H400; H410	N; R50/53	1	0.043	25	0.00172	0.074
Fluorene	NR	>500	0.025	0.047	H400; H410	N; R50/53	10	0.47	2.5	0.0188	1.88
Anthracene	NR	>500	0.001	0.13	H400; H410	N; R50/53	100	13	0.25	0.52	130
Phenanthrene	NR	>500	0.051	0.63	H400; H410	N; R50/53	10	6.3	2.5	0.252	12.4
Pyrene	NR	>500	0.00023	0.95	H400; H410	N; R50/53	1000	950	0.025	38	4130
Chrysene	NR	–	0.0007	0.81	H400; H410 ⁱ⁾	N; R50/53 ⁱ⁾	1000	810	0.025	32.4	1157
Benz[a]anthracene	NR	> 100 – < 500	0.0018	0.77	H400; H410 ⁱ⁾	N; R50/53 ⁱ⁾	100	77	0.25	3.08	428
Fluoranthene	NR	>500	0.0001	1.1	H400; H410	N; R50/53	1000	1100	0.025	44	11000
Benzo[a]pyrene	NR	> 100 – < 500	0.058	1.0	H400; H410 ⁱ⁾	N; R50/53 ⁱ⁾	10	10	2.5	0.4	17.2
Benzo[b]fluoranthene	NR	–	> water solubility	1.2	not classified ⁱ⁾	not classified ⁱ⁾	– ^{k)}	–	– ^{k)}	–	– ^{l)}
Benzo[k]fluoranthene	NR	–	0.00065	0.61	H400; H410 ⁱ⁾	N; R50/53 ⁱ⁾	1000	610	0.025	24.4	938
Dibenz[a,h]anthracene	NR	–	0.0018	0.17	H400; H410 ⁱ⁾	N; R50/53 ⁱ⁾	100	17	0.25	0.68	94.4
Benzo[ghi]perylene	NR	–	> water solubility	0.87	not classified	not classified	– ^{k)}	–	– ^{k)}	–	– ^{l)}
Indeno[1,2,3-cd]pyrene	NR	–	0.00027	0.91	H400; H410	N; R50/53	1000	910	0.025	36.4	3370
Total				9.2				4504		180	21280

^{a)} NR: not readily biodegradable; ^{b)} Based upon fish data only from Table 4.3.1 (–: no fish data available); ^{c)} Weight based percentage (data for binder pitch taken from Table 1.2.1); ^{d)} Classification of each individual PAH, based upon the criteria from Regulation EC 1272/2008 (EU, 2008b); ^{e)} This multiplying factor (M) is applied to give increased weight to highly toxic constituents of a mixture. The value of M depends on the lowest acute aquatic toxicity value (EU, 2008b); ^{f)} The weight based percentage of each individual PAH is multiplied by its specific multiplying factor to enable a classification of the mixture based on summation of classified components according to Regulation (EC) 1272/2008 (see text for details); ^{g)} The weight based percentage of each individual PAH is divided by its specific concentration limit (in %) to enable a classification of the mixture based on summation of classified components according to Directive 1999/45/EC (EU, 1999) (see text for details); ^{h)} The weight based percentage of each individual PAH is divided by its specific lowest acute aquatic toxicity value (E/LC₅₀ in mg/L) to enable calculation of an overall toxicity for the mixture (see text for details); ⁱ⁾ These compounds are classified in Annex VI of Regulation EC 1272/2008 as H400/H410 in Table 3.1 and as N;R50/53 in Table 3.2; ^{j)} The lowest acute aquatic toxicity value is >1 mg/L and thus no M-factor or concentration limit is applied; ^{k)} Acute toxicity reference value is higher than water solubility (and thus no M-factor or concentration limit); ^{l)} An absolute toxicity value could not be applied in this calculation.

JUSTIFICATION THAT ACTION IS REQUIRED ON A COMMUNITY-WIDE BASIS

1 CMR CLASSIFICATION

Harmonised classification and labelling for CMR and respiratory sensitisation is a Community-wide action under Article 36.1 (EU, 2008b). The proposal for classification as a human carcinogen (Category 1A carcinogen) is based on epidemiological data. The proposal for classification as Category 2 mutagen and Category 2 reproductive toxicant is based on the presence of impurities. No priority is normally given to such classification proposals. However, a search for safety data sheets containing a classification for CTPHT showed that most but not all companies used additional classifications besides the harmonised classification (including note H). Therefore, harmonised classification for mutagenicity and reproductive toxicity is proposed.

2 NON-CMR HEALTH CLASSIFICATION

The classification of CTPHT for several non-CMR hazard classes has been discussed by the TC-C&L and agreed. Such proposals now need a justification that action is required on a community-wide basis (article 36.3). Considering the CMR classification in category 1 and 2, such additional classifications have limited added value. Therefore, no classification is proposed for non-CMR health classes.

3 ENVIRONMENTAL CLASSIFICATION

The environmental classification of CTPHT has been discussed by the TC-C&L, but not concluded. The difficulties in the interpretation of the information regarding environmental hazards may result in different environmental classifications for CTPHT. Harmonisation of the environmental classification of this substance facilitates the safe use of the substance.

OTHER INFORMATION

This proposal for harmonized classification and labelling is mainly based on the data provided in the transitional dossier on CTPHT (Netherlands, 2008). Only on bioaccumulation properties additional data were used from the recent report by Bleeker & Verbruggen (2009).

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ANNEXES

ANNEX I: GLOSSARY

Carbonisation:	The destructive distillation of coal to produce coke and/or natural gas.
Coal Tar:	The condensation product of the destructive distillation of coal to produce coke and/or natural gas.
Coal Tar high temperature:	The condensation product of high-temperature (>700 °C) carbonisation of coal.
Coal Tar low temperature:	The condensation product of low-temperature (<700 °C) carbonisation of coal.
Coal Tar Oil:	Tar oils produced by the distillation of crude coal tar.
Coal Tar Pitch (CTP):	The solid fraction produced during the distillation of coal tars.
Coal Tar Pitch high temperature (CTPHT):	The solid fraction produced during the distillation of high temperature coal tars.
Coal Tar Pitch low temperature (CTPLT):	The solid fraction produced during the distillation of low temperature coal tars.
Coal Tar Pitch Volatiles (CTPV):	Volatiles released when coal tar pitch (CTP), coal tar or coal tar products are heated.
Coal Tar Pitch (Volatiles) CTP(Vs):	Coal tar pitch and coal tar pitch volatiles released when coal tar or coal tar products are heated.
Coal Tar Creosote:	Fractions or blends of coal tar oils, sometimes including coal tar pitch, that are used for timber preservation.
Condensate:	The product of condensation.
Condensation:	To condense a gas or vapour into a liquid by applying pressure, by cooling it down or both.
Distillate:	The product of distillation.
Distillation:	The extraction of the volatile components of a mixture by the condensation and collection of the vapours that are produced as the mixture is heated.

ANNEX II: MINUTES OF THE TCNES-MEETING OF 4-5 OCTOBER 2006

ECBI/13/07 Rev. 1

Ispra, 23 January 2007

Draft Summary Record**Technical Committee on Classification and Labelling of Dangerous Substances****Meeting on Health Effects of Existing Chemicals and General issues****Hotel Concorde, Arona, 4-5 October 2006**

C014	<i>Pitch, coal tar, high-temp. (NL)</i> <i>Issue for discussion: Complete classification proposal</i>	648-055-00-5 EC: 266-028-2 CAS: 65996-93-2	ECBI/47/06 and Add. 1 ECBI/87/06 Add. 1 (FR comments)
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Current Annex I classification: T; Carc. Cat.2; R45 Note H

Classification proposal: Xi; R41Xi; R43 Carc. Cat.1; R45 Muta. Cat.2; R46 Repro. Cat.2; R60–R61

Documents⁴:**ECBI/47/06** **NL, C&L proposal****ECBI/47/06Add. 1** **NL, Response to comments****ECBI/87/06 Add. 1** **F, Comments**

NL went through the proposal endpoint by endpoint saying that no classification was warranted for acute toxicity and skin irritation whereas Xi; R41 based on severe effects to the eye was warranted.

⁴ The proposal and comments documents are available at http://ecb.jrc.it/classlab/4706_NL_Coaltar-pitch.DOC, http://ecb.jrc.it/classlab/4706a1_NL_pitch_coal_tar.doc, and http://ecb.jrc.it/classlab/8706a1_F_comments.doc, respectively.

The TC C&L agreed to this part of the **NL** proposal.

In regard to skin sensitization R43 was proposed based on the content of B (a)P which was agreed by the **TC C&L**. After a brief discussion about splitting the entries based on B(a)P content it was agreed that a putative splitting could be done in the follow-up provided IND would support this committee with data of the individual batches. **The Group** agreed not to classify the substance for repeat-dose toxicity based on lack of data and to classify as Muta. Cat. 2 R46 based on its B(a)P content. The substance was also classified as Carc. Cat. 1 R45 based on its B(a)P content. Similarly R60-61 was applied for effects on fertility and development based on the B(a)P content.

Conclusion:

The TC C&L agreed to the following classification proposal: Carc. Cat. 1; R45 - Muta. Cat. 2; R46 -Repr. Cat. 2; R60-61 - Xi; R41 – R43, further Note H should be deleted. The labelling would be: Symbol: T; R-phrases: 45–46– 60–61–41–43 and S-phrases: 53-45.

Follow-up:

NL sent in comments, which, however, did not alter the conclusions taken at the meeting.

ANNEX III: SUMMARY OF HUMAN CARCINOGENICITY DATA

In the tables below relevant epidemiological data are presented per exposure scenario. The epidemiological publications summarised by IARC and HSE were not consulted individually.

Scenario 1: Production of CTP(ht) in coal tar distillation plants

In a review by the HSE (Armstrong *et al*, 2003) three cohort studies were identified, none of which contained data on exposure. The study by Hansen (1989) was not solely related to tar distillation but also to asphalt and roof felt processing. Statistically non-significant increased lung cancer risks were observed in all three studies and non-significant increased bladder cancer in two of the three studies. In one of the studies a non-significant reduced SMR for bladder cancer was observed. The studies are summarised in the table below. This table is mainly based on the review by Armstrong *et al* (2003).

Summary of the epidemiological studies on tar distillation (based on Armstrong *et al*, 2003)

Reference	Study population	Comparison group Reference population	Exposure groups observation period (of outcome) analysed by	Findings
Hansen (1989)	1320 Danish workers employed in the asphalt industry (tar distillation, asphalt and roof felt processing) for 10 years	unexposed Danish workers	exposed/non-exposed observation period: 1970-1980	non-significant increases in both lung and bladder cancer death. Standardised Mortality Ratio (SMR) men >45y: lung cancer: 143 (95% CI: 82-232) (16 cases) bladder cancer: 301 (95% CI: 98-703) (5 cases) SMR men > 45 y old + 5 y latency required: lung cancer: 152 (95%CI: 76-271) (11 cases) bladder cancer: 291 (95% CI: 60-851) (3 cases)
Swaen & Slagen (1997 cited in Armstrong <i>et al</i> , 2003)	907 tar distillery Dutch workers employed at least one half-year between January 1947 and January 1980.	national population	exposed/non-exposed observation period: 1947-1988	non-significant increased SMR for lung cancer: 118 (95%CI: 87-157) (48 cases). non-significant reduced SMR for bladder cancer : 55 (95%CI: 6-2001) (2 cases)
Maclaren & Hurley (1987 cited in Armstrong <i>et al</i> , 2003)	255 British tar distillery workers employed on 1 January 1967 31 December to 1983	regional and national population	exposed/non-exposed observation period:1967-1983	non-significant increases in both lung and bladder cancer deaths SMR lung cancer: 160 (p=0.08) (12 cases) SMR bladder cancer: 429 (p=0.03) (3 cases)

Scenario 2: Use as a binder for electrodes*Sub-scenario 2i: in the aluminum industry (studies on aluminum production workers)*

Several studies among aluminum production workers in Canadian, French, Italian, Norwegian, US and Russian industries have been published. These studies are summarised in the table below. This table is mainly based on the review of Ronneberg and Langmark (1992), complemented with information from the IARC (IARC, 1984, 1987) and HSE (HSE, 1993; Armstrong *et al*, 2003). Assuming that these reviews contain the most important issues with respect to the studies, the original data were not consulted, with the exception of five more recent publications not included in the reviews (Armstrong *et al*, 1994; Ronneberg & Andersen, 1995; Tremblay *et al*, 1995; Cullen *et al*, 1996; Ronneberg *et al*, 1999; Romundstad *et al*, 2000a; Romundstad *et al*, 2000b).

The lung and bladder have been the most commonly identified sites for excess cancer in populations of aluminum production workers. In Canadian studies dose-response relations were found for bladder and lung cancer. The Norwegian studies have shown inconsistent results. Excess risk of stomach, kidney, prostate, pancreas, lymphatic and haemopoietic cancer and leukaemia were noted in several studies among aluminum production workers.

The IARC concluded that there is sufficient evidence that certain exposures occurring during aluminum production cause cancer and that pitch volatiles have fairly consistently been suggested in epidemiological studies as being possible causative agents (IARC, 1987).

Summary of studies on aluminum production workers (based on IARC, 1984; HSE, 1993)

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Canadian studies				
Theriault <i>et al</i> (1984 cited in Ronneberg & Langmark, 1992)	85 cases of bladder cancer diagnosed between 1970 and 1979, currently or previously employed for at least one year in one of the five aluminum plants in Quebec (using both Soderberg and prebake pots)	225 controls, currently or previously employed for at least one year in one of the five aluminum plants in Quebec	four cumulative exposure categories (relative exposure intensity of the job (0.00, 0.25, 0.50, 0.75, or 1.00) multiplied by the number of years on the job) cases diagnosed between 1970 and 1979 matched by plant, year of birth, year of first employment, and employment period at the time that the case was diagnosed	Odds Ratio (OR) bladder cancer: low BaP years: 3.4 (44 cases) moderate BaP years: 6.8 (27 cases) high BaP years: 12.4 (8 cases) all workers: 4.5 (85 cases) OR bladder cancer: Soderberg workers: 2.3 (95% CI: 1.4-3.8) prebake potroom workers: 1.5 (95% CI: 0.9-2.6) carbon plant workers: 0.7 (95% CI: 0.4-1.6)

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Armstrong <i>et al</i> (1986)	see above	see above	estimates of time-weighted exposure levels ranged from 0.0-0.1 to 1.4-3.5 mg/m ³ BSM see above matched by plant, year of birth, year of first employment, employment period at the time that the case was diagnosed and smoking history	assuming a linear relationship between exposure and bladder cancer risk and a latency of at least 10 year: Relative Risk (RR) bladder cancer increased by 18 % (95% CI: 7-41) for each year's exposure to 1 mg/m ³ BSM RR bladder cancer increased by 23% (95% CI: 9-52) for each year's exposure to 10 µg/m ³ BaP
Gibbs <i>et al</i> (1985)	5406 male workers employed in 1950 at 2 Söderberg smelter plants in Quebec	population of the province	four cumulative exposure categories (relative exposure intensity of the job multiplied by number of years exposed) observation period: 1950-77 no information on smoking: death rates were calculated from age and sex specific rates of the province	Standardised Mortality Ratio (SMR) bladder cancer (# cases) unexposed, low, intermediate, high and all workers: 28(1), 61(3), 188(3), 667(6) and 161 (12) (p>0.05) SMR lung cancer (# cases) unexposed, low, intermediate, high and all workers: 102 (30), 97(42), 172 (27), 271 (32), 143 (101) (p<0.05) SMR lung cancer (# cases) ancer of stomach and oesophagus: 153 (50) (p<0.05) Hodgkin's disease: 179 (5) (p>0.05) leukemias: 112 (9/)(p>0.05) pancreas cancer: 105 (14) (p>0.05) larynx cancer: 131 (7) (p>0.05) brain cancer: 109 (8/)(p>0.05)
Armstrong <i>et al</i> (1994)	338 lung cancer cases, employed for at least one year between 1950 and 1970 in a manual job in an aluminum plant in Quebec (using both Söderberg and prebake pots).	1138 controls, randomly sampled from the entire cohort of workers employed for at least one year between 1950 and 1970 in an aluminum plant in Quebec.	cumulative exposure (estimated PAH level based on industrial hygiene data x job years) observation period: 1950-1988 adjusted for smoking	smoking-adjusted lung cancer rate ratios (RR) (95% Confidence interval (95% CI)) <1, 1-9, 10-19, 20-29 and ≥30 BSM mg/m ³ -years: 1.00, 1.15 (0.84-1.59), 2.25 (1.50-3.38), 1.90 (1.22-2.97) and 2.08 (1.30-3.33) smoking-adjusted lung cancer rate ratios (95% CI) <10, 10-99, 100-199, 200-299, ≥300 BaP µg/m ³ -years: 1.00, 1.48 (1.09-2.00), 2.23 (1.46-3.39), 2.10 (1.40-3.15) and 1.87 (1.05-3.33) The data were compatible with a linear relation with cumulative exposure (RR=1 + 0.031 mg/m ³ -years BSM, while a curved relation (RR=1 + 0.098 mg/m ³ -years BSM ^{0.7}) fitted somewhat better

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Tremblay <i>et al</i> (1995)	138 bladder cancer cases, from the same cohort as described above	414 controls, randomly sampled from the same cohort as described above	see above	<p>smoking-adjusted bladder cancer rate ratios (RR) (95% CI) <1, 1-9, 10-19, 20-29 and ≥ 30 BSM mg/m³-years: 1.00, 1.67 (0.89-3.16), 3.93 (1.85-8.49), 7.31 (3.56-14.99) and 5.18 (2.47-10.89).</p> <p>smoking-adjusted bladder cancer rate ratios (95% CI) <10, 10-99, 100-199, 200-299, ≥ 300 BaP $\mu\text{g}/\text{m}^3$-years: 1.00, 1.97 (1.10-3.51), 6.24 (3.00-12.97), 6.66 (3.43-12.99), 4.36 (2.10-9.17)</p> <p>The data were best described by a linear model using BaP cumulative exposure on a lag time before diagnosis of 10 years (RR=1 + 0.0166 $\mu\text{g}/\text{m}^3$-years BaP).</p>
<i>et al</i> (1991)	4213 men employed for at least 5 years between 1954 and 1985 in a Söderberg plant in British Columbia.	the population of the same province in Canada.	<p>four cumulative exposure categories (estimated BSM level based on recent plant monitoring x job years)</p> <p>observation period: 1970-1985</p>	<p>Standardised Incidence Ratio (SIR) bladder cancer (number of cases) unexposed, low, intermediate, high and all workers: 1.0 (4), 0.4 (1), 1.3 (2), 5.0 (9) (p<0.01) and 1.7 (16) (p<0.05)</p> <p>SIR lung cancer (number of cases) unexposed, low, intermediate, high and all workers: 0.7 (11), 1.0 (9), 1.1 (7), 1.3 (10) and 0.97 (37)</p> <p>SIR non-Hodgkin's lymphomas (number of cases) unexposed, low, intermediate, high and all workers: 0.4 (1), - (0), 2.6 (3), 2.3 (3) (p<0.05) and 1.1 (7)</p>
Norwegian studies				
Andersen, 1982 cited in Ronneberg and Langmark, 1992 and IARC, 1984a	7410 men working for at least 18 months prior to 1970 in 4 aluminum plants in Norway (using both Söderberg and prebake pots)	national population	<p>old plant workers/new plant workers/non-aluminum workers</p> <p>observation period: 1953-1970</p> <p>no information on smoking</p>	<p>Observed/Expected (O/E) lung cancer mortality all workers: 57/35.9 (p<0.05) old plant workers: 2.0 (27 cases) old plant workers >15 years employed: 2.1 (11 cases) old plant unhooded pots: 20/12.5</p> <p>O/E bladder cancer mortality all workers: 26/21.8 old plant workers: 18/10.7 old plant unhooded pots: 13/6.1 (p<0.05) new plant workers: 8/11.1</p> <p>O/E leukemia mortality all workers: 17/12.6 old plant workers: 9/5.9</p> <p>O/E kidney cancer mortality all workers: 18/14.8 O/E cancer of other sites all workers: 104/94.4</p>

