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ANNEX XV TRANSITIONAL DOSSIER

SUBMITTED BY:	The Netherlands
DATE:	November 2008

SUBSTANCE NAME:	Coal Tar Pitch, High Temperature
IUPAC NAME:	Coal Tar Pitch, High Temperature
EC NUMBER:	266-028-2
CAS NUMBER:	65996-93-2

PREFACE

Coal tar pitch, high temperature (CTPHT) was a priority substance on the 3rd priority list in the framework of Council Regulation (EEC) 793/93 (EC, 1993). The work on this substance was not completed within this regulation, therefore in accordance with Article 136(3) of the REACH regulation (EC, 2006b) a transitional dossier has been made by the Netherlands. The Annex XV restriction proposal format has been used on request of the ECHA. It is decided to include all information from the previous dangerous substances regulation for completeness and transparency reasons.

The knowledge and data regarding the potential risk(s) of CTPHT to the environment and human health that are reported in this transitional dossier (see Section B) are copied from the human health and environment risk assessment reports (The Netherlands, 2008ab), which were both endorsed under Regulation 793/93. In addition, the risk reduction strategy document with respect to workers (The Netherlands, 2008c) is included (see Section H.1). This risk reduction strategy document was endorsed at the 15th RRSM in April 2008. The risk assessment for the environment was not finished until May 2008, which prevented the preparation of the risk reduction strategy for the environment in time to finalize the work on CTPHT in the framework of Council Regulation (EEC) 793/93. Conform Article 136(3c) of the REACH regulation (EC, 2006b) this dossier considers how environmental risks should be addressed. This risk reduction strategy is a new part in this dossier (incorporated in Sections B.11 - E)

Considerations about the risk assessment of CTPHT

In the human health and environment risk assessment reports (The Netherlands, 2008ab) exposure to Polycyclic Aromatic Hydrocarbons (PAHs) is often used as an indicator of exposure to CTPHT, based on the following argumentation, which is copied from these risk assessment reports.

Coal tar pitch, high temperature (CTPHT) 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), in the air (IARC, 1985). Since PAHs are among the major components of CTPHT, and some individual PAHs are proven animal carcinogens, PAH levels are considered as a suitable indicator of exposure to CTPHT (WATCH, 2000). Existing exposure information suggests that the airborne concentration of benzo(a)pyrene correlates well with the concentration of total PAHs for most workplaces. Based on these findings and the availability of exposure data, the Working group on Assessment of Toxic Chemicals (WATCH) from the Health and Safety Executive (HSE, UK) has pinpointed benzo(a)pyrene as the most suitable marker for assessing exposure to PAHs[1] for coal tar pitch volatile (CTPV) industries (WATCH, 2000). As such, in conducting this exposure assessment to CTPHT, exposure to benzo(a)pyrene has been adopted as the primary indicator in the way described in Section B.9.

The database on possible health hazards induced by CTPHT is rather limited, and it is, therefore, hardly possible to perform a full risk 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 striking hazard associated with CTPHT. This is attributed to the presence of the PAHs in CTPHT, as indicated above. 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 effect,

[1] These are 11 PAHs identified by HSE as having the greatest carcinogenic potential of the PAH family of compounds.

quite possibly even to zero. Therefore, in view of the limited database, it is decided that this transitional dossier will focus on the carcinogenic and mutagenic properties, using the best-studied PAH benzo(a)pyrene as a guidance substance (see for further information Sections B.5, B.5.8, and B.10.1.1).

For the environmental risk assessment the use of one single marker substance has little meaning. It is well realised that each of the many substances that constitute coal tar pitch may be relevant for the receiving environment. It is however the Rapporteur's opinion that the assessment should be focused on the risk of the emission of polycyclic aromatic hydrocarbons (PAHs) only, since this was the main reason to put CTPHT on the 3rd priority list (EC, 1993). Moreover, based on the available information, it is only for the EPA 16 homocyclic PAHs that sufficient effect and exposure data are available. It is for this reason that the risk assessment of CTPHT for the environment is restricted to this group of PAHs and regards them as representative for the total emission of PAH, accepting that the potential risk of CTPHT might be underestimated.

Since so many unintentional sources contribute to the total emission of PAHs into the environment (see Section B.8.2.2), which by extension are not related to production and use of CTPHT, the risk assessment will only be focussed on the PAHs that is emitted by producers and the downstream users of CTPHT on a local scale. To put this emission into perspective, the calculated local concentrations will be related to the background levels measured in urban and rural areas.

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A. PROPOSAL

A.1 Proposed restriction(s)

A.1.1 The identity of the substance(s)

Chemical name: Coal Tar Pitch, High Temperature

IUPAC Name: Coal Tar Pitch, High Temperature

EC Number: 266-028-2

CAS Number: 65996-93-2[2]

A.1.2 Conditions of restriction(s)

No restrictions are proposed for CTPHT.

A.2 Summary of the justification

A.2.1 Identified hazard and risk

CTPHT is a priority substance on the 3rd priority list (EC, 1993). CTPHT was selected as a priority substance because of the risk of the emission of polycyclic aromatic hydrocarbons (PAHs).

For all worker scenarios there is a need for limiting the risks (risk reduction measures which are already being applied shall be taken into account) The risk is related to skin sensitisation, mutagenicity and carcinogenicity (conclusion iii).

For consumers no exposure was identified and thus no risks are expected.

Table A.2.1 gives the overall results of the risk characterisation for environment and human exposed via the environment. For all sites and scenarios there is a need for limiting the risks (risk reduction measures which are already being applied shall be taken into account) (conclusion iii).

Table A.2.1. Summary results of risk characterisation of CTPHT for the environment and human indirectly exposed via the environment.

	Air [a)]	Water [b)]	Sediment [b)]	Terrestrial [b)]	Human via environment [b,c)]	Secondary poisoning [a)]	Exposure scenario
Production of CTPHT	x	iii	iii	ii	iii	x	site specific
Primary aluminium production [d)]	x	iii	iii	ii	iii	x	site specific
Graphite electrode production	x	ii [e)]	ii [e)]	ii	iii	x	generic
Ferro-alloy industry applying electric arc furnaces with Søderberg electrodes	x	iii	iii	ii	iii	x	generic
Regional background	x	iii	iii	ii	iii	x	

[a)] No Predicted No Effect Concentration (PNEC) available, so no risk characterisation possible (indicated by x); [b)] ii = there is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already, iii = there is a need for limiting the risks, risk reduction measures which are already applied shall be taken into account; [c)] Most critical endpoint; [d)] Significantly higher PEC than regional levels for some of the sites, but there are large differences in emissions and related risks; [e)] No emissions to water.

[2] The Rapporteur notices that the CAS registry number is not used by CAS. The effect may be that the 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 some relevant data are not found with these searches and therefore not discussed in this Transitional Dossier.

It is important to notice that also for the regional background a risk is identified. For most scenarios at risk the exposure lies in the same order of magnitude as the regional background. Significantly higher concentrations are calculated for the primary anode production.

For CTPHT production risks are identified at 1 site and for primary aluminium production risks are identified at 12 primary aluminium plants and at 1 anode production site.

Highest concern is identified for humans exposed via the environment.

A.2.2 Justification that action is required at Community-wide basis

Risk assessment of PAHs involves many (diffuse) sources, which are unlikely to be adequately controlled by the individual Member States and thus appears to require Community-wide action. However, the production and use of CTPHT only adds a small contribution to the emission of PAHs. In addition, the local scale of CTPHT related risks suggests that implementation of IPPC and national legislation of the Member States appears to be sufficient to adequately control these risks, indicating that there is no clear basis for Community-wide action for CTPHT risks other than adjustment of the BAT reference document for the IPPC legislation. It should be emphasized, however, that this conclusion only holds for risks associated with CTPHT. For a proper risks assessment of PAHs in general, Community-wide action appears to be inevitable, because of the many (diffuse) sources of PAHs.

In addition, for workers it is proposed to establish at Community level occupational exposure limit values for CTPHT according to Directive 98/24/EEC (EC, 1998) or Directive 2004/37/EC (EC, 2004b) as appropriate.

A.2.3 Justification that the proposed restriction is the most appropriate measure

No restrictions are proposed for CTPHT.

It should be noted, however, that for workers it is proposed to establish at community level occupational exposure limit values for CTPHT according to Directive 98/24/EEC (EC, 1998) or Directive 2004/37/EC (EC, 2004b) as appropriate.

In addition, to enhance the effectiveness of the IPPC legislation, it is suggested that a revision of the BAT reference document should incorporate a refinement of emission limits, which includes a limit for the total PAH concentrations, next to the limits for the individual PAHs that are included at present. In addition it is suggested to incorporate limits per time unit next to the limits per production unit that are included at present.

B. INFORMATION ON HAZARD AND RISK

The information in this section is copied from the Risk Assessment Reports for Coal Tar Pitch, High Temperature for Human Health and Environment (The Netherlands, 2008ab).

B.1 Identity of the substance(s) and physical and chemical properties

B.1.1 Name and other identifiers of the substance(s)

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 (EC, 1976b) 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, 2001). Some of the coal tar pitches present in these lists are shown in Table B.1.1.

Table B.1.1 Different types of pitches as defined in official listings.

CAS number	Product	List
61789-60-4	Pitch	EC (1976b)
65996-93-2	Pitch, coal tar, high temperature	EC (1976b); US-EPA (2001)
92061-94-4	Residues (coal tar), pitch distillation	EC (1976b); US-EPA (2001)
94114-13-3	Pitch, coal tar, high temperature, secondary	EC (1976b); US-EPA (2001)
121575-60-8	Pitch, coal tar, high temperature, heat treated	EC (1976b); US-EPA (2001)

The feedstock for the production of coal tar pitch high temperature (CTPHT) is tar, coal, high-temperature with CAS # 65996-89-6. This "substance" is defined in EC (1976b) 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 composed primarily of a complex mixture of condensed ring aromatic hydrocarbons and may contain minor amounts of phenolic compounds and aromatic nitrogen bases'. Coal tars are condensation products obtained during the production of coke an/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.

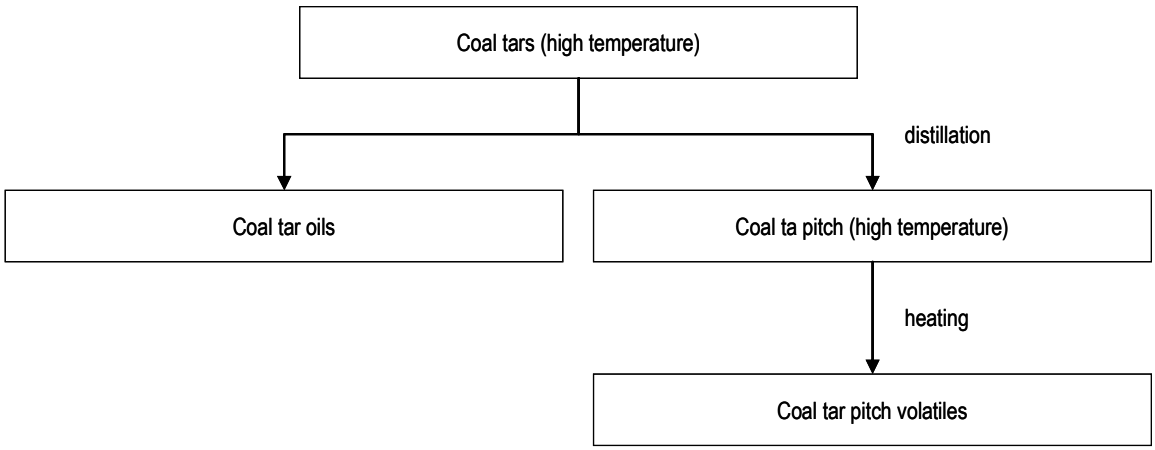


Figure B.1.1. Origin of high temperature coal tar products.

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 it's products are heated (HSDB, 2004). Figure B.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 J.2).

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

CAS Number: 65996-93-2[3]

EINECS Number: 266-028-2

IUPAC Name: not applicable

Molecular formula: not applicable; coal tar pitch 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

B.1.2 Composition of the substance(s)

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).

B.1.2.1 Purity/Impurities, Additives

The content of the sixteen EPA PAHs in pitch used for impregnation and binding is presented in Table B.1.2. Most relevant for the risk assessment is the composition for binder pitch, as it is the

[3] The Rapporteur notices that the CAS registry number is not used by CAS. The effect may be that the 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 some relevant data are not found with these searches and therefore not discussed in this Transitional Dossier.

main source for the production of anodes and electrodes (see Section B.2). The structural formulae of the 16 EPA PAHs are shown in Figure B.1.2 (those marked with * belong to the Borneff 6).

Table B.1.2. PAH content in CTPHT (16 EPA PAH and other aromatic hydrocarbons).

	Impregnation Pitch (mg/kg)	Binder Pitch (mg/kg)
Aromatic hydrocarbons		
Ethylbenzene	n.d.	n.d.
m-/p-Xylene	n.d.	n.d.
1,2,4-Trimethylbenzene	n.d.	n.d.
3- Ethyltoluene	n.d.	n.d.
1,3,5-Trimethylbenzene	n.d.	n.d.
1,2,3-Trimethylbenzene	n.d.	n.d.
Indene	n.d.	n.d.
1,2,4,5-Tetramethylbenzene	n.d.	n.d.
Naphthalene*	n.d.	n.d.
2,4,6- Trimethylbenzene	n.d.	n.d.
2- Methylnaphthalene	n.d.	n.d.
1- Methylnaphthalene	n.d.	n.d.
Biphenyl	n.d.	n.d.
Dimethylnaphthalenes	n.d.	n.d.
Acenaphthylene [a)]	n.d.	n.d.
Acenaphthene [a)]	390	432
Fluorene [a)]	144	472
2-Methylfluorene	50	112
1-Methylfluorene	n.d.	61
Phenanthrene [a)]	3874	6299
Anthracene [a)]	737	1311
3-Methylphenanthrene	n.d.	n.d.
2-Methylphenanthrene	n.d.	n.d.
2-Methylanthracene	n.d.	n.d.
Cyclopenta(def)phenanthrene	918	821
4-Methylphenanthrene	n.d.	n.d.
1-Methylphenanthrene	n.d.	n.d.
Fluoranthene [a)]	17389	10789
Acephenanthrylene	828	386
Pyrene [a)]	14849	9449
Benzo(a)fluorene	4509	1974
Benzo(b)fluorene	4306	2456
Benz(a)anthracene [a)]	15008	7715
Chrysene [a)]	14041	8053
Benzo(b)fluoranthene [a)]	17408	12131
Benzo(k)fluoranthene [a)]	8704	6065
Benzo(e)pyrene	11891	8976
Benzo(a)pyrene [a)]	12924	10021
Perylene	5014	3167

	Impregnation Pitch (mg/kg)	Binder Pitch (mg/kg)
Dibenz(a,h)anthracene [a)]	2209	1749
Indeno(123-cd)pyrene [a)]	11106	9061
Benzo(ghi)perylene [a)]	9945	8664
Anthantrene	4581	3464
Tar acids / phenolics		
Phenol	n.d.	n.d.
o-Cresol	n.d.	n.d.
m-/p-Cresol	n.d.	n.d.
2,6-Xylenol	n.d.	n.d.
2,5-Xylenol	n.d.	n.d.
3,5-Xylenol	n.d.	n.d.
3,4-Xylenol	n.d.	n.d.
4-Isopropylphenol	n.d.	n.d.
2,3,5-Trimethylphenol	n.d.	n.d.
3,4,5-Trimethylphenol	n.d.	n.d.
Tar bases / nitrogen-containing heterocycles		
Benzonitrile	n.d.	n.d.
o-Tolunitrile	n.d.	n.d.
m-Tolunitrile	n.d.	n.d.
p-Tolunitrile	n.d.	n.d.
Quinoline	n.d.	n.d.
Isoquinoline	n.d.	n.d.
Indole	n.d.	n.d.
Chinaldine	n.d.	n.d.
Acridine	242	264
Carbazole	1556	1664
Sulfur-containing heterocycles		
Thionaphthene	n.d.	n.d.
Dibenzothiophene	269	438
Oxygen-containing heterocycles / furans		
Dibenzofuran	n.d.	215
Total	162,892	116,209

Method used is DIN 51920 (DIN, 1984) for softening point and ISO 6998 (ISO, 1997) for coking value. [a)] These are the 16 EPA PAH.

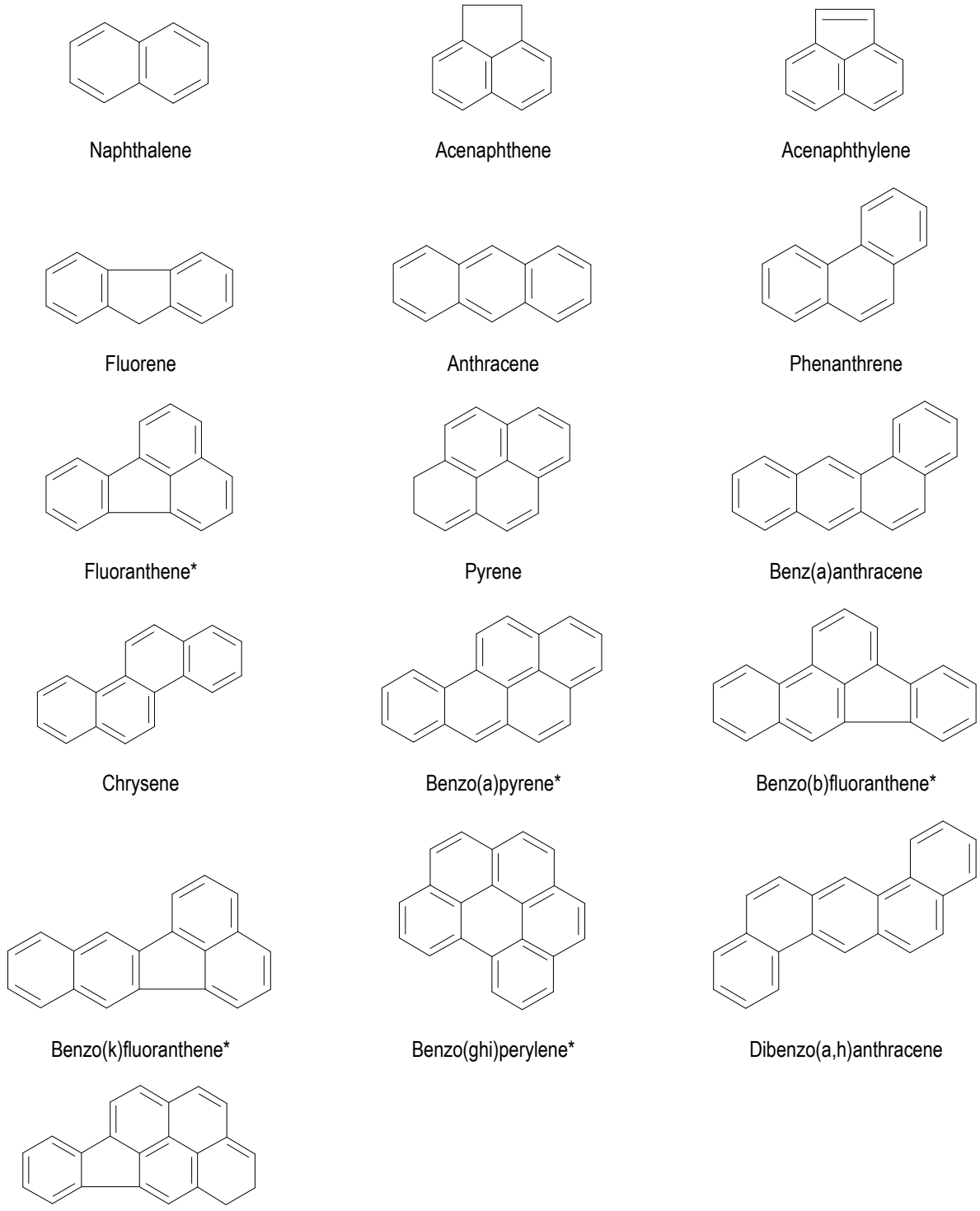
B.1.3 Physico-chemical properties

The physico-chemical characteristics of pitch are presented in Table B.1.3. Because of the importance for the classification and labelling, the water solubility of CTPHT is discussed in detail. In Table B.1.4 and in Table B.1.5 the relevant data on water solubility of pitch are summarized. In Table B.1.6 the physico-chemical properties of the individual EPA PAHs are presented.

B.1.3.1 Water solubility

The lowest values were found after elution from a percolation column (Table B.1.4, No. 1 and 2) and after settling following mechanical agitation (Table B.1.4, No. 5). In the first case, the surface of the 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 pitch (Table B.1.4: 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 B.1.4, No. 7). The highest value found was about 140 µg/L at an elevated temperature (Table B.1.4, No. 4). Overall, the data show high consistency taking into account the different test and analytical conditions.



PAH marked with * belong to the Borneff 6.

Figure B.1.2. Structural formulae of polycyclic aromatic hydrocarbons covered in this risk assessment report.

B.1.3.2 Multiple elution

Within the scope of a comprehensive analytical programme on the availability of PAH from pitch in water (Rütgers VFT, 1999ab), 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. 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. The following table 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 B.1.4, 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 B.1.4, compare No. 2 with No. 1, No. 6, or No. 7).

Table B.1.3. Physico-chemical properties of CTPHT.

REACH ref Annex, §	Property	IUCLID section	Value	Comment/reference
VII, 7.1	Physical state	3.1	black solid	at 20°C and 101.3 KPa
VII, 7.2	Melting point	3.2	65-150 °C	softening range (CCSG, 2006a)
VII, 7.3	Boiling point	3.3	>360 °C	at 1013 hPa
VII, 7.4	Relative density	3.4 density	1.15-1.40 g/m^3	at 20 °C (ASTM, 2004; CCSG, 2006a)
VII, 7.5	Vapour pressure	3.6	<0.1 <10	at 20 °C at 200 °C (OECD, 1995; CCSG, 2006a)
VII, 7.6	Surface tension	3.10	--	not applicable
VII, 7.7	Water solubility	3.8	~0.040 mg/L	16 EPA PAHs, at a loading of 10 g/L at 22 °C (Rütgers VFT 1999ab)
VII, 7.8	Partition coefficient n-octanol/water (log value)	3.7 partition coefficient	--	not applicable
VII, 7.9	Flash point	3.11	>250 °C	(ISO, 2002; CCSG, 2006a)
VII, 7.10	Flammability	3.13	non flammable	84/449/EEC (EC, 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
VII, 7.11	Explosive properties	3.14	not explosive	(CCSG, 2006a)
VII, 7.12	Self-ignition temperature		>450 °C	at 1013 hPa (DIN, 2003; CCSG, 2006a)
VII, 7.13	Oxidising properties	3.15	not oxidizing	(CCSG, 2006a)

Table B.1.4. 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-PAH	Elution column	Rütgers VFT, 1999ab
2	Granules	100	27	25	EPA-PAH	Elution column	Rütgers VFT, 2000
3	Molten pitch	42	91	22-25	EPA-PAH	Hot injection into the water phase and 24-h settling	Rütgers VFT, 1997
4	Powder	10	137	30	EPA-PAH	24-h stirring, membrane filtration (0.45 µm)	UBA, 1997
5	Powder	10	22	30	EPA-PAH	24-h stirring, 24-h settling, centrifugation	UBA, 1997
6	Powder	10	67	22	EPA-PAH	8-d stirring, membrane filtration (0.45 µm)	UBA, 1999
7	Powder	10	54-75	22	EPA-PAH	8-d stirring (6x), 6-fold repetitive water extraction, membrane filtration (0.45 µm)	UBA, 1999

[a] WSF = Water-Soluble Fraction based on the16 EPA PAHs.

Table B.1.5. 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-PAH	Content in 10 g Pitch (rounded values)		Concentration (µg/L) (rounded values)		
	(µg)	(%)	1st cycle	15th cycle	39th cycle
Naphthalene	100	0.001	1.5	0.8	0.1
Acenaphthylene	n.d.	n.d.	n. d.	n.d.	n.d.
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
Dibenzo(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(123-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 (1999ab).