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Ronneberg and Andersen, 1995	1137 men hired between 1922 and 1975, employed for at least 6 months at a time in a prebake aluminum smelter in Norway	national male population	semi-quantitative exposure estimates (from job exposure matrix) observation period: 1953-1991 smoking increased at the most only slightly the incidence of lung cancer and undetectably the incidence of bladder cancer	Standardised Incidence Ratio (SIR) (95% CI) workers <3 years employed cancer all sites: 1.5 (1.23-1.88), p<0.01 lung cancer: 2.65, p<0.01, 20 cases skin cancer: 3.09, p<0.05, 5 cases stomach cancer: 1.55, 9 cases prostate cancer: 1.55, 16 cases SIR (95% CI) workers ≥3 years employed cancer of all sites: 0.93 (0.77-1.11) bladder cancer: 1.58, 14 cases
Ronneberg <i>et al</i> , 1999	5908 workers of a Norwegian aluminum smelter (using both Söderberg and prebake pots)	national male population	cumulative exposure (exposure intensity (2-1700 µg/m ³ PAH x duration of each job held in the smelter) observation period: 1953-1993 no associations were observed between exposure to asbestos and lung cancer, between heat and kidney cancer, or between magnetic fields and cancer of the brain or lymphatic and haematopoietic tissue.	Standardised Incidence Ratio (SIR) short-term workers (95% CI) cancer all sites: 1.07 (0.94-1.12) lung cancer: 1.52 (1.09-2.06) pleural mesothelioma 3.86 (0.80-11.27) SIR production workers (95% CI) cancer all sites: 1.04 (0.94-1.16) malignant melanomas: 0.35 (0.10-0.90) cancer of the lip: 2.04 (0.93-3.87) cancer of the rectum: 1.41 (0.92-2.09) SIR maintenance workers (95% CI) cancer all sites: 1.18 (0.85-1.60) lung cancer: 2.11 (1.01-3.87) lymphatic & haematopoietic tissues cancer: 2.39 (0.96-4.92) bladder cancer: 2.00 (0.65-4.67) cancer of the prostate: 1.58 (0.72-3.00) a dose-response relation between cumulative PAH exposure attained 30 years before observation and an increased incidence of bladder and lip cancer was observed among production workers a dose-response relation between cumulative PAH exposure attained 30 years before observation and lung cancer was observed among maintenance workers

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Romundstad et al, 2000a	5627 workers employed for at least 6 months two Norwegian aluminum reduction plants	national male population	cumulative exposure (exposure intensity ($2 > 3000 \mu\text{g}/\text{m}^3 \text{PAH} \times$ duration of each job held in the smelter) observation period: 1954 or 1957-1995 adjusted for smoking, no associations were observed between exposure to asbestos and lung cancer or between magnetic fields and lymphatic and haematopoietic cancer.	Standardised Incidence Ratio (SIR) (95% CI) bladder cancer (30 year lag period): $50 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 1.29 (0.83-1.92) $50-500 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 1.04 (0.21-3.03) $500-2000 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 1.16 (0.32-2.97) >2000 $\mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 4.08 (1.32-9.51)SIR (95% CI) pancreatic cancer (10 year lag):$50 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 0.43 (0.05-1.57)$50-500 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 1.14 (0.14-4.11)>500 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 0.81 (0.81-3.36)SIR (95% CI) lung cancer (20 year lag period):$50 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 0.94 (0.60-1.14)$50-500 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 1.72 (0.92-2.95)$500-2000 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}$: 0.78 (0.34-1.54)>2000 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 0.28 (0.03-1.02)$
Romundstad, 2000b	11103 workers employed for more than 3 years in six Norwegian aluminum plants	national population and local county	cumulative exposure (exposure intensity ($0 - 3400 \mu\text{g}/\text{m}^3 \text{PAH} \times$ duration of each job held in the smelter) observation period: 1953-1996 subanalysis of 3 plants with adjustment for smoking, a weak association was found between exposure to fluoride and bladder cancer, little evidence for association of fluorides with kidney, pancreas or lung cancer.	Rate ratio (95% CI) for bladder cancer (30 year lag time): 0 $\mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1 (ref)0-499 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.0 (0.7-1.9)500-1999 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.3 (0.8-2.0)> 2000 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 2.0 (1.1-2.8)Rate ratio (95% CI) for lung cancer (30 year lag time):0 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.0 (ref)0-499 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.0 (0.6-1.6)500-1999 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.1 (0.7-1.7)> 2000 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 0.4 (0.1-1.0)Rate ratio (95% CI) for pancreatic cancer (30 year lag time):0 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.0 (ref)0-499 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.0 (0.4-2.7)500-1999 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.4 (0.6-3.3)> 2000 \mu\text{g}/\text{m}^3 \text{PAH}\cdot\text{y}</math>: 1.5 (0.5-4.6)$

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
US studies				
Milham, 1979 cited in HSE, 1993 and Armstrong <i>et al</i> , 2003	2103 workers employed for 3 years or more including at least 1 year between 1946 and 1962 in a Washington state prebake plant	US national population	exposed/non-exposed observation period: 1946-1976 death rates by age, sex and year-specific mortality rate no information on smoking	Standardised Mortality Ratio (SMR) all workers: cancer of all causes: 0.86 respiratory cancer: 1.17 (O/E: 35/29.8, not stat. significant) pancreas cancer: 1.80 (O/E: 9/5, not stat. significant) prostate cancer: 1.62 (O/E: 8/5, not stat. significant) lymphatic & haemopoietic cancer: 1.84 (O/E:7/2.2, p<0.05) emphysema: 2.04 (O/E: 14/6.9, p<0.05) SMR exposed workers: respiratory cancer: 1..29 (O/E: 16/12.4, not statistically significant) lymphosarcomareticulosarcoma: 6.43 (O/E: 6/0.9, p<0.05) emphysema: 2.12 (O/E: 6/2.8, p<0.05) non-neoplastic respiratory disease: 1.73, (O/E: 17/9.9, p<0.05) non-exposed workers SMR cancer: respiratory cancer: 1.09 (O/E: 19/17.4) (not statistically significant) pancreas cancer: 2.38 (O/E: 7/3, p<0.05) being brain neoplasms: 6.75 (O/E: 5/0.7, p<0.05)
Rockette and Arena, 1983 cited in Ronneberg and Langmark, 1992 and Armstrong <i>et al</i> , 2003	21,829 men who were employed in 14 aluminum plants for at least 5 years between 1949 and 1977 (using both Söderberg and prebake pots)	?national population	length of employment and work area observation period 1950-1977	Observed/Expected Ratios (number of deaths) mortality prebake plant workers: all causes: 0.91 (2433) benign and unspecified neoplasms: 14/7.2 ≥15 years employed: lung cancer: 1.0 (30) (not stat. sign.) pancreas cancer : 2.2 (12 deaths) (p<0.05) kidney cancer: 19/13 (not stat. sign.), leukaemia: 1.7 to 2.0 fold increase ≥ 25 years employed in caron area: lung cancer: 3.75 (6) O/E Ratio (number of death) Söderberg plant workers all causes: 0.79 (923) Hodgkin's disease: 1.8 (6) Stomach cancer: 1.1 (35) <15 years employed: leukaemia: 2.5 (9 deaths) (p<0.05) bladder cancer: 1.0 (2) (not stat. sign.) lung cancer: 0.8 (23) (not stat. sign.) ≥15 years employed: bladder cancer: 2.0 (6) (not stat. sign.) lung cancer: 1.0 (19) (not stat. sign.) bladder, and pancreatic cancer: 1.7 to 2.0 fold increase

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Enterline, 1982 cited in HSE, 1993	deaths of male US aluminum workers from one company during the period 1947 to 1959 (based on insurance company records)	US population (same age and time periods and with 1950-1958 group life insurance policy holders in industries without rate hazards)	aluminum workers/non-aluminum workers observation period: 1946-1953	No non-accidental excess mortality was reported for the aluminum workers. Standardised Mortality Ratio (SMR) lung cancer: 0.79 compared with US males. Results were difficult to interpret because of limited data.
Equitable environmental Health Incl., 1977 cited in HSE, 1993	23033 men working 5 years or more during the period 1946 to 1973	US population	aluminum workers/non-aluminum workers observation period: 1946-1977	No excess respiratory cancer mortality was found when comparing mortality with that of US males. Relative Risk for Søderberg potroom workers employed ≥ 30 years: 2.2. Results were difficult to interpret because job histories were not closely defined and a lost to follow-up of 10%.
Cullen et al, 1996	25 cases of pituitary adenomas among employees of all US factories of one company diagnosed between 1989 and 1994	125 controls, randomly selected from the unified health insurance data base	technology, job type and duration of employment observation period: 1989-1994 mean age, sex distribution, duration of employment, ratio of active to retired workers were comparable in cases and controls	No strong evidence was found for increased risk of pituitary adenoma among aluminum workers. Results were difficult to interpret because of the relative crude exposure classification scheme used and possible selection bias (controls had to have filed at least one health insurance claim in 1992)
France study				
Mur <i>et al</i> , 1987 cited in HSE, 1993 and Armstrong <i>et al</i> , 2003	6455 workers employed for at least one year in the period 1950-1976 in one of the 11 aluminum plants in France (both Søderberg and prebake plants)	national population	aluminum workers/non-aluminum workers observation period: 1950-1976	Increased mortality for cancer of the lung, bladder, pancreas, liver and brain, and for leukaemia (not statistically significant). Based on very few deaths, the excess of lung cancer deaths appeared not to be associated with specific work areas and limited to workers with a short duration of employment. Results were difficult to interpret because of lack of information accompanying the Standardised Mortality Ratio (SMR) values, a lost to follow-up of 29%, uncertainties in occupational histories, and the allocation of numbers to each cause of deaths.

Reference	Study population	Comparison group Reference population	Exposure groups Observation period (of outcome) Correction for confounding and effect modification	Findings
Moulin <i>et al</i> , 2000	2133 workers employed for at least 1 year between 1950 and 1994 in a French aluminum plant	regional population	job type and duration of employment observation period: 1968-1994 adjusted for gender, age and calendertime	Standardised Mortality Ratio (SMR) (95% confidence interval) workers all causes of death: 0.81 (0.72-0.90) lung cancer: 0.63 (0.38-0.98) bladder cancer: 1.77 (0.71-3.64) psychoses and neuro-degenerative diseases: 2.39 (0.88-2.51) workshops where PAH exposure was likely: all causes of death: 0.84 (0.74-0.95) lung cancer: 0.69 (0.39-1.15) bladder cancer: 2.15 (0.79-4.68) psychoses and neuro-degenerative diseases: 2.39 (0.88-5.21)
Italian study				
Giovannazie and D'Andrea, 1981 cited in Ronneberg and Langmark, 1992	494 men employed between 1965 and 1979 in a Söderberg reduction plant in Italy	regional population	potroom workers/ regional population observation period: 1965-1979	increased mortality in potroom workers, due to cardiovascular diseases, liver cirrhosis and lung cancer.
USSR studies				
Konstantinov and Kuz'minykh, 1971 cited in IARC, 1984a	workers of two aluminum production plants in the USSR	regional population	SoderbergSöderberg workers/regional population observation period: 1956-1966	Excesses of all cancers, lung cancer and skin cancer was observed Results were difficult to interpret because of limited information and no raw data.
Konstantinov <i>et al</i> , 1974 cited in IARC, 1984a	potroom workers in three aluminum plants	regional population	SoderbergSöderberg workers/ regional population	Results were difficult to interpret because of the absence of information both on the study population and the reference population.

Sub-scenario 2ii: Use as a binder and impregnation of electrodes

In a review by Armstrong et al (2003) three papers (reporting 3 cohort studies and one case control study) were identified on carbon workers. One paper from China includes workers of six carbon plants (not further specified) and one aluminum reduction plant (working potroom and carbon department). Although part of this cohort falls under sub- scenario 2i, the study is described under this scenario, assuming most workers were involved in the use of CTPHT as a binder and impregnation of electrodes. The other two papers describe workers in carbon (graphite) electrode plants in Italy and France. The studies are summarised in the table below. This table is mainly based on the review by Armstrong et al (2003).

In one of the available studies on the use of CTPHT as a binder and impregnation of electrodes, a statistically significant increased lung cancer risk was observed (Liu *et al*, 1997). In the other studies non-significant increases in lung and bladder cancer risks were observed (Moulin *et al*, 1989; Donato *et al*, 2000).

Summary of the epidemiological studies on the use of CTPHT as a binder and impregnation of electrodes (based on Armstrong *et al*, 2003)

Reference	Study population	Comparison group Reference population	Exposure groups observation period (of outcome) correction for confounding and effect modification	Findings
Liu et al, 1997	6635 chinese carbon workers employed for more than 15 years in seven factories including one aluminum plant and 6 carbon plants	11470 other steel workers in employed in rough rolling mills	4 exposure categories observation period: 1971-1985 smokers and non-smokers were also analysed separately	A significant positive relation was found for lung cancer mortality, with a Standardised Mortality Ratio (SMR) of 2.16 (statistically significant, $p < 0.01$) SMR lung cancer non-smokers: 3.00 ($p < 0.01$) SMR lung cancer high exposure: 5.34 ($p < 0.01$)
Donato et al, 2000	1006 workers employed for at least one year between 1945 and 1966 in a carbon graphite electrode plant in Italy	national population	duration of employment observation period: 1955-1966	Non significant increased risks of lung and bladder cancer
Moulin et al, 1989	1302 carbon workers employed at plant A in 1975 and 1115 employed at plant B in 1957	local and national population	duration of exposure observation period plant A: 1975-1985 observation period plant B: 1957-1984 smoking adjusted for plant A only	Plant A: Non significant Standardised Incidence Ratios (SIRs) below 1 for lung and bladder cancer Plant B Non significant Standardised Mortality Ratio (SMR) above 1 for lung and bladder cancer.

Occupational Scenario 3: Use as a binder in Asphalt industry and in Roofing

Several studies among asphalt workers have been published. An overview of these studies is given in the table below. This overview is mainly based on the review of Partanen & Boffetta (1994), who examined and combined the results of 20 epidemiologic studies conducted on asphalt workers and roofers, complemented with information from the IARC (1985) and more recent publications by Stern (2000), Boffetta *et al* (2003), Randem *et al* (2004) and Armstrong *et al* (2003). Assuming that the review of Partanen and Boffetta, the IARC document and the meta-analysis of Armstrong *et al* (2003; 2004) contain the most important issues with respect to the evaluated epidemiological studies, original data of these studies were not consulted.

Most of the studies evaluated by Partanen and Boffetta (1994), the IARC (1985) and Armstrong *et al* (2003) have limitations, with respect to power, lack of exposure data, or failure to control for confounding. In roofers, some studies with smoking-adjusted results suggest an excess lung cancer risk unexplained by tobacco smoking. Since roofers work with hot pitch they have probably been exposed to great amounts of carcinogenic PAHs. However, the data were insufficient to specifically address the carcinogenicity of the different exposures encountered in roofing (and other asphalt workers), including coal tar derived exposures.

Summary of studies on asphalt workers and roofers (based on Partanen & Boffetta, 1994)

Reference	Study population	Exposure groups observation period (for outcome) correction for confounding and effect modification	Findings
Cohort studies			
Hammond <i>et al</i> , 1976 cited in Partanen and Boffetta, 1994 and Armstrong <i>et al</i> , 2003	5339 US roofers and waterprooferers	> 20 years of employment 12 years of follow-up no smoking data available.	Standardised Mortality Ratio (SMR) all cancer: 1.5 95% CI (1.3-1.6); 315 death SMR lung cancer: 1.6 (1.3-1.9); 99 deaths SMR cancer of buccal cavity, pharynx, larynx, and esophagus: 2.0 (1.3-2.8); 31 deaths SMR stomach cancer: 1.7 (1.1-2.5); 24 deaths Lung cancer SMRs increased with year since joining the union.
Menck <i>et al</i> , 1976 cited in Partanen and Boffetta, 1994	2000 US roofers	roofers vs other occupations observation period: 1968-1973	Standardised Mortality Ratio (SMR) lung cancer: 5.0 (2.5-8.9); 11 deaths
Maizlish <i>et al</i> , 1988 cited in Partanen and Boffetta, 1994	1570 deaths	California Highway Maintenance Workers (HMW) observation period: 1970-1983	Proportional Mortality Ratio (PMR) cancer of digestive organs: 1.5 (1.0-2.2); 25 deaths PMR stomach cancer: 2.2 (0.8-5.0); 6 deaths PMR prostate cancer: 2.3 (0.9-4.7); 7 deaths
Povarov <i>et al</i> , 1988 cited in Partanen and Boffetta, 1994	workers employed for at least 3 years during 1974-1984 in the production of hot-lay asphalt concrete in Estonia	hot-lay asphalt production workers (BaP concentrations 0.2-0.7 µg/100 m ³ were reported on worksites) observation period: not available, 10369 person years	all workers Standardised Mortality Ratio (SMR) all cancers: 1.5 (0.9-2.4); 17 deaths workers 40-64 years of age SMR lung cancer: 2.1, p < 0.05

Reference	Study population	Exposure groups observation period (for outcome) correction for confounding and effect modification	Findings
Bender et al, 1989 cited in Partanen and Boffetta, 1994	4849 US HMW with at least 1 year of employment in Minnesota	HMW (workers did not use coal tar products for 50 years in Minnesota) observation period: 1945-1984, 96567 person-years of follow-up analysed by urban/rural residence, age at death, calendar year at death, age started work, year started work, years worked, and latency	Standardised Mortality Ratio (SMR) leukemia: 4.3 (1.7-8.8); 7 deaths SMR respiratory cancer: 0.7 > 40 years of employment SMR kidney and bladder cancer: 2.9 (1.2-6.0); 7 deaths SMR mouth & pharynx cancer: 11.1 (1.3-40.1); 2 deaths workers starting work 1955-1964 at age > 40 years SMR prostatic cancer: 3.0 (1.5-5.4) 11 deaths
Hansen, 1989; Hansen et al, 1991 cited in Partanen and Boffetta, 1994 and Armstrong et al, 2003	679 Danish mastic asphalt workers	mastic asphalt workers (average asphalt fume concentration, weighted of 12-months, was estimated to be close to the Danish TWA of 5 mg/m ³) observation period: 1959-1986, 6692 person-years of follow-up	Minimum latency 15-20 years Standardised Incidence Ratio (SIR) all cancer: 2.0 (1.6-2.5); 74 cases SIR cancer of oral cavity: 11.1 (1.45-40.1); 2 cases SIR cancer of esophagus: 7.0 (1.4-20.4); 3 cases SIR cancer of the rectum: 3.2 (1.3-6.6); 7 cases SIR cancer of the lung: 3.4 (2.3-5.0) 27 cases excess of respiratory and digestive cancers persisted after correction for urbanisation and smoking habits Standardised Mortality Ratio (SMR) all cancers: 2.3 (1.7-2.9); 62 deaths SMR lung cancer: 2.9 (1.9-4.3); 25 deaths SMR non-pulmonary cancers: 2.0 (1.4-2.8); 37 deaths
Engholm et al, 1991 cited in Partanen and Boffetta, 1994	2572 Swedish pavers and 704 roofers	pavers and roofers 11.5 years of follow-up	pavers: Standardised Incidence Ratio (SIR) stomach cancer: 2.1 (0.9-4.1); 8 cases SIR lung cancer: 1.2 (0.5-2.4); 8 cases roofers: SIR lung cancer: 3.6 (0.6-8.0); 4 cases
Hrubec et al., 1992 cited in Partanen and Boffetta, 1994	52 deaths	roofers and slaters (US) observation period: 1954-1980 adjusted for smoking	Standardised Mortality Ratio (SMR) respiratory cancer: 3.0 (1.2-7.7); 4 deaths SMR multiple myeloma: 8.0; 1 death
Minder et al, 1992	National population of Switzerland	roofers observation period: 1979-1982	Proportional Mortality Ratio (PMR) mouth and pharynx cancer: 3.3 (1.2-7.2); 5 deaths
Pukkala et al, 1992 cited in Partanen and Boffetta, 1994	National population of Finland	Finish asphalt workers observation period: 1971-1985 adjusted for social class and age	Standardised Incidence Ratio (SIR) lung cancer: 2.7 (1.6-4.1) 20 cases

Reference	Study population	Exposure groups observation period (for outcome) correction for confounding and effect modification	Findings
Milham et al, 1993 cited in Partanen and Boffetta, 1994	7266 deaths	US Graders, pavers, operators and excavators (Washington State) observation period: 1950-1989 co-exposure to crystalline silica was suggested	Proportional Mortality Ratio (PMR) lung cancer: 1.2 (1.1-1.3); 558 deaths
Swaen and Slagen (1997 cited in Armstrong et al, 2003)	866 Dutch roofers employed at least one half-year between January 1947 and January 1980.	national population observation period: 1947-1980	Mortality from cancer of the lungs and trachea was higher than expected, but not statistically significant. In addition the roofers had experienced an excess mortality rate from external causes.
Boffetta et al, 2003;	29820 European male workers exposed to bitumen in road paving, asphalt mixing and roofing and 32245 ground and building construction workers and 17757 workers not classifiable as bitumen workers from Denmark, Finland, France, Germany, Israel, the Netherlands, Norway and Sweden	national population observation period: 1953-2000 confounding from exposure to carcinogens in other industries, tobacco smoking, and other lifestyle factors cannot be ruled out.	Standardised Mortality Ratio (SMR) (95% confidence interval) total cohort: all causes: 0.92 (0.90-0.94) workers in road paving, asphalt mixing and roofing: lung cancer: 1.17 (1.04-1.30) increased risk of cancer of the head and neck ground and building construction workers: lung cancer: 1.01 (0.89-1.15)
Boffetta Boffetta et al, 2004	22362 male asphalt workers employed for more than one season in jobs entailing exposure to bitumen in Denmark, Finland, Norway and Sweden	national population confounding from exposure to carcinogens in other industries, tobacco smoking, and other lifestyle factors cannot be ruled out.	Standardised Incidence Ratio (SIR) (95% confidence interval) all cancers: 0.89 (0.86-0.94) lung cancer: 1.21 (1.07-1.36) no trend according to time since first employment relative risk (95% confidence interval) relative risk (95% confidence interval) > 30 year vs 1-14 years employed bladder cancer: 1.85 (0.90-3.78)
case-control studies			
Schoenberg et al, 1987 cited in Partanen and Boffetta, 1994	736 lung cancer cases + 900 population controls (US)	roofers and slaters (ever employed) case ascertainment: 1967-1976 adjusted for smoking	Odds Ratio (OR) lung cancer: 1.7 (0.7-4.4); 13 cases

Reference	Study population	Exposure groups observation period (for outcome) correction for confounding and effect modification	Findings
Vineis et al, 1988 cited in Partanen and Boffetta, 1994	2973 lung cancer cases + 3210 controls (data from five US case-control studies)	roofers and asphalt workers case ascertainment: 1974-1981 adjusted for age, birth cohort and smoking	OR lung cancer: 1.4 (0.9-2.3); 45 cases
Zahm et al, 1989 cited in Partanen and Boffetta, 1994	4431 lung cancer cases + 11326 controls (Missouri, US)	occupation at the time of diagnosis case ascertainment: 1980-1985 adjusted for age, cigarette smoking and time of diagnosis	Roofers: Odds Ratio (OR) all lung cancers: 2.1 (0.6-8.2); 6 cases OR squamous cell carcinoma lung: 2.6 (0.5-12.7); 3 cases OR adenocarcinoma lung: 1.5 (0.1-13.3); 1 case OR other/mixed lung cancers: 2.9 (0.4-18.0) 2 cases Pavers, surfacers and operators: OR all lung cancers: 0.9 (0.6-1.5); 32 cases OR squamous cell carcinoma lung: 1.1 (0.6-2.1); 14 cases OR small cell carcinoma lung: 0.7 (0.2-1.9); 5 cases OR adenocarcinoma lung: 0.5 (0.2-1.5); 4 case OR other/mixed lung cancers: 1.2 (0.5-2.5) 9 cases
Morabia et al, 1992 cited in Partanen and Boffetta, 1994	1793 lung cancer cases + 3228 controls (US)	roofers and slaters case ascertainment: 1980-1989 adjusted for age, gender, geographical area, questionnaire version, and cigarette smoking	Odds Ratio (OR) lung cancer: 2.1 (0.7-6.2); 7 cases
Risch et al, 1988 cited in Partanen and Boffetta, 1994	781 bladder cancer cases and 781 matched population controls (Alberta and Southern Ontario, Canada)	exposure to "tar, asphalt" case ascertainment: 1979-1982 matched on birth year and area of residence	Odds Ratio (OR) bladder cancer at least 6 months of exposure: 1.4 (0.8-2.7) 8-28 years exposure before diagnose: 3.1 (1.2-9.7) any 10-year exposure period: 2.0 (1.1-5.0)
Mommsen et al, 1982; 1984 cited in Partanen and Boffetta, 1994	121 bladder cancer patients and 259 population controls (Danish)	work with "kerosene or asphalt" (based on job titles) case ascertainment: 1977-1979	Odds Ratio (OR) 3.1 (0.9-11.0); 9 cases somewhat attenuated after adjustment for additional occupational factors
Jensen et al, 1988 cited in Partanen and Boffetta, 1994	96 cases of renal pelvis tumors + 294 matched hospital controls in Danish Baltic island of Sjaelland	asphalt and tar workers case ascertainment: 1979-1982 adjusted for lifetime tobacco consumption	Odds Ratio renal pelvis and ureter cancer: 5.5 (1.6-19.6) 9 cases

Reference	Study population	Exposure groups observation period (for outcome) correction for confounding and effect modification	Findings
Siemiatycki et al, 1991 cited in Partanen and Boffetta, 1994	4576 hospital-based cases and controls in Montreal	any versus substantial exposure to asphalt case ascertainment: 1979-1981 adjusted for age, family income, tobacco smoking, ethnic origin, alcohol consumption (lung cancer for ethnic origin, alcohol index and respondent –proxy/self-)	substantial exposure Odds Ratio (OR) stomach cancer: 2.0 (1.0-4.1) 7 cases OR colon cancer: 1.0 OR lung cancer: 0.7, 13 cases French Canadians: OR prostate cancer: 3.0 (1.0-9.0) 5 cases OR bladder cancer: 2.2 (1.0-4.9) 8 cases OR non-Hodgkin's lymphoma: 1.5 (0.4-5.1) 2 cases any asphalt exposure OR colon cancer: 1.6 (1.1-2.5) 22 cases OR lung cancerL 0.9, 30 cases French Canadians: OR non-Hodgkin's lymphoma: 2.0 (1.0-4.0) 7 cases OR bladder cancer: 2.2 (1.0-4.9) 8 cases

ANNEX IV: SUMMARY OF AQUATIC TOXICITY DATA

Acute toxicity of naphthalene (CASnr. 91-20-3) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /l)	Exp time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Chlorella vulgaris</i>		N	S	-	am	-	-	24 h	EC50	cell number	33000	Kauss & Hutchinson, 1975
<i>Chlorella vulgaris</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	19000	Hutchinson et al, 1980
<i>Chlamydomonas angulosa</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	9600	Hutchinson et al, 1980
<i>Nitzschia palea</i>		Y	S	-	am	7.6	-	4 h	EC50	assimilation ¹⁴ C	2820	Millemann et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	-	-	-	-	-	14 d	EC50	standing crop	25000	Gaur, 1988
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	7.6	-	4 h	EC50	assimilation ¹⁴ C	2960	Millemann et al, 1984
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC50	growth, area under the curve	68210	Djomo et al, 2004
Mollusca												
<i>Physa gyrina</i>	7.5 mm, 0.057 g	Y	Sc	-	nw	7.8	140	48 h	LC50	mortality	5020	Millemann et al, 1984
Crustacea												
<i>Daphnia magna</i>		Y	S	-	tw	8.0-8.6	-	48 h	LC50	mortality	4100	Crider et al, 1982
<i>Daphnia magna</i>	1.5 mm, 4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	17000	Bobra et al, 1983
<i>Daphnia magna</i>	<24 h	N	Sc	≥97%	rw	-	-	48 h	EC50	immobility	2194	Munoz & Tarazona, 1993
<i>Daphnia magna</i>	adult, mixed age	N	S	-	nw	7.6±0.2	134±16	48 h	LC50	mortality	22600	Eastmond et al, 1984
<i>Daphnia magna</i>	24 h	Y	Sc	-	nw	7.8	140	48 h	EC50	immobility	2160	Millemann et al, 1984
<i>Daphnia magna</i>	4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	4700	Abernethy et al, 1986
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	1664	Bisson et al, 2000
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 + 2 +1 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	<24 h	N	Sc	-	rw	7.4-9.4	173	48 h	LC50	mortality	8600	LeBlanc, 1980
<i>Daphnia magna</i>	<24 h	N	Sc	-	rw	7.4-9.4	173	48 h	NOEC	mortality	600	LeBlanc, 1980
<i>Daphnia magna</i>		-	-	-	-	-	-	48 h	LC50	mortality	24100	Parkhurst et al, 1981
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	nw	-	160-180	48 h	EC50	immobility	4663	Smith et al, 1988
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	rw	-	160-180	48 h	EC10	immobility	1900	Smith et al, 1988
<i>Daphnia pulex</i>	1.9-2.1 mm	N	Sc	-	nw	7.5	-	96 h	LC50	mortality	1000	Trucco et al, 1983
<i>Daphnia pulex</i>	neonates	Y	S	-	tw	6.8-7.5	43-48	48 h	LC50	mortality	3400	Geiger & Buikema, 1981, 1982
<i>Gammarus minus</i>	adult	Y	Sc	-	nw	-	-	48 h	LC50	mortality	3930	Millemann et al, 1984
Insecta												
<i>Chironomus attenuatus</i>	4 th instar	N	S	-	tw	7.9-8.3	-	24 h	LC50	mortality	13000	Darville & Wilhm, 1984
<i>Chironomus tentans</i>	4 th instar	Y	Sc	-	nw	7.8	140	48 h	EC50	immobility	2810	Millemann et al, 1984
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	>99%	DSW	-	-	96 h	LC50	mortality	600	Bleeker et al, 2003
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	>99%	DSW	-	-	96 h	LC50	mortality	650	Bleeker et al, 2003
<i>Somatochlora cingulata</i>		N	-	-	nw	-	-	96 h	LC50	mortality	1000-2500	Correa & Coler, 1983
<i>Tanytarsus dissimilis</i>	4 th instar	N	S	-	tw	7.9-8.3	-	48 h	LC50	mortality	16000	Darville & Wilhm, 1984
Pisces												
<i>Abramis brama</i>		N	S	technical	nw	7.8-8.1	-	24 h	LC50	mortality	10000	Frumin et al, 1992
<i>Oncorhynchus kisutch</i>	fry 1 g	Y	CF	-	nw	-	-	96 h	LC50	mortality	2100	Moles et al, 1981
<i>Oncorhynchus kisutch</i>	fry 0.3 g	Y	CF	-	nw	-	-	96 h	LC50	mortality	3220	Moles, 1980
<i>Oncorhynchus mykiss</i>	fry, 13-21 d	N	S	≥95%	nw	-	160-190	96 h	LC50	mortality	1800	Edsall, 1991
<i>Oncorhynchus mykiss</i>	fry, 13-21 d	N	S	≥95%	nw	-	160-190	96 h	LC50	mortality	6100	Edsall, 1991
<i>Oncorhynchus mykiss</i>	fry, 13-21 d	N	S	≥95%	nw	-	160-190	96 h	LC50	mortality	2600	Edsall, 1991
<i>Oncorhynchus mykiss</i>	fry, 13-21 d	N	S	≥95%	nw	-	160-190	96 h	LC50	mortality	4400	Edsall, 1991
<i>Oncorhynchus mykiss</i>	fry, 13-21 d	N	S	≥95%	nw	-	160-190	96 h	LC50	mortality	5500	Edsall, 1991
<i>Oreochromis mossambicus</i>		N	R	-	-	-	-	96 h	LC50	mortality	7900	Dangé, 1986
<i>Oreochromis mossambicus</i>	4-5 mo	N	S	technical	nw	7.8-8.1	-	24 h	LC50	mortality	22000	Frumin et al, 1992
<i>Pimephales promelas</i>	34 d	Y	CF	98%	-	7.4	44	96 h	LC50	mortality	6140	Broderius et al, 1995; Geiger et al, 1985
<i>Pimephales promelas</i>	31-35 d	Y	CF	98%	nw	6.9-7.7	44.9	96 h	LC50	mortality	6080	Holcombe et al, 1984
<i>Pimephales promelas</i>	1-2 mo, 0.27 g, 28 mm	Y	Sc	-	nw	7.8	140	96 h	LC50	mortality	1990	Millemann et al, 1984
<i>Pimephales promelas</i>	0.9 g, 46 mm	Y	CF	-	nw	7.9-8.0	535-596	96 h	LC50	mortality	7900	DeGraeve et al, 1982

Chronic toxicity of naphthalene to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /l)	Exp time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Chlorella vulgaris</i>		N	S	-	am	-	-	24 h	EC10	cell number	3900	Kauss & Hutchinson, 1975
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	>4270	Bisson et al, 2000
<i>Pseudokirchneriella subcapitata</i>		N	-	-	-	-	-	14 d	EC10	standing crop	13000	Gaur, 1988
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC10	growth, area under the curve	7270	Djomo et al, 2004
Macrophyta												
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	32000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1600	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	280	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	1000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC1-3	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-13	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC2-21	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC2-42	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC4-7	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC3-13	chlorophyll content	2000	Ren et al, 1994
Cyanophyta												
<i>Anabaena flos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	≥25000	Bastian & Toetz, 1982
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	514	Bisson et al, 2000
<i>Daphnia pulex</i>	<24 h	Y	R	-	tw	6.9-7.5	41-50	lifetime	NOEC	reproduction, growth	≥600	Geiger & Buikema, 1982
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	10 d	LC50	mortality	2720	Lee et al, 2001
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	14 d	LC50	mortality	2130	Lee et al, 2001
Insecta												
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juvenile	Y	R	>98%	nw	8.1-8.3	165-250	5 d	EC50	immobility	1587	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juvenile	Y	R	>98%	nw	8.1-8.3	165-250	10 d	EC50	immobility	1141	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juvenile	Y	R	>98%	nw	8.1-8.3	165-250	10 d	LC50	mortality	1757	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juvenile	Y	R	>98%	nw	8.1-8.3	165-250	28 d	LC50	mortality	1266	Landrum et al, 2003
<i>Tanytarsus dissimilis</i>	4 th instar	Y	CF	-	tw	7.7-8.0	132-190	life-cycle	NOEC	egg hatching, adult emergence	<500	Darville & Wilhm, 1984
Pisces												
<i>Micropterus salmoides</i>	eggs 2-4d post spawn	Y	CF	-	am	7.41-8.1	86.8-116.3	7 d incl. 4 post-hatch	LC50	mortality	680	Milleman et al, 1984
<i>Micropterus salmoides</i>	eggs 2-4d post spawn	Y	CF	-	am	7.4-8.1	86.8-116.3	7 d incl. 4 post-hatch	LC50	mortality	510	Black et al, 1983
<i>Micropterus salmoides</i>	eggs 2-4d post spawn	Y	CF	-	am	7.4-8.1	86.8-116.3	7 d incl. 4 post-hatch	LC10	mortality	37	Black et al, 1983
<i>Oncorhynchus kisutch</i>	fry, 1 g	Y	CF	-	nw	-	-	40 d	NOEC	weight/length (increase)	370	Moles et al, 1981
<i>Oncorhynchus kisutch</i>	fry, 1 g	Y	CF	-	nw	-	-	40 d	EC50	weight (wet/dry; increase)	770	Moles et al, 1981
<i>Oncorhynchus kisutch</i>	fry, 1 g	Y	CF	-	nw	-	-	40 d	EC10	weight (wet/dry; increase)	520	Moles et al, 1981
<i>Oncorhynchus kisutch</i>	fry, 1 g	Y	CF	-	nw	-	-	40 d	EC50	length (increase)	840	Moles et al, 1981
<i>Oncorhynchus kisutch</i>	fry, 1 g	Y	CF	-	nw	-	-	40 d	EC10	length (increase)	460	Moles et al, 1981
<i>Oncorhynchus mykiss</i>	eggs 20 min post fertilization	Y	CF	-	nw	7.41-8.10	86.8-116.3	27 d incl. 4 post-hatch	LC50	mortality	120	Milleman et al, 1984
<i>Oncorhynchus mykiss</i>	eggs 20 min post fertilization	Y	CF	-	nw	7.4-8.1	86.8-116.3	27 d incl. 4 post-hatch	LC50	mortality	110	Black et al, 1983
<i>Oncorhynchus mykiss</i>	eggs 20 min post fertilization	Y	CF	-	nw	7.4-8.1	86.8-116.3	27 d incl. 4 post-hatch	LC10	mortality	20	Black et al, 1983
<i>Oncorhynchus mykiss</i>	3.9 g, 93 mm embryo/larvae	Y	CF	-	nw	7.9-8.0	535-596	30 d	LC50	mortality	1600	DeGraeve et al, 1982
<i>Pimephales promelas</i>	embryo/larvae	Y	CF	-	nw	7.9-8.0	535-596	30 d	NOEC	length, weight	450	DeGraeve et al, 1982
<i>Pimephales promelas</i>	embryo/larvae	Y	CF	-	nw	7.9-8.0	535-596	30 d	NOEC	hatchability	450	DeGraeve et al, 1982
<i>Pimephales promelas</i>	embryo/larvae	Y	CF	-	nw	7.9-8.0	535-596	30 d	EC10	hatchability	2900	DeGraeve et al, 1982
<i>Pimephales promelas</i>	embryo/larvae	Y	CF	-	nw	7.9-8.0	535-596	30 d	NOEC	mortality	1800	DeGraeve et al, 1982
<i>Sarotherodon mossambicus</i>	18±3 g	N	R	-	tw	7.6±0.3	235	12 w	NOEC	growth	2300	Dange & Masurekar, 1982