B.1.4 Justification for grouping

Not applicable

Table B.1.6. Physico-chemical 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 (kg/L)	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	208-96-8	$C_{12}H_8$	154.2	96	278	3910 [b]	4.00 [f]	$3.3 \cdot 10^{-1}$ [i]	0.899	14.3 [m]
Acenaphthylene	91-20-3	$C_{12}H_{10}$	150.2	92	279	16100 [b]	3.62 [g]	$4.8 \cdot 10^{-1}$ [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]	$8.3 \cdot 10^{-2}$ [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]	$9.4 \cdot 10^{-4}$ [j]	1.283	4.3 [m]
Phenanthrene	85-01-8	$C_{14}H_{10}$	178.2	100.5	340	974 [a]	4.57 [e]	$2.6 \cdot 10^{-2}$ [j]	0.980	3.7 [m]
Fluoranthene	206-44-0	$C_{16}H_{10}$	202.3	108.8	375	200 [a]	5.20 [e]	$1.2 \cdot 10^{-3}$ [i]	1.252	1.1 [o]
Pyrene	129-00-0	$C_{16}H_{10}$	202.3	156	360	125 [a]	4.98 [f]	$1.0 \cdot 10^{-3}$ [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]	$7.6 \cdot 10^{-6}$ [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]	$5.7 \cdot 10^{-7}$ [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]	$7.3 \cdot 10^{-7}$ [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]	$3.3 \cdot 10^{-6}$ [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]	$1.3 \cdot 10^{-7}$ [l]	-	0.043 [o,r]
Benzo(ghi)perylene	191-24-2	$C_{22}H_{12}$	276.3	277	545 [j]	0.14 [a]	6.22 [e]	$1.4 \cdot 10^{-8}$ [k]	1.329	0.027 [o,r]
Dibenzo(a,h)anthracene	53-70-3	$C_{22}H_{14}$	278.4	266.6	524	0.82 [b]	6.50 [f]	$3.7 \cdot 10^{-10}$ [k]	1.282	$1.3.10^{-4}$ [q]
Indeno(123-cd)pyrene	193-39-5	$C_{22}H_{12}$	276.3	163.6	536	0.1 [c]	6.58 [g]	$1.7 \cdot 10^{-8}$ [l]	-	0.046 [q]

The data presented in the table were taken form 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(123-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.

B.2 Manufacture and uses

B.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' (EC, 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 B.2.1. 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 B.2.1 presents the market data for coal tar pitch within the European Union.

Figure B.2.1 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.

Table B.2.1. 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

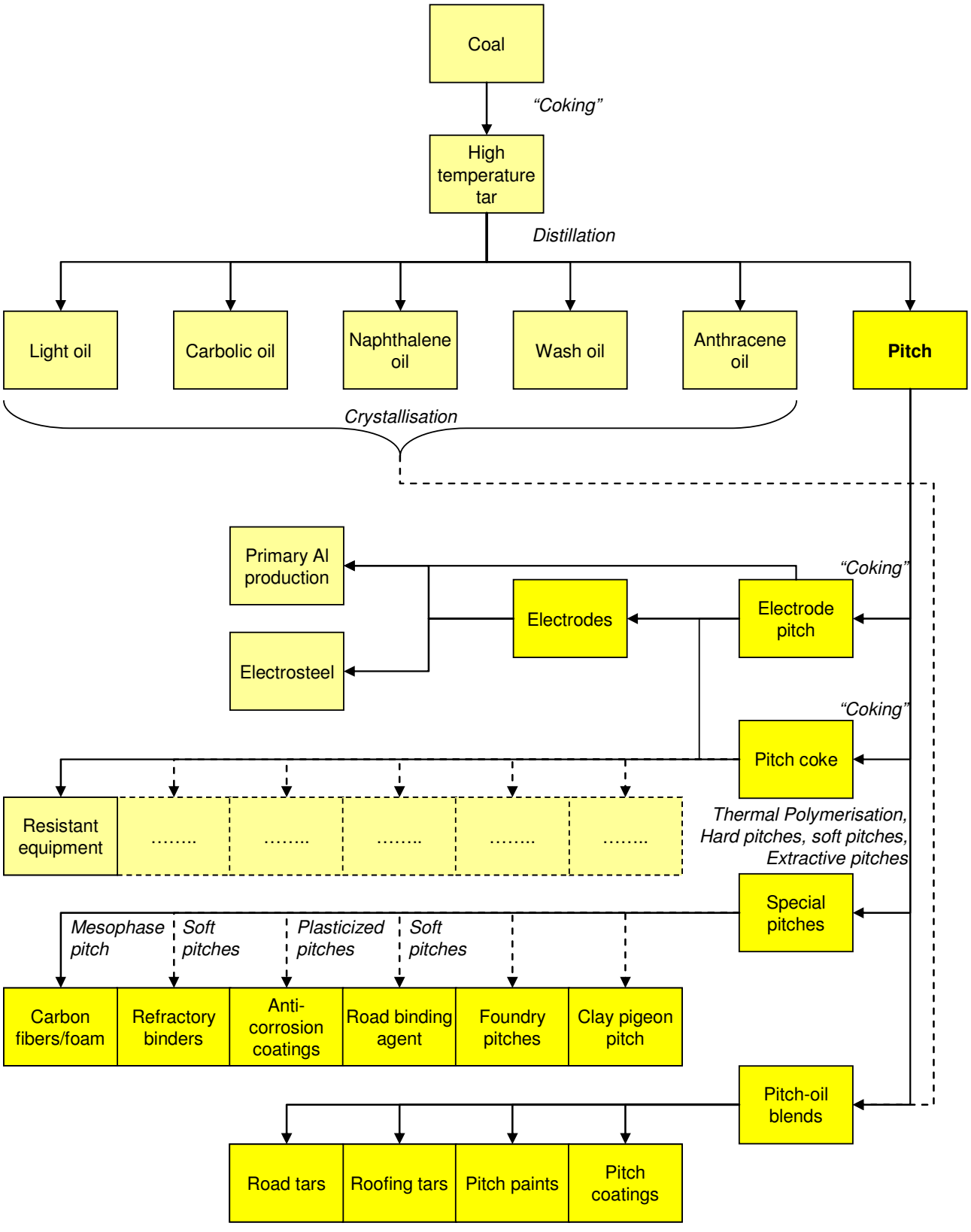


Figure B.2.1. Schematic overview of the production of CTPHT and the subsequent processing and formulation possibilities.

B.2.2 Uses

B.2.2.1 Introduction

Coal tar pitch is mainly used as a binding agent in the production of carbon electrodes, anodes and Søderberg 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 B.2.2.

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

Table B.2.2. 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.

B.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 Søderberg 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 Søderberg 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, 2001a). 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, 2001a).

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.

B.2.2.3 Use as binding agent in the production of other products

B.2.2.3.1 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, 2001a). 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 Section B.2.2.3.3. High purity graphite is produce in a similar way but the graphitising process is used to remove included impurities such as metals.

B.2.2.3.2 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 & Granitzkin, 2002).

B.2.2.3.3 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).

B.2.2.3.4 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.

B.2.2.3.5 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., 2008). 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.

B.2.2.3.6 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.

B.2.2.3.7 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 B.2.2 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.

<u>Physically drying pitch coatings</u>

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.

<u>Pitch combination coatings</u>

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.

B.2.2.4 Application of carbon and graphite electrodes

B.2.2.4.1 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 Søderberg (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, 2001a). 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, 2001a).

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 Søderberg technology in the total amount of primary aluminium produced within the European Union is 10 percent, see Table B.2.3.

The Søderberg 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 B.2.3. 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 Søderberg production in Europe; [c] Vertical Stud Søderberg; [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.

B.2.2.4.2 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 of 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 B.2.4. 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
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 B.2.4.

Table B.2.5 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 B.2.5).

B.2.3 Uses advised against by the registrants

Not applicable.

B.2.4 Description of targeting

Not relevant.

Table B.2.5. 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.

B.3 Classification and labelling

B.3.1 Classification in Annex I of Directive 67/548/EEC

Human Health

Classification Carc. Cat. 2, R45

Labelling T

R45

S53-45

Note H

It should be noted that a more extended classification proposal for human health was agreed by TC C&L in October 2006. This proposal, in which Note H was deleted, has been forwarded to ECHA and most probably will be agreed in the 1st ATP to Annex VI of CLP, once entered into force. The proposal reads as follows:

Classification Carc. Cat. 1, R45; Muta. Cat. 2, R46; Repr. Cat. 2, R60-61; Xi, R41 R43

Labelling T

R45, R46, R60-61, R41, R43

S53-45

B.3.2 Self classification(s) and labelling

Next to the (extended) classification from Section B.3.1, the following classification proposal for environment was agreed by TC NES, but not yet discussed in TC C&L:

Classification N, R50/53

Labelling N

R50/53

Justification

CTPHT is a complex mixture containing many compounds, such as homo- and heterocyclic aromatic hydrocarbons. As a consequence, it is very difficult to classify CTPHT on the basis of the individual compounds. In addition, not all the substances can be analyzed when diluted in water. Furthermore, the different CTPHT components influence each others 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, 2002). 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.

Studies were available in which the WAF concept was applied in the preparation of aqueous extracts of CTPHT. The most important short-coming is that the tests 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 limited data is available on the preparation and aquatic toxicity

testing of WAFs of CTPHT. Hence, it proved to be impossible to draw any definitive conclusions on the aquatic classification and labelling of CTPHT taking the preferred WAF approach.

Table B.3.1. Aquatic hazard classification of CTPHT.

16 EPA-PAH	Content in CTPHT (% w/w)	Lowest acute aquatic toxicity value (E/LC_{50} or EC_{10}) in mg/L	Proposed classification of CTPHT [b)]
Naphthalene	0.0095	> 4.3	not classified
Acenaphthylene	0	1.8	not classified
Acenaphthene	0.043	0.58	not classified
Fluorene	0.052	0.025	R52/53
Phenanthrene	0.69	0.051	N; R51/53
Anthracene	0.14	0.001	N; R50/53
Fluoranthene	1.3	0.0001	N; R50/53
Pyrene	1.6	0.00023	N; R50/53
Benz(a)anthracene	0.81	0.0018	N; R50/53
Chrysene	0.77	0.0007	N; R50/53
Benzo(b)fluoranthene	1.2	> water solubility [c)]	not classified
Benzo(k)fluoranthene	0.59	0.00065	N; R50/53
Benzo(a)pyrene	1.0	0.058	N; R51/53
Dibenzo(a,h)anthracene	0.15	0.0018	N; R51/53
Benzo(ghi)perylene	0.83	> water solubility [c)]	not classified
Indeno(123-cd)pyrene	0.81	0.00027	N; R50/53
Total	11	-	N; R50/53

[a)] Weight based percentage; [b)] Classification based upon the Dangerous Preparations Directive (EC, 1999b); [c)] Acute toxicity reference value is higher than water solubility.

Given the considerations mentioned above, it was decided to base the aquatic classification and labelling of CTPHT upon the rules laid down in the Dangerous Preparations Directive (DPD) (1999/45/EEC; EC, 1999b), which is the first choice as a suitable alternative in this case. CTPHT is considered as a 'preparation' in this perspective. In the last Adaptation to Technical Progress of the DPD (2006/8/EC; EC, 2006a) concentration limits are provided for classification of preparations containing substances that are very toxic to the aquatic environment (N;R50/53). The 16 individual EPA PAHs were analysed with respect to their acute aquatic effects data and the lowest available EC_{50} or LC_{50} was chosen as a point of departure for aquatic hazard classification. The effects data are described in more detail in Section B.7.1.These lowest acute toxicity data were linked to the concentration of each specific PAH in CTPHT (data for binder pitch taken from Table B.1.2). Finally, for each PAH an individual conclusion is drawn on the aquatic hazard classification of CTPHT. The results are summarised in Table B.3.1 show that for seven of the individual PAHs present in CTPHT, the conclusion is that CTPHT as a 'preparation' should be classified with N;R50/53. For three PAHs the classification would be N;R51/53, for one PAH (*i.e.* fluorene) R52/53 is concluded and for five PAHs the conclusion is 'not classified'. The latter is 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 in combination with low content of the specific PAH in CTPHT (*i.e.* naphthalene, acenaphthylene and acenaphthene). It should be noted that this classification proposal is based on the 16 EPA PAHs, whereas CTPHT also contains other compounds that could be classifiable.

Evaluation of Water-Accommodated Fraction (WAF) data

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*), 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/cm^2, 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 LC_{50} value was between 100 and 1000 mg/L for all species. With the dilution method the LC_{50} was > 1000 mg/L for *O. latipes* (other species were not tested).

Additional information is available concerning the solubility of pulverized CTPHT (see Section B.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) 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 was equal at loadings of 100 and 10,000 mg/L, it is assumed that at both concentrations an excess of PAHs is available, in other words, that dissolution in time is not limited by the availability of PAHs from the amount of pitch applied. 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 LC_{50} for fluoranthene (*i.e.* 0.1 µg/L) exceeds the concentration in the force percolate 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.

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.

B.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.

B.4.1 Degradation

B.4.1.1 Atmospheric degradation (or fate in the atmosphere)

Photolysis in the troposphere results in the formation of reactive hydroxyl and nitrate (NO_3) radicals and ozone (O_3), which reacts as oxidizing agent with organic compounds, like PAHs. These radical and ozone reactions comprise mainly degradation of gas-phase PAHs (Calvert *et al.*, 2002). Particle-associated PAHs are expected to degrade in air predominantly via direct photolysis by light with a wavelength < 290 nm (Kamens *et al.*, 1988), although reaction with ozone will also occur (Peters & Seifert, 1980; Grosjean *et al.*, 1983; Pitts *et al.*, 1986; Coutant *et al.*, 1988).

In the atmosphere PAHs are partitioned between the gas and particle phases, with the gas-particle partitioning depending on a number of factors, including the liquid-phase (or sub-cooled liquid-phase) vapour pressure of the PAH at the ambient atmospheric temperature, the surface area of the particles per unit volume of air, and the nature of the particle (Wania & Mackay, 1996; Pankow, 1987; Bidleman, 1988). As a first approximation, chemical compounds with liquid-phase vapour pressure of $P_L < 10^{-5}$ Pa at the ambient atmospheric temperature are present in the particle phase, and those with values of $P_L > 10^{-2}$ Pa at the ambient atmospheric temperature are essentially totally in the gas phase (Arey & Atkinson, 2003). As shown in measurements performed in Norway (Oslo), Germany (Bayreuth) and California USA (Torrance), the 2-4 ring PAHs with vapour pressures $\geq 10^{-4}$ Pa are largely gas-phase species, whereas PAHs with 4 rings or more, with vapour pressure $< 10^{-4}$ Pa are particle-associated (see Table B.4.1).

Table B.4.1. Summary of gas-particle phase partitioning.

PAH (number of rings)	Vapour pressure (Pa) [a]	Observed % in particulate phase			
Naphthalene (2)	$1.1 \cdot 10^{1}$	0 [b]			0 [e]
Acenaphthylene (3)	$1.3 \cdot 10^{-1}$				
Acenaphthene (3)	$4.0 \cdot 10^{-1}$				
Fluorene (3)	$1.1 \cdot 10^{-1}$	0 [b]			
Anthracene (3)	$8.7 \cdot 10^{-4}$	3 [b]			0.5 [e]
Phenanthrene (3)	$2.0 \cdot 10^{-2}$	3 [b]	12.4 [c]	1.9 [d]	0.4 [e]
Fluoranthene (4)	$6.0 \cdot 10^{-3}$	54 [b]	49.7 [c]	19.1 [d]	5.9 [e]
Pyrene (4)	$4.4 \cdot 10^{-4}$	57 [b]	61.4 [c]	29.6 [d]	7.5 [e]
Benz(a)anthracene (4)	$2.1 \cdot 10^{-6}$	97 [b]	89.4 [c]	62.7 [d]	
Chrysene (4)	$1.4 \cdot 10^{-6}$	99 [b]			
Benzo(b)fluoranthene(5)	$1.0 \cdot 10^{-6}$	100 [b]	92.2 [c]	92.3 [d]	
Benzo(a)pyrene (5)	$5.3 \cdot 10^{-8}$	100 [b]	100 [c]	100 [d]	98.3 [e]
Perylene (5)	$1.8 \cdot 10^{-8}$	100 [b]			90.0 [e]
Dibenzo(a,c]anthracene (5)	$5.7 \cdot 10^{-9}$	100 [b]			
Dibenzo(a,h)anthracene (5)	$4.9 \cdot 10^{-9}$	100 [b]	100 [c]	100 [d]	
Benzo(ghi)perylene (6)	$1.0 \cdot 10^{-8}$	100 [b]	100 [c]	100 [d]	

Summary as given in EC (2001b). [a] Vapour pressures taken from Neiderfellner *et al.* (1997) and Oja & Suuberg (1998); [b] Measurements made in Oslo, January/February 1979 (Thrane & Mikalsen, 1981); [c] Annual mean measurements made in Bayreuth, Germany, May 1995-April 1996 (Horstmann & McLachlan, 1998); [d] Summer mean measurements made in Bayreuth, Germany, May-October 1995 (Horstmann & McLachlan, 1998); [e] Measurements made in Torrance, California, February 1986 (Arey *et al.*, 1987).

Virtually all organic compounds react with OH-radicals, while alkenes also generally react at a significant rate with NO_3- or O_3-radicals (Atkinson, 1994). Among the semi-volatile PAHs, acenaphthylene, and to a lesser extent phenanthrene and perhaps pyrene, undergo 'alkene-like' reactions. NO_3-radical concentrations are highly variable because their formation requires the presence of both O_3 and NO_2. Furthermore, NO_3-radical reaction is only a night-time loss process because the NO_3-radical rapidly photolyses. Although reaction with NO_3 is generally not an

important degradation pathway for PAHs the yield of nitro-PAHs from certain PAHs make night-time NO_3 chemistry an important source of ambient nitro-PAHs (Arey & Atkinson, 2003). The calculated lifetimes are inversely proportional to the concentration of OH, NO_3 or O_3 assumed. The reaction rates of PAH with airborne OH-radicals measured under standard conditions shows that most of the calculated half-lives are one day or less. Table B.4.2 shows the representative lifetimes of some 2- to 4-ring PAHs with respect to gas-phase reaction with OH- and NO_3-radicals and O_3. From these half-lives the reaction rate constants for reaction with atmospheric OH-radicals (K_{OH}) used in the risk assessment are calculated. For the other PAH the reported K_{OH} in literature are used (Table B.4.3).

Table B.4.2. Representative lifetimes of some 2- to 4-ring PAHs with respect to gas-phase reaction with hydroxyl (OH) radicals, nitrate (NO_3) radicals and ozone (O_3).

PAH (number of rings)	Representative lifetime with respect to reaction with							
	OH [a,b]				NO_3 [a,c]		O_3 [a,d]	
	Summer		Winter					
Naphthalene (2)	12	hours	2.7	days	6.0	years	>80	days
Acenaphthylene (3)	2.6	hours	13	hours	24	minutes	43	minutes
Acenaphthene (3)	3.5	hours	18	hours	4.8	hours	>30	days
Fluorene (3)	1.8	days	9	days				
Anthracene (3)	2.1	hours	10	hours				
Phenanthrene (3)	9.0	hours	1.9	days				
Fluoranthene (4)	5.6	hours	1.2	days	340	days		
Pyrene (4)	5.6	hours	1.2	days	120	days		

Lifetimes as given in EC (2001b). [a] Lifetimes calculated using rate coefficients summarised by Atkinson & Arey (1994) and Brubaker & Hites(1998); [b] 24 hour-average summer and winter OH concentrations of $1 \cdot 10^6$ molecule/cm^3 (0.04 pptv) and $2 \cdot 10^5$ molecule/cm^3 (0.008 pptv) assumed for boundary layer UK (Collins *et al.*, 1995); [c] 24 hour-average NO_3 concentration of $1.2 \cdot 10^8$ molecule/cm^3 (5 pptv) assumed for boundary layer UK based on typical night-time values (Carslaw *et al.*, 1997). Note that the NO_3 concentration is very variable and may be significantly suppressed under polluted conditions. The reaction also requires the presence of NO_2, which is assumed to be present at a concentration of $2.5 \cdot 10^{11}$ molecule/cm^3 (10 ppbv), based on the average southern UK level (PORG, 1997); d) Typical UK background O_3 concentration of $7.5 \cdot 10^{11}$ molecule/cm^3 (30 ppbv) assumed (PORG 1997).

Table B.4.3. Selected reaction rate constants for reaction with atmospheric OH radicals molecule.

PAH compound	K_{OH} (cm^3·molecule^{-1}·s^{-1})	Reference
Naphthalene	$2.16 \cdot 10^{-11}$	EC (2001b)
Acenaphthene [a]	$1.00 \cdot 10^{-10}$	EC (2001b)
Acenaphthylene	$4.20 \cdot 10^{-9}$	EC (2001b)
Fluorene	$1.30 \cdot 10^{-11}$	EC (2001b)
Anthracene	$1.30 \cdot 10^{-10}$	EC (2001b)
Phenanthrene	$3 \cdot 10 \cdot 10^{-11}$	EC (2001b)
Fluoranthene	$5.00 \cdot 10^{-11}$	EC (2001b)
Pyrene	$5.00 \cdot 10^{-11}$	EC (2001b)
Benz(a)anthracene	$1.22 \cdot 10^{-10}$	Slooff *et al.* (1989)
Chrysene	$8.00 \cdot 10^{-11}$	SRC (2004)
Benzo(a)pyrene	$5.00 \cdot 10^{-11}$	SRC (2004)
Benzo(b)fluoranthene	$1.86 \cdot 10^{-11}$	SRC (2004)
Benzo(k)fluoranthene	$5.36 \cdot 10^{-11}$	SRC (2004)
Benzo(ghi)perylene	$8.69 \cdot 10^{-11}$	SRC (2004)
Dibenzo(a,h)anthracene	$5.00 \cdot 10^{-11}$	SRC (2004)
Indeno(123-cd)pyrene	$6.46 \cdot 10^{-11}$	SRC (2004)

[a] The reaction rate is recalculated so it matches the reaction rate constant for reaction with NO_3 radicals, see Table B.4.5.

Under environmental conditions, PAH of higher molecular mass are almost completely adsorbed onto fine particles, which reduces the degradation rate markedly. In a study of the rate of 18 PAH on 15 types of fly ash, carbon black, silica gel, and alumina, the PAH were stabilized, depending on the colour, which is related to the carbon content: the higher the carbon content, the more stable the PAH. The authors suggested that the radiation energy is adsorbed by the organic matter of particulates, and PAH therefore do not achieve the excited state in which they can be degraded (Behymer & Hites, 1988). Table B.4.4 shows the representative lifetimes of some particle adsorbed PAHs with respect to photolysis. In comparison, the half-lives for direct photolysis of various PAH adsorbed onto silica gel are in the range of hours (Vu Duc & Huynh, 1991). A two layer model has been proposed for the behaviour of naturally occurring PAH on airborne particulate matter, in which photo oxidation takes place in the outer layer, and much slower, 'dark' oxidation takes place in the inner layer (Valerio *et al.*, 1987). This model is in line with the results of Kamens *et al.* (1991), who reported that PAH on highly loaded particles degrade more slowly than those on particles with low loads. As PAH occur mainly on particulate matter with a high carbon content, their degradation in the atmosphere is slower than that of PAH in the vapour phase under laboratory conditions or adsorbed on synthetic material like alumina and silica gel that have no or a low carbon content.

Table B.4.4. Representative lifetimes of some surface-adsorbed PAHs with respect to photolysis under conditions representative of a cloudless sky over the southern UK.

PAH (number of rings)	Classification of ash [a]															
	"White group"				"Red group"				"Grey group"				"Black group"			
	Summer		Winter		Summer		Winter		Summer		Winter		Summer		Winter	
Fluoranthrene (4)	7.7	hr	23	hr	1.5	day	4.5	day	1.9	day	5.7	day	1.2	day	3.6	day
Pyrene (4)	3.7	hr	11	hr	17	hr	2.1	day	1.6	day	4.8	day	1.1	day	3.3	day
Benz(a)anthracene (4)	15	min	45	min	6.6	hr	20	hr	17	hr	2.1	day	1.1	day	3.3	day
Chrysene (4)	10	hr	1.3	day	2.6	day	7.8	day	2.3	day	6.9	day	1.0	day	3.0	day
Benzo(a)pyrene (5)	15	min	45	min	8.0	hr	1.0	day	18	hr	2.3	day	20	hr	2.5	day
Perylene (5)	24	min	1.2	hr	7.0	hr	21	hr	18	hr	2.3	day	21	hr	2.6	day
Benzo(ghi)perylene (6)	1.9	hr	5.7	hr	2.0	day	6.0	day	2.5	day	7.5	day	23	hr	2.9	day

Lifetimes as reported by EC (2001b). [a] The classification of the ash into four groups depends on the relative contents of 10 elements, which influences the colour of the substrate (Behymer & Hites, 1988). The photolysis lifetimes measured in that study have been scaled to provide values representative of 24-hour averaged conditions in the boundary layer over the southern UK.