Acute toxicity of naphthalene to marine organisms

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp time	Criterion	Test endpoint	Value (µg/L)	Reference
Mollusca												
<i>Callinectes sapidus</i>	adult, 50-227 g	Y	CF	analytical	am	-	10	48 h	LC50	mortality	2900	Sabourin, 1982
<i>Callinectes sapidus</i>	adult, 50-227 g	Y	CF	analytical	am	-	20	48 h	LC50	mortality	2100	Sabourin, 1982
<i>Callinectes sapidus</i>	adult, 50-227 g	Y	CF	analytical	am	-	30	48 h	LC50	mortality	2000	Sabourin, 1982
<i>Mytilus edulis</i>		N	R	≥98%	nw	-	33	48 h	EC50	feeding, filtration	922	Donkin et al., 1989, 1991
Annelida												
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	32	96 h	LC50	mortality	3800	Rossi & Neff, 1978
Crustacea												
<i>Artemia salina</i>	nauplii	Y	S	≥98%	am	8.5-8.7	32	24 h	EC50	immobility	3190	Foster & Tullis, 1984
<i>Artemia salina</i>	nauplii	N	Sc	≥97%	-	-	30	24 h	LC50	mortality	11000	Abernethy et al., 1986
<i>Cancer magister</i>	1 st instar zoeae	Y	S	analytical	nw	-	30	96 h	LC50	mortality	>2000	Caldwell et al., 1977
<i>Cancer magister</i>	newly hatched zoeae	Y	F	-	nw	7.5	-	36 h	LC100	mortality	8-12	Sanborn & Malins, 1977
<i>Calanus finmarchicus</i>	adult	Y	Sc	>97%	nw	-	33	96 h	LC50	mortality	2400	Falk-Petersen et al., 1982
<i>Calanus finmarchicus</i>	adult	Y	Sc	>97%	nw	-	33	96 h	LC10	mortality	2200	Falk-Petersen et al., 1982
<i>Elasmopus pectenicus</i>	adult	N	Rc	-	nw	-	30	96 h	LC50	mortality	2680	Lee & Nicol, 1978b
<i>Eualis suckleyi</i>	1 g	Y	CF	-	nw	-	-	96 h	LC50	mortality	1390	Rice & Thomas, 1989
<i>Eurytemora affinis</i>	adult	Y	Sc	≥99%	nw	-	20	24 h	LC50	mortality	3800	Ott et al., 1978
<i>Hemigrapsus nudus</i>		N	CF	-	nw	-	28-29	8 d	LC50	mortality	1100	Gharrett & Rice, 1987
<i>Hemigrapsus nudus</i>		N	CF	-	nw	-	28-29	8 d	LC50	mortality	2100	Gharrett & Rice, 1987
<i>Hemigrapsus nudus</i>		N	CF	-	nw	-	28-29	8 d	LC50	mortality	2800	Gharrett & Rice, 1987
<i>Neomysis americana</i>		Y	CF	-	am	-	-	96 h	LC50	mortality	1280	Smith & Hargreaves, 1983
<i>Neomysis americana</i>		Y	CF	-	am	-	-	96 h	LC50	mortality	850	Smith & Hargreaves, 1983
<i>Neomysis americana</i>		Y	R	-	am	-	-	96 h	LC50	mortality	1420	Hargreaves et al., in press cited in Smith & Hargreaves, 1983
<i>Neomysis americana</i>		Y	R	-	am	-	-	96 h	LC50	mortality	800	Hargreaves et al., in press cited in Smith & Hargreaves, 1983
<i>Palaemonetes pugio</i>		Y	S	-	nw	-	20	24 h	LC50	mortality	2600	Anderson et al., 1974
<i>Palaemonetes pugio</i>		-	-	-	-	-	-	48 h	LC50	mortality	2350	Tatem & Anderson., 1973
<i>Palaemonetes pugio</i>	adult	N	S	high purity	am	8.1±0.1	20	96 h	LC50	mortality	2350	Tatem et al., 1978
<i>Pandalus goniurus</i>	0.8 g, 6 cm	Y	S	-	nw	-	26-28	96 h	LC50	mortality	2160	Korn et al., 1979
<i>Pandalus goniurus</i>	0.8 g, 6 cm	Y	S	-	nw	-	26-28	96 h	LC50	mortality	1020	Korn et al., 1979
<i>Pandalus goniurus</i>	0.8 g, 6 cm	Y	S	-	nw	-	26-28	96 h	LC50	mortality	971	Korn et al., 1979
<i>Pandalus platyceros</i>	larvae stages I and IV	Y	F	-	nw	7.5	-	36 h	LC100	mortality	8-12	Sanborn & Malins, 1977
<i>Parhyale hawaiiensis</i>	adult	N	S	-	nw	-	30	24 h	LC50	mortality	15000	Lee & Nicol, 1978a
<i>Parhyale hawaiiensis</i>	adult	N	S	-	nw	-	30	24 h	LC10	mortality	12000	Lee & Nicol, 1978a
<i>Parhyale hawaiiensis</i>	adult	N	Sc	-	nw	-	30	24 h	LC50	mortality	6000	Lee & Nicol, 1978a
<i>Parhyale hawaiiensis</i>	adult	N	Sc	-	nw	-	30	24 h	LC10	mortality	3700	Lee & Nicol, 1978a
<i>Penaeus aztecus</i>		Y	S	-	nw	-	20	24 h	LC50	mortality	2500	Anderson et al., 1974
<i>Penaeus aztecus</i>	juvenile, 0.3 g	N	S	high purity	am	8.1±0.1	20	96 h	LC50	mortality	2500	Tatem et al., 1978
Echinodermata												
<i>Strongylocentrotus droebachiensis</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC50	mortality	1000	Falk-Petersen et al., 1982
<i>Strongylocentrotus droebachiensis</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC10	mortality	940	Falk-Petersen et al., 1982
<i>Strongylocentrotus droebachiensis</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC50	mortality	580	Saethre et al., 1984
<i>Strongylocentrotus droebachiensis</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC10	mortality	570	Saethre et al., 1984
Pisces												
<i>Cyprinodon variegatus</i>		Y	S	-	nw	-	20	24 h	LC50	mortality	2400	Anderson et al., 1974
<i>Fundulus heteroclitus</i>	8.2 cm	Y	R	-	nw	7.6	15	96 h	LC50	mortality	5300	DiMichele & Taylor, 1978
<i>Gadus morhua</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC50	mortality	1200	Falk-Petersen et al., 1982
<i>Gadus morhua</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC10	mortality	1000	Falk-Petersen et al., 1982
<i>Gadus morhua</i>	eggs, ELS	Y	S	>97%	nw	-	33	96 h	LC10	mortality	>700	Saethre et al., 1984
<i>MetaPenaeus monocyclus</i>	juvenile	N	R	-	nw	7.5	17.5	96 h	LC50	mortality	5087	Deshmukh et al., 1985
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	-	nw	-	28	96 h	LC50	mortality	1200	Moles & Rice, 1983
<i>Oncorhynchus gorbuscha</i>	fry, 0.35 g, 3.5 cm	Y	S	-	nw	-	26-28	96 h	LC50	mortality	1370	Korn et al., 1979
<i>Oncorhynchus gorbuscha</i>	fry, 0.35 g, 3.5 cm	Y	S	-	nw	-	26-28	96 h	LC50	mortality	1840	Korn et al., 1979
<i>Oncorhynchus gorbuscha</i>	fry, 0.35 g, 3.5 cm	Y	S	-	nw	-	26-28	96 h	LC50	mortality	1240	Korn et al., 1979
<i>Oncorhynchus gorbuscha</i>	fry	Y	CF	-	nw	-	-	48 h	LC50	mortality	961	Rice & Thomas, 1989
<i>Oncorhynchus gorbuscha</i>	fry, 4.5-5.5 cm	Y	S	-	nw	-	27	24 h	LC50	mortality	920	Thomas & Rice, 1979

Chronic toxicity of naphthalene (CASnr. 91-20-3) to marine organisms

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Champia parvula</i>	female	N	R	-	am	-	30	14 d	NOEC	growth, reproduction (nr. of cystocarps)	1300	Thursby et al, 1985
<i>Champia parvula</i>	female	N	R	-	am	-	30	14 d	EC50	growth	2200	Thursby et al, 1985
<i>Champia parvula</i>	female	N	R	-	am	-	30	14 d	EC10	growth	850	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	14 d	NOEC	growth	1300	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	14 d	NOEC	growth	<695	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	11 d	NOEC	reproduction (nr. of tetrasporangia)	695	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	14 d	EC50	growth	1900	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	14 d	EC10	growth	1400	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	14 d	EC50	growth	1000	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	14 d	EC10	growth	470	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	11 d	EC50	reproduction (nr. of tetrasporangia)	1300	Thursby et al, 1985
<i>Champia parvula</i>	tetrasporophyte	N	R	-	am	-	30	11 d	EC10	reproduction (nr. of tetrasporangia)	900	Thursby et al, 1985
Crustacea												
<i>Cancer magister</i>	zoeeae, Alaska	Y	CF	analytical	nw	-	29-34	40 d	NOEC	larval development	21	Caldwell et al, 1977
<i>Cancer magister</i>	zoeeae, Oregon	Y	CF	analytical	nw	-	29-34	60 d	NOEC	larval development	≥170	Caldwell et al, 1977
<i>Cancer magister</i>	zoeeae, Alaska	Y	CF	analytical	nw	-	29-34	40 d	NOEC	mortality, growth	>130	Caldwell et al, 1977
<i>Cancer magister</i>	zoeeae, Oregon	Y	CF	analytical	nw	-	29-34	60 d	NOEC	mortality, growth	>170	Caldwell et al, 1977
<i>Eurytemora affinis</i>	adult	Y	Rc	≥99%	nw	-	20	lifetime, 15 d	NOEC	life time, nr. of eggs, brood size<14	136	Ott et al, 1978
<i>Rhithropanopeus harrissi</i>	zoeeae	N	R	-	am	-	5, 15, 25	zoecal development until molting to emgalops	NOEC	mortality	≥500	Laughlin & Neff, 1979
Pisces												
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	-	nw	-	28	5 w	NOEC	wet weight (increase)	120	Moles & Rice, 1983
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	-	nw	-	28	5 w	EC50	wet weight (increase)	700	Moles & Rice, 1983
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	-	nw	-	28	5 w	C10	wet weight (increase)	260	Moles & Rice, 1983
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	≥	nw	-	28	5 w	NOEC	length (increase)	560	Moles & Rice, 1983
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	≥	nw	-	28	5 w	EC50	length (increase)	950	Moles & Rice, 1983
<i>Oncorhynchus gorbuscha</i>	325 mg, 32 mm	Y	CF	≥	nw	-	28	5 w	EC10	length (increase)	390	Moles & Rice, 1983

Acute toxicity of acenaphthylene (CASnr: 208-96-8) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	1800	Bisson et al, 2000
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	>1024	Wernersson, 2003
Pisces												
<i>Oryzias latipes</i>	-	-	-	-	-	-	-	48 h	LC50	mortality	11000	Yoshioka et al, 1986
<i>Oryzias latipes</i>	-	-	R	-	dtw	7.2	40	96 h	LC50	mortality	6400	Yoshioka & Ose, 1993

Chronic toxicity of acenaphthylene (CASnr: 208-96-8) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	82	Bisson et al, 2000
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	64	Bisson et al, 2000

Acute toxicity of acenaphthene (CASnr: 83-32-9) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Mollusca												
<i>Aplexa hypnorum</i>	adult	Y	CF	99%	nw	7.5-7.6	43.3	96 h	LC50	mortality	>2040	Holcombe et al, 1983
Crustacea												
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	958	Bisson et al, 2000
<i>Daphnia magna</i>	<24 h	N	Sc	-	nw	7.4-9.4	173	48 h	LC50	mortality	41000	LeBlanc, 1980
<i>Daphnia magna</i>	<24 h	N	Sc	-	nw	7.4-9.4	173	48 h	NOEC	mortality	600	LeBlanc, 1980
<i>Daphnia magna</i>	<24 h	N	Sc	≥97%	nw	-	-	48 h	EC50	immobility	1275	Munoz & Tarazona, 1993
<i>Daphnia magna</i>	12±12 h 1 st instar	N	S	-	nw	7.7	154.5	48 h	EC50	immobility	3450	Randall & Knopp, 1980
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	>1024	Wernersson, 2003
Pisces												
<i>Oryzias latipes</i>	-	-	R	-	dtw	7.2	40	48 h	LC50	mortality	23000	Yoshioka & Ose, 1993
<i>Pimephales promelas</i>	2 w	Y	CF	-	nw	7.4	35	96 h	LC50	mortality	608	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	0.16 g	Y	CF	99%	nw	7.5-7.6	43.3	96 h	LC50	mortality	1600	Holcombe et al, 1983
<i>Ictalurus punctatus</i>	5.0 g	Y	CF	99%	nw	7.5-7.6	43.3	96 h	LC50	mortality	1720	Holcombe et al, 1983
<i>Oncorhynchus mykiss</i>	1.3 g	Y	CF	99%	nw	7.2-7.4	45.8	96 h	LC50	mortality	670	Holcombe et al, 1983
<i>Salmo trutta</i>	0.16 g	Y	CF	99%	nw	7.2-7.4	45.8	96 h	LC50	mortality	580	Holcombe et al, 1983
<i>Lepomis macrochirus</i>	0.32-1.2 g	N	Sc	>80%	nw	6.5-7.9	32-48	96 h	LC50	mortality	1700	Buccafusco et al, 1981

Chronic toxicity of acenaphthene (CASnr: 83-32-9) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Cyanophyta												
<i>Anabaenaeflos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	≥4500	Bastian & Toetz, 1982
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	38	Bisson et al, 2000
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	42	Bisson et al, 2000
Pisces												
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	NOEC	fork length, wet weight	332	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC50	fork length	2400	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC10	fork length	220	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC50	wet weight	510	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC10	wet weight	190	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	NOEC	fork length, wet weight	345	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC50	fork length	1400	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC10	fork length	590	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC50	wet weight	760	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	EC10	wet weight	440	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	NOEC	mortality	509	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	LC50	mortality	690	Cairns & Nebeker, 1982
<i>Pimephales promelas</i>	ELS, embryos ≥48 h	Y	CF	-	nw	7.4	35	96 h	LC10	mortality	590	Cairns & Nebeker, 1982

Acute toxicity of acenaphthene (CASnr: 83-32-9) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity(‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Mollusca												
<i>Mytilus edulis</i>		N	R	≥98%	nw	-	33	48 h	EC50	feeding filtration	382	Donkin et al, 1989, 1991
Pisces												
<i>Cyprinodon variegatus</i>	8-15 mm, 14-28 d	N	S	>80%	nw	-	10-31	96 h	EC50	mortality	2200	Heitmuller et al, 1981
<i>Cyprinodon variegatus</i>	8-15 mm, 14-28 d	N	S	>80%	nw	-	10-31	96 h	NOEC	mortality	1000	Heitmuller et al, 1981
<i>Cyprinodon variegatus</i>	adult	Y	IF	-	nw	-	25	96 h	LC50	mortality	3100	Ward et al, 1981

Chronic toxicity of acenaphthene (CASnr: 83-32-9) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Notes
Pisces												
<i>Cyprinodon variegatus</i>	ELS embryo	Y	IF	-	nw	7.9-8.3	25±3	4h after fertilization – hatching	NOEC	hatching	970	175
<i>Cyprinodon variegatus</i>	ELS embryo	Y	IF	-	nw	7.9-8.3	25±3	4h after fertilization – hatching	EC50	hatching	1300	92, 175
<i>Cyprinodon variegatus</i>	ELS embryo	Y	IF	-	nw	7.9-8.3	25±3	4h after fertilization – hatching	EC10	hatching	760	92, 175
<i>Cyprinodon variegatus</i>	ELS embryo	Y	IF	-	nw	7.9-8.3	25±3	28 d after hatching	NOEC	mortality	520	175
<i>Cyprinodon variegatus</i>	ELS embryo	Y	IF	-	nw	7.9-8.3	25±3	28 d after hatching	EC50	mortality	860	92, 175
<i>Cyprinodon variegatus</i>	ELS embryo	Y	IF	-	nw	7.9-8.3	25±3	28 d after hatching	EC10	mortality	610	92, 175

Acute toxicity of anthracene (CASnr: 120-12-7) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Chlorella vulgaris</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	530	Hutchinson et al, 1980
<i>Chlamydomonas angulosa</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	240	Hutchinson et al, 1980
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC50	growth	>40000	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC50	growth rate	3.9	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC50	growth rate	6.6	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC50	growth rate	5.3	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC50	growth rate	12.1	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC50	growth rate	37.4	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC50	primary production	3.3	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC50	primary production	5.9	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC50	primary production	4.9	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC50	primary production	8.1	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC50	primary production	24	Gala & Giesy, 1992
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC50	growth, area under the curve	1040	Djomo et al, 2004
Macrophyta												
<i>Lemna gibba</i>	-	Y	S	-	am	-	-	4 h	EC50	Chl a fluorescence	6600	Huang et al, 1997b
<i>Lemna gibba</i>	-	Y	S	-	am	-	-	4 h	EC10	Chl a fluorescence	590	Huang et al, 1997b
<i>Lemna gibba</i>	-	Y	S	-	am	-	-	4 h	EC50	Chl a fluorescence	6300	Huang et al, 1997b
<i>Lemna gibba</i>	-	Y	S	-	am	-	-	4 h	EC10	Chl a fluorescence	110	Huang et al, 1997b
<i>Lemna gibba</i>	-	Y	S	high	am	-	-	6 h	EC50	Chl a fluorescence	1800	Mallakin et al, 2002
<i>Lemna gibba</i>	-	Y	S	high	am	-	-	6 h	EC50	Chl a fluorescence	1000	Mallakin et al, 2002
<i>Lemna gibba</i>	-	Y	S	high	am	-	-	6 h	EC50	electron transport	90	Mallakin et al, 2002
<i>Lemna gibba</i>	-	Y	S	high	am	-	-	6 h	EC50	electron transport	50	Mallakin et al, 2002
<i>Lemna gibba</i>	-	Y	S	high	am	-	-	6 h	EC50	t _{1/2} photosynthetic activity	1200	Mallakin et al, 2002
Mollusca												
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	24 h	LC50	mortality	>16.6	Weinstein & Polk, 2001
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	8 h	LC50	mortality	2.84	Weinstein & Polk, 2001
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	16 h	LC50	mortality	2.01	Weinstein & Polk, 2001
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	24 h	LC50	mortality	1.93	Weinstein & Polk, 2001
Crustacea												
<i>Daphnia magna</i>	mature	N	S	analytical	tw	-	-	2 h	LC50	mortality	20	Kagan et al, 1985, 1987
<i>Daphnia magna</i>	4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	36	Abernethy et al, 1986
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>25	Bisson et al, 2000
<i>Daphnia magna</i>	<24 h	N	Sc	≥97%	rw	-	-	48 h	EC50	immobility	95	Munoz & Tarazona, 1993
<i>Daphnia magna</i>	1.5 mm, 4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	3000	Bobra et al, 1983
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	5.66	Wernersson, 2003
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	rw	-	160-180	48 h	EC50	immobility	754	Smith et al, 1988
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	rw	-	160-180	48 h	EC10	immobility	80	Smith et al, 1988
<i>Daphnia pulex</i>	adult	Y	S	99.9	nw	7.2	350	24+0.5 h	EC50	immobility	1	Allred & Giesy, 1985
<i>Daphnia pulex</i>	adult	Y	S	99.9	nw	7.2	350	24+1 h	EC50	immobility	5.1	Allred & Giesy, 1985
<i>Daphnia pulex</i>	adult	Y	S	99.9	nw	7.2	350	24+1 h	EC50	immobility	20	Allred & Giesy, 1985
<i>Daphnia pulex</i>	adult	Y	S	99.9	nw	7.2	350	24+0.75 h	EC50	immobility	13	Allred & Giesy, 1985
<i>Daphnia pulex</i>	adult	Y	S	99.9	nw	7.2	350	24+0.75 h	EC50	immobility	11	Allred & Giesy, 1985
<i>Daphnia pulex</i>	adult	Y	S	99.9	nw	7.2	350	24+0.75 h	EC50	immobility	20	Allred & Giesy, 1985
Insecta												
<i>Aedes aegypti</i>	3 rd instar	Y	R	-	am	-	-	48 h	LC50	mortality	26.8	Oris et al, 1984
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	anal	-	-	-	<24 h	LC50	mortality	150	Kagan et al, 1985, 1987