B.4.1.2 Aquatic degradation (incl. sediment)

B.4.1.2.1 Abiotic

PAH are chemically stable, with no functional groups that results in hydrolysis. Under environmental conditions, therefore, hydrolysis does not contribute to the degradation of PAH (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 carries) 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.

B.4.1.2.2 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 (Coates *et al.*, 1997; Thierrin *et al.*, 1993), 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 CO_2 and H_2O (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 risk assessment it is decided to use the suggested mean half-lives by Mackay *et al.* (1992).

B.4.1.3 Degradation 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 (Kastner *et al.*, 1994; McGillivray & Shiarias, 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; Baudgrasset *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; Kastner *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 (Banerjee *et al.* 1995; Bossert & Bartha, 1986), 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 (Carmichael & Pfaender, 1997; Herbes & Schwall, 1978). 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 B.4.5). 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 B.4.5. 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 risk assessment 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 B.4.1.4).

B.4.1.4 Summary of environmental degradation

On the basis of model calculations, Mackay *et al.* (1992) ranked the 16 EPA PAH according to their persistence in, water, soil and sediment in different classes (Table B.4.6) which correspond to a specific half-live in these compartments (Table B.4.7). For the risk assessment these values are used.

Table B.4.6. 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
Dibenzo(a,h)anthracene	5	7	8
Indeno(123-cd)pyrene	5	7	8

[a)] classified based on information from literature by the rapporteur.

Table B.4.7. 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).

B.4.2 Environmental distribution

B.4.2.1 Adsorption

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.*, 1998).

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 B.4.1).

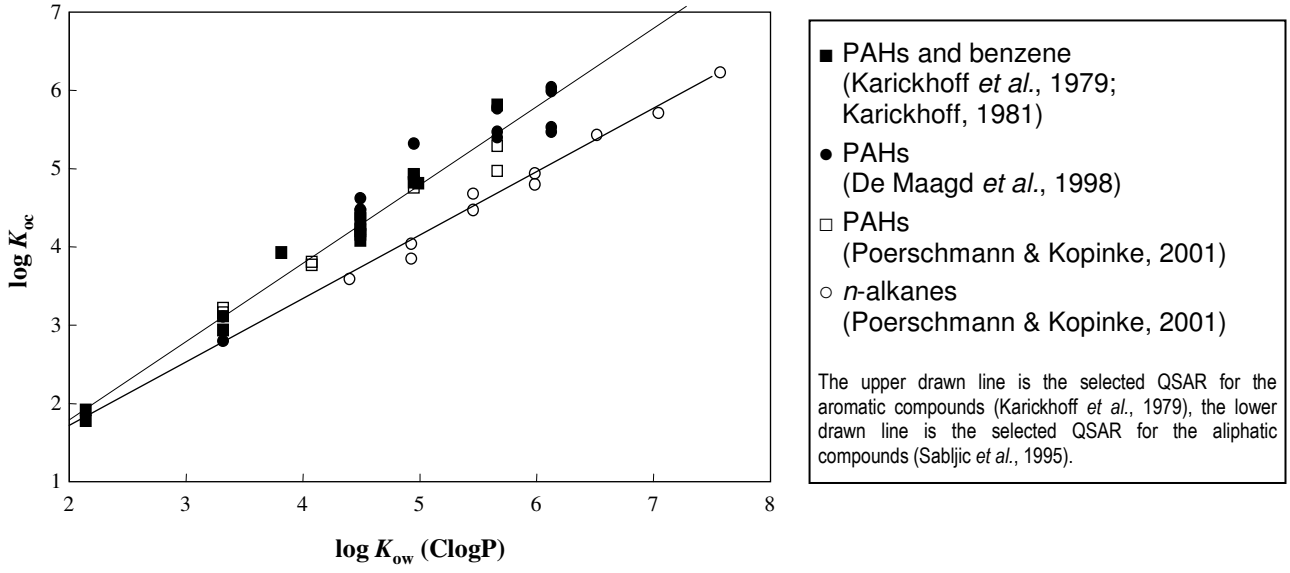


Figure B.4.1. Organic carbon-water partition coefficients as a function of log K_{OW}.

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):

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

From several studies (Burgess *et al.*, 2004; Lohmann *et al.*, 2004; Vinturella *et al.*, 2004; Jonker & Koelmans, 2001) 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 B.4.2). 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 µg/L (benzo(ghi)perylene) to 34800 µg/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.

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.

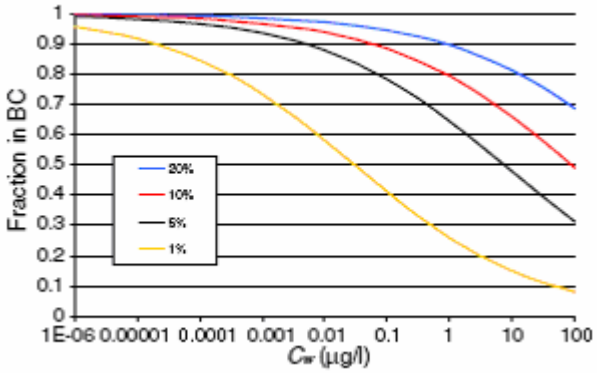
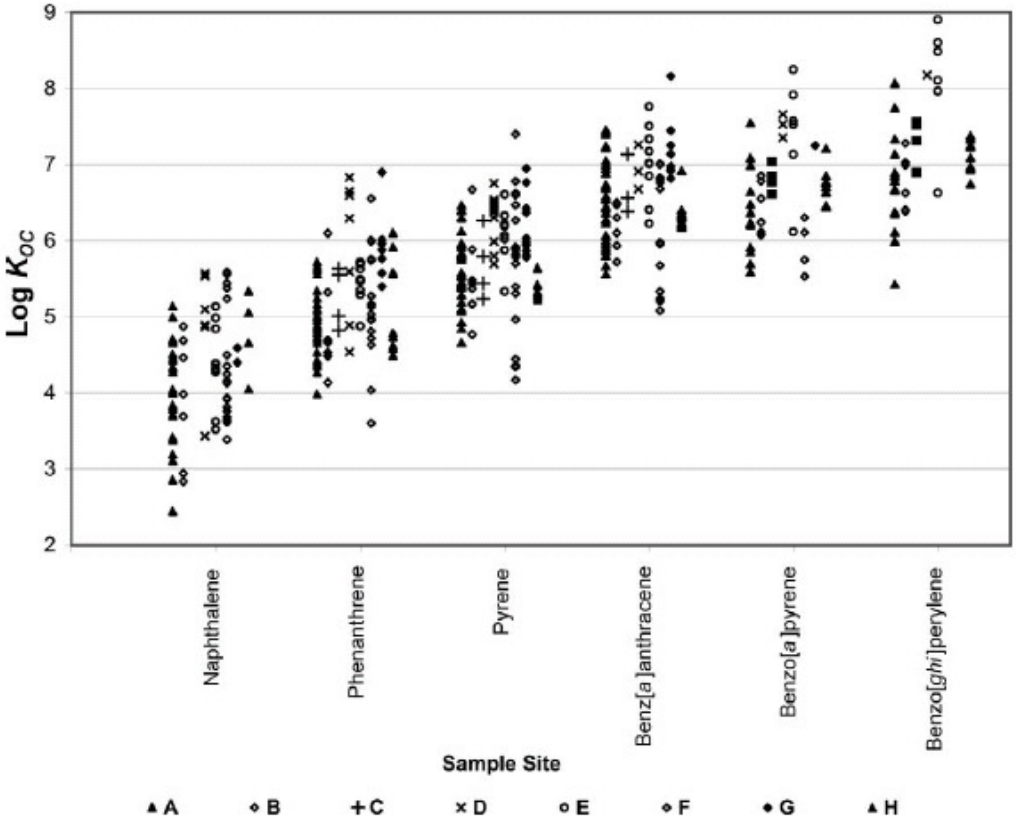


Figure reproduced from Koelmans *et al.* (2006).

Figure B.4.2. 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).

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 B.4.3), 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*

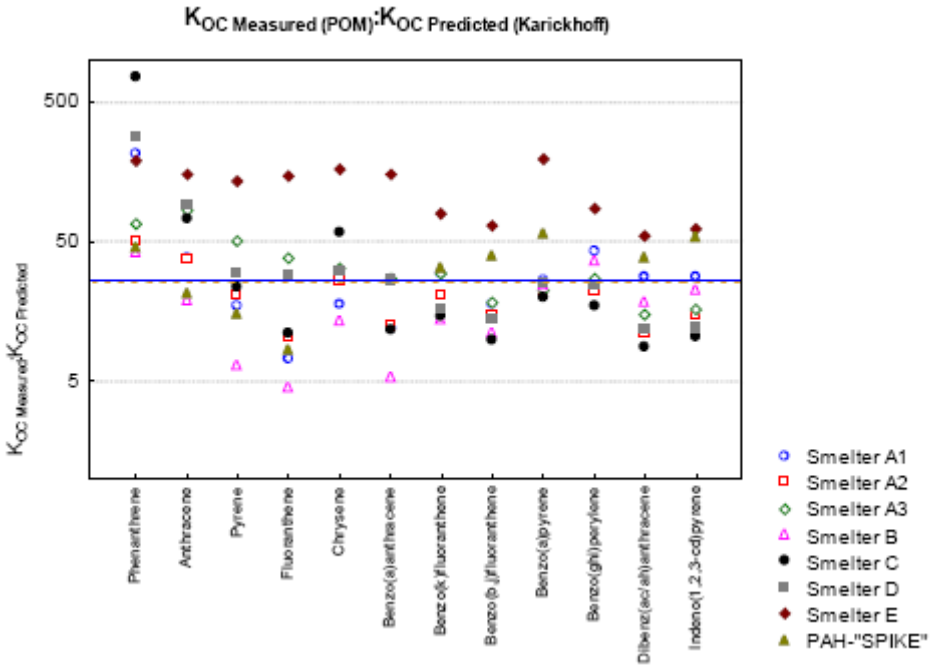
al. (1979), whereas the median value was an order of magnitude higher. It was concluded that the dependence of K_{OC} values on the site location was significant for some sites; however it did not appear to be related to the likely source of PAHs. This was illustrated by the difference between the two 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 B.4.3). 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.



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 B.4.3. Log sediment organic carbon-water partitioning coefficients (K_{OC}) values for several polycyclic aromatic hydrocarbons at different industrial sites.

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_ds were much higher than predicted from free-energy relationships, following Karickhoff *et al.* 1979 (see Figure B.4.4). 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 risk assessment. 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.



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 B.4.4. 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.

Table B.4.8. 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
Dibenzo(a,h)anthracene	6.50	6.29
Indeno(123-cd)pyrene	6.58	6.37

[a)] Values are based on the equation of Karickhoff *et al.* (1979).

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 transitional dossier 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 B.4.8).

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.*, 2002c). 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 risk assessment.

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 amorphic 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 risk assessment 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-dimethyl-phenanthrene) 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.

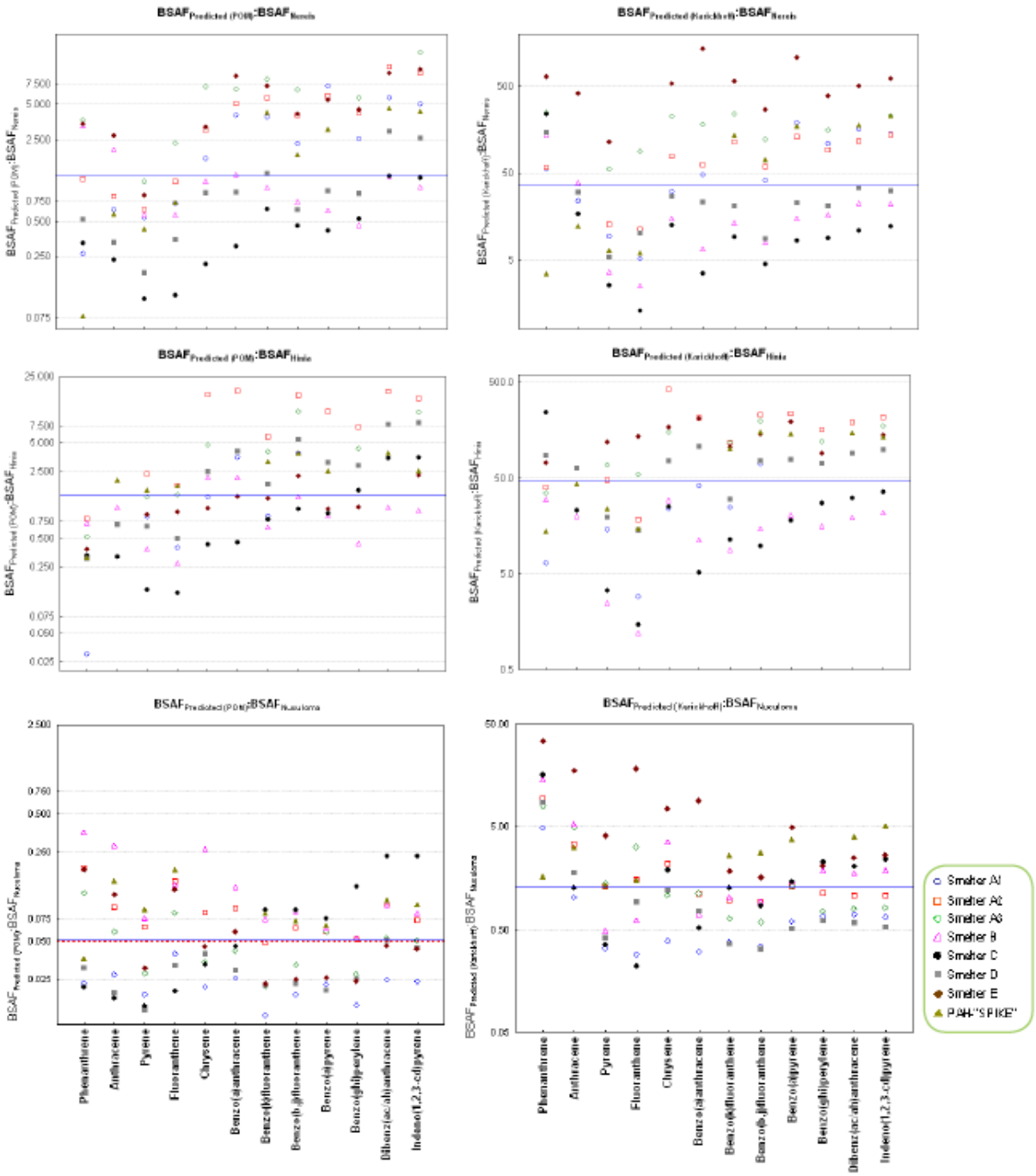
In order to investigate bio-availability of PAHs associated with coal tar pitch, Ruus *et al.* (2007) evaluated in the same study as mentioned above the bioaccumulation of PAHs from sediments outside several Nordic aluminium smelters in a mesocosm experiment using the polychaete *Nereis diversicolor*, 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 B.4.5), 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.



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_{OW}s 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 B.4.5. 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).

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 risk assessment.

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 risk assessment 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, it was decided not to include a correction for binding to soot-like materials in the risk assessment.

B.4.2.2 Precipitation

Most PAHs reach the soil via deposition from the atmosphere. Wild & Jones (1995) estimated emissions of 11 PAHs to the UK atmosphere to total about 700 tonnes/year and the average deposition rate to be 8.4 g/ha/y. PAHs are removed from the atmosphere by dry and wet deposition. The deposition rate depends on the occurrence of the PAHs, in gaseous form or bound to particles. The reaction at which PAHs are distributed over these two phases varies per PAH compound and also depends on the emission source (Slooff *et al.*, 1989). In general the low-molecular PAHs are mainly in the gas phase while the high molecular PAHs are bound to particles (see Section B.4.1.1). Dry deposition results from the direct impaction on land and water of airborne gaseous and particulate PAHs. Since particles are deposited mostly by gravitational settling, the higher molecular weight PAHs have higher dry deposition velocities than the gas phase PAHs which are deposited mainly by diffusion. Sheu *et al.* (1996) reported dry-deposition-velocity ranges of gas phase PAHs between 0.001 and 0.010 cm/s, only the lower molecular-weight naphthalene and acenaphthylene had a significant fraction of dry deposition flux contributed by the gas phase. The upper dry-deposition level is equal to the one recommended by the EU TGD and is therefore used for the exposure assessment.

All the remaining higher molecular-weight PAHs had more than 94.5% of their dry deposition flux resulting from the particle phase. In conformity, Meharg *et al.* (1998) observed that the transport distance of PAHs decreased with increasing molecular weight. As especially gaseous PAHs are relatively short-lived, in the order of a few to tens of hours (Mackay *et al.*, 1992), in dry air gaseous PAHs may not travel very far from the emission sources, to a distance of a few to tens of kilometres. Wet deposition is brought to the surface by precipitating hydrometeors (raindrops and snow flakes). The lifetime of PAHs in the aqueous phase is in the order of tens to hundreds of hours (Mackay *et al.*, 1992). Cloud droplets accumulate gaseous and particulate PAHs as they travel over long distances. Wet deposition of PAHs may originate from regional urban/industrial sources hundreds to thousand kilometres away. In the vicinity of urban/industrial areas, dry deposition predominates; in more remote areas, wet deposition predominates (Golomb *et al.*, 1997).

McVetty & Hites (1988) studied the total flux of PAH for a remote lake over the course of a year by sampling PAH concentrations in atmospheric particulate and gas phases, precipitation, and sediment. The authors found that particle-bound PAH have washout ratios 2 orders of magnitude greater than PAH present primarily in the gas phase. It was found that dry deposition of aerosol particles accounted for circa 90% of the flux of each PAH species with the remainder of the input flux from wet deposition. The flux of PAH from the lake due to evaporation is greater than that due to condensation from the gas phase to the lake. For more volatile PAH evaporation accounts for 80% of the flux from the lake. Particle size also affects the removal rate of the associated PAH from the atmosphere by dry and wet deposition (Main & Friedlander, 1990). This information mentioned above is in conformity with the estimated distribution using EUSES 2.0, which was used for the current risk assessment.

B.4.2.3 Volatilisation

The Henry law constants for PAHs rank from 49 Pa m^3/mol for naphthalene to 0.007 Pa m^3/mol for dibenzo(a,h)anthracene (see Table B.1.6). 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 observation 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 risk assessment the Henry law constants shown in Table B.1.6 were used.

B.4.2.4 Distribution in wastewater treatment plants

The distribution of the 16 EPA PAHs in sewage treatment plants has been calculated using the model SIMPLETREAT integrated to EUSES (EC, 2004a) based on the K_{OC} values and the Henry's low constants presented in Table B.4.8 and Table B.1.6, respectively. They are presented as an example in Table B.4.9.

Table B.4.9. Estimation of removal of the 16 EPA in a sewage treatment plant.

PAH compound	% to air	% to water [a)]	% 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
Dibenzo(a,h)anthracene	0.0	8.3	91.7	0.0	91.7
Indeno(123-cd)pyrene	0.0	8.3	91.7	0.0	91.7

Values according to EUSES 2.0. [a)] % to water is equal to parameter F_{stp} used in Section B.9.5.3.1 (Calculation of predicted environmental concentrations).

B.4.3 Bioaccumulation

Since bioaccumulation of PAHs is influenced by biotransformation both processes are discussed together.

B.4.3.1 Aquatic organisms

As stated in WHO (1998), for substances like PAH, long exposure times are necessary to achieve equilibrium conditions, so that results obtained under non-equilibrium conditions can result in underestimates of the BCF. For this reason, BCFs calculated as the ratio between the rates of uptake ($k_u = k_1$) and depuration ($k_e = k_2$) have to be preferred, as these can be determined with a relative short exposure time and because constant concentrations of compounds like benzo(a)pyrene are very difficult to maintain over a longer period. There are different methodologies used: (1) "flow-through method in which the concentration in water is kept constant and the concentration in fish is studied as a function of time, (2) "Banerjee method" in which fish are exposed in a static system and the decreasing concentration in water is used to derive the rate constants on basis of a mass balance and (3) adjusted Banerjee method" in which the concentration in both water and fish is determined as a function of time. In contrast to the Banerjee method this last method gives the possibility to determine the biotransformation rate as well (De Maagd, 1996).

According to OECD (1996) animals should be fed during the exposure phase. In case animals are not fed, metabolites may accumulate in the gall bladder, due to a low extraction of bile to the intestinal tract. Consequently, when the BCF study is performed with unfed animals and no distinction is made between the parent compound and metabolites, this study is considered not valid.

As the BCF is defined as the concentration in the animal divided by the concentration in water at steady state, steady state situations in soil or sediment systems can be described with two parameters: (1) the bioaccumulation factor (BAF), defined as the concentration in the animal divided by the concentration in the interstitial water and (2) the biota to sediment/soil accumulation factor (BSAF), defined as the concentration in the animal divided by the concentration in sediment/soil (Belfroid *et al.*, 1996). According to the Equilibrium Partitioning Theory, when equal affinities for lipids and sediment organic carbon can be assumed, BSAFs should be around 1.7. However, for very hydrophobic compounds such as PAHs, both BCFs and BSAFs show a curvilinear relationship with log K_{OW}. Up to log K_{OW} = 5 or 6, BCF usually increases with log K_{OW} and BCF stays more or less constant, but at higher log K_{OW}s BSAF decrease and BCFs tend to level off and can even decrease considerably (Thomann *et al.*, 1992; Fisk *et al.*, 1998; Baussant *et al.*, 2001). This curvilinear relationship is explained through variations in compound lipid solubility, slow desorption, lower actual bioavailable water concentrations than what is estimated using conventional models, effect of molecular size (and thus reduced membrane passage of the larger molecules) metabolism, and elimination into faeces (Thomann *et al.*, 1992; Fisk *et al.*, 1998; Meador, 2003).

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 (Sijm & Opperhuizen, 1989; Lech & Vodicnik, 1985). 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 stuchburyl*) 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).

The BCF values in the different vertebrate and invertebrate species are shown in Table B.4.10 and Table B.4.11, respectively. Below a description will be given of the most relevant BCF studies in fish, crustaceans and mussels.

In the study by Djomo *et al.* (1996), the uptake and elimination of four radiolabelled PAHs by zebra fish (*Brachydanio rerio*) was studied. The used experimental design differs at some points from the OECD guideline 305 (OECD, 1996). The study was performed in the presence of sediment in a static system. The four tested PAHs were for more than 90% sorbed to this sediment containing 0.95% organic carbon. Only one concentration was tested. This concentration was much higher than the natural background concentration of the sediment. The sediment/water system was allowed to equilibrate for 48 hours, which is claimed to be enough to reach equilibrium between the two phases.

The loading of the fish is at the upper limit of the recommended range from the OECD 305 guideline, *i.e.* 1.0 gram of fish per litre water. However, water concentrations were measured using LSC (Liquid Scintillation Counting) and were nearly constant during the exposure period, which may be attributed to the buffering capacity of the sediment present in the test system. The uptake period lasted for 30 days. Fish that were exposed for 24 hours to the PAHs were used in a depuration experiment in clean water. Uptake and elimination rate constants were determined by measuring total ^{14}C counts in fish bodies using LSC. The BCF could be determined as the quotient of these two constants. The resulting BCFs are 10400 L/kg for anthracene, 13400 L/kg for phenanthrene, 4300 L/kg for pyrene, and 3600 L/kg for benzo(a)pyrene.

However, there are two reasons why the BCFs determined can not be considered as reliable.

First, after a strong initial uptake, the uptake curves showed a decrease in the concentration in fish after 1 day to a more or less constant level after 20 days. Therefore, the uptake rate constants that are determined from the initial part of the curve (not specified, but probably from the 7 time points in the first 24 hours), could be erroneous and are probably too high. If the constant levels from 20 to

30 days are used and the BCF is calculated as the quotient of the concentrations in fish and water, the resulting BCFs are 33 L/kg for anthracene, 8 L/kg for phenanthrene, 11 L/kg for pyrene, and 60 L/kg for benzo(a)pyrene. The difference between these values and the values determined as the quotient of k_1 and k_2, is too large to consider the study to be reliable. Second, all concentrations in water and fish were from total radioactivity determined by liquid scintillation counting (LSC). This may lead to an overestimation of the BCF, because metabolites of the PAHs formed in the fish contribute to the total radioactivity as well as the parent compound. For these reasons the BCF values that are deduced from this study are considered to be not valid (Validity = 3).