<i>Aedes aegypti</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	<1	Borovsky et al, 1987
<i>Aedes taeniorhynchus</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	260	Borovsky et al, 1987
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	>99%	DSW	-	-	96 h	LC50	mortality	2.5	Bleeker et al, 2003
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	>99%	DSW	-	-	96 h	LC50	mortality	110	Bleeker et al, 2003
<i>Culex quinquefasciatus</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	37	Borovsky et al, 1987
Pisces												
<i>Lepomis macrochirus</i>	juvenile 0.78±0.05 g, 3.11±0.05 cm	Y	CF	tech.	tw	7.7	326	5 d	LC50	mortality	1.27	McCloskey & Oris, 1991
<i>Lepomis macrochirus</i>	juvenile 0.78±0.05 g, 3.11±0.05 cm	Y	CF	tech.	tw	7.7	326	5 d	LC50	mortality	7.97	McCloskey & Oris, 1991
<i>Lepomis macrochirus</i>	juvenile 0.78±0.05 g, 3.11±0.05 cm	Y	CF	tech.	tw	7.7	326	5 d	LC50	mortality	3.74	McCloskey & Oris, 1991
<i>Lepomis macrochirus</i>	juvenile 0.78±0.05 g, 3.11±0.05 cm	Y	CF	tech.	tw	7.7	326	5 d	LC50	mortality	8.27	McCloskey & Oris, 1991
<i>Lepomis macrochirus</i>	juvenile 0.78±0.05 g, 3.11±0.05 cm	Y	CF	tech.	tw	7.7	326	5 d	LC50	mortality	7.47	McCloskey & Oris, 1991
<i>Lepomis macrochirus</i>	juvenile 0.78±0.05 g, 3.11±0.05 cm	Y	CF	tech.	tw	7.7	326	5 d	LC50	mortality	6.78	McCloskey & Oris, 1991
<i>Lepomis macrochirus</i>	juvenile 0.5-1 g, 2-3 cm	Y	CF	tech.	tw	8.20±0.2	328	6 d	LC50	mortality	2.78	Oris & Giesy, 1985; Oris et al, 1984
<i>Lepomis spec. (macrochirus)</i>	juvenile 0.5-1 g, 2-3 cm	Y	CF	tech.	tw	8.20±0.2	328	6 d	LC50	mortality	11.92	Oris & Giesy, 1985; Oris et al, 1984
<i>Lepomis spec. (macrochirus)</i>	juvenile 0.5-1 g, 2-3 cm	Y	CF	tech.	tw	8.20±0.2	328	6 d	LC50	mortality	18.23	Oris & Giesy, 1985; Oris et al, 1984
<i>Lepomis spec. (macrochirus)</i>	juvenile 0.5-1 g, 2-3 cm	Y	CF	tech.	tw	8.20±0.2	328	6 d	LC50	mortality	26.47	Oris & Giesy, 1985; Oris et al, 1984
<i>Pimephales promelas</i>	5 cm, 0.8 g	N	S	anal.	-	-	-	~ 24 h	LC50	mortality	360	Kagan et al, 1985

Chronic toxicity of anthracene (CASnr: 120-12-7) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	7.8	Bisson et al, 2000
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC10	growth	290	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC10	growth rate	1.5	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC10	growth rate	2.5	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC10	growth rate	2.3	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC10	growth rate	8.7	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	EC10	growth rate	7.8	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC10	primary production	1.7	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC10	primary production	2.7	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC10	primary production	2.2	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC10	primary production	2.5	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	EC10	primary production	3.9	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	NOEC	growth rate	1.42	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	NOEC	growth rate	2.35	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	NOEC	growth rate	<5.03	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	NOEC	growth rate	5.93	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	34 h	NOEC	growth rate	6.2	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	NOEC	primary production	1.36	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	NOEC	primary production	2.26	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	NOEC	primary production	<4.87	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	NOEC	primary production	5.75	Gala & Giesy, 1992
<i>Pseudokirchneriella subcapitata</i>		Y	R	99.90%	am	7.5	-	36 h	NOEC	primary production	2.81	Gala & Giesy, 1992
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC10	growth, area under the curve	10	Djomo et al, 2004
Macrophyta												
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	1400	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	140	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	420	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	150	Ren et al, 1996
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1300	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	470	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	790	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	370	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	480	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	110	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC49-100	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC57-100	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC75-100	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC100	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC6-30	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC43-61	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC100	growth	2000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1100	Huang et al, 1995

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	680	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	470	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	110	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	200	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	67	Huang et al, 1995
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC96	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC100	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	LOEC	growth inhibition	60	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	EC10	growth inhibition	45	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	EC50	growth inhibition	1300	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	LOEC	growth inhibition	10	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	EC10	growth inhibition	40	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	EC50	growth inhibition	800	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	LOEC	growth inhibition	10	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	EC10	growth inhibition	15	Mallakin et al, 1999
<i>Lemna gibba</i>	-	N	R	-	am	-	-	8 d	EC50	growth inhibition	300	Mallakin et al, 1999
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	>3.4	Bisson et al, 2000
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	population growth	3.4	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	population growth	7.1	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	population growth	2.2	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	population growth	2.5	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	population growth	2.2	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	population growth	4.7	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	population growth	1.9	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	population growth	3.2	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC50	population growth	10	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	fecundity	4.5	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	fecundity	3.3	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	fecundity	2.2	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	fecundity	2	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	NOEC	fecundity	1.9	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	analytical	am	8.1	230	21 d	EC10	fecundity	1.5	Foran et al, 1991
<i>Daphnia magna</i>		Y	R	technical	nw	8.1±0.2	230	21 d	NOEC	reproduction (total number of young)	<21	Holst & Giesy, 1989
<i>Daphnia magna</i>		Y	R	technical	nw	8.1±0.2	230	21 d	EC10	reproduction (total number of young)	52	Holst & Giesy, 1989
<i>Daphnia magna</i>		Y	R	technical	nw	8.1±0.2	230	21 d	NOEC	reproduction (total number of young)	<19	Holst & Giesy, 1989
<i>Daphnia magna</i>		Y	R	technical	nw	8.1±0.2	230	21 d	EC50	reproduction (total number of young)	44	Holst & Giesy, 1989
<i>Daphnia magna</i>		Y	R	technical	nw	8.1±0.2	230	21 d	EC10	reproduction (total number of young)	19	Holst & Giesy, 1989
<i>Hyalella azteca</i>	7-14 d	Y	R	98%	nw	7.79-8.88	140-170	10 d	LC50	mortality	5.6	Hatch & Burton, 1999
Insecta												
<i>Chironomus tentans</i>	8-10 d	Y	R	98%	nw	7.79-8.88	140-170	10 d	LC50	mortality	5.6	Hatch & Burton, 1999
<i>Chironomus riparius</i>	<24 h	Y	S	>99%	DSW	8.4	200	28 d	LC50	mortality	1.8	Bleeker et al, 2003
<i>Chironomus riparius</i>	<24 h	Y	S	>99%	DSW	8.4	200	28 d	NOEC	emergence	<0.53	Bleeker et al, 2003
Pisces												
<i>Pimephales promelas</i>	eggs	Y	CF	analytical	-	8	184	6 w	NOEC	hatching	6.7	Hall & Oris, 1991
<i>Pimephales promelas</i>	eggs	Y	CF	analytical	-	7.9	191	9 w	NOEC	survival	12	Hall & Oris, 1991

Acute toxicity of anthracene (CASnr: 120-12-7) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Artemia salina</i>	nauplii	N	Sc	≥97%	-	-	30	24 h	LC50	mortality	>50	Abernethy et al, 1986
<i>Artemia salina</i>	<1 d	N	S	analytical	-	-	-	3 h	LC50	mortality	20	Kagan et al, 1985, 1987
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC50	immobility	34	Peachey & Crosby, 1996
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC10	immobility	22	Peachey & Crosby, 1996
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC50	immobility	5.2	Peachey & Crosby, 1996
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC10	immobility	1.7	Peachey & Crosby, 1996
<i>Mysidopsis bahia</i>	24-48 h	Y	S	-	nw	-	30	48 h	LC50	mortality	535	Pelletier et al, 1997
<i>Mysidopsis bahia</i>	24-48 h	Y	S	-	nw	-	30	48 h	LC50	mortality	3.6	Pelletier et al, 1997
Mollusca												
<i>Mulinexa lateralis</i>	embryo/larval	Y	S	-	nw	-	30	48 h	L(E)C50	survival/development	4260	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	embryo/larval	Y	S	-	nw	-	30	48 h	L(E)C50	survival/development	6.47	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juv. 1-1.5 mm	Y	S	-	nw	-	30	96 h	LC50	mortality	>13300	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juv. 1-1.5 mm	Y	S	-	nw	-	30	96 h	LC50	mortality	68.9	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juv. 1-1.5 mm	Y	S	-	nw	-	30	96 h	EC50	growth	>13300	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juv. 1-1.5 mm	Y	S	-	nw	-	30	96 h	EC50	growth	>82.8	Pelletier et al, 1997

Acute toxicity of phenanthrene (CASnr: 85-01-8) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Chlorella vulgaris</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	1200	Hutchinson et al, 1980
<i>Chlamydomonas angulosa</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	940	Hutchinson et al, 1980
<i>Nitzschia palea</i>		Y	S	-	am	7.6	-	4 h	EC50	assimilation	14C	Millemann et al, 1984
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	7.6	-	4 h	EC50	assimilation	14C	Millemann et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	>96%	am	8.1-9.0	-	3 d	EC50	growth rate	2021	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		N	S	>96%	am	8.1-8.4	-	2 d	EC50	growth rate	2028	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	S/Sc	>96%	am	8.4-9.0	-	2 d	EC50	growth rate	1228	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-9.0	-	2 d	EC50	growth rate	663	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-7.3	-	2 d	EC50	growth rate	180	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-8.4	-	3 d	EC50	growth rate	324	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-7.3	-	2 d	EC50	growth rate	302	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-8.2	-	3 d	EC50	growth rate	333	Halling-Sørensen et al, 1996
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC50	growth, area under the curve	50240	Djomo et al, 2004
Crustacea												
<i>Daphnia magna</i>	mature	N	S	analytical	tw	-	-	2 h	LC50	mortality	450	Kagan et al, 1987
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	24 h	EC50	immobility	854	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48 h	EC50	immobility	731	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48+2 h	EC50	immobility	725	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	24 h	EC50	immobility	678	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48 h	EC50	immobility	604	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48+2 h	EC50	immobility	273	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	Y	S	-	am	-	-	24 h	EC50	immobility	269	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	Y	S	-	am	-	-	48 h	EC50	immobility	199	Verrhiest et al, 2001
<i>Daphnia magna</i>	1.5 mm, 4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	1200	Bobra et al, 1983
<i>Daphnia magna</i>	<24 h	N	Sc	≥97%	rw	-	-	48 h	EC50	immobility	383	Munoz & Tarazona, 1993
<i>Daphnia magna</i>	adult, mixed age	N	S	-	nw	7.6±0.2	134±16	48 h	LC50	mortality	843	Eastmond et al, 1984
<i>Daphnia magna</i>	24 h	Y	Sc	-	nw	7.8	140	48 h	EC50	immobility	700	Millemann et al, 1984
<i>Daphnia magna</i>	4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	210	Abernethy et al, 1986
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>400	Bisson et al, 2000
<i>Daphnia magna</i>	-	-	-	-	-	-	-	48 h	LC50	mortality	1000	Parkhurst et al, 1981
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	378	Wernersson, 2003
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	rw	-	160-180	48 h	EC50	immobility	350	Smith et al, 1988
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	rw	-	160-180	48 h	EC10	immobility	140	Smith et al, 1988
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	rw	-	hard	48 h	EC50	immobility	734	Passino & Smith, 1987
<i>Daphnia pulex</i>	1.9-2.1 mm	N	Sc	-	nw	7.5	-	96 h	LC50	mortality	100	Trucco et al, 1983
<i>Daphnia pulex</i>	neonates	Y	S	-	tw	7.2	43	48 h	LC50	mortality	1140	Geiger & Buikema, 1981, 1982
<i>Gammarus minus</i>	adult	Y	Sc	-	nw	-	-	48 h	LC50	mortality	460	Millemann et al, 1984
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	10 d	LC50	mortality	232	Lee et al, 2001
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	10 d	LC50	mortality	235	Lee et al, 2001
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	14 d	LC50	mortality	225	Lee et al, 2001

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Insecta												
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	analytical	-	-	-	<24 h	LC50	mortality	500	Kagan et al, 1987
<i>Chironomus tentans</i>	4 th instar	Y	Sc	-	nw	7.8	140	48 h	EC50	immobility	490	Milleman et al, 1984
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	99.5%	DSW	-	-	96 h	LC50	mortality	41	Bleeker et al, 2003
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	99.5%	DSW	-	-	96 h	LC50	mortality	160	Bleeker et al, 2003
Pisces												
<i>Oncorhynchus mykiss</i>	fry (Arlee), 13-21 d	N	S	≥95%	nw	-	160-190	96 h	LC50	mortality	3200	Edsall, 1991
<i>Pimephales promelas</i>	larvae	Y	R	high	tw	-	-	96 h	NOEC	mortality	≥10	Oris & Giesy, 1987

Chronic toxicity of phenanthrene (CASnr: 85-01-8) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Cyanophyta												
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	320	Bastian & Toetz, 1982
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	EC10	growth	450	Bastian & Toetz, 1982
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	26	Bisson et al, 2000
<i>Pseudokirchneriella subcapitata</i>		N	S	>96%	am	8.1-9.0	-	3 d	EC10	growth rate	803	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		N	S	>96%	am	8.1-8.4	-	2 d	EC10	growth rate	720	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	S/Sc	>96%	am	8.4-9.0	-	2 d	EC10	growth rate	110	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-9.0	-	2 d	EC10	growth rate	139	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-7.3	-	2 d	EC10	growth rate	10	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-8.4	-	3 d	EC10	growth rate	50	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-7.3	-	2 d	EC10	growth rate	24	Halling-Sørensen et al, 1996
<i>Pseudokirchneriella subcapitata</i>		Y	Sc	>96%	am	7.0-8.2	-	3 d	EC10	growth rate	37	Halling-Sørensen et al, 1996
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC10	growth, area under the curve	4910	Djomo et al, 2004
Macrophyta												
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	3200	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	590	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1900	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	880	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	880	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	240	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC32-59	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC29-32	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC33-73	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC42-100	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC16-22	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC18-25	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC40	growth	2000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	3000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	530	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	2200	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	730	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	710	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	270	Huang et al, 1995
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC16	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC28	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		Y	R	99%	am	-	-	8 d	EC50	growth	>5000	McConkey et al, 1997
<i>Lemna gibba</i>		Y	R	99%	am	-	-	8 d	EC50	growth	3480	McConkey et al, 1997
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	13	Bisson et al, 2000
<i>Daphnia magna</i>		Y	R	-	-	-	-	19-21 d	NOEC	reproduction	75	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	R	-	-	-	-	19 d	EC50	reproduction	42-75	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	R	-	-	-	-	21 d	EC50	reproduction	~130	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	R	-	-	-	-	19 d	NOEC	mortality	75	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	R	-	-	-	-	19 d	LC50	mortality	120	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	IF	-	-	-	-	21 d	NOEC	reproduction	21	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	IF	-	-	-	-	21 d	EC50	reproduction	50	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	IF	-	-	-	-	21 d	NOEC	mortality	66	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	IF	-	-	-	-	21 d	LC50	mortality	130	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia magna</i>		Y	IF	-	-	-	-	21 d	NOEC	length	38	Hooftman & Evers-de Ruiter, 1992d
<i>Daphnia pulex</i>	<24 h	Y	R	-	tw	6.9-7.5	41-50	lifetime	NOEC	reproduction, growth	110	Geiger & Buikema, 1982
<i>Daphnia pulex</i>	neonates	Y	R	≥97%	rw	-	160-200	16 d	NOEC	reproduction, growth	<60	Savino & Tanabe, 1989
<i>Daphnia pulex</i>	neonates	Y	R	≥97%	rw	-	160-200	16 d	EC10	reproduction, growth	31	Savino & Tanabe, 1989
<i>Daphnia pulex</i>	neonates	Y	R	≥97%	rw	-	160-200	16 d	EC50	reproduction, growth	79	Savino & Tanabe, 1989

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
<i>Daphnia pulex</i>	neonates	Y	R	≥97%	rw	-	160-200	16 d	EC10	growth	41	Savino & Tanabe, 1989
<i>Daphnia pulex</i>	neonates	Y	R	≥97%	rw	-	160-200	16 d	EC50	growth	100	Savino & Tanabe, 1989
Insecta												
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juv.	Y	R	>98%	nw	8.1-8.3	165-250	2 d	EC50	immobility	295	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juv.	Y	R	>98%	nw	8.1-8.3	165-250	5 d	EC50	immobility	74.3	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juv.	Y	R	>98%	nw	8.1-8.3	165-250	10 d	EC50	immobility	38.2	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juv.	Y	R	>98%	nw	8.1-8.3	165-250	10 d	LC50	mortality	168.4	Landrum et al, 2003
<i>Diporeia</i> spp.	1-2 mm, 5-11 m, juv.	Y	R	>98%	nw	8.1-8.3	165-250	28 d	LC50	mortality	95.2	Landrum et al, 2003
<i>Chironomus riparius</i>	<24 h	Y	S	>99%	DSW	8.4	200	28 d	LC50	mortality	55	Bleeker et al, 2003
<i>Chironomus riparius</i>	<24 h	Y	S	>99%	DSW	8.4	200	28 d	LOEC	emergence	43	Bleeker et al, 2003
Pisces												
<i>Brachydanio rerio</i>	ELS	Y	R	-	-	-	-	28 d	NOEC	length	14	Hoofman & Evers-de Ruiter, 1992d
<i>Brachydanio rerio</i>	ELS	Y	R	-	-	-	-	28 d	NOEC	weight	24	Hoofman & Evers-de Ruiter, 1992d
<i>Brachydanio rerio</i>	ELS	Y	R	-	-	-	-	28 d	NOEC	mortality/hatching	≥ 240	Hoofman & Evers-de Ruiter, 1992d
<i>Micropterus salmoides</i>	eggs 2-4 d post spawning	Y	CF	-	am	7.4-8.1	86.8-116.3	7 d incl. 4 post-hatch	LC50	mortality	180	Black et al, 1983
<i>Micropterus salmoides</i>	eggs 2-4 d post spawning	Y	CF	-	am	7.4-8.1	86.8-116.3	7 d incl. 4 post-hatch	LC10	mortality	10	Black et al, 1983
<i>Micropterus salmoides</i>	eggs 2-4 d post spawning	Y	CF	-	rw	7.41-8.1	86.8-116.3	7 d incl. 4 post-hatch	LC50	mortality	250	Milleman et al, 1984
<i>Oncorhynchus mykiss</i>	eggs 20 min post fertilization	Y	CF	-	rw	7.4-8.1	86.8-116.3	27 d incl. 4 post-hatch	LC50	mortality	40	Black et al, 1983
<i>Oncorhynchus mykiss</i>	eggs 20 min post fertilization	Y	CF	-	rw	7.4-8.1	86.8-116.3	27 d incl. 4 post-hatch	LC10	mortality	28	Black et al, 1983
<i>Oncorhynchus mykiss</i>	eggs 20 min post fertilization	Y	CF	-	rw	7.41-8.1	86.8-116.3	27 d incl. 4 post-hatch	LC50	mortality	30	Milleman et al, 1984
<i>Oncorhynchus mykiss</i>	ELS	N	S	98%	tw	8.25	0.25	22 d	NOEC	mortality/hatching/abnormalities	<500	Hawkins et al, 2002
<i>Oncorhynchus mykiss</i>	fry, 4 d	N	CF	>98%	tw/nw	8.2	120	60 d	LC50	mortality	100-200	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, 4 d	N	CF	>98%	tw/nw	8.2	120	60 d	EC50	mortality	67	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, 4 d	N	CF	>98%	tw/nw	8.2	120	60 d	EC10	mortality	46	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, 4 d	N	CF	>98%	tw/nw	8.2	120	60 d	NOEC	length	<44	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, newly hatched to 7 d	N	CF	>98%	tw/nw	8.2	120	60 d	NOEC	length	38	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, newly hatched to 7 d	N	CF	>98%	tw/nw	8.2	120	60 d	EC50	length	71	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, newly hatched to 7 d	N	CF	>98%	tw/nw	8.2	120	60 d	EC10	length	37	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, 4 d	N	CF	>98%	tw/nw	8.2	120	60 d	NOEC	weight	44	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, <7 d	N	CF	>98%	tw/nw	8.2	120	60 d	NOEC	weight	38	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, <7 d	N	CF	>98%	tw/nw	8.2	120	60 d	EC50	weight	63	Passino-Reader et al, 1995
<i>Oncorhynchus mykiss</i>	fry, <7 d	N	CF	>98%	tw/nw	8.2	120	60 d	EC10	weight	33	Passino-Reader et al, 1995