The bioaccumulation of 5 PAHs in fathead minnows (*Pimephales promelas*) was studied in a static experimental set-up according to the so-called 'adjusted Banerjee method' (De Maagd, 1996; chapter 4). This study was designed to quantify the role of biotransformation in the bioaccumulation process. PAHs were added to tap water by a generator column. Fish (7-11) of on average 0.52 g were added to an aquarium with 1.5 L of water. The concentrations of the parent compounds in both water and fish in the static systems were analyzed using HPLC during 48 hours on 7 to 11 points in time. Fish were fed daily until two days prior to the experiment. In the modelling of the concentrations, the amount of fish and the volume of the water were adjusted every time a sample was taken. The bioaccumulation with and without biotransformation was determined by running parallel tests with and without the addition of piperonyl butoxide (PBO), a known biotransformation inhibitor for substrates binding to the site of cytochrome P450-isoenzymes. To distinguish between loss due to abiotic processes and biotransformation controls without fish were used as well.

The uptake rate determined from the concentration in fish was not in accordance with the uptake rate determined from the decrease of concentration in the water phase. Therefore, the recovery of anthracene, fluoranthene, and benz(a)anthracene from fish exposed via water had to be slightly adjusted downwards in comparison with homogenized fish spiked with the PAHs dissolved in hexane (18, 16, and 43% respectively). For benz(a)anthracene this adjustment of the recovery did not result in an good fit of the data and therefore the uptake was also estimated on the concentration in the fish only. For phenanthrene the increase in fish and decrease in water concentration was in accordance with the recovery of phenanthrene determined from spiked fish homogenate (+3%). Only the estimated recovery of naphthalene appeared to be significantly higher from fish exposed via PAHs taken up from water than from homogenized fish spiked with naphthalene in hexane. In the latter case the recovery was only about 16%, which was attributed to volatilisation during the extraction process. Estimated recovery of naphthalene to fit both the decrease in the aqueous concentration and increase in the concentration in fish was 35%. The calculated BCF values in the absence of PBO were 300 L/kg for naphthalene, 6800 L/kg for phenanthrene and anthracene, 3400 for fluoranthene and 200 L/kg for benz(a)anthracene. Only for benz(a)anthracene, biotransformation, which was not completely inhibited by PBO, significantly influenced the bioaccumulation process. No effect of biotransformation was observed for naphthalene, phenanthrene, and anthracene, while the uptake of fluoranthene could be better modelled if biotransformation was taken into account. The amount of fish in the aquarium (more than 3 grams/L) is three times more than the upper limit of what is recommended in OECD guideline 305. However, in the modified Banerjee method this amount of fish is necessary to reduce the water concentration in such a way that the uptake can be modelled from the concentration in water (Validity = 2).

In a second study by De Maagd *et al.* (1998) with fathead minnows benz(a)anthracene was tested in a flow-through study. In this study fish were exposed to a constant concentration of about 8.7 ± 3.4 µg/L obtained by passing tap water through a generator column. Fish were fed daily until two days prior to the experiment. For each exposure time an aquarium containing 15 fish was used. Six exposure times were used with the longest exposure time lasting for 14 days. At the end of each exposure time three fish were sampled. In each group one fish was sampled at a shorter

intermediate exposure time, which increases the number of sampling times up to 12. For each exposure time, the rest of the fish were transferred to an aquarium with clean water and charcoal filters to follow the elimination of the compound. From these aquaria one fish was sampled at 11 different time points. Concentrations of the parent compound in fish were analysed using HPLC. On basis of the steady state concentrations from 10 hours exposure time and higher and the kinetic modelling of the uptake during the whole bioconcentration experiment similar BCF values of 265 and 262 L/kg were found, respectively. The BCF study determined in this flow-through study is comparable to the value that was determined in the static test according to the "adjusted Banerjee method". (Validity = 2).

In another study with fathead minnows (Weinstein & Oris, 1999), juvenile fish (48 hours post-hatching) were exposed in a static system to four concentrations of fluoranthene in tap water in a dish containing 150 ml solution, prepared by a generator column. Apart from the four concentrations of fluoranthene in tap water, the accumulation was also determined at four different amounts of dissolved humic acid, each with four concentrations of fluoranthene. The concentration of the parent compound in water and fish were determined after one day of exposure using HPLC. The accumulation experiments showed a consistent picture of the bioaccumulation with the BCF decreasing exponentially with the concentrations of dissolved organic matter at all fluoranthene concentrations. At one dissolved humic matter concentration the BCF values were not significantly different. The BCF value in tap water without dissolved humic matter was 9054 ± 555 L/kg. (Validity = 2).

A similar test setup as that from the Maagd and co-workers was used by De Voogt *et al.* (1991). However, in this case the Banerjee method was used, but not the adjusted Banerjee method, which implies that the concentrations in fish were not determined. Solutions of the PAHs in 1:1 tap water:deionized water were prepared by the generator column technique. The tested concentrations were about one tenth of the experimental LC_{50}. Guppies (*Poecilia reticulata*; 0.135 g) were added to test vessels with 3 litres of water at a load of 1 to 4 gram fish per litre. Water samples were taken at 8 time points during 48 hours. Controls without fish were run to check the loss of compound due to other factors than accumulation in fish. It is not clear if the fish were fed or not. Concentrations in water were analyzed using HPLC.

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

At the end of the experiment, six fish were transferred to a vessel with clean water. The concentration of the parent compound in fish was determined with HPLC in duplicate at three time points in addition to the concentration in fish just after the end of the static experiment. With these 4 time points a depuration rate constant could be determined which can be compared with the depuration rate calculated according to the Banerjee method. The depuration rates for fluorene and anthracene determined directly from the concentration in fish were significantly different from those determined from the kinetic model applied to the water concentrations from the static test. For pyrene both values agreed well. Only in the case of anthracene the two-fold difference in depuration rate constant can be explained by neglecting the role of metabolism in the depuration constant in the kinetic approach of the Banerjee method. For fluorene the actual depuration rate constant is six-fold lower instead of higher than the one estimated from the kinetic approach.

For comparison also a regular semi-static (renewal) bioaccumulation test was carried out. In this test, 2 litre of solution in the test vessels was renewed every 12 hours for pyrene and anthracene and every 24 hours for fluorene. Eight to ten guppies were placed in each test vessel leading to a load of

about 0.5 to 0.7 gram fish per litre. For pyrene two guppies were sampled on days 1, 3, and 7. For fluorene and anthracene two guppies were samples on days 1, 2, and 4. PAHs were analyzed using HPLC. The BCF was determined by dividing the final concentration in fish by the average concentration in water during the last renewal period. These BCF values are 1050 L/kg for fluorene, 4550 L/kg for anthracene and 11300 L/kg for pyrene. (Validity = 2; preferred values).

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

Spacie *et al.* (1983) exposed bluegills (*Lepomis macrochirus)* for 4 hours to ^{14}C anthracene and ^{14}C benzo(a)pyrene, after which depuration was measured. Juvenile bluegills (0.1-0.6 g; max. 0.5 g fish/L) were kept in 5 L static systems with reconstituted natural water (pH 7.4-7.6; temperature 23-24 °C). To minimize photodegradation, all experimental procedures were performed under gold fluorescent lights ($\lambda \geq 500$nm). Fish were not fed 4 hours prior to the start of the experiment. In a preliminary test, tanks without fish lost only 4.5% of initial ^{14}C activity after 16 hours. In the same preliminary test, it was shown that when using longer exposure times than 4 hours, uptake rates declined because the fish had extracted a significant portion of the anthracene from the water. It is unclear how many replicate tanks were used in the final test. Figures show error bars however, which could be from replicate fish from one tank, or from replicate tanks. Water and fish were analyzed for total 14C activity using LSC. Metabolites and parent compounds were separated by TLC, and measured separately using 14C activity. Extracts were also analysed using HPLC. Uptake rate constants were determined from fish body burdens, which were determined at 1, 2, and 4 hours after the start of the exposure phase. Seven different exposure concentrations for anthracene (0.7-16.6 µg/L) resulted in similar uptake rate constants (k_u) which ranged from 36 to 42 hr-1. Elimination half-lives were 17 hours for anthracene and 67 hours for benzo(a)pyrene, and were calculated from the fraction total 14C remaining in the fish. HPLC analysis revealed that after 4 hours, 7.9% of anthracene and 89% of benzo(a)pyrene had been metabolized. BCFs were calculated using rate constants from results from the lowest exposure concentration using total 14C activity (parent and metabolite), and were 900 for anthracene and 4900 for benzo(a)pyrene. However, after 4 hours of exposure, only 7.9% of anthracene was present as a metabolite. Thus, for anthracene the BCF largely reflects the parent compound. For benzo(a)pyrene, however, 89% of the compound is present as metabolites after 4 hours of exposure. (Validity = 2 for anthracene; Validity = 3 for benzo(a)pyrene) The BCF values calculated using rate constants (k_u/k_e) based on total 14C activity were multiplied by the percentage of parent material present in the fish at the end of 4h exposure. The obtained estimates are the product of the kinetic data for total radioactivity and the percentage of metabolites after 4 hours. The BCF values determined in this way are 675 for anthracene and 490 for benzo(a)pyrene. However, for benzo(a)pyrene this percentage metabolites is large and strongly increasing over time. Therefore the estimation of the BCF can not be considered as reliable for benzo(a)pyrene. For anthracene the difference of 25% between the two reported BCF values can not be explained from the percentage metabolites of 7.9% after 4 hours and therefore also this value should be considered as not reliable. (BCF corrected for metabolites validity 3).

Linder *et al.* (1985) exposed rainbow trout (*Salmo gairdneri*/*Oncorhynchus mykiss*) for 72 h to 14C anthracene, after which a 144h depuration phase was employed. 15 Sexually immature fish (10 grams) were kept in 15 L static-renewal systems with a mixture of well water and dechlorinated tap water (pH 8.3, temperature 12 °C). Fish were not fed for 5 days prior to the start of the depuration experiment. Per exposure treatment two replicate aquaria and one control were used. A 16h light:

8h dark photoperiod was maintained; although all aquaria were covered with dark lids to reduce anthracene photodecomposition, light still penetrated the transparent glass walls of the aquaria. Analyzed exposure concentrations were approximately 0.05 mg/L, a concentration which is at the solubility limit of anthracene. In a preliminary experiment it was determined that in an 18h period no detectable biodegradation or volatilization occurred. Water was sampled 8 times per 24h replacement interval; two fish per replicate were removed every 24 hours for tissue residue analysis at the uptake experiment, and every 48 hours at the depuration experiment. Besides total 14C counts using LSC, water and fish samples were fractionated using HPLC to determine metabolite distributions. BCFs were calculated from (1) fish anthracene levels divided by the geometric means of the water concentration and (2) uptake rates (14C anthracene in exposure waters during the first 4 hours of exposure) and depuration rates (^{14}C appearance in depuration waters combined with anthracene concentrations in fish at the start of the depuration phase). Depuration rates were lower than reported literature values of Spacie *et al.* (1983). According to Linder *et al.* (1985) this could be due to the fact that in their experiment the fish were not fed for five days before the start of the elimination phase. Moreover, the fact that metabolites may still have been in the body is not taken into account, which may also be a cause for the low depuration rates and thus the high BCFs for the parent compound. The average non-steady state BCFs (based on only the parent compound) calculated using concentrations were after 24 hours: BCF = 200; after 48 hours: BCF = 575; after 72 hours: 779. This shows that steady state was not reached within 72 hours of exposure. Steady-state BCFs (calculated with kinetics) were on average 9100. Uptake rates reflected only the parent compound; depuration rates reflected only metabolites since no parent compound was measured in the water phase. No internal metabolism rate was determined. It is not clear if and how results from the control aquaria were used. Therefore, this kinetically determined steady-state BCF can not be considered as reliable.

Fish concentrations other than at the start of the depuration experiment were not reported. Important deviations from OECD guideline 305 (OECD, 1996) are that the fish were not fed two days before and during the experiment, and that the amount of fish in the exposure tanks (10 grams/L) is 10 times more than what is recommended by OECD guideline 305 (OECD, 1996). (Validity = 3 for steady state and non-steady state BCFs).

Freitag *et al.* (1985) determined concentration factors in activated sludge, algae and fish. Experimental protocols are described in detail in other publications (Freitag et al, 1982; Korte *et al.*, 1978). Golden ide (*Leuciscus idus melanotus*; 1.5g) were exposed to 14C labelled PAHs for 3 days, with a constant concentration of 0.05 mg/L. Five fish were kept in static systems with 10 litres of tap water diluted with de-ionised water. pH was about 7, temperature 20-25 °C. The test is not performed in the dark (so breakdown may have occurred), no controls are mentioned. Fish were not fed during the test. Water concentrations are measured daily for total 14C activity using LSC. Transformation products were determined with undefined chromatography techniques, but it is unclear if and how these results were used. Bioconcentration is calculated using the average aquatic exposure to the chemicals. Because the 14C method in this case does not distinguish between the parent compound and its metabolites, the accumulation factor is a measure of both (*i.e.* the BCF for the parent compound is overestimated). Median water measured concentrations for anthracene are reported in Freitag *et al.* (1982) and are 26 µg/L, nominal concentrations were 50 µg/L. In Freitag *et al.* (1982), the same BCF is reported for anthracene as in Freitag *et al.* (1985). Photomineralization is also measured and reported for all tested chemicals, except for anthracene. (Validity = 4).

Barrows *et al.* (1980) exposed bluegill sunfish (*Lepomis macrochirus*; wwt: 0.37 g) to 14C acenaphthene and 32 other chemicals, for a maximum period of 28 days or until equilibrium was reached, after which a depuration phase was employed. Acenaphthene exposure was 28 days. 100 fish were kept in 15 L intermittent flow systems with diluted well water (pH ranged from 6.3-7.9 over all tests, temperature 16 °C). Fish were fed three times a week during the test. Water and fish

were sampled at days 0, 1, 2, 4, 7, 10, 14, 21 and 28 during the exposure phase and analyzed for total 14C residues using LSC, in exposure aquaria as well as in controls. The mean exposure concentration for acenaphthene was 9 µg/L. During the depuration phase, fish were sampled on days 1, 2, 4, and 7. No distinction is made between the parent compound and its metabolites. No reference is made on photolytic degradation and measures to prevent this.

BCFs were calculated as the quotient of the mean chemical concentration measured in fish tissues during equilibrium divided by the mean measured chemical concentration in water during the entire exposure period. The BCF for acenaphthene is reported to be 387; with a half-life in tissues of less than 1 day. Because it was an intermittent flow system, exposure water concentrations can be expected to have been stable; the presence of metabolites and photolytic breakdown products will not have had a large effect on the results. However, the 14C fish tissue concentration comprises both the parent compound and metabolites. It is unclear if and how much metabolites are present, although with a half-life of less than 1 day metabolism can be expected to have been present. In the absence of better data, the BCF of 387 could be used as a worst case estimate for acenaphthene. (Validity = 2).

Johnsen *et al.* (1989) determined BCFs for 14C benzo(a)pyrene in Atlantic salmon (*Salmo salar*) by measuring rate constants for uptake and depuration. 30 fish (2 grams) were kept in 100 L tanks with filtered lake water (pH 6.6, temperature 6 °C) for an uptake period of 48 hours, after which a 96 hour depuration period started. Fish and water were sampled after 4, 8, 12, 24, 36 and 48 hours during uptake, and fish were sampled after 0, 24, 48 and 72 hours during depuration. Total 14C activity in the fish was measured using LSC. No distinction was made between parent compound and metabolites. A light period of 12 hours per day was employed, but to prevent photolytic breakdown a low-intensity artificial lamp was used. The fish were not fed during the experiment. It is not clear how many replicate systems were used. No control aquaria without fish were used to investigate the stability of the test concentrations over time. BCFs are calculated from uptake and depuration rate constants, which were estimated using measured concentrations in Atlantic salmon (k_u = 3.17; k_e = 0.0015; BCF = 2310). Time to reach steady state is 64 days, which is reported to be because of the low temperatures used in the study. This may then also have affected the depuration rate constant, and thus not reflect a BCF at higher temperature. Because uptake and depuration rates were calculated from total 14C counts, including metabolites, the BCF does not reflect the parent compound only. (Validity = 3).

Jimenez *et al.* (1987) exposed bluegill sunfish (*Lepomis macrochirus*) to 14C benzo(a)pyrene in flow-through systems. 70 Fish (10-15g) were exposed in 65 L systems with a flow rate of 200 ml/min at two temperatures (13 and 23 °C) and with or without food. Uptake lasted for 48 hours, depuration for 96 hours. At intervals, groups of fish were sampled. It is not clear how many replicates were used. Because a flow-through system was used, a control system to check water concentrations was not needed. No reference is made on the light conditions during the experiment and possible photolytic breakdown, but the use of a flow-through system circumvents problems with photolytic breakdown. Fish amounts in the exposure aquaria exceeded OECD guideline 305 (OECD, 1996) recommendations. Total ^{14}C concentrations were analysed at several time intervals in water and fish samples using LSC. Relative proportions of parent compound and metabolites were determined using HPLC. Exposure water concentrations were corrected for the presence of metabolites. Uptake rates were determined for the parent compound, and depuration rates for total ^{14}C activity. Experiments were performed with fish that were not fed, and with fish that were fed during the study. BCFs for fed fish were 377-608; the BCF for unfed fish was 3208. The reason for this difference is that metabolites are probably accumulating in the gall bladder in unfed fish, because extraction of bile to the intestinal tract is low in this case. As the BCF values are based on the total radio activity, they can be considered as reliable (Validity = 3), though a value of 600 could be considered as a upper limit for benzo(a)pyrene.

Petersen & Kristensen (1998) measured bioaccumulation of ^{14}C labelled naphthalene, phenanthrene, pyrene and benzo(a)pyrene in eggs of zebra fish (*Brachydanio rerio*) and larvae of zebra fish (*Brachydanio rerio*), cod (*Gadus morhua*), herring (*Clupea harengus*) and turbot (*Scophthalmus maximus*). Eggs and larvae were exposed in semi-static systems with renewal every 24 hours in climate rooms of 27 °C for zebra fish, 15 °C for turbot, 6.5 °C for cod and 7.5 °C for herring. Natural seawater was collected from a depth of about 30 meters. Up to approximately 200 organisms were placed in 1-L glass beakers. During the first 8 hours of exposure, water samples were analysed for total ^{14}C activity using LSC every hour, after that twice per 24 hours. During the depuration experiments, samples were taken six times the first 24 hours, and twice per 24 hours after that. The duration of exposure and depuration periods varied in accordance with specific survival time of the yolk sac larvae of each of the investigated species. BCFs were calculated by dividing the uptake rate constant by the elimination rate constants and by dividing fish concentrations by water concentrations. BCFs were similar between eggs and larvae of zebra fish, because the low rate of uptake in eggs is compensated by lower elimination rates. Because biotransformation of PAHs in early life stages is thought to be insignificant when compared to adults, BCFs may be higher in these early life stages than in adults. BCFs are reported on a dry weight basis (Validity = 3).

Jonsson *et al.* (2004) exposed the fish *Cyprinodon variegatus* for 36 days to PAHs in a continuous flow system with seawater, followed by 8 days of depuration. PAHs studied were naphthalene, phenanthrene, pyrene, and 5 alkylated PAHs, and exposure was performed with a mixture of the PAHs at two different exposure concentrations. Mean measured levels for total PAHs in the exposure chambers were 7.57 and 72.31 µg/L. Adult fish (mean weight 2.47 g and mean length 4.7 cm) were exposed in seawater of 34 ‰ and 25 °C and were fed daily with raw, peeled shrimp throughout the experiment. Water and fish samples were taken at several time intervals during the study and analyzed using GC-MS. Two to six individual fish were pooled, after removal of the whole gallbladder. Bile was analyzed separately. Besides the parent compound, also a selection of metabolites was analyzed. BCF ratios were determined as the ratio between k_u and k_e, and as the concentration in fish divided by the concentration in water. These two types of BCF values were very similar. Maximum tissue levels were measured within 7 days of exposure. The reported BCFs reflect the tissue concentrations without gall bladder, but parent compound concentrations in bile can be expected to be very low, this does not influence BCFs. BCFs range from 714 to 999 for naphthalene, from 700 to 2229 for phenanthrene, and from 50 to 145 for pyrene. (Validity = 1).

McCarthy & Jimenez (1985) exposed bluegill sunfish (*Lepomis macrochirus*) to ^{14}C naphthalene and ^{14}C benzo(a)pyrene in the presence and absence of dissolved humic material. Fish were 4-12 grams wet weight, and were not fed 24 hours before exposure and during the experiment. Naphthalene exposure concentrations were 7.8 and 0.78 nmol/mL. Benzo(a)pyrene exposure concentrations declined exponentially from 4 to approximately 2 pmol/mL over the exposure period. The exposure duration was 24 hours (naphthalene) or 48 hours (benzo(a)pyrene) in a flow-through system with undefined filtered flowing water (23 °C), after which a depuration period of 36 hours (naphthalene) or 96 hours (benzo(a)pyrene) was deployed. At several time intervals, fish and water were sampled and analyzed for radioactivity using LSC. Parent compounds and metabolites were also determined using HPLC. BCFs were calculated using rate constants.

For naphthalene, at both exposure concentrations virtually all of the radioactivity present in the fish was parent compound. Thus, the BCF for naphthalene reflects the parent compound and is similar for the high exposure and low exposure experiments (310 and 320, respectively). In contrast, for benzo(a)pyrene most of the radioactivity present in the fish during depuration was caused by metabolites. The BCF for benzo(a)pyrene (2657) reflects parent compound and metabolites, and the BCF of parent compound alone would be much lower. The ‘true’ BCF for the parent compound only is reported to be 30. Similar to the value derived by Spacie *et al.* (1983) this value can only be

seen as an indication of a low BCF value for benzo(a)pyrene, based on parent compound, but the correction results only in an estimation of the BCF. (Validity = 3 for steady state and non-steady state BCFs).

Melancon & Lech (1978) exposed rainbow trout (*Oncorhynchus mykiss*) to ^{14}C-naphthalene and ^{14}C-methylnaphthalene. Groups of seven fingerlings (9 to 11 grams) were exposed for 8 hours to 0.005 mg/L or 0.023 mg/L naphthalene in a static system at 2.5 grams fish per litre. Fish were sacrificed at various times during exposure and during a subsequent 24-hr elimination period in fresh flowing water. Also, fingerlings were exposed for four weeks in a continuous flow system to 0.02 mg/L naphthalene at about 5 grams fish/L. Groups of five fish were removed at intervals for the determination of tissue levels of ^{14}C. After four weeks, the remaining fish were transferred to fresh flowing water to follow the elimination. Water concentrations were monitored during both exposure and elimination periods. The fish were fed twice per week and sampling was usually done at least two days after feeding. The presence of parent compounds and metabolites was determined by TLC.

Total ^{14}C fish levels were found to be 22 to 340 times the initial water level of naphthalene at the 0.005 mg/L 8h exposure, and 24 to 585 times the initial water level at the 0.023 mg/L exposure. 21% of naphthalene was found to be present as polar compounds in the muscle after 24 hours of exposure. (Validity = 3).

Cho *et al.* (2003) exposed larvae of the fathead minnow (*Pimephales promelas*) to fluoranthene with and without methyl *tert*-butyl ether (MTBE) to determine if MTBE enhances bioaccumulation and toxicity of fluoranthene. Larvae (4 days post-hatching) were exposed to 20 µg/L fluoranthene and 0 or 40 µg/L MTBE under simulated sunlight for 24 hours in flow-through systems with dechlorinated and carbon-filtered tap water (22 °C; pH 7.6, hardness 248 mg/L $CaCO_3$). After 24 hours of exposure, the fish were moved to clean water for the elimination phase. Fish were provided with a small amount of food (brine shrimp) for 30 minutes per day. Fish were removed for analysis at several time intervals during exposure and elimination. Concentrations of fluoranthene in fish and water were measured using reverse-phase HPLC. Toxicity was tested during 96 hours exposure in the same systems. BCFs were determined using rate constants. The presence of MTBE caused 37% higher uptake rates and 30% lower elimination rates, resulting in a BCF that was twice as high (29208) as when no MTBE was present (BCF = 14836). (Validity = 1).