Acute toxicity of phenanthrene (CASnr: 85-01-8) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Neanthes arenaceodentata</i>	emergent juvenile	N	S	98%	am	-	30	96 h	LC50	mortality	51	Emery & Dillon, 1996
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	32	96 h	LC50	mortality	600	Rossi & Neff, 1978
Crustacea												
<i>Artemia salina</i>	nauplii	N	Sc	≥97%	-	-	30	24 h	LC50	mortality	680	Abernethy et al, 1986
<i>Artemia salina</i>	nauplii	Y	S	≥98%	am	8.5-8.7	32	24 h	EC50	immobility	520	Foster & Tullis, 1984
Mollusca												
<i>Mytilus edulis</i>		N	R	≥98%	nw	-	33	48 h	EC50	feeding filtration	148	Donkin et al, 1989, 1991
Pisces												
<i>Cyprinodon variegatus</i>	fry, 6-8 d	N	R	-	am	-	18	96 h	LC50	mortality	478	Moreau et al, 1999

Chronic toxicity of phenanthrene (CASnr: 85-01-8) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Rhithropanopeus harrissi</i>	zoeae	N	R	-	am	-	5	zoeal development until molting to emgalops	NOEC	mortality	100	Laughlin & Neff, 1979
<i>Rhithropanopeus harrissi</i>	zoeae	N	R	-	am	-	15, 25	zoeal development until molting to emgalops	NOEC	mortality	150	Laughlin & Neff, 1979
Annelida												
<i>Neanthes arenaceodentata</i>	immature adult	N	S	98%	am	-	30	14 d	LC50	mortality	501	Emery & Dillon, 1996
<i>Neanthes arenaceodentata</i>	emergent juvenile	N	S	98%	am	-	30	8 w	NOEC	growth, fecundity and number of emergent juveniles, time to egg deposition	<20	Emery & Dillon, 1996

Acute toxicity of fluoranthene (CASnr: 206-44-0) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Scenedesmus subspicatus</i>		Y	S	-	am	8.3	-	7 d	EC50	growth (rate)	192	Sepic et al, 2003
<i>Scenedesmus subspicatus</i>		Y	S	-	am	8.3	-	7 d	EC50	biomass	229	Sepic et al, 2003
Annelida												
<i>Lumbriculus variegatus</i>	adult	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>178	Spehar et al, 1999
<i>Lumbriculus variegatus</i>	adult	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	1.2	Spehar et al, 1999
<i>Stylaria lacustris</i>		N	S	-	nw	6.5-7.3	4-18	48 h	NOEC	mortality	>220	Suedel & Rodgers, 1996
<i>Stylaria lacustris</i>		N	S	-	nw	6.5-7.3	4-18	48 h	LC50	mortality	>220	Suedel & Rodgers, 1996
Macrophyta												
<i>Lemna minor</i>	2 frond	Y	-	98%	nw	7.10-8.42	83.9-85.8	96 h	EC50	growth	>166	Spehar et al, 1999
<i>Lemna minor</i>	2 frond	Y	-	98%	nw	7.10-8.42	83.9-85.8	96 h	EC50	growth	>159	Spehar et al, 1999
Coelenterata												
<i>Hydra americana</i>	nonbudding	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	70	Spehar et al, 1999
<i>Hydra americana</i>	nonbudding	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	2.2	Spehar et al, 1999
Crustacea												
<i>Ceriodaphnia dubia</i>	<12 h	Y	R	>99%	nw	8.18±0.04	57.07±4.14	48 h	LC50	mortality	45	Oris et al, 1991
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>112	Bisson et al, 2000
<i>Daphnia magna</i>	mature	N	S	analytical	tw	-	-	2 h	LC50	mortality	4	Kagan et al, 1985, 1987
<i>Daphnia magna</i>	24 h	Y	S	-	am	7.8	-	24 h	EC50	immobility	190	Sepic et al, 2003
<i>Daphnia magna</i>		N	S	-	nw	6.5-7.3	4-18	48 h	NOEC	mortality	85	Suedel & Rodgers, 1996
<i>Daphnia magna</i>		N	S	-	nw	6.5-7.3	4-18	48 h	LC50	mortality	105.7	Suedel & Rodgers, 1996
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48 h	EC30	immobility	180	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48+2 h	EC50	immobility	20.2	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	24 h	EC50	immobility	63.3	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48 h	EC50	immobility	34.4	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48+2	EC90	immobility	18	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	Y	S	-	am	-	-	24 h	EC50	immobility	30.7	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	Y	S	-	am	-	-	48 h	EC50	immobility	13.1	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	Y	R	98%	nw	7.10-8.42	169-219	48 h	LC50	mortality	117	Spehar et al, 1999
<i>Daphnia magna</i>	<24 h	Y	R	98%	nw	7.10-8.42	169-219	48 h	LC50	mortality	1.6	Spehar et al, 1999
<i>Daphnia magna</i>	<24 h	N	Sc	-	rw	7.4-9.4	173	48 h	LC50	mortality	320000	
<i>Daphnia magna</i>	<24 h	N	Sc	-	rw	7.4-9.4	173	48 h	NOEC	mortality	<8800	
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	5.01	Wernersson, 2003
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24 h	EC50	immobility	196	Wernersson & Dave, 1997
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24+2+2 h	EC50	immobility	35	Wernersson & Dave, 1997
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	LC50	mortality	75.22	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	LC10	mortality	73.7	Barata & Baird, 2000
<i>Daphnia magna</i>	eggs	Y	S	98%	am	-	hard	1 instar, 3-4 d	LC50	mortality	58.64	Barata & Baird, 2000
<i>Daphnia magna</i>	eggs	Y	S	98%	am	-	hard	1 instar, 3-4 d	LC10	mortality	35.74	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC50	feeding	37.83	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC10	feeding	19.51	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	NOEC	feeding	10	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC50	number of offspring	51.53	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC10	number of offspring	31.37	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	NOEC	number of offspring	30	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC50	brood mass	43.85	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC10	brood mass	17.8	Barata & Baird, 2000

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC50	body mass	104.38	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	EC10	body mass	13.54	Barata & Baird, 2000
<i>Daphnia magna</i>	8-9 d female	Y	S	98%	am	-	hard	1 instar, 3-4 d	NOEC	body and brood mass	20	Barata & Baird, 2000
<i>Gammarus pseudolimnaeus</i>	adult	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	108	Spehar et al, 1999
<i>Hyalella azteca</i>	7-14 d	Y	R	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	44	Spehar et al, 1999
<i>Hyalella azteca</i>		N	S	-	nw	6.5-7.3	4-18	48 h	NOEC	mortality	<74	Suedel & Rodgers, 1996
<i>Hyalella azteca</i>		N	S	-	nw	6.5-7.3	4-18	48 h	LC50	mortality	92.2	Suedel & Rodgers, 1996
<i>Hyalella azteca</i>		N	S	-	nw	7.7-8.4	160-180	24 h	LC50	mortality	>500	Werner & Nagel, 1997
Mollusca												
<i>Physella virgata</i>	adult	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>178	Spehar et al, 1999
<i>Physella virgata</i>	adult	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	82	Spehar et al, 1999
Insecta												
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	analytical	-	-	-	<24 h	LC50	mortality	12	Kagan et al, 1985, 1987
<i>Aedes aegypti</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	10	Borovsky et al, 1987
<i>Aedes taeniorhynchus</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	48	Borovsky et al, 1987
<i>Chironomus tentans</i>		N	S	-	nw	6.5-7.3	4-18	48 h	NOEC	mortality	>250	Suedel & Rodgers, 1996
<i>Chironomus tentans</i>		N	S	-	nw	6.5-7.3	4-18	48 h	LC50	mortality	>250	Suedel & Rodgers, 1996
<i>Culex quinquefasciatus</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	45	Borovsky et al, 1987
<i>Ophiogemphus spec.</i>	nymph	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>178	Spehar et al, 1999
<i>Ophiogemphus spec.</i>	nymph	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>110	Spehar et al, 1999
Pisces												
<i>Lepomis macrochirus</i>	juvenile	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>117	Spehar et al, 1999
<i>Lepomis macrochirus</i>	juvenile	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	12.3	Spehar et al, 1999
<i>Lepomis macrochirus</i>	0.32-1.2 g	N	Sc	>80%	rw	6.5-7.9	32-48	96 h	LC50	mortality	4000	Buccafusco et al, 1981
<i>Oncorhynchus mykiss</i>	30-50 d	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>91	Spehar et al, 1999
<i>Oncorhynchus mykiss</i>	30-50 d	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	7.7	Spehar et al, 1999
<i>Pimephales promelas</i>	larvae (0-48 h)	Y	CF	-	tw	7.12±0.6	284.9±10.4	96 h	LC50	mortality	9.46	Diamond et al, 1995
<i>Pimephales promelas</i>	larvae (0-48 h)	Y	CF	-	tw	7.12±0.6	284.9±10.4	96 h	LC50	mortality	6.83	Diamond et al, 1995
<i>Pimephales promelas</i>	5 cm, 0.8 g	N	S	analytical	-	-	-	~ 24 h	LC50	mortality	200	Kagan et al, 1985
<i>Pimephales promelas</i>	5 d	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	>212	Spehar et al, 1999
<i>Pimephales promelas</i>	30-50 d	Y	CF	98%	tw	7.10-8.42	46.5-61.7	96 h	LC50	mortality	12.2	Spehar et al, 1999

Chronic toxicity of fluoranthene (CASnr: 206-44-0) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	8.6	Bisson et al, 2000
Annelida												
<i>Stylaria lacustris</i>		N	S	-	nw	6.4-7.2	72-80	10 d	LC50	mortality	>137	Suedel & Rodgers, 1996
<i>Stylaria lacustris</i>		N	S	-	nw	6.4-7.2	72-80	10 d	NOEC	mortality	115	Suedel & Rodgers, 1996
Cyanophyta												
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	<38	Bastian & Toetz, 1982
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	EC10	growth	220	Bastian & Toetz, 1982
Macrophyta												
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	10000	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC10	root growth	590	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	4900	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC10	root growth	470	Ren et al, 1996
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	20000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	130	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	860	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	110	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	780	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC27-47	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-39	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC20-55	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC14-100	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-7	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC11-15	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC90	growth	2000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	7500	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	210	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	2100	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	120	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	94	Huang et al, 1995

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC57	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC58	growth	2000	Huang et al, 1997ab
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	1.17	Bisson et al, 2000
<i>Ceriodaphnia dubia</i>	<12 h	Y	R	>99%	rw	8.18±0.04	57.07±4.14	7 d	NOEC	reproduction	57	Oris et al, 1991
<i>Ceriodaphnia dubia</i>	<12 h	Y	R	>99%	rw	8.18±0.04	57.07±4.14	7 d	EC50	reproduction	38.4	Oris et al, 1991
<i>Ceriodaphnia dubia</i>	<12 h	Y	R	>99%	rw	8.18±0.04	57.07±4.14	7 d	NOEC	reproduction	32	Oris et al, 1991
<i>Ceriodaphnia dubia</i>	<12 h	Y	R	>99%	rw	8.18±0.04	57.07±4.14	7 d	EC50	reproduction	28.5	Oris et al, 1991
<i>Daphnia magna</i>		Y	R	98%	nw	7.10-8.42	169-219	21 d	NOEC	mortality	73.2	Spehar et al, 1999
<i>Daphnia magna</i>		Y	R	98%	rw	7.10-8.42	169-219	21 d	NOEC	growth	17	Spehar et al, 1999
<i>Daphnia magna</i>		Y	R	98%	rw	7.10-8.42	169-219	21 d	NOEC	growth	1.4	Spehar et al, 1999
<i>Daphnia magna</i>	<48 h	Y	S	-	nw	7.0±0.5	120±20	10 d	EC50	immobility	102.6	Suedel et al, 1993
<i>Daphnia magna</i>	<48 h	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	immobility	91.6	Suedel et al, 1993
<i>Daphnia magna</i>	<48 h	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	immobility	64.1	Suedel et al, 1993
<i>Daphnia magna</i>	<48 h	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	immobility	42.7	Suedel et al, 1993
<i>Daphnia magna</i>		N	S	-	nw	8.1-8.4	100-130	10 d	NOEC	mortality	90	Suedel & Rodgers, 1996
<i>Daphnia magna</i>		N	S	-	nw	6.4-7.2	72-80	10 d	NOEC	mortality	75	Suedel & Rodgers, 1996
<i>Daphnia magna</i>		N	S	-	nw	8.1-8.4	100-130	10 d	LC50	mortality	102.6	Suedel & Rodgers, 1996
<i>Daphnia magna</i>		N	S	-	nw	6.4-7.2	72-80	10 d	LC50	mortality	110.5	Suedel & Rodgers, 1996
<i>Diporeia</i> sp.		Y	S	-	nw	8.2	165	10 d	LC50	mortality	>388	Kane Driscoll et al, 1997b
<i>Diporeia</i> sp.		Y	S	-	nw	8.2	165	10 d	LC50	mortality	>273	Kane Driscoll et al, 1997b
<i>Diporeia</i> sp.		Y	S	-	nw	8.2	165	10 d	NOEC	mortality	66	Kane Driscoll et al, 1997b
<i>Diporeia</i> sp.		Y	S	-	nw	8.2	165	10 d	NOEC	mortality	<63	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>		N	S	-	nw	8.1-8.4	100-130	10 d	NOEC	mortality	18	Suedel & Rodgers, 1996
<i>Hyalella azteca</i>		N	S	-	nw	6.4-7.2	72-80	10 d	NOEC	mortality	<24	Suedel & Rodgers, 1996
<i>Hyalella azteca</i>		N	S	-	nw	8.1-8.4	100-130	10 d	LC50	mortality	30.3	Suedel & Rodgers, 1996
<i>Hyalella azteca</i>		N	S	-	nw	6.4-7.2	72-80	10 d	LC50	mortality	60.6	Suedel & Rodgers, 1996
<i>Hyalella azteca</i>	2-3 w	Y	S	-	nw	8.2	165	10 d	NOEC	mortality	14	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>	2-3 w	Y	S	-	nw	8.2	165	10 d	NOEC	mortality	44	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>	2-3 w	Y	S	-	nw	8.2	165	10 d	LC10	mortality	55	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>	2-3 w	Y	S	-	nw	8.2	165	10 d	LC10	mortality	65	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>	2-3 w	Y	S	-	nw	8.2	165	10 d	LC50	mortality	114	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>	2-3 w	Y	S	-	nw	8.2	165	10 d	LC50	mortality	97.3	Kane Driscoll et al, 1997b
<i>Hyalella azteca</i>	0.355-0.5 mm	Y	R	-	nw	8.2±0.18	259±23	10 d	LC10	mortality	56	Wilcoxon et al, 2003
<i>Hyalella azteca</i>	0.355-0.5 mm	Y	R	-	nw	8.2±0.18	259±23	10 d	LC10	mortality	8.0	Wilcoxon et al, 2003
<i>Hyalella azteca</i>	0.355-0.5 mm	Y	R	-	nw	8.2±0.18	259±23	10 d	LC10	mortality	1.1	Wilcoxon et al, 2003
<i>Hyalella azteca</i>	0.355-0.5 mm	Y	R	-	nw	8.2±0.18	259±23	10 d	LC50	mortality	83.1	Wilcoxon et al, 2003
<i>Hyalella azteca</i>	0.355-0.5 mm	Y	R	-	nw	8.2±0.18	259±23	10 d	LC50	mortality	13.8	Wilcoxon et al, 2003
<i>Hyalella azteca</i>	0.355-0.5 mm	Y	R	-	nw	8.2±0.18	259±23	10 d	LC50	mortality	2.22	Wilcoxon et al, 2003
<i>Hyalella azteca</i>	7-14 d	Y	R	98%	nw	7.79-8.88	140-170	10 d	LC50	mortality	7.3	Hatch & Burton, 1999
<i>Hyalella azteca</i>	7-14 d	Y	R	98%	nw	7.79-8.88	140-170	10 d	LC50	mortality	71	Hatch & Burton, 1999
<i>Hyalella azteca</i>	0.6-1.0 mm (2-3 w)	Y	S	-	nw	7.0±0.5	120±20	10 d	EC50	immobility	44.9	Suedel et al, 1993
<i>Hyalella azteca</i>	0.6-1.0 mm (2-3 w)	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	immobility	44.7	Suedel et al, 1993
<i>Hyalella azteca</i>	0.6-1.0 mm (2-3 w)	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	immobility	54	Suedel et al, 1993
<i>Hyalella azteca</i>	0.6-1.0 mm (2-3 w)	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	immobility	32.4	Suedel et al, 1993
Insecta												
<i>Chironomus tentans</i>	8-10 d	Y	R	98%	nw	7.79-8.88	140-170	10 d	LC50	mortality	12.6	Hatch & Burton, 1999
<i>Chironomus tentans</i>	10-12 d	Y	S	-	nw	7.0±0.5	120±20	10 d	EC50	growth	31.9	Suedel et al, 1993
<i>Chironomus tentans</i>	10-12 d	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	growth	61	Suedel et al, 1993
<i>Chironomus tentans</i>	10-12 d	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	growth	50.6	Suedel et al, 1993
<i>Chironomus tentans</i>	10-12 d	Y	S	-	nw	6.5-8.5	100-140	10 d	EC50	growth	30.4	Suedel et al, 1993
<i>Chironomus tentans</i>		N	S	-	nw	8.1-8.4	100-130	10 d	NOEC	mortality	30	Suedel & Rodgers, 1996
<i>Chironomus tentans</i>		N	S	-	nw	6.4-7.2	72-80	10 d	NOEC	mortality	20	Suedel & Rodgers, 1996
<i>Chironomus tentans</i>		N	S	-	nw	8.1-8.4	100-130	10 d	NOEC	mortality	37.8	Suedel & Rodgers, 1996
<i>Chironomus tentans</i>		N	S	-	nw	6.4-7.2	72-80	10 d	LC50	mortality	23.6	Suedel & Rodgers, 1996
<i>Chironomus riparius</i>	larvae, 24 h post-hatch	Y	S	98%	rw	8.2	82	11 d	LC50	mortality	64.1	Stewart & Thompson, 1995
<i>Chironomus riparius</i>	larvae, 24 h post-hatch	Y	S	98%	rw	8.2	82	11 d	LC50	mortality	70.5	Stewart & Thompson, 1995
<i>Chironomus riparius</i>	larvae, 24 h post-hatch	Y	S	98%	rw	8.2	82	11 d	LC50	mortality	61.5	Stewart & Thompson, 1995
<i>Chironomus riparius</i>	larvae, 24 h post-hatch	Y	S	98%	rw	8.2	82	11 d	LC50	mortality	86.1	Stewart & Thompson, 1995
<i>Chironomus riparius</i>	larvae, 24 h post-hatch	Y	S	98%	rw	8.2	82	28 d	NOEC	total emergence, emergence time/ onset	4390	Stewart & Thompson, 1995
Pisces												
<i>Brachydanio rerio</i>	ELS	Y	IF	96%	rw	7.3-7.8	210	41 d	NOEC	mortality	47	Hoofman & Evers-de Ruyter, 1992a
<i>Brachydanio rerio</i>	ELS	Y	IF	96%	rw	7.3-7.8	210	41 d	NOEC	length	4.4	Hoofman & Evers-de Ruyter, 1992a
<i>Brachydanio rerio</i>	ELS	Y	IF	96%	rw	7.3-7.8	210	41 d	EC10	length	18	Hoofman & Evers-de Ruyter, 1992a
<i>Brachydanio rerio</i>	ELS	Y	IF	96%	rw	7.3-7.8	210	41 d	NOEC	weight	16	Hoofman & Evers-de Ruyter, 1992a
<i>Brachydanio rerio</i>	ELS	Y	IF	96%	rw	7.3-7.8	210	41 d	EC10	weight	21	Hoofman & Evers-de Ruyter, 1992a
<i>Brachydanio rerio</i>	ELS	Y	IF	96%	rw	7.3-7.8	210	41 d	LC100	mortality	130	Hoofman & Evers-de Ruyter, 1992a