Wang & Wang (2006) examined the toxicokinetics of benzo(a)pyrene and the trophic transfer in a marine planktonic food chain comprising phytoplankton, copepods, and fish. Influences of food quality and quantity and different routes of exposure (aqueous and food) were also examined. Here, only uptake in water-only experiments will be discussed. Copepods (*Acartia ecrythraea*) were collected from the field, while mangrove snappers (fish; *Lutjanus argentimaculatus*; 2-3 cm and 0.5-0.7 g wwt) were purchased from a local fish farm. Filtered seawater was spiked with 1.3 ng/L ^{3}H benzo(a)pyrene and exposure was performed in the dark in static systems. Copepods were exposed for 8 hours, during which water samples were taken at regular time intervals. Copepod samples were taken at the end of the exposure period. Fish were exposed for 4 hours and fish were sampled every hour. Radioactivity of the samples was quantified using LSC. Depuration was performed in renewal systems and lasted 48 hours for the copepods, which were sampled at regular time intervals. Rate constants were calculated with the assumption that all metabolites of benzo(a)pyrene were immediately excreted out of the body. Because this assumption may not be valid, BCFs obtained in this study reflect parent compound and metabolites. No depuration experiment was performed for the fish with water-only exposure. Given the reported half life of the depuration experiment after food exposure, a depuration rate and thus a BCF can be calculated for the fish. The results section of this article does not seem to be very reliable. (Validity = 3).

For most of the 5 and 6-ring PAHs only BCF values for crustaceans and molluscs are available and no values for fish. Remarkably highest BCF values were measured for phenanthrene, anthracene, pyrene, benz(a)anthracene and benzo(a)pyrene in the amphipod *Pontopereia hoyi*, which has a 20-50% lipid content by wet weight and no capacity for biotransformation (Landrum, 1988). It is however also obvious that for all PAHs a large variation within the different taxonomic groups was observed, fish included. In view of the high BCF values of molluscs, it is considered relevant not only to assess the risk for fish eating birds and mammals but for mollusc eating birds and mammals as well. Thus, molluscs may be an important species in determining bioaccumulation potential of PAHs. Because they do not metabolise PAHs, their BCF values may be higher than BCF values for fish. Consequently, mollusc-eating birds may therefore be at higher risk than fish-eating birds. Thus, the mussel-studies mentioned in Table B.4.10, are also reviewed.

McLeese & Burridge (1987) determined PAH accumulation in the clam *Mya arenaria*, the mussel *Mytilus edulis*, the shrimp *Crangon septemspinosa*, and the polychaete worm *Nereis virens*. Groups of the invertebrates were exposed for 4 days in seawater containing a mixture of five PAHs (phenanthrene, fluoranthene, pyrene, triphenylene, and perylene) in continuous flow-systems. After 4 days, exposure was terminated, and the animals were maintained in flowing seawater at 10 °C for two weeks. Animals and water were sampled daily during the exposure period, and animals were sampled at days 1, 2, 4, 7 and 14 during the depuration period. Samples were analyzed using HPLC. Measured concentrations in water and animals were used to calculate k_u and k_e, which were subsequently used to calculate BCFs. For clam, mussel, shrimp and polychaete BCFs for phenanthrene were 1280, 1240, 210, and 400 respectively; for fluoranthene 4120, 5920, 180, and 720 respectively; for pyrene 6430, 4430, 225, and 700, respectively; for triphenylene 5540, 11390, 270, and 2560, respectively, and for perylene 10000, 10500, 175, and 180, respectively. The study is very well documented. (Validity = 1).

Weinstein & Polk (2001) determined phototoxicity of anthracene and pyrene to glochidia (larvae) of the mussel *Utterbackia imbecillis*. To assess the intrinsic potency of the compounds, tissue residues were also analyzed. Mature glochidia were removed from parent mussels and used immediately. Glochidia were exposed during 4 hours in static systems, with reconstituted water (pH 8.09; temperature 22.8 °C). Five replicate chambers were used for four different test concentrations and contained between 20 and 30 glochidia. Ambient laboratory lighting was used, so photolytic breakdown of PAHs may have occurred. Concentrations in water and glochidia were quantified using HPLC at the beginning and after 8 hours of exposure in the toxicity tests. But for BCF determination, only the initial water concentration was used. Thus, water concentrations may have been overestimated and BCFs underestimated. Tissue residues were quantified using a reverse-phase HPLC. Unpublished data from earlier experiments had shown that after 4 hours of exposure, steady state was reached. BCFs were calculated by dividing the 4h PAH tissue concentration by the initial water concentration. For anthracene, the mean BCF (over four different concentrations) was 346, and for pyrene the mean BCF was 1070. (Validity = 2).

Skarphéðinsdóttir *et al.* (2003) exposed mussels (*Mytilus edulis*) for 6 days to 17 µg/L radiolabelled benzo(a)pyrene under semi-static conditions, followed by 28 days of depuration in uncontaminated water. 100 mature mussels (length 21 mm) were placed on Petri dishes in glass aquaria filled with 14 L of natural seawater (6.7 ‰; 9 °C). Water was renewed every 24 hours. Three replicates were used. Control aquaria without mussels were not mentioned. Mussels were fed during the experiment and kept with 16 h daylight and 8 h dark, so photolytic breakdown of benzo(a)pyrene in the exposure water could have occurred. Mussels were sampled every 28 hours during the exposure phase and at different time intervals during the depuration phase. Total radioactivity was counted using LSC, no distinction was made between benzo(a)pyrene and possible metabolites. Benzo(a)pyrene concentrations were reported on a dry weight basis. Steady state was not reached during the exposure period. Uptake rates were not reported, but can be calculated from a figure with

tissue concentrations in time. Uptake rate is 40.85 L/kg/hr on a dry weight basis. Depuration half life is reported to be approximately 8 days, which can be recalculated into a depuration rate constant of 0.0036 h^{-1}. BCF can then be calculated by dividing the uptake rate by the depuration rate, and is 11347 L/kg. This value is based on dry weight. Assuming a dry to wet weight ratio of 0.1, the BCF based on wet weight will be around 1200. (Validity = 3).

Moy & Walday (1996) exposed the mussel *Mytilus edulis* to ^{14}C-benzo(a)pyrene in a continuous flow-through system with non-filtered seawater (11-15 °C; 32-35 ‰). The systems were illuminated for 12 hours per day. Mussel shell length was 40-50 mm and no additional food was provided during the experiment. Exposure duration was 14 days, with an average concentration of 1.36 µg/L benzo(a)pyrene. Samples were taken at various time intervals during exposure and the subsequent depuration phase (44 days), but no rates were reported. Sample activity was measured in a liquid-scintillation counter. The method does not differentiate between benzo(a)pyrene and its breakdown products. After 14 days of exposure, equilibrium was not reached and benzo(a)pyrene concentration in the mussels were 332 µg/kg. A BCF can be estimated by dividing the water concentration by the biota concentration (BCF = 244 L/kg). This BCF reflects total ^{14}C amounts and that equilibrium was not reached. (Validity = 3).

Bruner *et al.* (1994) exposed the zebra mussel (*Dreissena polymorpha*) in a static system to ^{13}H-labelled benzo(a)pyrene and pyrene. Zebra mussels were collected from unpolluted littoral zones and acclimated to the laboratory at least 1 week prior to experimentation. Two size classes of mussels were used (21 mm and 15 mm), and for the first size-class the experiments were performed for pre-spawning mussels (with high lipid contents) and post-spawning mussels (with low lipid content). Soft standard reference water (20 ± 2 °C, pH 8, hardness 40-48 mg/L $CaCO_3$) was used. Exposure was performed under gold light to minimize photodegradation. Mussels were fed daily with TetraMin prior to the experiment and during the elimination phase, but not during exposure. Exposure to 0.5 µg/L benzo(a)pyrene and 0.4 µg/L pyrene lasted for 6 hours, after which elimination lasted for 168 hours. Elimination was performed in static-renewal systems. Water and biota samples were taken at regular time intervals and counted with LSC. In a preliminary experiment, no biotransformation could be detected. BCFs were calculated using kinetic rate constants and ranged from 13000 to 35000 for pyrene and 41000 to 84000 for benzo(a)pyrene. (Validity = 2).

Gossiaux *et al.* (1996) exposed the zebra mussel (*Dreissena polymorpha*) in a static system to radiolabelled benzo(a)pyrene in combination with either hexachlorobiphenyl, pentachlorophenol or pyrene. Adult field-collected mussels with a shell length of 14-22 mm were fed daily prior to exposure and during the elimination phase, but not during uptake. The mussels were exposed for 6 hours in filtered lake water at field collection temperatures (4, 8, 10, 15, 20, 23, 24, or 28 °C), or after acclimatization from field temperatures to 4, 10, 12, 15, or 20 °C. In total a number of 23 experiments with benzo(a)pyrene and 10 experiments with pyrene were conducted. Exposure concentrations differed per experiment and ranged from 0.002-0.079 µg/L for benzo(a)pyrene and 0.0004-0.010 µg/L for pyrene. Contaminant concentrations in water and mussel samples were analyzed at regular time intervals using LSC. Elimination was followed during 15 days in static-renewal systems. In a preliminary experiment, no biotransformation could be detected. BCFs were calculated using kinetic rate constants and ranged from 37000 to 43000 for pyrene and 133000 to 142000 for benzo(a)pyrene. (Validity = 2).

Palmork & Solbakken (1981) exposed the horse mussel *Modiola modiolus* to ^{14}C phenanthrene in static systems. Field-collected mussels of both sexes (42 ± 12 g flesh weight) were fed krill during elimination, but were not fed during exposure. Exposure was during 48 hours in seawater (34 ‰) of 11.5-15 °C, depuration lasted for 28 days in flowing seawater of 9 °C. Water samples were analyzed for radioactivity at the beginning of the experiment, mussels were sampled at regular time intervals and dissected, after which radioactivity was determined using ‘standard methods’.

According to the results section, the exposure concentration was 20 µg/L and the mussel concentration at the end of exposure was 62 $\mu g/kg_{wwt}$, which corresponds to a BCF of 3.1. The overall description of the results and the study is rather vague. (Validity = 3).

Widdows *et al.* (1983) exposed the mussel *Mytilus edulis* to ^{14}C naphthalene in a static system with membrane-filtered seawater. Field-collected mussels (55-60 mm) were exposed to 7 µg/L ^{14}C naphthalene during 4 hours, after which depuration lasted for 192 hours. Mussels were sampled at regular time intervals and analyzed using LSC. Results show that after 4 hours a near-equilibrium is reached, and the bioaccumulation factor (BCF) is reported to be around 50. This is close to a BCF of 59 for *Ostrea edulis*, which was determined by Riley *et al.* (1981) and mentioned in the article. There is no mention of solvent controls, water sample results, feeding or no feeding, etc. (Validity = 4).

Richardson *et al.* (2005) exposed the green-lipped mussel (*Perna viridis*) to anthracene, fluoranthene, pyrene and benzo(a)pyrene in a renewal system for 20 days. Field collected mussels of similar length (101 ± 5 mm) were fed continuously with green algae during all phases of the experiment. Mussels were exposed in natural seawater, which was filtered trough charcoal and fine sand for 24 hours before use. Exposure concentrations for each individual PAH were 1 µg/L. Depuration lasted for 10 days. Water and biota samples were taken at regular time intervals and analyzed using GC/FID. Water samples were filtered so particulate and dissolved phase fractions could also be analyzed. BCFs were calculated using rate constants using concentrations in the dissolved phase. Water temperature fluctuated from 17.5-20.5 °C, salinity and pH were 28.2 ± 2 ‰ and 7.9 ± 0.2, respectively.

Mussel weight and lipid content did not change during the experiment, mortality was only 4%. PAHs were mainly present in the particulate water phase. Reported BCFs are based on lipid weight instead of wet weight, making comparison with other data difficult. (Validity = 4).

Other invertebrate BCFs were determined by Evans & Landrum. (1989). The amphipod *Pontoporeia hoyi* and the mysid *Mysis relicta* were exposed to radiolabelled benzo(a)pyrene in filtered natural water (4 °C). Experiments with *P. hoyi* (8.3 mg) were conducted in 200 mL flow through chambers, where 25-40 amphipods were placed and water was passed through at 100 mL/h. Experiments were conducted under low level red light or in the dark. Experiments with *M. relicta* (43 mg) were performed in 3L static systems with 10-15 individuals per aquarium. Animals were not fed during the experiment. Animals and water were sampled after 1, 2, 4, and 6 h of exposure and analysed for ^{14}C activity using LSC. Elimination experiments were performed for 10-26 days in 6L aquaria containing unfiltered lake water, and for *P. hoyi* also sieved lake sediment. Animals were removed for analysis at several time intervals. Previous experiments showed that *P. hoyi* did not metabolize benzo(a)pyrene during the uptake period, thus measured radioactivity is assumed to be parent compound activity only. Water concentrations declined on average with 20.7% during the course of the uptake experiment in the *P. hoyi* experiments and with 11.0% for the *M. relicta* experiments. Uptake and depuration rate constants were determined several times, at different seasons, and it was shown that both uptake and depuration rates varied per season.

Uptake rate constants varied from 35-183 $L/kg^{/}h$ for *P. hoyi* and 20-50 L $kg^{-1}h^{-1}$ for *M. relicta*. Depuration rate constants were 0.0012-0.0028 h^{-1} for *P. hoyi* and 0.0037-0.0055 h^{-1} for *M. relicta*. Mean BCF was 48582 for *P. hoyi* and 8496 for *M. relicta*. (Validity = 2).

Laurén & Rice (1985) exposed the crab *Hemigrapsus nudus* to ^{14}C-naphthalene in a static system with seawater for 12 hours, followed by a depuration period of 156 hours. The crabs (adult males, 3-4 cm carapace width, intermolt C_4 stage) were starved three days prior to the experiment. Exposure was during 12 hours in seawater of 7 °C, containing 3.95 mg/L ^{14}C naphthalene. Samples were taken at regular time intervals, dissected into digestive gland, muscle, antennal gland and gills, and analysed using LSC. After 12 hours of exposure and 12 and 156 hours of depuration, samples

were also taken to determine metabolite concentrations. When expressed in terms of a 'standard crab', the whole body BCF can be calculated from the reported concentration data to be 15.3 L/kg. After 12 hours, only 4% of total ^{14}C activity was present as metabolites. However, results show that after 12 hours, equilibrium was not reached. (Validity = 3).

The BSAF values for fish have recently been reviewed by Van der Oost *et al.* (2003) and are presented in Table B.4.12. The values range from 0.01 for the total PAH in eel (Van der Oost *et al.*, 1991) to 10 for phenanthrene in brown bullhead (Baumann & Harshbarger, 1995). All lipid weight:organic matter based BSAF values were much lower than one indicating a reduced uptake, possibly due to lowered bioavailability, or an increased clearance. BSAF values of PAHs in sunfish declined with increasing K_{OW}, probably due to low gut assimilation efficiency and increased metabolism (Thomann & Komlos, 1999). The BSAF values for marine invertebrates have recently been evaluated by Meador (2003). Invertebrates from all types of studies (field and laboratory) exhibited values close to the theoretical maximum, but mostly for the LPAHs, which achieve steady-state or equilibrium partitioning between tissue and the exposure matrix relatively rapidly. It appears that most values are relatively constant in the range 0.2-0.4 (see Table B.4.13).

In general, BSAFs in benthic invertebrates for PAHs appear to be one order of magnitude below those expected at equilibrium partitioning and those reported for non-metabolized compounds like PCBs.

Table B.4.10. Bioaccumulation factors in fish and mussels for the various PAHs.

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
Naphthalene	Mollusca					
	Mytilus edulis	50	4	S	equilibrium (total)	Widdows *et al.* (1983)
	Fish					
	Brachydanio rerio (eggs)	1820 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (eggs)	1738 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	1778 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	1259 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Pimephales promelas	302	2	S	k_1/k_2 (parent)	De Maagd (1996)
	Leuciscus idus melanotus	30	4	S	k_1/k_2 (unclear)	Freitag *et al.* (1982)
	Cyprinodon variegatus	895 [e]	1	F	k_1/k_2 (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	999 [f]	1	F	k_1/k_2 (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	692 [e]	1	F	equilibrium (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	714 [f]	1	F	equilibrium (parent)	Jonsson *et al.* (2004)
	Lepomis macrochirus	310 [f]	2	F	k_1/k_2 (total=parent)	McCarthy & Jimenez (1985)
	Lepomis macrochirus	320 [e]	2	F	k_1/k_2 (total=parent)	McCarthy & Jimenez (1985)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
	Oncorhynchus mykiss	22-340 [e]	3	S	k_1/k_2 (total)	Melancon & Lech (1978)
	Oncorhynchus mykiss	24-585 [f]	3	S	k_1/k_2 (total)	Melancon & Lech (1978)
Acenaphthene	Fish					
	Lepomis macrochirus	387	2	S	equilibrium (total)	Barrows *et al.* (1980)
Acenaphthylene	-	-				
Fluorene	Fish					
	Poecilia reticulata	2230	3	S	k_1/k_2 (parent)	De Voogt *et al.* (1991)
	Poecilia reticulata	1050 [g]	2	R	equilibrium (parent)	De Voogt *et al.* (1991)
	Poecilia reticulata	3500	2	S	equilibrium (parent)	De Voogt *et al.* (1991)
Anthracene	Mollusca					
	Utterbackia imbecillis (larvae)	345	2	R	equilibrium (parent)	Weinstein & Polk (2001)
	Perna viridis	380189 [h]	3	R	k_1/k_2 (parent)	Richardson *et al.* (2005)
	Fish					
	Brachydanio rerio	10400	3	S	k_1/k_2 (total)	Djomo *et al.* (1996)
	Lepomis macrochirus	900	2	S	k_1/k_2 (total)	Spacie *et al.* (1983)
	Lepomis macrochirus	675	3	S	k_1/k_2 (corrected)	Spacie *et al.* (1983)
	Oncorhynchus mykiss	9000-9200	3	R	k_1/k_2 (parent)	Linder *et al.* (1985)
	Oncorhynchus mykiss	779	3	R	equilibrium (parent)	Linder *et al.* (1985)
	Pimephales promelas	6760	2	S	k_1/k_2 (parent)	De Maagd (1996)
	Poecilia reticulata	7260	3	S	k_1/k_2 (parent)	De Voogt *et al.* (1991)
	Poecilia reticulata	4550 [g]	2	R	equilibrium (parent)	De Voogt *et al.* (1991)
	Poecilia reticulata	6000	2	S	equilibrium (parent)	De Voogt *et al.* (1991)
	Leuciscus idus melanotus	910	4	S	k_1/k_2 (unclear)	Freitag *et al.* (1982)
Phenanthrene	Mollusca					
	Mytilus edulis	1240	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Mya arenaria	1280	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Modiola modiolus	3.1	3	S	equilibrium (parent)	Palmork & Solbakken (1981)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
	Fish					
	Brachydanio rerio (eggs)	9120 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (eggs)	12303 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	7943 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	6309 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Gadus morhua (larvae)	10715 [c]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Gadus morhua (larvae)	14454 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Clupea harengus (larvae)	20893 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Clupea harengus (larvae)	21380 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Scophthalmus maximus (larvae)	11220 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Scophthalmus maximus (larvae)	11482 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio	13400 [d]	3	S	k_1/k_2 (total)	Djomo *et al.* (1996)
	Pimephales promelas	6760	2	S	k_1/k_2 (parent)	De Maagd (1996)
	Leuciscus idus melanotus	1760	4	S	k_1/k_2 (unclear)	Freitag *et al.* (1982)
	Cyprinodon variegatus	810 [e]	1	F	k_1/k_2 (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	2229 [f]	1	F	k_1/k_2 (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	700 [e]	1	F	equilibrium (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	1623 [f]	1	F	equilibrium (parent)	Jonsson *et al.* (2004)
Fluoranthene	Mollusca					
	Mytilus edulis	5920	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Mya arenaria	4120	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Perna viridis	245471 [h]	3	R	k_1/k_2 (parent)	Richardson *et al.* (2005)
	Fish					
	Pimephales promelas	9054	2	S	equilibrium (parent)	Weinstein & Oris (1999)
	Pimephales promelas	3388	2	S	k_1/k_2 (parent)	De Maagd (1996)
	Pimephales promelas (larvae)	14836	1	F	k_1/k_2 (parent)	Cho *et al.* (2003)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
Pyrene	Mollusca					
	Mya arenaria	6430	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Mytilus edulis	4430	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Utterbackia imbecillis (larvae)	1054	2	R	equilibrium (parent)	Weinstein & Polk (2001)
	Dreissena polymorpha	16000 [i]	2	S	k_1/k_2 (total=parent)	Bruner *et al.* (1994)
	Dreissena polymorpha	13000 [j]	2	S	k_1/k_2 (total=parent)	Bruner *et al.* (1994)
	Dreissena polymorpha	35000 [k]	2	S	k_1/k_2 (total=parent)	Bruner *et al.* (1994)
	Dreissena polymorpha	43000 [l]	2	S	k_1/k_2 (total=parent)	Gossiaux *et al.* (1996)
	Dreissena polymorpha	37000 [m]	2	S	k_1/k_2 (total=parent)	Gossiaux *et al.* (1996)
	Perna viridis	891251 [h]	4	R	k_1/k_2 (parent)	Richardson *et al.* (2005)
	Fish					
	Brachydanio rerio (eggs)	10000 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (eggs)	30200 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	54954 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	53703 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Gadus morhua (larvae)	60256 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Gadus morhua (larvae.)	85114 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Clupea harengus (larvae)	97724 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Clupea harengus (larvae)	128825 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio	4300	3	S	k_1/k_2 (total)	Djomo *et al.* (1996)
	Poecilia reticulata	4810	3	S	k_1/k_2 (parent)	De Voogt *et al.* (1991)
	Poecilia reticulata	11300 [g]	2	R	equilibrium (parent)	De Voogt *et al.* (1991)
	Poecilia reticulate	2700	2	S	equilibrium (parent)	De Voogt *et al.* (1991)
	Cyprinodon variegatus	145 [e]	1	F	k_1/k_2 (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	97 [f]	1	F	k_1/k_2 (parent)	Jonsson *et al.* (2004)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
	Cyprinodon variegatus	50 [e]	1	F	equilibrium (parent)	Jonsson *et al.* (2004)
	Cyprinodon variegatus	53 [f]	1	F	equilibrium (parent)	Jonsson *et al.* (2004)
Benz(a)anthracene	Fish					
	Leuciscus idus melanotus	350	4	S	NS (unclear)	Freitag *et al.* (1985)
	Pimephales promelas	200-265	2	S	k_1/k_2 (parent)	De Maagd (1996); De Maagd *et al.* (1998)
Chrysene	-	-				
Benzo(a)pyrene	Mollusca					
	Mytilus edulis	12000 [d]	3	S	k_1/k_2 (total)	Skarphéðinsdóttir *et al.* (2003)
	Mytilus edulis	244	3	F	equilibrium (total)	Moy & Walday (1996)
	Dreissena polymorpha	84000 [i]	2	S	k_1/k_2 (total=parent)	Bruner *et al.* (1994)
	Dreissena polymorpha	41000 [j]	2	S	k_1/k_2 (total=parent)	Bruner *et al.* (1994)
	Dreissena polymorpha	77000 [k]	2	S	k_1/k_2 (total=parent)	Bruner *et al.* (1994)
	Dreissena polymorpha	133000 [n]	2	S	k_1/k_2 (total=parent)	Gossiaux *et al.* (1996)
	Dreissena polymorpha	142000 [o]	2	S	k_1/k_2 (total=parent)	Gossiaux *et al.* (1996)
	Perna viridis	169824 [h]	4	R	k_1/k_2 (parent)	Richardson *et al.* (2005)
	Fish					
	Brachydanio rerio (eggs)	20893 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (eggs)	290000 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	331131 [d]	3	R	equilibrium (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (larvae)	436516 [d]	3	R	k_1/k_2 (total = parent)	Petersen & Kristensen (1998)
	Brachydanio rerio (adult)	3600	3	S	k_1/k_2 (total)	Djomo *et al.* (1996)
	Leuciscus idus melanotus	480	4	S	k_1/k_2 (unclear)	Freitag *et al.* (1982)
	Salmo salar	2310	3	S	k_1/k_2 (total)	Johnsen *et al.* (1989)
	Lepomis macrochirus	3208	3	F	k_1/k_2 (total); unfed	Jimenez *et al.* (1987)
	Lepomis macrochirus	608	2	F	k_1/k_2 (total); fed	Jimenez *et al.* (1987)
	Lepomis macrochirus	4900	3	F	k_1/k_2 (total)	Spacie *et al.* (1983)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
	Lepomis macrochirus	2657	3	F	k_1/k_2 (total)	McCarthy & Jimenez (1985)
	Lepomis macrochirus	30	3	F	k_1/k_2 (parent)	McCarthy & Jimenez (1985)
	Lutjanus argentimaculatus	3.28	3	S	k_1/k_2 (total)	Wang & Wang (2006)
Benzo(b)fluoranthene	-	-		-	-	
Benzo(k)fluoranthene	-	-				
Benzo(ghi)perylene	-	-				
Dibenzo(a,h)anthracene	Fish					
	Leuciscus idus melanotus	10	4	S	k_1/k_2 (unclear)	Freitag *et al.* (1982)

a) Reliability score: 1-reliable without restrictions, 2-reliable with restrictions, 3-unreliable, 4-not assignable; b) S: static exposure system, F: flow-through system, R: static renewal system; c) k1/k2: uptake rate/depuration rate, total: total compound concentration (including transformation products), parent: parent compound concentration, NS, not steady state; d) based on dry weights; e) low exposure concentrations; f) high exposure concentrations; g) preferred by the author; h) based on lipid weights; i) 21 mm size class with high lipid content; j) 21 mm size class with low lipid content; k) 15 mm size class; l) average BCF obtained from 4 experiments at ambient field temperatures (individual BCFs were 33000, 22000, 77000, and 39000); m) average BCF obtained from 6 experiments after acclimatization to lab temperatures (individual BCFs were 32000, 48000, 41000, 39000, 24000, and 39000);n) average BCF obtained from 11 experiments at ambient field temperatures (individual BCFs were 77000, 49000, 191000, 167000, 132000, 165000, 150000, 197000, 40000, 24000, and 273000); o) average BCF obtained from 12 experiments after acclimatization to lab temperatures (individual BCFs were 190000, 83000, 61000, 197000, 220000, 116000, 40000, 147000, 215000, 270000, 107000, and 62000).

Table B.4.11. Bioaccumulation factors in Oligochaeta, Polychaeta, Crustacea and Insecta for the various PAHs.

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
Naphthalene	Crustacea					
	Daphnia magna	20-50			k_1/k_2	McCarthy *et al.* (1985)
	Daphnia pulex	131 (10844)			k_1/k_2 (NS)	Southworth *et al.* (1978)
	Daphnia pulex	1032-1424			k_1/k_2	Trucco *et al.* (1983)
	Diporeia sp.	473		R	k_1/k_2	Landrum *et al.* (2003)
	Hemigrapsus nudus	15.3	3	S	equilibrium (total)	Laurén & Rice (1985)
	Insecta					
	Somatochlora cingulata	524			equilibrium	Correa & Coler (1983)
Acenaphthene	-	-				
Acenaphthylene	-	-				
Fluorene	Oligochaeta					
	Lumbriculus variegatus	395		R	equilibrium	Ankley *et al.* (1997)
	Crustacea					
	Daphnia magna	506		S	equilibrium	Newsted & Giesy (1987)
Anthracene	Oligochaeta					
	Lumbriculus variegatus	1369		R	equilibrium	Ankley *et al.* (1997)
	Stylodrilus heringianus	5051		F	k_1/k_2	Frank *et al.* (1986)
	Crustacea					
	Daphnia magna	511		F	k_1/k_2	McCarthy *et al.* (1985)
	Hyalella azteca	1800-10985		F	k_1/k_2	Landrum & Scavia (1983)
	Pontoporeia hoyi	16857-39727		F	k_1/k_2	Landrum (1982; 1988)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
Phenanthrene	Oligochaeta					
	Stylodrilus heringianus	5055		F	k_1/k_2	Frank *et al.* (1986)
	Crustacea					
	Crangon septemspinosa	210	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Daphnia magna	323-600		S	NS	Eastmond *et al.* (1984); Newsted & Giesy (1987)
	Daphnia pulex	325		F	k_1/k_2	Southworth *et al.* (1978)
	Diporeia sp.	8889		R	k_1/k_2	Landrum *et al.* (2003)
	Pontoporeia hoyi	28145		F	k_1/k_2	Landrum (1988)
	Polychaeta					
	Nereis virens	500	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
Fluoranthene	Polychaeta					
	Nereis virens	720	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Crustacea					
	Crangon septemspinosa	180	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Diporeia sp.	6256		S	kinetic model	Kane Driscoll *et al.* (1997b)
	Hyalella azteca	7827		S	kinetic model	Kane Driscoll *et al.* (1997b)
Pyrene	Oligochaeta					
	Stylodrilus heringianus	6588		F	k_1/k_2	Frank *et al.* (1986)
	Polychaeta					
	Nereis virens	700	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Crustacea					
	Crangon septemspinosa	225	1	F	k_1/k_2 (parent)	McLeese & Burridge (1987)
	Daphnia pulex	2702		S	k_1/k_2	Southworth *et al.* (1978)
	Diporeia sp.	21916		R	k_1/k_2	Landrum *et al.* (2003)
	Pontoporeia hoyi	16600		F	k_1/k_2	Landrum (1988)
Benz(a)anthracene	Crustacea					
	Daphnia magna	2920		S	k_1/k_2	McCarthy *et al.* (1985)
	Daphnia pulex	10109		S	k_1/k_2	Southworth *et al.* (1978)
Chrysene	Crustacea					
	Daphnia magna	6088		S	equilibrium	Newsted & Giesy (1987)
Benzo(a)pyrene	Oligochaeta					
	Stylodrilus heringianus	7048		F	k_1/k_2	Frank *et al.* (1986)
	Insecta					
	Chironomus riparius	607		S	equilibrium	Clements *et al.* (1994)
	Hexagenia limbata	5879		F	k_1/k_2	Landrum & Poore (1988)
	Crustacea					
	Daphnia magna	2440-5770		S/F	k_1/k_2	Leversee *et al.* (1981); McCarthy *et al.* (1985)

Compound	Species	BCF	R [a]	Test system [b]	Type [c]	References
	Pontoporeia hoyi	73000		S	k_1/k_2	Landrum (1988)
		48582	2	F	k_1/k_2 (total=parent)	Evans & Landrum (1989)
	Mysis relicta	8496	2	F	k_1/k_2 (total=parent)	Evans & Landrum (1989)
	Acartia erythraea	25	3	S	k_1/k_2 (total)	Wang & Wang (2006)
Benzo(b)fluoranthene	-	-				
Benzo(k)fluoranthene	Crustacea					
	Daphnia magna	13225		S	equilibrium	Newsted & Giesy (1987)
Benzo(ghi)perylene	Crustacea					
	Daphnia magna	28288		S	equilibrium	Newsted & Giesy (1987)
Dibenzo(a,h)anthracene	Crustacea					
	Daphnia magna	50119		S	equilibrium	Newsted & Giesy (1987)

[a] Reliability score: 1-reliable without restrictions, 2-reliable with restrictions, 3-unreliable, 4-not assignable; [b] S: static exposure system, F: flow-through system, R: static renewal system; [c] k_1/k_2: uptake rate/depuration rate, total: total compound concentration (including transformation products), parent: parent compound concentration, NS, not steady state.

Table B.4.12. Biota-sediment accumulation factors (BSAFs) of polycyclic aromatic hydrocarbons in fish.

Species	Mean fish-sediment concentration ratios (range) of compounds										Dimensions [a]	Ref [b]
	fluorene	phenan-threne	fluoran-thene	pyrene	chrysene	ΣPAH 2 [c]	ΣPAH 3 [c]	ΣPAH 4 [c]	ΣPAH 5 [c]	ΣPAH 6 [c]	ΣPAH	
Antarctic fish *Notothenia gibberifrons*											0.24-1.25	DW:DW	1
Brown bull-head *Ictalurus nebulosus*		2-10	2-6	2-5	1-2						1-6	FW:DW	2
											0.01-0.10	LW:OM	3
Eel *Anguilla anguilla*						0.24-2.9	0.091-1.6	0.01-0.4	0.003-0.06	0.02-0.20	0.04-0.56	LW:OM	4
Killifish *Fundulus heteroclitus*	0.05-0.36	0.004-0.05	0.0005-0.001	0.0003-0.001	0.0001-0.001						0.001-0.012	DW:DW	5
Lake trout *Salvelinus namaycush*		0.00011	0.00016	0.0071	0.00033							LW:OM	6
Pike *Esox lucius*											0.02-0.09	LW:OM	3
Roach *Rutilus rutilus*											0.02-0.13	LW:OM	3
						0.5-2.3	0.1-0.6	0.02-0.14	0.01-0.06	0.01-0.13		LW:OM	4
Sunfish *Lepomis macrochirus*											0.00001-0.8	LW:OM	7

Table as summarized by Van der Oost *et al.* (2003). [a] DW, dry weight; FW, fresh weight; LW, lipid weight; OM, organic matter or organic carbon; [b] References (see Van der Oost *et al.*, 2003): 1: McDonald *et al.* (1995), 2: Baumann & Harshbarger (1995a), 3: Van der Oost *et al.* (1991a), 4: Van der Oost *et al.* (1994), 5: Elskus & Stegeman (1989), 6: Burkhard & Lukasewycz (2000), 7: Thomann & Komlos (1999); [c] number of aromatic rings.

Table B.4.13. Bioaccumulation factors for PAHs accumulated by marine invertebrates.

Species	Feeding type [a]	Area/type [b]	Number of PAHs	Total PAH (ng/g DW) [c]	BAF (DW)	BSAF (range)	BSAF (mean) [d]	Time (days) [e]	Ref. [f]
Mytilus edulis	FF	Western Baltic Sea/F	Several	88-3880	0.02-53	0.01-0.59	0.17		1
Macomona liliana	DF	Northern New Zealand/F	9 PAHs	18-203	0.09-1.7	0.04-0.13	0.10		2
Austrovenus stutchburyi	FF	Northern New Zealand/F	9 PAHs	9-47	0.01-0.58	0.002-0.05	0.04		2
Macoma inquinata	DF/FF	Northwest USA/LS	3 PAHs			0.6-2.4	1.3	60	3
Abarenicola pacifica	DF	Northwest USA/LS	3 PAHs			1.5-3.7	2.4	60	3
Macoma balthica	DF/FF	Chesapeake Bay, USA/LS	chrysene, naphthalene			0.17-0.78	0.17	12	4
Rhepoxynius abronius	omn	New York, USA/LF	24 PAHs			0.001-0.5	0.052	10	5
Armandia brevis	DF	New York, USA/LF	24 PAHs			0.002-0.9	0.18	10	5
Yoldia limatula	DF	New York, USA/LF	several			1-3		35	6
Palaemonetes pugio	omn	Louisiana USA/LS	Benzo(a)pyrene, phenanthrene			nd-1.6	0.23	14	7
Rangia cuneata	FF	Louisiana USA/LS	Benzo(a)pyrene, phenanthrene			nd-1.5	0.42	14	7
Ampelisca abdita	sus	Rhode Island USA/LS	Benz(a)anthracene, Benzo(a)pyrene			0.13-0.15	0.14	10	8
Macoma nasuta	DF/FF	Northwest USA/LS	Benzo(a)pyrene		0.25			7	9
Eohaustorius washingtonianus	detr	Northwest USA/LS	Benzo(a)pyrene		3.0			7	9
Rhepoxynius abronius	omn	Northwest USA/LS	Benzo(a)pyrene		1.5			7	9
Macoma nasuta	DF/FF	Northwest USA/LF	16 PAHs		0.06-0.19			28	9
Eohaustorius washingtonianus	detr	Northwest USA/LF	16 PAHs		0.1-0.45			7	9
Rhepoxynius abronius	omn	Northwest USA/LF	16 PAHs		0.09-05			7	9
Nereis virens	omn	California and New Jersey/LF	Fluoranthene			0.8-3.3		15	10
Macoma nasuta	DF/FF	California and New Jersey/LF	Fluoranthene			0.6-3.8		15	10
Abarenicola pacifica	DF	Northwest USA/LS	Benzo(a)pyrene			1.1-2.3		68	11
Potamocorbula amurensis	FF	San Francisco USA/F	18 PAHs	130-860		0.6-5.4	0.3		12
Tapes japonica	FF	San Francisco USA/F	18 PAHs	95-450		0.007-2.7	0.15		12
Polychaetes (several spp.)	DF/omn	San Francisco USA/F	18 PAHs	310-1790		0.04-2.0	0.2		12
Macoma nasuta	DF/FF	Los Angeles, USA/LF	5 PAHs			0.05-1.0	0.4	28	13
Leptocheirus plumulosus	detr/sus	Chesapeake Bay, USA/LS	Fluoranthene				0.32	26	14

Species	Feeding type [a]	Area/type [b]	Number of PAHs	Total PAH (ng/g DW) [c]	BAF (DW)	BSAF (range)	BSAF (mean) [d]	Time (days) [e]	Ref. [f]
Corophium volutator	detr	Netherlands/LF	8 PAHs			0.5-1.7		25	15
Stichopus tremulus	DF	Norway/F	12 PAHs	237-797		0.004-0.67	0.18		16
Chlamys septemradiata	FF	Norway/F	12 PAHs	75-304		0.007-0.43	0.11		16
Ascidia sp.	FF	Norway/F	12 PAHs	193-671		0.0040.47	0.09		16
Arenicola marina	DF	Netherlands/LF	7 PAHs	370-3100	0.76			60-90	17
Macoma balthica	DF/FF	Chesapeake Bay, USA/F	14 PAHs			0.5-8.1	2.5		18
Nereis succinea	omn	Chesapeake Bay, USA/F	14 PAHs			0.41-6.0	2.4		18
Streblospio benedicti	DF	South Carolina, USA/F	3 PAHs	860-2000	0.2-1.4	0.08-0.4	0.22		19
Schizopera knabeni	DF	Louisiana, USA/LS	Fluoranthene			0.51-0.80	0.62	1	20
Coullana sp.	omn	Louisiana, USA/LS	Fluoranthene			0.22-0.67	0.43	1	20
Mytilus sp.	FF	Baltic and Mediterranean	14 PAHs	25-2420		0.001-7.4	0.52		21

Table as summarized by Meador (2003). [a] Feeding types are: DF, deposit feeder; FF, filter feeder; omn, omnivore; sus, suspension feeder; detr, detritivore. [b] Area/type list, the area found or where research was conducted and type of exposure (F, samples from field; L, exposures in laboratory to field contaminated sediment; LS, exposures in laboratory to spiked sediments). [c] Values are ranges of total PAHs found in tissues. [d] Mean BSAFs includes all PAHs and locations studied, excluding site CB, which produced BSAF values of 106 (*M. balthica*) and 98 (*N. succinea*). An approximate conversion of wet weight to dry weight concentration is ([PAH] dry weight = [PAH] wet weight × 5). [e] Time is the exposure period; no value is given for field collections. All analyses conducted with whole organisms or the soft tissue of clams. [f] References (see Meador, 2003): 1, Baumard *et al.* (1999a); 2, Hickey *et al.* (1995); 3, Augenfeld *et al.* (1982); 4, Foster *et al.* 1987; 5, Meador *et al.* (1995); 6, Lamoureux & Brownawell (1999); 7, Mitra *et al.* (2000); 8, Fay *et al.* (2000); 9, Varanasi *et al.* (1985); 10, Brannon *et al.* (1993); 11, Weston (1990); 12, Maruya *et al.* (1997); 13, Ferraro *et al.* (1990); 14, Kane Driscoll *et al.* (1998); 15, Kraaij *et al.* (2001); 16, Naes *et al.* (1999); 17, Kaag *et al.* (1997); 18, Foster & Wright (1988); 19, Ferguson & Chandler (1998); 20, Lotufo (1998); 21, Baumard *et al.* (1999b).

B.4.3.2 Terrestrial organism

According to the Technical Guidance Document (EC, 2003b), the bioconcentration in earthworm can be described as a hydrophobic portioning 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):

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

where for $\rho_{earthworm}$ by default a value of 1 (kg_{wwt}/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 risk assessment, which are given in Table B.4.14.

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.*, 1996b). 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.*, 1996b).

B.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 (Broman *et al.*, 1990; Neff, 1979; 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 (Clements *et al.*, 1994; Hellou *et al.*, 1991; Lemaire *et al.*, 1993; Niimi & Dookran, 1989). 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; Van Hattum *et al.*, 1996; Van Hattum *et al.*, 1998; Den Besten *et al.*, 1995).

Table B.4.14. 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
Dibenzo(a,h)anthracene	6.50	38000
Indeno(123-cd)pyrene	6.58	46000

[a)] Calculations based on the equation described in the Technical Guidance Document (EC, 2003b).

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.

B.5 Human health hazard assessment

As already indicated 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 striking hazard associated with CTPHT (see Section B.5.8.2). This is attributed to the presence of PAHs in CTPHT, for which benzo(a)pyrene has been chosen as exposure indicator (see Section B.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 CTP-PAH profiles (see Section B.9), these data were originally grouped per scenario, using benzo(a)pyrene as a scenario-specific effect indicator. However, as is indicated in Section B.5.8.2, 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.

B.5.1 Toxicokinetics

There were no data available on the toxicokinetics of CTPHT. From reviews on selected homocyclic polycyclic aromatic hydrocarbons, the following summarising data can be taken (Montizaan *et al.*, 1989; WHO, 1998).

Polycyclic aromatic hydrocarbons (PAHs) are lipophilic compounds and can be absorbed via the respiratory and gastrointestinal tract and the skin. As to the respiratory tract, uptake, retention, and clearance depend, amongst others, on whether they are particle-bound in aerosols or not. Non-particle-bound PAHs or "pure" PAHs may be inhaled as volatiles or as dry or wet aerosol. These "pure" PAHs disappear rapidly from the respiratory tract in a concentration-dependent process. At concentrations in the lungs of micrograms or more, the clearance and/or metabolism half-time of benzo(a)pyrene is less than 1 hour to 1 day, while this half-time is 1 day or more at benzo(a)pyrene concentrations in the nanogram range. However, these figures may differ considerably for other PAHs.

After inhalation as dry or wet aerosol, the "pure" PAHs will be partly metabolised already in the nasopharynx, dependent on the aerosol particle size. In rats, uptake of benzo(a)pyrene by direct diffusion through lung epithelium has been demonstrated. Following intratracheal instillation in rats with a ligated oesophagus, more than 50% of the amount of pure benzo(a)pyrene administered appeared outside the lungs within 24 hours. However, normally, clearance of PAHs from the upper respiratory tract may occur mainly via the gastrointestinal tract through mucociliary action and subsequent ingestion.

Retention and clearance of particle-bound PAHs depend on particle size-the smaller the particles, the more extensive the PAHs elute from the particles and are subsequently cleared and/or metabolised-and on the weight ratio PAH: carrier-the higher the ratio, the faster the clearance. Depending on these factors, clearance of benzo(a)pyrene generally occurs between 10 hours and 14 days. Assuming that 70% of PAH is released from smaller particles-a relative low percentage of all particles-in the deeper pulmonary parts of the lungs and 10% from the larger particles-a relatively high percentage-in the nasopharyngial and tracheobronchial parts, and that differences in particle retention on one hand counterbalance differences in PAH elution rate on the other hand, the PAH fraction actually absorbed from the lungs was estimated to be 20% of all particle-bound PAHs in outdoor air, being independent of particle size. However, the PAH fraction absorbed from pitch particles might be different. Free hydrocarbons are more rapidly cleared from the lungs than PAHs adsorbed onto particles.

As to the gastrointestinal tract, non-particle-bound PAHs were rapidly absorbed in rodents. In rats, relatively insoluble PAH (*e.g.* benzo(a)pyrene, 7,12-dimethylbenz(a)anthracene, anthracene) were substantially excreted via bile whereas more soluble compounds (*e.g.* phenanthrene, dimethylnaphthalene, 3-methylcholanthrene) were also readily excreted without bile. Metabolites excreted in the bile may return into the portal circulation (enterohepatic cycle).

Human and experimental animal data showed that non-particle-bound PAHs can penetrate the skin. Considerable differences were noted between the different PAH. In mice, hardly any dibenzo(a,h)anthracene had been taken up after 16 days, whereas the uptake of benzo(a)pyrene was 100% (Heidelberger & Weiss, 1951 cited in WHO, 1998). Also in mice, the percentage of dermal uptake of benzo(a)pyrene was found to be inversely related to the dose. Of doses of 1.25 or 12.5 $\mu g/cm^2$ (about 0.09 or 0.9 mg/kg_{bw}), approximately 82-83% had been taken up after 24 hours while of a dose of 125 $\mu g/cm^2$ (about 9 mg/kg_{bw}), approximately 41 and 93% were taken up after 24 hours and 1 week, respectively (Sanders *et al.*, 1984 cited in Montizaan *et al.*, 1989). Based on data on

PAHs, absorption of PAH components of coal tar products after dermal exposure may be limited by binding and/or metabolism in the skin, thus leaving less for systemic absorption (ATSDR, 1995). Excretion of (metabolites of) PAHs following dermal application may be detected in hours or days, and is increased due to an improved absorption by solubilization of the compounds in a fat or oil mixture prior to application. Due to the variable composition of coal tar and coal tar pitch, the predictive value of dermal absorption studies conducted with pure PAHs is limited. A further problem with the use of individual PAHs to estimate absorption of these compounds from a coal tar matrix is that individual PAHs differ in their rates of absorption. The concentrations of ten different PAHs were measured after topical application of coal tar to a blood-perfused pig-ear (Van Rooij *et al.* 1995). There was a variation of accumulations of the various PAHs in the perfused blood, ranging between 830 pmol/ cm^2 for phenanthrene to <4 pmol/cm^2 for benzo(b)fluoranthene, benzo(a)pyrene, and indeno(1,2,3,-cd)pyrene. The authors calculated absorption for the individual PAH from dermally applied coal tar during an 8 h shift of around 1 % for the higher molecular weight PAH dibenzo(a,h)anthracene and indeno(1,2,3,-cd)pyrene to over 30% for the lower molecular weight PAHs fluorene and phenanthrene, and for benzo(a)pyrene about 1-3%. These data show that different components of coal tar are absorbed at different rates, and that using a single PAH to represent absorption of the mixture is likely to over- or under-estimate the absorption of other components (ATSDR, 2002).

After absorption, PAHs are widely distributed throughout the organism to almost all organs, especially the lipid-rich ones. They can cross the placenta and reach foetal tissues. The metabolism of PAHs can take place in the liver, respiratory tract, and the skin. It is very complex leading to a variety of metabolites from a limited number of reaction types, although only a few metabolites are toxicologically relevant. Generally, the first step is a cytochrome-P450-mediated epoxidation by the mono-oxygenase enzyme arylhydrocarbonhydroxylase. Rearrangement or hydration and subsequent reactions lead to phenols, diols, diolepoxides, phenol-epoxides, and tetraols. Most metabolic processes result in detoxification conjugates with sulphuric acid, glucuronic acid, or glutathione that are excreted in urine and faeces. However, some pathways yield specific diolepoxides, which are reactive compounds capable of binding to DNA and initiating tumour formation. Generally, the metabolism appears to be qualitatively similar with respect to cell or tissue type. However, between cell types within one tissue, between different tissues, and between different organisms (species), large quantitative variations may occur caused by the inducibility and availability of enzyme systems. Since the latter show wide variations between individuals of one species also, a quite different pattern of reactive metabolites may result which may directly affect the susceptibility for carcinogenic action of PAHs.

B.5.1.1 Summary of toxicokinetics

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 are, 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%) 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 risk assessment.

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, 2003b): 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 not possible to quantify the extent of this likely overestimation of the inhalation and oral absorption rates.

It is emphasized, however, that the default absorption values are not used for consumer risk assessment, because of the absence of relevant identified exposures, nor for worker risk assessment, because both hazard- and exposure assessments are based on comparable worker scenarios, *i.e.* include the combined specific inhalation and dermal exposure conditions.

B.5.2 Acute toxicity

B.5.2.1 Studies in animals

B.5.2.1.1 In vivo studies

Acute toxicity data following single oral and dermal exposure to CTP (not further specified) or CTPHT were available and are summarised in Table B.5.1. There were no data following single exposure by inhalation.

Table B.5.1. Acute toxicity data of CTP.

Route	Species	LD_{50}/LC_{50}	Unit	Reference
Oral	rat (Wistar)	3,300	mg/kg_{bw}	Contox (1991b)
Oral	rat (Sprague-Dawley)	> 5,000	mg/kg_{bw}	Solorzano *et al.* (1993)
Oral	rat	6,200	mg/kg_{bw}	ACCCI (1992)
Oral	rat	> 15,000	mg/kg_{bw}	Steinhauser (1997)
Dermal	rat (Wistar)	> 5,000	mg/kg_{bw}	Contox (1991a)
Dermal	rat (Sprague-Dawley)	> 400	mg/kg_{bw}	Solorzano *et al.* (1993)

Inhalation

No inhalation studies were available.