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
<i>Brachydanio rerio</i>	ELS	Y	IF		nw	7.8-8.2		28 d	LC100	mortality	240	Hooffman & Evers-de Ruiter, 1992b
<i>Pimephales promelas</i>		Y	CF	-	tw	7.12±0.6	284.9±10.4	14 w	NOEC	number of eggs	<7.9	Diamond et al, 1995
<i>Pimephales promelas</i>		Y	CF	-	tw	7.12±0.6	284.9±10.4	11 w	NOEC	survival of hatchlings	<6.2	Diamond et al, 1995
<i>Pimephales promelas</i>	ELS	Y	CF	98%	tw	7.10-8.42	46.5-61.7	32 d	NOEC	growth	10.4	Spehar et al, 1999
<i>Pimephales promelas</i>	ELS	Y	CF	98%	tw	7.10-8.42	46.5-61.7	32 d	NOEC	growth	1.4	Spehar et al, 1999

Acute toxicity of fluoranthene (CASnr: 206-44-0) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Monopylephorus rubroniveus</i>		Y	R	98	nw	8.1±0.1	29.0±1.9	72 h	LC50	mortality	>120.4	Weinstein et al, 2003
<i>Monopylephorus rubroniveus</i>		Y	R	98	nw	8.1±0.1	29.0±1.9	72 h	LC50	mortality	0.7	Weinstein et al, 2003
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	-	32	LC50	mortality	500	Rossi & Neff, 1978
<i>Neanthes arenaceodentata</i>	adult	Y	R	98%	nw	-	30-32	96 h	LC50	mortality	>127	Spehar et al, 1999
Crustacea												
<i>Ampelisca abdita</i>	juvenile	Y	R	98%	nw	-	30-32	96 h	LC50	mortality	67	Spehar et al, 1999
<i>Ampelisca abdita</i>		N	S	-	nw	7.7-8.4	25	24 h	LC50	mortality	>100	Werner & Nagel, 1997
<i>Artemia salina</i>	<1 d	N	S	anal.	-	-	-	3 h	LC50	mortality	40	Kagan et al, 1985, 1987
<i>Homarus americanus</i>	larvae	Y	R	98%	nw	-	30-32	96 h	LC50	mortality	317	Spehar et al, 1999
<i>Homarus americanus</i>	larvae	N	R	98%	nw	-	30-32	96 h	LC50	mortality	13	Spehar et al, 1999
<i>Homarus americanus</i>	larvae	N	R	98%	nw	-	30-32	96 h	LC50	mortality	22	Spehar et al, 1999
<i>Mysidopsis bahia</i>	24-48 h	Y	S	-	nw	-	30	48 h	LC50	mortality	63.8	Pelletier et al, 1997
<i>Mysidopsis bahia</i>	24-48 h	Y	S	-	nw	-	30	48 h	LC50	mortality	5.32	Pelletier et al, 1997
<i>Mysidopsis bahia</i>	<24 h	Y	CF	98%	nw	-	30-32	96 h	LC50	mortality	31	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	1.4	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	1.7	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	58	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	12	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	12	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	2.8	Spehar et al, 1999
<i>Mysidopsis bahia</i>	<24 h	N	CF	98%	nw	-	30-32	96 h	LC50	mortality	1.7	Spehar et al, 1999
<i>Palaemonetes spec.</i>	3 d	Y	R	98%	nw	-	30-32	96 h	LC50	mortality	142	Spehar et al, 1999
<i>Palaemonetes spec.</i>	3 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	22	Spehar et al, 1999
<i>Palaemonetes spec.</i>	3 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	6.6	Spehar et al, 1999
<i>Ampelisca abdita</i>		N	S	-	nw	7.7-8.4	31	24 h	LC50	mortality	>100	Werner & Nagel, 1997
<i>Rhepoxynius abronius</i>		Y	R	-	nw	-	28	96 h	LC50	mortality	>70	Boese et al, 1997
<i>Rhepoxynius abronius</i>		Y	R	-	nw	-	28	96+1 h	LC50	mortality	14	Boese et al, 1997
<i>Rhepoxynius abronius</i>		Y	R	-	nw	-	28	96+1 h	EC50	reburial	63	Boese et al, 1997
<i>Rhepoxynius abronius</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	<5	Boese et al, 1997
<i>Eohaustorius estuarius</i>		Y	R	-	nw	-	28	96 h	LC50	mortality	>70	Boese et al, 1997
<i>Eohaustorius estuarius</i>		Y	R	-	nw	-	28	96+1 h	LC50	mortality	66	Boese et al, 1997
<i>Eohaustorius estuarius</i>		Y	R	-	nw	-	28	96+1 h	EC50	reburial	>70	Boese et al, 1997
<i>Eohaustorius estuarius</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	7	Boese et al, 1997
<i>Leptocheirus plumulosus</i>		Y	R	-	nw	-	20	96 h	LC50	mortality	>98	Boese et al, 1997
<i>Leptocheirus plumulosus</i>		Y	R	-	nw	-	28	96+1 h	LC50	mortality	69	Boese et al, 1997
<i>Leptocheirus plumulosus</i>		Y	R	-	nw	-	28	96+1 h	EC50	reburial	51	Boese et al, 1997
<i>Leptocheirus plumulosus</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	20	Boese et al, 1997
<i>Grandidierella japonica</i>		Y	R	-	nw	-	28	96 h	LC50	mortality	36	Boese et al, 1997
<i>Grandidierella japonica</i>		Y	R	-	nw	-	28	96+1 h	LC50	mortality	26	Boese et al, 1997
<i>Grandidierella japonica</i>		Y	R	-	nw	-	28	96+1 h	EC50	reburial	27	Boese et al, 1997
<i>Grandidierella japonica</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	19	Boese et al, 1997
<i>Corophium insidiosum</i>		Y	R	-	nw	-	28	96 h	LC50	mortality	85	Boese et al, 1997
<i>Corophium insidiosum</i>		Y	R	-	nw	-	28	96+1 h	LC50	mortality	32	Boese et al, 1997
<i>Corophium insidiosum</i>		Y	R	-	nw	-	28	96+1 h	EC50	reburial	54	Boese et al, 1997
<i>Corophium insidiosum</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	20	Boese et al, 1997
<i>Excirolana vancouverensis</i>		Y	R	-	nw	-	28	96 h	LC50	mortality	>70	Boese et al, 1997
<i>Excirolana vancouverensis</i>		Y	R	-	nw	-	28	96+1 h	LC50	mortality	>70	Boese et al, 1997
<i>Excirolana vancouverensis</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	>70	Boese et al, 1997
<i>Excirolana vancouverensis</i>		Y	R	-	nw	-	28	96+1+1 h	EC50	reburial	73	Boese et al, 1997
Mollusca												
<i>Macomona lilliana</i>	0.5-2 mm juvenile	Y	S		nw		34	96 h	EC50	reburial	153	Ahrens et al, 2002
<i>Macomona lilliana</i>	0.5-2 mm juvenile	Y	S		nw		34	96 h	NOEC	reburial	50	Ahrens et al, 2002

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	EC50	reburial	46	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	NOEC	reburial	10	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	EC50	reburial	49	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	NOEC	reburial	14	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	EC50	reburial	48	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	EC50	reburial	207	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	EC50	reburial	12	Ahrens et al, 2002
<i>Macomona liliana</i>	0.5-2 mm juvenile	Y	S		nw	-	34	96 h	EC50	reburial	51	Ahrens et al, 2002
<i>Mulinex lateralis</i>	embryo/larval	Y	S	-	nw	-	30	48 h	L(E)C50	survival/development	58.8	Pelletier et al, 1997
<i>Mulinex lateralis</i>	embryo/larval	Y	S	-	nw	-	30	48 h	L(E)C50	survival/development	1.09	Pelletier et al, 1997
<i>Mulinex lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	LC50	mortality	3310	Pelletier et al, 1997
<i>Mulinex lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	LC50	mortality	1.8	Pelletier et al, 1997
<i>Mulinex lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	EC50	growth	900	Pelletier et al, 1997
<i>Mulinex lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	EC50	growth	>0.81	Pelletier et al, 1997
<i>Mulinex lateralis</i>	embryo/larval	Y	S	98%	nw	-	30-32	96 h	LC50	mortality	>127	Spehar et al, 1999
<i>Mulinex lateralis</i>	embryo/larval	N	S	98%	nw	-	30-32	96 h	LC50	mortality	2.8	Spehar et al, 1999
<i>Mytilus edulis</i>		N	R	≥98%	nw	-	33	48 h	EC50	feeding filtration	80	
Echinodermata												
<i>Arbacia punctulata</i>	embryo/larval	Y	S	98%	nw	-	30-32	96 h	LC50	mortality	>127	Spehar et al, 1999
<i>Arbacia punctulata</i>	embryo/larval	N	S	98%	nw	-	30-32	96 h	LC50	mortality	3.9	Spehar et al, 1999
<i>Arbacia punctulata</i>	embryo/larval	N	S	98%	nw	-	30-32	96 h	LC50	mortality	3.9	Spehar et al, 1999
Pisces												
<i>Cyprinodon variegatus</i>	8-15 mm, 14-28 d	N	S	>80%	nw	-	10-31	96 h	LC50	mortality	>560000	Heitmuller et al, 1981
<i>Cyprinodon variegatus</i>	42 d	Y	R	98%	nw	-	30-32	96 h	LC50	mortality	>127	Spehar et al, 1999
<i>Cyprinodon variegatus</i>	42 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	159	Spehar et al, 1999
<i>Cyprinodon variegatus</i>	42 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	172	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	Y	R	98%	nw	-	30-32	96 h	LC50	mortality	616	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	30	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	13	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	620	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	103	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	49	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	30	Spehar et al, 1999
<i>Menidia beryllina</i>	21 d	N	R	98%	nw	-	30-32	96 h	LC50	mortality	13	Spehar et al, 1999
<i>Pleuronectes americanus</i>	28 d	Y	S	98%	nw	-	30-32	96 h	LC50	mortality	>188	Spehar et al, 1999
<i>Pleuronectes americanus</i>	28 d	N	S	98%	nw	-	30-32	96 h	LC50	mortality	0.1	Spehar et al, 1999

Chronic toxicity of fluoranthene (CASnr: 206-44-0) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Corophium spicorne</i>	0.5-1 mm	Y	S		nw	-	28	10 d	LC50	mortality	23.9	Swartz et al, 1990
<i>Mysidopsis bahia</i>		Y	CF	98%	nw	-	30-32	31 d	NOEC	reproduction	11.1	Spehar et al, 1999
<i>Mysidopsis bahia</i>		N	CF	98%	nw	-	30-32	31 d	NOEC	reproduction	0.6	Spehar et al, 1999
<i>Rhepoxynius abronius</i>		Y	S	-	nw	-	28	10 d	EC4	mortality	77	Boese et al, 1999
<i>Rhepoxynius abronius</i>		Y	S	-	nw	-	28	10 d	EC3	mortality	77	Boese et al, 1999
<i>Rhepoxynius abronius</i>		Y	S	-	nw	-	28	10 d	EC4	reburial	77	Boese et al, 1999
<i>Rhepoxynius abronius</i>		Y	S	-	nw	-	28	10 d	EC100	reburial	77	Boese et al, 1999
<i>Rhepoxynius abronius</i>	0.5-1 mm	Y	S	-	nw	-	28	10 d	LC50	mortality	11.1	Swartz et al, 1990

Acute toxicity of chrysene (CASnr: 218-01-9) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>1.3	Bisson et al, 2000
<i>Daphnia magna</i>	mature	N	S	analytical	tw	-	-	2 h	LC50	mortality	1900	Kagan et al, 1987
<i>Daphnia magna</i>	neonates	<24 h	N	S	-	am	8	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates	<24 h	N	S	-	am	8	24+2+1 h	EC50	immobility	>1024	Wernersson, 2003
Insecta												
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	analytical	-	-	-	<24 h	LC50	mortality	1700	Kagan et al, 1987

Chronic toxicity of chrysene (CASnr: 218-01-9) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	>1	Bisson et al, 2000
Cyanophyta												
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	640	Bastian & Toetz, 1982
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	EC10	growth	440	Bastian & Toetz, 1982
Macrophyta												
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC5	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC74	growth	2000	Huang et al, 1997ab
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	>0.09	Bisson et al, 2000
<i>Daphnia magna</i>	<24 h	Y	IF	99-100%	rw	7.3-8.1	212	21 d	NOEC	mortality	≥1.4	Hoofman, 1991
<i>Daphnia magna</i>	<24 h	Y	IF	99-100%	rw	7.3-8.1	212	21 d	NOEC	reproduction	≥1.4	Hoofman, 1991
Pisces												
<i>Brachydanio rerio</i>	ELS	Y	IF		rw	7.8-8.2		28 d	NOEC	mortality, hatchability, length, weight	>0.91	Hoofman & Evers-de Ruiter, 1992b

Acute toxicity of chrysene (CASnr: 218-01-9) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	32	96 h	LC50	mortality	>1000	Rossi & Neff, 1978
Crustacea												
<i>Artemia salina</i>	<1 d	N	S	analytical	-	-	-	3 h	LC50	mortality	3000	Kagan et al, 1987

Acute toxicity of benzo[a]anthracene (CASnr: 56-55-3) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC50	growth	>40000	Cody et al, 1984
Crustacea												
<i>Daphnia pulex</i>	1.9-2.1 mm	N	Sc	-	nw	7.5	-	96 h	LC50	mortality	10	Trucco et al, 1983
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>9.1	Bisson et al, 2000
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2*1 h	EC50	immobility	3.37	Wernersson, 2003

Chronic toxicity of benzo[a]anthracene (CASnr: 56-55-3) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	1.2	Bisson et al, 2000
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC10	growth	18	Cody et al, 1984
Cyanophyta												
<i>Anabaenaflos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	8.3	Bastian & Toetz, 1982
Macrophyta												
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC70	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC100	growth	2000	Huang et al, 1997ab
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	>8.7	Bisson et al, 2000

Acute toxicity of benzo[b]fluoranthene (CASnr: 205-99-2) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>1.1	Bisson et al, 2000
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24 h	EC50	immobility	>1024	Wernersson & Dave, 1997
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24+2*2 h	EC50	immobility	4.2	Wernersson & Dave, 1997

Chronic toxicity of benzo[b]fluoranthene (CASnr: 205-99-2) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae <i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	>1	Bisson et al, 2000
Crustacea <i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	>1.083	Bisson et al, 2000

Acute toxicity of benzo[ghi]perylene (CASnr: 191-24-2) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea <i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>0.2	Bisson et al, 2000
Pisces <i>Pimephales promelas</i>	larvae	Y	R	high	tw	-	-	96 h	LC20	mortality	>0.15	Oris & Giesy, 1987

Chronic toxicity of benzo[ghi]perylene (CASnr: 191-21-2) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae <i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	>0.16	Bisson et al, 2000
Crustacea <i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	0.082	Bisson et al, 2000
Pisces <i>Brachydanio rerio</i>	ELS	Y	IF	-	rw	7.8-8.2	-	28 d	NOEC	mortality, hatchability, length, weight	>0.16	Hooftman & Evers-de Ruiter, 1992b
Macrophyta <i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC13	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC27	growth	2000	Huang et al, 1997ab

Acute toxicity of benzo[k]fluoranthene (CASnr: 207-08-9) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea <i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>1.1	Bisson et al, 2000
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48 h	EC30	immobility	>1	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48+2 h	EC50	immobility	>1	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48 h	EC50	immobility	>1	Verrhiest et al, 2001
<i>Daphnia magna</i>	<24 h	N	S	-	am	7.8	250	48+2 h	EC90	immobility	>1	Verrhiest et al, 2001

Chronic toxicity of benzo[k]fluoranthene (CASnr: 207-08-9) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae <i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	>1	Bisson et al, 2000
Crustacea <i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	>1.08	Bisson et al, 2000
Pisces <i>Brachydanio rerio</i>	ELS	Y	IF	-	rw	7.8-8.2	-	28 d	NOEC	length, weight	<0.58	Hooftman & Evers-de Ruiter, 1992b
<i>Brachydanio rerio</i>	ELS	Y	IF	-	rw	7.8-8.2	-	28 d	LC52	length, weight	0.58	Hooftman & Evers-de Ruiter, 1992b
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	NOEC	mortality	0.35	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	LC50	mortality	0.65	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	LC10	mortality	0.62	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	NOEC	length	<0.19	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	EC50	length	0.86	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	EC10	length	0.17	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	NOEC	weight	0.35	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	EC50	weight	0.50	Hooftman & Evers-de Ruiter, 1992c
<i>Brachydanio rerio</i>	ELS	Y	IF	100%	rw	7.9-8.2	206	42 d	EC10	weight	0.31	Hooftman & Evers-de Ruiter, 1992c

Acute toxicity of benzo[a]pyrene (CASnr: 50-32-8) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC50	growth	>13000	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC50	growth	40	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC50	growth	2.8	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	15	Schoeny et al, 1988
<i>Scenedesmus acutus</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	5	Schoeny et al, 1988
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC50	growth, area under the curve	1.48	Djomo et al, 2004
(Euglenophyta)												
<i>Euglena gracilis</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	>4000	Schoeny et al, 1988
(Heterokontophyta)												
<i>Ochromonas malhamensis</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	>4000	Schoeny et al, 1988
Cyanophyta												
<i>Anabaena flosaquae</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	>4000	Schoeny et al, 1988
<i>Ankistrodesmus braunii</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	1300	Schoeny et al, 1988
<i>Chlamydomonas reinhardtii</i>		N	S	≥99%	am	-	-	72 h	EC50	growth	>4000	Schoeny et al, 1988
Bacteria												
<i>Escherichia coli</i>		N	S	98	-	-	-	48 h	EC2	growth	0.96	Jamroz et al, 2003
<i>Escherichia coli</i>		N	S	98	-	-	-	48 h	EC2	growth	0.96	Jamroz et al, 2003
Crustacea												
<i>Daphnia magna</i>	neonates <48 h	N	S	-	am	-	-	48 h	LC50	mortality	250	Atienzar et al, 1999
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>2.7	Bisson et al, 2000
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24 h	EC50	immobility	40	Wernersson & Dave, 1997
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24+2+2 h	EC50	immobility	8.6	Wernersson & Dave, 1997
<i>Daphnia magna</i>	neonates	<24 h N	S	-	am	8	250	24 h	EC50	immobility	59.7	Wernersson, 2003
<i>Daphnia magna</i>	neonates	<24 h N	S	-	am	8	250	24+2+1 h	EC50	immobility	1.16	Wernersson, 2003
<i>Daphnia pulex</i>	1.9-2.1 mm	N	Sc	-	nw	7.5	-	96 h	LC50	mortality	5	Trucco et al, 1983
Insecta												
<i>Aedes aegypti</i>	<8h, 1 st instar	N	S	anal	tw	-	-	<24 h	LC50	mortality	8	Kagan & Kagan, 1986
<i>Aedes aegypti</i>	<8h, 1 st instar	N	S	anal	tw	-	-	<24 h	LC50	mortality	1.2	Kagan & Kagan, 1986