As part of a study on the carcinogenic effects following intratracheal instillation of CTPHT in male and female Wistar rats, 36 out of a group of 190 received only one instillation of about 0.65, 13.7, or 20.0 mg/animal, suspended in physiological saline (particle size distribution: 90% <10 µm; 75% <5 µm), and were killed after one, two, or four weeks to study acute effects (no data on group size). A control group given a charcoal powder suspension was included as well. Both CTPHT and charcoal powder induced a similar spectrum of acute inflammatory reactions in the respiratory system, varying from accumulation of eosinophilic proteinaceous materials in the respiratory lumen to infiltration of the mucosa by neutrophils, lymphocytes, and macrophages (Chang *et al.*, 1992) (see also Section B.5.8).

Dermal

When CTP (not further specified) was applied to the clipped skin of rats (Wistar; n=5/sex/group) under occlusion for 24 hours (after which the material was removed) at doses of 0, 2000, 3500, and 5000 mg/kg_{bw}, no mortality was observed in any of the experimental groups. No clinical signs or behavioural or macroscopic changes were seen in any of the exposed animals. The dermal LD_{50} was concluded to be >5000 mg/kg_{bw} (Contox, 1991a).

No effects (mortality, clinical signs, behaviour, body weight, macroscopy) were seen following application of 400 mg/kg_{bw} CTP (not further specified) to the occluded clipped skin of rats (Sprague-Dawley; n=3/sex/group) for 24 hours (washed off thereafter) (Solorzano *et al.*, 1993).

Oral

When rats (Wistar; n=5/sex/group) were orally (gavage) given CTP (not further specified) at doses of 0, 2000, 3000, or 5000 mg/kg_{bw}, mortality occurred in 0/10, 0/10, 4/10, and 8/10 animals, respectively. Most animals died 72 to 96 hours after administration. These animals showed piloerection, decreased food intake, and general apathy. At necropsy, congestion of the digestive tract was seen. No effects were observed in the surviving animals. In this experiment, the LD_{50} was calculated to be 3300 mg/kg_{bw} (Contox, 1991b).

A single oral (gavage) dose of 200 mg/kg_{bw} CTP (not further specified) did not induce mortality or any other effect in rats (Sprague-Dawley; n=5/sex). When dosed with 5000 mg/kg_{bw} (Sprague-Dawley; n=4/sex), the only effects observed were slight piloerection lasting for 30 hours after administration and (not specified) macroscopic duodenal changes in the majority of the male animals (Solorzano *et al.*, 1993).

Other oral LD_{50}s presented in data sheets without experimental details were 6200 and >15,000 mg/kg_{bw} for rats (ACCCI, 1992; Steinhauser, 1997).

B.5.2.2 Studies in humans

No human data were available.

B.5.2.3 Summary of acute toxicity

No inhalation studies were available.

From acute oral and dermal toxicity studies conducted according to EU guidelines (and resulting in LD_{50}s of $\geq$ 3300 mg/kg_{bw}), it is concluded that CTPHT does not need classification and labelling according to EC criteria (EC-Directive 2001/59/EC; EC, 2001c) for these exposure routes.

B.5.3 Irritation

B.5.3.1 Skin

B.5.3.1.1 Studies in animals

Exposure to roofing CTPV followed by irradiation to UV light was stated to be phototoxic to the skin of guinea pigs (Emmett, 1986).

Since there were no specific irritation toxicity studies with CTPHT available, skin irritation observed after repeated exposures in the dermal carcinogenicity studies (see also Section B.5.8) are discussed underneath.

After repeated dermal exposure of 40% solutions of CTP (not further specified) in benzene painted on the hairy skin of white mice (n=49; strain and sex not reported), once weekly, for 19 months, the painted skin lost its hair after the first application. Skin changes in the treated areas were fairly similar in all experimental groups, among animals which did not develop tumours. These were atrophy or focal hyperplasia of the epidermis and the epithelium of the follicles, partial or complete atrophy of the skin papillae, and hyperkeratosis. There were also acute and chronic inflammatory phenomena observed. Animals of the control group were painted with pure benzene, and they developed epidermic atrophy, focal hyperplasia, atrophy of the hair follicles and sebaceous glands (characteristic effects of this substance) (Kireeva, 1968).

Repeated dermal 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 hyperplasia of the epidermis, frequently accompanied by inflammatory infiltration of the dermis, and on several occasions ulceration with formation of small abscesses. The mean survival time of the exposed animals was 31 weeks, whereas control animals survived to an average of 82 weeks (Wallcave *et al.*, 1971).

Since both dermal carcinogenicity studies described above do not focus on dermal irritation, these effects (or the lack of them) are not adequately described in the publications. In addition, the test substance in both studies is dissolved in benzene, which itself is classified as a skin irritant (R38). Therefore, the relevance of these studies for the evaluation of dermal irritation is rather limited.

B.5.3.1.2 Studies in humans

Hodgson & Whiteley (1970 cited in IARC, 1985) studied 144 workers of a patent-fuel plant where coal dust and CTP (no further specification given) were fused by steam heat to form fuel ovoids. Pitch warts (clinically and histologically keratoacanthomas) occurred, 26% of which regressed spontaneously. Tar keratoses among pitch-tar workers exposed to CTP (no further definition given) may occur after exposure to the causal agents has been discontinued (Gotz, 1976 cited in IARC, 1985). Crow (1970 cited in IARC, 1985) described 54 cases of acne due to pitch fumes (from CTP, not further specified). An eruption of pure comedones (morphologically distinguishable from those seen in chloracne) occurred, particularly on the malar region of the face. These generally healed rapidly but sometimes remained and could recur more than ten years later. Folliculitis of the thighs and forearms was common.

Cutaneous photosensitivity from coal-tar pitch has been described by a number of authors. Pitch ‘smarts’, intense burning sensations, occur on direct exposure to sunlight; erythema usually follows (IARC, 1985). Emmet (1986) performed on-site surveys of more then 50 roofers. Most roofers complained of skin and lip irritation from pitch, ranging in severity from a burning sensation upon exposure to the sun to extensive bulla formation. The burning often starts within an hour of beginning exposure to pitch and sunlight, and continues to worsen as long as exposure continues. Erythema and blistering might follow the onset of burning, particularly in fair-skinned individuals. Slight burning might also occur if pitch gets under gloves, but this is greatly accentuated on subsequent sun exposure (Emmett, 1986).

B.5.3.2 Eye

B.5.3.2.1 Studies in animals

Roofing CTPV produced phototoxic keratoconjunctivitis in New Zealand white rabbits. Treatment with the pitch distillate (10 µL) alone produced slight transient effects such as vasodilatation of the lids, tearing, and slight mucosal discharge at five and 24 hours after application in two out of six eyes while marked photophobia, conjunctival irritation, corneal swelling, opalescence, and corneal ulceration were seen in all six eyes after treatment followed by irradiation to UV light. The changes were most pronounced after 24 hours, and had resolved after 96 and 120 hours. UV light alone did not have such effects (Emmett, 1986; Grant, 1986; IARC, 1985). In addition, Grant (1986) refers to a paper from D’Asaro Biondo (1933 cited in Grant, 1986) in which pitch was found to cause irritation and injury to the eyes of rabbits.

B.5.3.2.2 Studies in humans

Occupational exposure to (undefined) coal tar pitch (fumes, volatiles) have been reported to cause irritation and, if repeatedly, chemosis of the conjunctiva, ulceration and infiltration of the cornea, accompanied with the presence of pus (hypopyon), and adherent leukoma. Furthermore, chronic exposure to dust resulted in deep staining of the cornea in the palpebral fissure, conjunctival discolouration and irritation, and deformities of the lower lid. Phototoxic effects-burning sensation

of the eyes aggravated by sunlight-have been documented as well (Emmett, 1986; Grant, 1986; IARC, 1985).

B.5.3.3 Respiratory tract

B.5.3.3.1 Studies in animals

As part of a study on the carcinogenic effects following intratracheal instillation of CTPHT in male and female Wistar rats, 36 out of a group of 190 received only one instillation of about 0.65, 13.7, and/or 20.0 mg CTP/animal, suspended in physiological saline (particle size distribution: 90% <10 µm; 75% <5 µm), and were killed after one, two, or four weeks to study acute effects (no data on group size). A control group given a charcoal powder suspension was included as well. Both CTPHT and charcoal powder induced a similar spectrum of acute inflammatory reactions in the respiratory system, varying from accumulation of eosinophilic proteinaceous materials in the respiratory lumen to infiltration of the mucosa by neutrophils, lymphocytes, and macrophages (Chang *et al.*, 1992) (see also Section B.5.8).

B.5.3.3.2 Studies in humans

No data available.

B.5.3.4 Summary of irritation

Combined exposure to CTPV and UV radiation was phototoxic to the skin of guinea pigs. No other dermal irritation studies with CTPHT are available and the dermal effects observed after repeated exposures in the dermal carcinogenicity studies were of limited relevance for dermal irritation, since benzene (a known skin irritant) was used as vehicle and dermal irritation (or the lack of irritation) was not adequately described.

Among workers exposed to CTP (fumes) dermal effects including pitch warts (clinical and histological keratoacathomas), tar keratoses, acne (morphologically distinguishable from those seen in chloracne) and folliculitis of the thighs and forearms were observed. Among roofers, with combined exposure of CTP and sunlight, skin and lip irritation were observed, ranging in severity from a burning sensation upon exposure to the sun to extensive bulla formation. However, from the available human data it is not possible to tell if the observed dermal effects are caused by irritation and sensitisation.

Skin effects were observed in animals and humans after repeated exposure to CTP(V) or combined exposure to CTP(V) and sunlight. However, from the available animal and human data it is not possible to tell if the observed dermal effects are caused by irritation and sensitisation (photosensitisation or sensitisation after repeated exposure), therefore classification of CTPHT for skin irritation is not possible.

In view of the human data on occupation exposure to CTP (fumes, volatiles and dust, not further specified) which show eye irritation and, after repeated exposure, chemosis of the conjunctiva, ulceration and infiltration of the cornea, deep staining of the cornea, and conjunctival discolouration and irritation, classification as ´irritant´ with ´risk of serious damage to eyes´ (Xi, R41) is proposed. Sunlight aggravated irritating effects of CTP(V) on the eyes and skin.

B.5.4 Corrosivity

No human or animal data on the potential corrosivity of CTPHT required as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967) were available.

B.5.5 Sensitisation

No experimental data on the potential sensitising properties of CTPHT required as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967) were available. Skin effects were observed in

animals and humans after repeated exposure to CTP(V) or combined exposure to CTP(V) and sunlight (see Section B.5.3.1). However, from the available animal and human data it is not possible to tell if the observed dermal effects are caused by irritation and sensitisation (photosensitisation or sensitisation after repeated exposure), therefore classification of CTPHT for skin sensitisation is not possible based on these studies. However, it should be noted that one of the components of CTPHT (benzo(a)pyrene) is a sensitizer (WHO, 1998).

B.5.5.1 Summary of sensitisation

The available data set does not meet the basic requirements as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967). According to the Directive 1999/45/EC relating to the classification, packaging and labelling of dangerous preparations (EC, 1999b), preparations containing more than 1% of a skin sensitising substance need to be classified as a skin sensitizer. Since CTPHT may contain up to 1.5% benzo(a)pyrene (a skin sensitizer) it is proposed to classify CTPHT as a skin sensitizer (R43).

B.5.6 Repeated dose toxicity

B.5.6.1 Studies in animals

B.5.6.1.1 In vivo studies

Apart from one oral study of limited significance (see below), no repeated dose toxicity studies addressing effects other than carcinogenicity were available to the rapporteur.

Oral

Outbreaks of coal tar pitch poisoning in grazing swine and pigs resulting in mortality and histological liver changes, reported in the 1920s and 1930s, instigated experiments in which groups of 9-week-old pigs were orally (diet and/or capsule) given powdered clay pigeon remnants (consisting of CTP, finely ground limestone, calcimine), powdered clay pigeon plus lead, or commercial CTP (not further specified). Two additional groups given lead and a normal diet, respectively, were included. In the first group (n=5) given a total dose of 57 g powdered remnants during five subsequent days, all animals died within eight to 20 days. At autopsy, jaundice, excessive serous fluid in the abdomen, oedematous visceral lymph nodes, and marked degenerative liver changes were found in four out of five animals. In the remaining fifth animal, there were no gross liver effects. Treatment with a total dose of 57 g of powdered remnants together with a total dose of 25 g of lead (exposure period: 5 days) caused the death of four out of five animals within 22 days. At autopsy, animals showed hepatic lesions. No such lesions were seen in the surviving animal sacrificed after 60 days.

In the last experimental group, liquid coal tar was administered by capsule at doses of 3 g/day, for five (n=3) or two (n=2) days. All three animals dosed for five days died within 10 to 18 days showing diffuse degenerative changes in the liver. One of the pigs dosed for two days died (at day 38). At autopsy, there were no gross liver lesions, but pseudomelanosis of the entire colon was seen. In the remaining animal sacrificed after 60 days, an extensive moist, proliferative dermatitis of unknown origin, but no liver changes were reported. No gross pathology changes were seen in the diet-control group. In the animals given lead alone, one out of five animals died within 30 days showing a marked haemorrhagic gastritis. No gross lesions were reported in the surviving animals (Graham *et al.,* 1940).

B.5.6.1.2 Repeated dose toxicity observed in carcinogenicity studies

Since apart from this above reported study no repeated dose toxicity studies addressing effects other than carcinogenicity were available to the rapporteur, other effects observed than carcinogenicity, following repeated exposures in the carcinogenicity studies are also shortly discussed underneath,

though some may be preneoplastic in nature or at least related to the carcinogenic effects of CTPHT (see also Section B.5.8).

Inhalation

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, or 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 B.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. All reported histological dose-dependent changes appear related to the carcinogenic response, found (*i.e.* hyperplastic, metaplastic, and dysplastic) were mainly found located in the bronchiolo-alveolar areas, and dose-dependent as to severity (Chang *et al.*, 1992). As the focus was on carcinogenic effects, no NOAEL for non-carcinogenic effects could be derived from this study.

Dermal

After repeated dermal exposure of 40% solutions of CTP in benzene painted on the hairy skin of white mice (n=49; strain and sex not reported), once weekly, for 19 months, the painted skin lost its hair after the first application. Skin changes in the treated areas were fairly similar in all experimental groups, among animals which did not develop tumours. These were atrophy or focal hyperplasia of the epidermis and the epithelium of the follicles, partial or complete atrophy of the skin appillae, and hyperkeratosis. There were also acute and chronic inflammatory phenomena observed. Animals of the control group were painted with pure benzene, and they developed epidermic atrophy, focal hyperplasia, atrophy of the hair follicles and sebaceous glands only (characteristic effects of this substance) (Kireeva, 1968).

Repeated dermal 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 hyperplasia of the epidermis, frequently accompanied by inflammatory infiltration of the dermis on several occasions ulceration with formation of small abscesses. The mean survival time of the exposed animals was 31 weeks, whereas control animals survived to an average of 82 weeks (Wallcave *et al.*, 1971).

Both repeated dermal application studies reported local effects on the skin. In these studies CTPHT was however applied in benzene, a substance which also causes local skin effects and the focus in the studies was on local dermal carcinogenicity. Therefore, these studies are not suitable for establishing a dermal NOAEL for local effects. In addition, regarding systemic effects only mortality was reported. Therefore, these studies are also not considered acceptable for derivation of a systemic NOAEL.

Oral

In a study conducted by Culp *et al.* (1998), the tumorgenicity 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 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 as equivalent to 0, 36, 120, and 360 mg/kg_{bw}) (both coal tar doses corrected for observed reductions in food consumption). Benzo(a)pyrene was fed at doses of 0, 5, 25, and 100

ppm (calculated by the rapporteur 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}) 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).

In conclusion:

- regarding coal tar mixtures, based on the increased mortality and liver weights and decreased food consumption and body weights were observed in mice from a dose level of 360 mg/kg_{bw}/day. A dose level of 120 mg/kg_{bw}/day was considered a NOAEL.
- regarding benzo(a)pyrene, a NOAEL of 0.6 mg/kg_{bw}/day was established for benzo(a)pyrene (at the higher dose levels a lower survival rate was observed).

In a study conducted by 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. 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 relatedly 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 (Kroese *et al.*, 2001). Based on a dose related decrease in survival after exposure to benzo(a)pyrene, a LOAEL of 3 mg/kg_{bw}/day was established.

Oral administration of acenaphthene, fluoranthene, fluorene, pyrene, and anthracene, resulted in NOAELs of 175 mg/kg_{bw}/day acenaphthene for hepatotoxicity; 125 mg/kg_{bw}/day fluoranthene for nephropathy, increased relative liver weights, and haematological and clinical effects; 125 mg/kg_{bw}/day fluorene for altered haematological parameters; 75 mg/kg_{bw}/day pyrene for nephropathy; and 1000 mg/kg_{bw}/day anthracene (highest dose tested) (WHO, 1998).

B.5.6.1.3 Repeated dose toxicity with high-boiling coal liquid

Since apart from one oral study of limited significance and some carcinogenicity studies discussed above no other repeated dose toxicity studies with CTPHT were available, some studies with coal tar derived material and high-boiling coal liquid are described underneath.

In a study by Springer *et al.* (1986b) Fischer rats were exposed to 30, 140, and 690 mg/m^3 of a high-boiling coal liquid aerosol (heavy distillate (HD), the highest-boiling material derived from the solvent refined coal-II (SRC-II) process) for 6 hours/day, 5 days/week for 5 or 13 weeks. Rats exposed to concentrations of high-boiling coal liquid aerosol of 30 mg/m^3 for 5 or 13 weeks showed histiocytosis of the lung tissue. At higher dose levels more serious effects were observed. Based on the available data no NOAEC could be established. The LOAEC was 30 mg/m^3.

In another study by Spinger *et al.* (1987) CD-1 mice were exposed to aerosol concentration of 0, 30, 140, and 690 mg/m^3 of heavy distillate, a high-boiling coal liquid form the solvent-refined coal (SRC)-II process. Relative liver weights were significantly increased and subtle changes in liver histology (a slight increase in cytoplasmic basophilia, slightly more variability in hepatocellular size, the presence of hepatomegalocytes, increased variability in nuclear size, minimal loss of

cording and lobular pattern, and minimal scattered focal necrosis) were noted in mice exposed to 690 mg/m^3 (females) or 140 mg/m^3 (males) of a high-boiling coal liquid aerosol for 6 hours/day, 5 days/week for 13 weeks (Springer *et al.* 1987). No changes in weight or histology were observed for female mice exposed to 30 or 140 mg/m^3 or male mice exposed to 30 mg/m^3 for 13 weeks (Springer *et al.* 1987). Therefore, the NOAEC in this study was 30 mg/m^3.

In a study by Weyand *et al.* (1994) B6C3F1 mice were 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 (MGP) residue (a by-product of coal gasification, coal-tar like material) for 94 or 185 days. No adverse effects of the treatment were observed in any dose groups (NOAEL of 462 mg/kg/day (males) and 344 mg/kg/day (females)).

B.5.6.2 Studies in humans

Inhalation

The effect on lung function (forced vital capacity-FVC, forced expiratory volume in 1 second-FEV_1, forced respiratory flow rate from 25% to 75% of FVC in 1 second-FEF_{25-75}) due to exposure to respiratory irritants including phosphorus oxides, fluorides, and CTPV has been investigated in 131 workers of a Canadian phosphorus rock refinery. Workers had been employed from three to 46 years. Data on at least four annual pulmonary function determinations and on smoking history were available. During the study, maximal concentrations measured were about 2.2, 4.2, and 0.1 mg/m^3 for phosphorus pentoxide, fluorides, and CTPV, respectively. After adjustment for smoking and age, no residual significant effect of exposure to these irritants was found at regression analysis of the longitudinal and cross-sectional lung function data. Statistically significant reductions in the lung function parameters were found in smokers but not in non-smokers or former smokers. However, after adjustment for smoking, no statistically significant changes remained that could be ascribed to factors such as years of exposure to the chemical compounds mentioned (Dutton *et al.*, 1993).

B.5.6.3 Summary of repeated dose toxicity

Regarding animals, since apart from one oral study of limited significance in pigs, no repeated dose toxicity studies with CTPHT addressing effects other than carcinogenicity were available to the rapporteur, the available data set does not meet the basic requirements as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967)and no NOAEL for non-carcinogenic effects could be derived from these studies.

In humans no statistical significant effects on lung function parameters were found in a group of phosphorus rock refinery workers exposed at the time of study to about 0.1 mg/m^3 CTPV in addition to other substances (including phosphorus pentoxide (about 2.2 mg/m^3) and fluorides (about 4.2 mg/m^3).

In addition, animal data was available on high-boiling coal liquid (LOAEC of 30 mg/m^3 in rats regarding semichronic inhalation exposure), and Manufactured Gas Plant (MGP) residue (a coal-tar like material) (NOAEL of 462 mg/kg/day (male mice; oral exposure) and 344 mg/kg/day (female mice; oral exposure). These, however, are not considered representative for establishing a NOAEL value for risk characterisation of CTPHT.

B.5.7 Mutagenicity

In vitro and *in vivo* genotoxicity tests with CTP or CTPV are summarised in Table B.5.2 and Table B.5.3, respectively.

B.5.7.1 Studies *in vitro*

B.5.7.1.1 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).

B.5.7.1.2 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/m^3 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 B.5.2. Genotoxicity of CTP or CTPV *in vitro.*

Assay	Compound	Species	Result	Reference
Bacteria				
Bacterial gene mutation test	CTP	*S. typhimurium* (TA 98, 100)	Positive +S9 in TA98, negative -S9 in TA98 and +S9 and -S9 in TA100	Solorzano *et al.* (1993)
Bacterial gene mutation test	Cyclohexane extracts of dust samples in iron foundries (using CTP)	*S. typhimurium* (TA 98 100)	Positive +S9	Schimberg *et al.* (1980)
Bacterial gene mutation test	Condensates of fumes generated from CTP by heating to 232 or 316 ˚C	*S. typhimurium* (TA 98)	Positive +S9	Machado *et al.* (1993)
Bacterial gene mutation test	DMSO extract of (unspecified) CTP	*S. typhimurium* (TA 98, 100, 1537)	Positive +S9, negative -S9	IARC (1985)
Bacterial gene mutation test	Dichloromethane extract of roofing-tar pot emissions	*S. typhimurium* (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)
Yeast				
Mitotoc recombination	Dichloromethane extract of roofing-tar pot emissions	*S. cerevisiae* D3	Negative without metabolic activation	IARC (1985)
Mammalian cells				
DNA fragmentation	Dichloromethane extract of roofing-tar pot emissions	Syrian hamster embryo cells	Negative without metabolic activation	IARC (1985)

Assay	Compound	Species	Result	Reference
Gene Mutation	Dichloromethane extract of roofing-tar pot emissions	BALB/c3 3T3 cells (oubain 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)
Human body fluids				
Bacterial gene mutation test	Human urine sample, occupational exposure in an aluminium reduction pant to a.o. CTPV	*S. typhimurium* (TA 98 100)	Positive with and without metabolic activation	Heussner *et al.* (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	*S. typhimurium* (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	*S. typhimurium* (TA 98 100)	Positive with and without metabolic activation	WHO (1998)
Bacterial gene mutation test	Human expectorate sample, occupational exposed workers of coke plant and aluminium (Søderberg potrooms) plant	*S. typhimurium* (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) (ATSDR, 2002; WHO, 1998).

B.5.7.2 Studies *in vivo*

B.5.7.2.1 In vivo*: animal 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 $B6C3F_1$ mice fed up to 2 g coal tar/100 g food for 28 days (Culp & Beland, 1994; Culp *et al.*, 1996ab). DNA adducts were detected in liver, lung, and forestomach by ^{32}P-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) (ATSDR, 2002; WHO, 1998).

B.5.7.2.2 In vivo*: 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/m^3) and 20.5 mg/m^3 (range: 0.13-1212 mg/m^3, respectively, *vs.* 0.700 mg/m^3 (0.120-3.830 mg/m^3) in controls -, some differences in individual PAH among which benzo(a)pyrene (0.068 and 0.219 mg/m^3, 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 µg/m^3) and 0.11 µg/m^3 (range: 0.01-0.37 µg/m^3) for workers and 3/5 controls (no detectable levels in 2 other ones), respectively. Median Benzo(a)pyrene levels were 1 µg/m^3 (range: 0.02-24 µg/m^3) for the workers and 0.004 and 0.02 µg/m^3 for the 2 controls with measurable levels. Levels of the sum of 7 gas phase congeners ranged from 0.01 to 131 µg/m^3 (median: 16.3 µg/m^3) in workers to 0.008 to 0.41 µg/m^3 (median: 0.20 µg/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) (ATSDR, 2002; WHO, 1998).