Chronic toxicity of benzo[a]pyrene (CASnr: 50-32-8) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Macrophyta												
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	9500	Ren et al, 1996
<i>Brassica napus</i>	seeds	N	R	-	am	-	-	6 d	EC50	root growth	620	Ren et al, 1996
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	6200	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1200	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	860	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	730	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	250	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-44	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-49	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC3-21	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC6-42	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC2-17	growth	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC22-51	chlorophyll content	2000	Huang et al, 1993
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC35	growth	2000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	>8000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	5600	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	560	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	160	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	130	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	54	Huang et al, 1995
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC21	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC99	growth	2000	Huang et al, 1997ab
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	0.78	Bisson et al, 2000
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC10	growth	4400	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC10	growth	10	Cody et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	≥99%	am	-	-	96 h	EC10	growth	0.96	Cody et al, 1984
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC10	growth, area under the curve	0.03	Djomo et al, 2004
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	72 h	EC10	growth rate	30	Djomo et al, 2004

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	0.503	Bisson et al, 2000
<i>Daphnia magna</i>	neonates <48 h	N	S	-	am	-	-	14 d	EC50	total number of young	30	Atienzar et al, 1999
<i>Daphnia magna</i>	neonates	<48 h N	S	-	am	-	-	14 d	EC10	total number of young	12.5	Atienzar et al, 1999
<i>Daphnia magna</i>	neonates	<48 h N	S	-	am	-	-	14 d	NOEC	total number of young	12.5	Atienzar et al, 1999
Pisces												
<i>Brachydanio rerio</i>	ELS	Y	IF	-	nw	7.8-8.2	-	28 d	NOEC	mortality, hatchability, length, weight	≥4.0	Hoofman & Evers-de Ruiter, 1992b
<i>Oncorhynchus mykiss</i>	ELS	Y	R	purified >99%	nw	6.85-7.10	-	36 d	NOEC	abnormalities	1.48	Hannah et al, 1982
<i>Oncorhynchus mykiss</i>	ELS	Y	R	purified >99%	nw	6.85-7.10	-	36 d	EC10	abnormalities	2.9	Hannah et al, 1982
<i>Oncorhynchus mykiss</i>	ELS	Y	R	purified >99%	nw	6.85-7.10	-	36 d	NOEC	length	<0.08	Hannah et al, 1982

Acute toxicity of benzo[a]pyrene (CASnr: 50-32-8) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	32	96 h	LC50	mortality	>1000	Rossi & Neff, 1978
Echinodermata												
<i>Strongylocentrotus purpuratus</i>	eggs and sperm	Y	S	99%	nw	7.88	33-34	48 h	NOEC	deformities	0.5	Hose et al, 1983

Chronic toxicity of benzo[a]pyrene (CASnr: 50-32-8) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Mollusca												
<i>Crassostrea gigas</i>	embryo/larval	N	S	97%	nw	-	32-33	48 h	NOEC	abnormal shell	1	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	97%	nw	-	32-33	48 h	EC50	abnormal shell	3.1	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	97%	nw	-	32-33	48 h	EC10	abnormal shell	1.1	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	97%	nw	-	32-33	48 h	NOEC	abnormal shell	0.5	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	97%	nw	-	32-33	48 h	EC50	abnormal shell	0.44	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	97%	nw	-	32-33	48 h	EC10	abnormal shell	0.22	Lyons et al, 2002
Pisces												
<i>Fundulus heteroclitus</i>	eggs	N	S	-	am	-	20	7 d	NOEC	EROD activity	0.25	Wassenberg et al, 2002
<i>Fundulus heteroclitus</i>	eggs	N	S	-	am	-	20	7 d	NOEC	deformities	<0.25	Wassenberg et al, 2002
<i>Psettichtys melanostichus</i>	eggs	Y	S	technical purified	am	7.1-7.5	25	6 d	NOEC	hatchability	<0.1	Hose et al, 1982

Acute toxicity of dibenzo[a,h]anthracene (CASnr: 53-70-3) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea												
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>0.35	Bisson et al, 2000
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24 h	EC50	immobility	496	Wernersson & Dave, 1997
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24+2+2 h	EC50	immobility	4.6	Wernersson & Dave, 1997
<i>Daphnia magna</i>	neonates	<24 h N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates	<24 h N	S	-	am	8	250	24+2+1 h	EC50	immobility	1.76	Wernersson, 2003
Pisces												
<i>Pimephales promelas</i>	larvae	Y	R	high	tw	-	-	96 h	NOEC	mortality	≥0.15	Oris & Giesy, 1987

Chronic toxicity of dibenzo[a,h]anthracene (CASnr: 53-70-3) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	0.14	Bisson et al, 2000
Macrophyta												
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC11	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC18	growth	2000	Huang et al, 1997ab
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	>0.032	Bisson et al, 2000

Acute toxicity of dibenzo[a,h]anthracene (CASnr: 53-70-3) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	32	96 h	LC50	mortality	>1000	Rossi & Neff, 1978

Acute toxicity of pyrene (CASnr: 129-00-0) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Chlorella vulgaris</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	330	Hutchinson et al, 1980
<i>Chlamydomonas angulosa</i>		N	S	-	am	6.5	-	3 h	EC50	photosynthesis	200	Hutchinson et al, 1980
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC50	growth, area under the curve	18.72	Djomo et al, 2004
Mollusca												
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	24 h	LC50	mortality	>28.2	Weinstein & Polk, 2001
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	8 h	LC50	mortality	7.71	Weinstein & Polk, 2001
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	16 h	LC50	mortality	3.35	Weinstein & Polk, 2001
<i>Utterbackia imbecilis</i>	glochidia	N	R	98%	rw	8.09	81.3	24 h	LC50	mortality	2.63	Weinstein & Polk, 2001
Crustacea												
<i>Daphnia magna</i>	4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	91	Abernethy et al, 1986
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	24.6	Bisson et al, 2000
<i>Daphnia magna</i>	1.5 mm, 4-6 d	N	Sc	≥97%	am	6.0-7.0	-	48 h	LC50	mortality	1800	Bobra et al, 1983
<i>Daphnia magna</i>	mature	N	S	analytical	tw	-	-	2 h	LC50	mortality	4	Kagan et al, 1985, 1987
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24 h	EC50	immobility	>1024	Wernersson & Dave, 1997
<i>Daphnia magna</i>	4 d	N	S	97%	nw	8	250	24+2+2 h	EC50	immobility	5.7	Wernersson & Dave, 1997
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	1.38	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	am	7.0±0.1	10	48 h	EC50	immobility	2.7	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	am	7.0±0.1	10	48 h	EC50	immobility	22	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	am	7.0±0.1	50	48 h	EC50	immobility	4.1	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	am	7.0±0.1	50	48 h	EC50	immobility	30	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	am	7.0±0.1	250	48 h	EC50	immobility	1.8	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	am	7.0±0.1	250	48 h	EC50	immobility	31	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	97	48 h	EC50	immobility	2.0	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	97	48 h	EC50	immobility	22	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	200	48 h	EC50	immobility	2.9	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	200	48 h	EC50	immobility	19	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	29	48 h	EC50	immobility	6.8	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	29	48 h	EC50	immobility	19	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	8.0	48 h	EC50	immobility	7.7	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	8.0	48 h	EC50	immobility	33	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	11	48 h	EC50	immobility	20	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	11	48 h	EC50	immobility	27	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	6.5	48 h	EC50	immobility	20	Nikkilä et al, 1999
<i>Daphnia magna</i>	neonates <24 h	N	S	98.7%	nw	7.0±0.1	6.5	48 h	EC50	immobility	27	Nikkilä et al, 1999
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	10 d	LC50	mortality	77.1	Lee et al, 2001
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	14 d	LC50	mortality	60.1	Lee et al, 2001
Insecta												
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	analytical	-	-	-	<24 h	LC50	mortality	20	Kagan et al, 1985, 1987
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	analytical	tw	-	-	<24 h	LC50	mortality	12	Kagan & Kagan, 1986
<i>Aedes aegypti</i>	<8 h, 1 st instar	N	S	analytical	tw	-	-	<24 h	LC50	mortality	9	Kagan & Kagan, 1986
<i>Aedes aegypti</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	35	Borovsky et al, 1987
<i>Aedes taeniorhynchus</i>	late-3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	60	Borovsky et al, 1987
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	98%	DSW	-	-	96 h	LC50	mortality	75	Bleeker et al, 2003
<i>Chironomus riparius</i>	1 st instar, <24 h	Y	S	98%	DSW	-	-	96 h	LC50	mortality	38	Bleeker et al, 2003
<i>Culex quinquefasciatus</i>	late 3 rd /4 th instar	N	S	-	-	-	-	24 h	LC50	mortality	37	Borovsky et al, 1987
<i>Diporeia sp.</i>	1-2 mm, juvenile 5-11 months	Y	R	>98%	nw	8.1-8.3	165-250	28 d	LC50	mortality	79.1	Landrum et al, 2003
Pisces												
<i>Pimephales promelas</i>	5 cm, 0.8 g	N	S	analytical	-	-	-	~24 h	LC50	mortality	220	Kagan et al, 1985

Chronic toxicity of pyrene (CASnr: 129-00-0) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S	-	am	-	215	72 h	EC10	growth	1.2	Bisson et al, 2000
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	7 d	EC10	growth, area under the curve	2.41	Djomo et al, 2004
<i>Scenedesmus subspicatus</i>		N	S	>98%	am	-	-	72 h	EC10	growth rate	8.4	Djomo et al, 2004
Cyanophyta												
<i>Anabaena flos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	≥120	Bastian & Toetz, 1982
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	2.1	Bisson et al, 2000
Macrophyta												
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	45000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	430	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	2600	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	270	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC14-21	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-29	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC11-39	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-34	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-5	growth	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC0-15	chlorophyll content	2000	Ren et al, 1994
<i>Lemna gibba</i>		Y	S	-	am	-	-	8 d	EC78	growth	2000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	>8000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	440	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	2800	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	690	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC50	growth	1000	Huang et al, 1995
<i>Lemna gibba</i>		Y	R	-	am	-	-	8 d	EC10	growth	230	Huang et al, 1995
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC24	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC46	growth	2000	Huang et al, 1997ab

Acute toxicity of pyrene (CASnr: 129-00-0) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Platynereis dumeralii</i>		N	S	-	nw	-	-	10 h	EC50	immobility	22	Peachey & Crosby, 1996
<i>Platynereis dumeralii</i>		N	S	-	nw	-	-	10 h	EC10	immobility	16	Peachey & Crosby, 1996
Crustacea												
<i>Artemia salina</i>	nauplii	N	Sc	≥97%	-	-	30	24 h	LC50	mortality	>99	Abernethy et al, 1986
<i>Artemia salina</i>	<1 d	N	S	analytical	-	-	-	3 h	LC50	mortality	8	Kagan et al, 1985, 1987
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC50	immobility	36	Peachey & Crosby, 1996
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC10	immobility	20	Peachey & Crosby, 1996
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC50	immobility	3.4	Peachey & Crosby, 1996
<i>Artemia salina</i>	nauplii	N	S	-	nw	-	-	10 h	EC10	immobility	1.8	Peachey & Crosby, 1996
<i>Amphilocus likelike</i>	larvae	N	S	-	nw	-	-	10 h	EC50	immobility	22	Peachey & Crosby, 1996
<i>Amphilocus likelike</i>	larvae	N	S	-	nw	-	-	10 h	EC10	immobility	8.4	Peachey & Crosby, 1996
<i>Mysidopsis bahia</i>	24-48 h	Y	S	-	nw	-	30	48 h	LC50	mortality	24.8	Pelletier et al, 1997
<i>Mysidopsis bahia</i>	24-48 h	Y	S	-	nw	-	30	48 h	LC50	mortality	0.89	Pelletier et al, 1997
Mollusca												
<i>Crassostrea gigas</i>	embryo/larval	N	S	98%	nw	-	32-33	48 h	NOEC	abnormal shell	25	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	98%	nw	-	32-33	48 h	EC50	abnormal shell	110	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	98%	nw	-	32-33	48 h	EC10	abnormal shell	32	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	98%	nw	-	32-33	48 h	NOEC	abnormal shell	0.5	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	98%	nw	-	32-33	48 h	EC50	abnormal shell	0.98	Lyons et al, 2002
<i>Crassostrea gigas</i>	embryo/larval	N	S	98%	nw	-	32-33	48 h	EC10	abnormal shell	0.93	Lyons et al, 2002
<i>Mulinexa lateralis</i>	embryo/larval	Y	S	-	nw	-	30	48 h	L(E)C50	survival/development	>11900	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	embryo/larval	Y	S	-	nw	-	30	48 h	L(E)C50	survival/development	0.23	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	LC50	mortality	>9454	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	LC50	mortality	1.68	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	EC50	growth	>9454	Pelletier et al, 1997
<i>Mulinexa lateralis</i>	juvenile 1-1.5 mm	Y	S	-	nw	-	30	96 h	EC50	growth	>0.91	Pelletier et al, 1997
<i>Mytilus edulis</i>	juvenile 1-1.5 mm	N	R	≥98%	nw	-	33	48 h	EC50	feeding filtration	>40	Donkin et al, 1989, 1991
Coelenterata												
<i>Fungia scutaria</i>	planulae	N	S	-	nw	-	-	10 h	EC50	immobility	32	Peachey & Crosby, 1996
<i>Fungia scutaria</i>	planulae	N	S	-	nw	-	-	10 h	EC10	immobility	26	Peachey & Crosby, 1996

Acute toxicity of fluorene (CASnr: 86-73-7) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Dunaliella bioculata</i>		N	S	purified	am	-	-	50-72 h	EC50	growth rate	15500	Heldal et al, 1984
<i>Pseudokirchneriella subcapitata</i>		N	S	98.6	-	-	-	96 h	EC50	CO ₂ incorporation, production	3400	Finger et al, 1985
<i>Pseudokirchneriella subcapitata</i>		N	S	98.6	-	-	-	7 d	EC50	cell number	2200	Finger et al, 1985
Crustacea												
<i>Daphnia magna</i>			S	98%		7.5	280	48 h	EC50	immobility	430	Mayer & Eilersieck, 1986
<i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	408	Bisson et al, 2000
<i>Daphnia magna</i>		Y	S	98.6	nw	7.2-7.4	270	48 h	EC50	immobility	430	Finger et al, 1985
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia magna</i>	neonates <24 h	N	S	-	am	8	250	24+2+1 h	EC50	immobility	>1024	Wernersson, 2003
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	nw	-	160-180	48 h	EC50	immobility	212	Smith et al, 1988
<i>Daphnia pulex</i>	<24 h	N	S	≥96%	nw	-	160-180	48 h	EC10	immobility	23	Smith et al, 1988
<i>Gammarus pseudolimnaeus</i>		Y	S	98.6	nw	7.2-7.4	270	96 h	LC50	mortality	600	Finger et al, 1985
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	10 d	LC50	mortality	525	Lee et al, 2001
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	10 d	LC50	mortality	452	Lee et al, 2001
<i>Hyalella azteca</i>	2-3 w	N	R	98%	nw	8.2	165	14 d	LC50	mortality	404	Lee et al, 2001
Insecta												
<i>Aedes aegypti</i>	<8h, 1 st instar	N	S	analytical	-	-	-	<24 h	LC50	mortality	2700	Kagan et al, 1987
<i>Chironomus plumosus</i>			S	98%		7.5	280	48 h	EC50	immobility	2350	Mayer & Eilersieck, 1986
<i>Chironomus riparius</i>	larvae	Y	S	98.6	nw	7.2-7.4	270	48 h	EC50	immobility	2350	Finger et al, 1985
<i>Hexagenia bilineata</i>	nymphs	Y	S	98.6	nw	7.2-7.4	270	120 h	LC50	mortality	5800	Finger et al, 1985
Mollusca												
<i>Mudalia potosensis</i>		Y	S	98.6	nw	7.2-7.4	270	96 h	LC50	mortality	5600	Finger et al, 1985
Pisces												
<i>Lepomis macrochirus</i>			S	98%		7.5	280	96 h	LC50	mortality	760	Mayer & Eilersieck, 1986
<i>Lepomis macrochirus</i>		Y	S	98.6	nw	7.2-7.4	270	96 h	LC50	mortality	910	Finger et al, 1985
<i>Oncorhynchus mykiss</i>		Y	S	98.6	nw	7.2-7.4	270	96 h	LC50	mortality	820	Finger et al, 1985
<i>Pimephales promelas</i>		Y	S	98.6	nw	7.2-7.4	270	96 h	LC50	mortality	>100000	Finger et al, 1985

Chronic toxicity of fluorene (CASnr: 86-73-7) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae												
<i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	820	Bisson et al, 2000
<i>Pseudokirchneriella subcapitata</i>		N	S	98.6	-	-	-	7 d	EC10	cell number	1400	Finger et al, 1985
<i>Pseudokirchneriella subcapitata</i>		N	S	98.6	-	-	-	7 d	NOEC	biomass, cell number, chlorophyll	1670	Finger et al, 1985
Cyanophyta												
<i>Anabaenaeflos-aqua</i>		Y	S	analytical	am	-	-	2 w	NOEC	growth	<110	Bastian & Toetz, 1982
<i>Anabaenaeflos-aqua</i>		Y	S	analytical	am	-	-	2 w	EC10	growth	430	Bastian & Toetz, 1982
Macrophyta												
<i>Chara</i> sp.	pre-emergence	N	S	98.6	nw	-	-	21 d	NOEC	weight	14000	Finger et al, 1985
<i>Chara</i> sp.	pre-emergence	N	S	98.6	nw	-	-	21 d	EC50	weight	20300	Finger et al, 1985
<i>Chara</i> sp.	21 d, post-emergence	N	S	98.6	nw	-	-	21 d	NOEC	weight	>35000	Finger et al, 1985
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC7	growth	2000	Huang et al, 1997ab
<i>Lemna gibba</i>		N	S	-	am	-	-	8 d	EC30	growth	2000	Huang et al, 1997ab
Insecta												
<i>Chironomus riparius</i>	larvae	Y	IF	98.6	nw	7.2-7.4	270	30 d	NOEC	emergence	290	Finger et al, 1985
<i>Diporeia</i> sp.	1-2 mm, juvenile, 5-11 m	Y	R	>98%	nw	8.1-8.3	165-250	28 d	LC50	mortality	542.7	Landrum et al, 2003
Crustacea												
<i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	25	Bisson et al, 2000
<i>Daphnia magna</i>		Y	IF	98.6	nw	7.2-7.4	270	21 d	NOEC	reproduction	62.5	Finger et al, 1985
Pisces												
<i>Lepomis macrochirus</i>	fingerlings, 0.74 g	Y	CF	98.6	nw	7.2-7.4	270	30 d	NOEC	growth	125	Finger et al, 1985
<i>Lepomis macrochirus</i>	fingerlings, 0.74 g	Y	CF	98.6	nw	7.2-7.4	270	30 d	NOEC	mortality	250	Finger et al, 1985

Acute toxicity of fluorene (CASnr: 86-73-7) to marine organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Salinity (‰)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Annelida												
<i>Neanthes arenaceodentata</i>		Y	S	≥98%	am	-	32	96 h	LC50	mortality	1000	Rossi & Neff, 1978
Crustacea												
<i>Artemia salina</i>	<1 d	N	S	analytical	-	-	-	3 h	LC50	mortality	3000	Kagan et al, 1987

Acute toxicity of indeno[1,2,3-cd]pyrene (CASnr: 193-39-5) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Crustacea <i>Daphnia magna</i>	<24 h	Y	S	-	am	7.8±0.2	250±30	48 h	EC50	immobility	>357	Bisson et al, 2000

Chronic toxicity of indeno[1,2,3-cd]pyrene (CASnr: 193-39-5) to freshwater organisms.

Species	Species properties	Analyzed	Test type	Substance purity	Test water	pH	Hardness (mg CaCO ₃ /L)	Exp. time	Criterion	Test endpoint	Value (µg/L)	Reference
Algae <i>Pseudokirchneriella subcapitata</i>		Y	S		am	-	215	72 h	EC10	growth	1.5	Bisson et al, 2000
Crustacea <i>Ceriodaphnia dubia</i>	<24 h	Y	R	-	nw	8.1±0.4	240±40	7 d	EC10	reproduction	0.27	Bisson et al, 2000

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