Table B.5.3 Genotoxicity of CTP or CTPV *in vivo*.

Endpoint	Compound	Species	Result	Reference
Human blood cells				
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 *et al.* (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 *et al.* (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 *et al.* (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 *et al.* (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 *et al.* (1998)
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 *et al.* (1999)
Aromatic DNA adducts (by ^{32}P-postlabelling)	Occupational exposure in potroom workers of an aluminium reduction plant	Human peripheral blood lymphocytes	No statistical significant differences between exposed and non-exposed	Carstensen *et al.* (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 *et al.* (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 *et al.* (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 *et al.* (1999a)

Endpoint	Compound	Species	Result	Reference
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)

B.5.7.3 Summary 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 (EC, 1967). However, numerous genotoxicity studies with coal tar, coal tar waste, coal tar products, and individual PAHs demonstrated the genotoxicity of these substances (ATSDR, 2002; WHO, 1998).

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 the Directive 1999/45/EC relating to the classification, packaging and labelling of dangerous preparations (EC, 1999b), preparations containing more than 0.1% of category 1 or 2 mutagens need to be classified as a category 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.

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 2 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 2 mutagen is proposed (T; R46).

B.5.8 Carcinogenicity

B.5.8.1 Studies in animals

Inhalation

Female rats (Wistar; n=72 group) were exposed to 0, 1.1, or 2.6 mg/m^3 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/m^3 aerosols contained among others 20 and 46 µg/m^3 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/m^3/h, respectively. Exposure to 2.6 mg/m^3 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 adenocarinomas were found. Tumour rates were 4.2 and 33.3% for the animals exposed to 1.1 mg/m^3 for 43 and 86 weeks, respectively, and 38.9 and 97.2% for the animals exposed to 2.6 mg/m^3 for 43 and 86 weeks, respectively (Heinrich *et al.*, 1986; Heinrich *et al.*, 1994ab).

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 B.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/m^3 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/m^3 benzo(a)pyrene, 16 hours/day, 5 days/week, for 15 months. The particle mass concentrations and the MMAD were 1.1 mg/m^3 and 0.1 µm, respectively, for the coal stove combustion product and 4.6 (±5.1) mg/m^3 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/m^3 of an aerosol generated by pyrolizing 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 ± 0.03 µm. They contained 0, 50, and 90 µg/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).

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).

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 tumorgenicity 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).

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 B.5.4). 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 B.5.5). 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$_{bw}$.

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 ^{32}P-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 B.5.4. 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.

Table B.5.5. 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 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 B.5.6). 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).

Table B.5.6. 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.

B.5.8.2 Studies in humans

B.5.8.2.1 Introduction

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). Several additional studies have been published (Armstrong *et al.*, 1994; Cullen *et al.*, 1996; Partanen & Boffetta, 1994; Ronneberg & Andersen, 1995; Ronneberg & Langmark, 1992; Stern *et al.*, 2000; Tremblay *et al.*, 1995; Armstrong *et al.*, 2004). Quantitative cancer risk estimates have been calculated by Armstrong *et al.* (1986; 1994), and Tremblay *et al.* (1995) attempted to quantify the relationship between exposure to CTPV in Søderberg 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. The summary is based on the publications by IARC and HSE. These publications were not consulted individually. More detailed tables with descriptions of the studies are included in Annex J.4. 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.

B.5.8.2.2 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. The studies are summarised in Table J.4.1 (see Annex J.4).

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) and HSE (HSE, 1993; Armstrong *et al.*, 2003). Five more recent publications not included in the reviews (Armstrong *et al.*, 1994; Cullen *et al.*, 1996; Ronneberg *et al.*, 1999; Ronneberg & Andersen, 1995; Tremblay *et al.*, 1995; Romundstad *et al.*, 2000ab; Armstrong 2003, 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 (see Table J.4.2, Annex J.4) 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. The studies are summarised in Table J.4.3 (Annex J.4).

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 (Donato *et al.*, 2000; Moulin *et al.*, 1989).

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 Table J.4.4 (Annex J.4). 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.

B.5.8.2.3 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 risk assessment 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 × 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 µg/m^3.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:

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

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 µg/m^3·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 µg/m^3·year cumulative benzo(a)pyrene (~ 40 years exposure to an average concentration of 2.5 µg/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 µg/m^3·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 µg/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 (Romundstad, 2000ab; Tremblay *et al.*, 1995). 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 µg/m^3·year cumulative benzo(a)pyrene exposure.

B.5.8.2.4 Summary of carcinogenicity

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 amongst others, 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.

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, 1993; Armstrong *et al.*, 2003). The IARC concluded that there is sufficient evidence that coal-tar pitches are carcinogenic in humans already in 1985 (IARC, 1985, 1987). Several additional studies have been published since including some attempting to derive quantitative cancer risk estimates. Armstrong *et al.* (2003; 2004) performed a meta-analysis which showed statistically increased overall relative risks for lung and bladder cancer risk for all CTPV exposure scenarios, and after exposure to PAHs an industry-specific increased relative risk for workers exposed in aluminium smelters.

Based on the available experimental and epidemiological data on the carcinogenicity of CTPHT and CTPVHT and the evaluation of these data by the IARC, CTPHT and CTPVHT has to be classified as a category 1 carcinogen (T; R45).

In Section A.1.1 it is concluded that in this transitional dossier focus will be on the carcinogenic and mutagenic properties, using the best-studied PAH benzo(a)pyrene as a guidance substance for

exposure to CTPHT in various worker exposure scenarios. In line with this, in the meta-analyses of Armstrong *et al.* (2003; 2004) on lung and bladder cancer benzo(a)pyrene was also used as exposure indicator. In addition, as this analysis has collected, analysed and summarised all available data on CTPHT exposures in a scientifically sound manner, it is considered to yield the best estimates of risks associated with CTPHT exposures in the various occupational scenarios.

It is noted though, that although this meta-analysis yields statistically stable estimates for relative risk values, they still harbour some uncertainties such as possible effects of possible carcinogenic co-exposures (for example, in aluminium smelters, very limited co-exposures are possible to some metals such as copper, chromium, nickel, manganese, cobalt; Healy *et al.*, 2001). Regarding confounding by smoking, borderline statistically significant higher estimates for the studies adjusted for smoking than for those that had not were observed (URR = 1.31, 95% confidence interval: 1.16-1.48 versus 1.16, 95% confidence interval: 1.11-1.21, respectively) (see Section B.5.8.2). The higher risk estimated from the studies that did control for smoking prove at least that the risk of cancer is not always overestimated when no adjustment is made.

Furthermore, the use of standard industry- and job-specific exposure values assigned to those studies without quantitative exposure data adds uncertainty to the estimates.

However, taking these uncertainties into account, the estimates based on the meta-analyses which include all evidence are considered the best estimates of the risk on lung and bladder cancer risk due to exposure of CTPHT. A meta-analysis is generally chosen as the best way not only to combine all available epidemiological evidence on an exposure-response association (besides pooling the raw data from the studies), but also to evaluate consistency of the association across the underlying studies and investigate sources of heterogeneity (if any). Single epidemiological studies may present extreme results, both in negative or positive direction (due to random error, possible uncontrolled biases, or particular circumstances applying to the study population or exposure conditions). In a state-of-the-art conducted meta-analysis, the meta-analyst carefully considers, based on all evidence, whether a summary risk estimate can reliably be derived. In the meta-analysis on lung cancer by Armstrong *et al.* (2003; 2004) it was concluded that this was the case. Therefore, the results of the meta-analysis are considered more reliable and closer to the true dose-response association than that of the individual studies. Moreover, the estimates are statistically much more stable and much less uncertain and therefore provide robust evidence, which is also in accordance with evidence from animal studies and IARC evaluations. Therefore, the relative risk value (URR) found for lung cancer in this meta-analysis is forwarded to the risk characterisation: an overall relative risk estimate (URR) of 1.20 (95% confidence interval: 1.11-1.29) per *unit* of 100 $\mu g/m^3 \cdot$year cumulative benzo(a)pyrene exposure.

Furthermore, for aluminium smelters, the only industry exposed to CTPVHT for which rather precise estimates could be established in the meta-analysis, the combined URR estimate was 1.16 (95% confidence interval: 1.05-1.28) for lung cancer. This value will be taken forward to the risk characterisation for aluminium smelters.

Regarding bladder cancer, for which the association with PAH exposure was less robust than the PAH-lung cancer association, the overall relative risk estimate (URR) of 1.33 (95% confidence interval: 1.17-1.51) per unit of 100 $\mu g/m^3 \cdot$year cumulative benzo(a)pyrene exposure is forwarded to the risk characterisation.

Furthermore, for aluminium smelters, the only industry exposed to CTPVHT for which rather precise estimates could be established in the meta-analysis, the combined URR estimate was 1.42 (95% confidence interval: 1.23-1.65) per unit of 100 $\mu g/m^3 \cdot$year cumulative benzo(a)pyrene exposure for bladder cancer. This value will be taken forward to the risk characterisation for aluminium smelters.

Furthermore, it is noted that lung cancer has a larger impact than bladder cancer on the excess life time risks (see Sections B.10.1.2.7 and B.10.1.4.3) and is therefore the most critical type of cancer with regard to exposure to CTPHT.

B.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.

B.5.9.1 Effects on fertility

B.5.9.1.1 Studies in animals

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/m^3 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/m^3 and higher. Examination of ovarian section showed a significant decrease in the amount of luteal tissue in rats exposed to 690 mg/m^3 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 (Creosote Council Europe, 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 17α-ethynylestradiol (positive control) once a day for 4 days. Treatment with 17α-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.

B.5.9.1.2 Studies in humans

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, 2004).

B.5.9.2 Developmental toxicity

B.5.9.2.1 Studies in animals

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. Embryotoxicity 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/m^3 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 (Creosote Council Europe, 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 (Creosote Council Europe, 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).

B.5.9.2.2 Studies in humans

Studies on developmental effects in humans are not available.

B.5.9.2.3 Summary of toxicity for reproduction

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/m^3 (NOAEC 30 mg/m^3). At the highest tested concentration (690 mg/m^3) 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) (Hackett *et al.* 1984; Zangar *et al.*, 1989; Springer *et al.* 1982, 1986b, 1987; Creosote Council Europe, 2004).

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 (category 2). According to the Directive 1999/45/EC relating to the classification, packaging and labelling of dangerous preparations (EC, 1999b), preparations containing more than 0.5% of a substance classified as toxic for reproduction in category 2 should be classified as a toxic for reproduction. For these reasons it is proposed to classify CTPHT as toxic to reproduction (category 2; T, R60/61).

Table B.5.7 Summary of reproductive toxicity studies with high-boiling coal liquid, coal tar derived products, and creosote.

Type of study	Species	Exposure	Results	Reference
Effects on fertility				
Repeated dose toxicity study-Inhalation	Mouse	0, 30, 140, or 690 mg/m^3 coal liquid aerosol; 6 hours/day; 5 days/week; 13 weeks	$NOAEL_{fertility}$: 140 mg/m^3	Springer *et al.*, 1987
Repeated dose toxicity study-Inhalation	Rat	0, 30, 140, or 690 mg/m^3 coal liquid aerosol; 6 hours/day; 5 days/week; 5 weeks	$NOAEL_{fertility}$: 690 mg/m^3	Springer *et al.*, 1986b
Repeated dose toxicity study-Inhalation	Rat	0, 30, 140, or 690 mg/m^3 coal liquid aerosol; 6 hours/day; 5 days/week; 13 weeks	$NOAEL_{fertility}$: 30 mg/m^3	Springer *et al.*, 1986b
Repeated dose toxicity study-Oral	Mouse	0, 51, 251, 462 mg/kg/day (males) 0, 42, 196, 344 mg/kg/day (females) MGP [a] residue for 94 and 185 days	$NOAEL_{fertility}$: 462 mg/kg/d $NOAEL_{fertility}$: 344 mg/kg/d	Weyand et al, 1994
Two-Generation Reproduction/ Fertility toxicity study-Oral	Rat	0, 25, 75, or 150 mg/kg/d creosote in corn oil by gavage; 56 days (F_0), ≥ 113 days (F_1)	$LOAEL_{fertility}$: 25 mg/kg/d $NOAEL_{maternal}$: 25 mg/kg/d	Creosote Council Europe, 2004
Oestrogen activity study-Oral	Mouse	0, 10, 50, 100 mg/kg coal tar creosote; once a day for 4 days	$NOAEL_{fertility}$: 100 mg/kg/d	Fielden *et al.*, 2000
Developmental toxicity				
Developmental toxicity study-halation	Rat	0, 17, 84, or 660 mg/m^3 coal liquid aerosol; 6 hours/day; on gestational days 12-16	$NOAEL_{development}$: 84 mg/m^3 $NOAEL_{maternal}$: 84 mg/m^3	Springer *et al.*, 1982
Developmental toxicity study-Oral	Rat	0, 90, 140, 180, 370, 740 mg/kg/day coal liquid on gestation days 12-16	$NOAEL_{development}$: 90 mg/kg/d $LOAEL_{maternal}$: 90 mg/kg/day	Hackett *et al.*, 1984
Developmental toxicity study-Oral	Rat	740 mg/kg/day coal liquid on gestation days 12-14	$NOAEL_{development}$: < 740 mg/kg/d $NOAEL_{maternal}$: < 740 mg/kg/d	Springer *et al.*, 1986a
Developmental toxicity study-Oral	Rat	0, 25, 50, or 175 mg/kg/d creosote in corn oil by gavage on gestational days 12-15.	$NOAEL_{development}$: 50 mg/kg/d $NOAEL_{maternal}$: 50 mg/kg/d	Creosote Council Europe, 2004
Developmental toxicity study-Oral	Rat	0, 25, 75, or 225 mg/kg/d creosote in corn oil by gavage on gestational days 12-15.	$NOAEL_{development}$: 75 mg/kg/d $NOAEL_{maternal}$: 75 mg/kg/d	Creosote Council Europe, 2004
Developmental toxicity study-Dermal	Mouse	0, 500, 1500 mg/kg coal liquid on gestation days 11-15	$LOAEL_{development}$: 500 mg/kg/d $LOAEL_{maternal}$: 500 mg/kg/d	Zangar *et al.*, 1989
Developmental toxicity study-Dermal	Pig	98.5% coal tar creosote 2-10 days before delivery	Fetotoxic without apparent maternal toxicity	Schipper, 1961

[a] Manufactured Gas Plant

B.5.10 Other effects

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

B.5.11 Derivation of DNEL(s)/DMEL(s) or other quantitative or qualitative measure for dose response

No DNEL(s)/DMEL(s) or other quantitative measure for dose response were derived. Based on several meta-analyses some qualitative measure for carcinogenicity could be derived (see Section B.5.8.2.4, summarized in Table B.5.8), which were forwarded to the risk characterisation (see Sections B.10.1.2.7 and B.10.1.4.3).

Table B.5.8. Relative risk values for lung and bladder cancer.

	relative risk value (95% confidence interval) [a]	
lung cancer	1.20	(1.11-1.29)
aluminium smelters [b]	1.16	(1.05-1.28)
bladder cancer	1.33	(1.17-1.51)
aluminium smelters [b]	1.42	(1.23-1.65)

[a] Values are expressed per unit of 100 µg/m^3·year. [b] Values for the specific type of cancer for aluminium smelters only.

B.6 Human health hazard assessment of physico-chemical properties

B.6.1 Explosivity

CTPHT is not explosive.

B.6.2 Flammability

CTPHT is not flammable.

B.6.3 Oxidising properties

CTPHT is not oxidising.

B.7 Environmental hazard assessment

In the effect assessment below the ecotoxicity data has been evaluated for the 16 EPA PAHs separately and subsequently PNEC for the individual PAHs have been derived. The data from both literature and other EU RARs are used. In the different sections the most decisive toxicity studies are described. In the tables presented in Annex J.3, an overview is given of all studies evaluated.

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. Therefore these effects are also considered in deriving the PNECs for aquatic species. It should be noted that the UV exposure levels of the selected studies did not exceed the UV levels under natural sun light conditions.

In the final risk characterisation the local concentration (C_{local}) for each PAH will be divided by its PNEC. Subsequently all C_{local}/PNEC ratios will be added to determine the risk of the 16 EPA PAHs as a whole (see Section B.9.5).

B.7.1 Aquatic compartment (including sediment)

B.7.1.1 Naphthalene

B.7.1.1.1 Fresh water environment

In the EU-RAR for naphthalene (Munn *et al.*, 2003) a PNEC of 2.4 µg/L was derived for water. This was based on the NOEC of 120 µg/L from a study with fry of the pink salmon (*Oncorhynchus gorbuscha*) tested in a flow-through system for 5 weeks (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 LC_{50} 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 LC_{50} 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 EC_{10} 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 EC_{10} 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 EC_{10}s 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. Therefore, the EC_{10} is considered to be useful in this case together with the less conservative assessment factor of 10 (see below).

B.7.1.1.2 Marine environment

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 *et al.*, 1982; Seathre *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.

B.7.1.1.3 Calculation of Predicted No Effect Concentration (PNEC)

Freshwater environment

For freshwater acute toxicity studies are available for algae, *Daphnia*, and fish. In the EU-RAR (Munn *et al.*, 2003) no chronic toxicity data for algae were available. Hence, an assessment factor of 50 was applied. Besides the EC_{10} that can be derived from the data after 1 day of exposure of *Chlorella vulgaris* to naphthalene, presented by Kauss & Hutchinson (1975), a well-performed algae toxicity study is now available for *Pseudokirchneriella subcapitata*, giving a EC_{50} value of > 4270 µg/L (Bisson *et al.*, 2000). Therefore, the use of an assessment factor of 10 instead of 50 seems to be justified. The lowest usable effect concentration for fresh water species is the EC_{10} from the ELS study with *Oncorhynchus mykiss* of 20 µg/L. The PNEC is thus 2.0 µg/L. This value is almost identical to the value derived in the EU-RAR for naphthalene (Munn *et al.*, 2003). However, an assessment factor of 10 has been used here instead of 50.

Marine environment

With four taxonomic groups for marine species, an assessment factor of 10 can be applied to the lowest NOEC. This is the NOEC for the development of larvae of *Cancer magister* of 21 µg/L. In this study only two exposure concentrations are used. Although well-performed, the statistical power of this test is limited. The PNEC for fresh water of 2.0 µg/L can therefore be used for both fresh water and marine species. It should be noted that in two studies with marine species effects were still observed at levels below the lowest NOEC of 20 µg/L. However, in both cases only one exposure concentration was used.

B.7.1.2 Anthracene

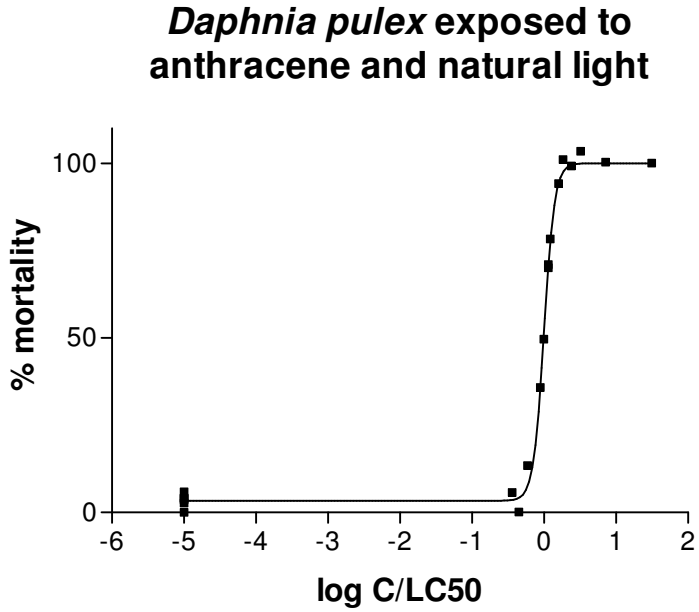
B.7.1.2.1 Fresh water environment

Anthracene is very phototoxic and toxic effects (LC_{50}s) 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 and Borovsky *et al.*, 1987). The UV-intensity of sunlight on a clear day was measured to be 4245 µW/cm^2 (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/cm^2.

Ultraviolet radiation in the most sensitive chronic toxicity studies was less harsh. 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/cm^2 with a ratio of UV-A:UV-B of 8:1. This resulted in NOECs or EC_{10}s for reproduction of 1.5-2.0 µg/L. 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. Algae were exposed to anthracene in static renewal set-up for 34-36 hours with 765 µW/cm^2 UV-A during the last 22-24 hours. UV-B was filtered out. The NOECs and EC_{10}s for growth rate and primary production ranged from 1.4 to 1.5 µg/L. In these three chronic studies experimental concentrations were measured.

In the study by Allred & Giesy (1985), adult *Daphnia pulex* were exposed to anthracene in the dark for 24 hours. Then they were exposed to full sunlight for half an hour. A dose-response relationship can not be easily determined, because only one exposure concentration does not result in 100% effects (see Figure B.7.1). The LC_{50} 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 LC_{50} is estimated and exposure concentrations are expressed as a ration of this LC_{50} for each light intensity, then a clear dose response relationship can be derived. For exposure to full sunlight, the LC_{50} is estimated to be 1.0 µg/L.

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/m^2 of the photosynthetically active radiation range. Cultures were aerated (0.1 or 2% CO_2) 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/m^2 was independent of the CO_2 level, whereas the growth inhibition at 33 and 12 W/m^2 was much greater in cultures aerated with 2% than with 0.1% CO_2.



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

Figure B.7.1. Dose response curve for *Daphnia pulex* exposed successively to anthracene and full sunlight.

B.7.1.2.2 Marine environment

For the marine environment EC_{10}s are available for the marine bacterium *Vibrio fisheri* for growth during 18 hours of exposure (El-Alawi *et al.*, 2001). The lowest EC_{10} is 23 µg/L. Acute toxicity data are available for bacteria, crustaceans and molluscs. The lowest value is for brine shrimp *Artesia salina*, exposed to anthracene for 10 hours of with irradiation by sunlight for the last eight hours (Peachy & Crosby, 1996). Similar to fresh water, this is the lowest effect concentration available. From the figures, the EC_{10} is estimated to be 1.7 µg/L. This is slightly higher than the EC_{50} for *Daphnia pulex*. For the brine shrimp, however, the exposure to sunlight is much longer, 8 hours instead of half an hour.

B.7.1.2.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

The lowest available chronic toxicity value is a NOEC between 1.5-2.0 µg/L for *Daphnia magna*, which would result in a PNEC between 0.1-0.2 µg/L. The most critical effect is however observed in acute study when the organisms are exposed to UV light. The observed EC_{50} value for *Daphnia pulex* was 1 µg/L (Allred & Giesy, 1985). There are two other studies that report values < 1 µg/L: a study with the midge *Chironomus riparius* by Bleeker *et al.* (2003) but this is a water-sediment exposure system and pore water concentrations are higher and another study with *Aedus aegypti* for which also EC_{50} values are reported much higher than 1 µg/L which were obtained under similar test conditions. The EC_{50} value observed for *D. magna* is therefore considered as the most reliable limit value for phototoxicity. Because of the steepness of the dose-response relationship (*i.e.* a low EC_{10}:EC_{50} ratio), it is considered appropriate to apply the same assessment factor of 10 to derive a PNEC for anthracene in fresh water of 0.1 µg/L.

Marine environment

The PNEC for fresh water species is also suitable for marine aquatic species, because marine crustaceans seem to be subject to phototoxicity as well as fresh water species. From the limited data the sensitivity of marine species is comparable with that of fresh water species. The lowest NOEC for marine bacteria with an assessment factor of 100 would lead to a value of 0.23 µg/L, which is

more than two times higher than the PNEC of 0.1 µg/L derived for fresh water. For this reason for the marine environment the same PNEC will be used. In the most recent draft EU RAR for anthracene, the same PNECs are derived.

B.7.1.3 Phenanthrene

B.7.1.3.1 Fresh water environment

For phenanthrene acute as well as chronic toxicity data are available for fresh water algae, crustaceans including daphnids, fish, and insects. In addition, chronic toxicity data are also available for protozoans, cyanobacteria, aquatic plants, and insects. For protozoans no effect concentration could be established (Rogerson *et al.*, 1983). For insects only 50% effect concentrations 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 EC_{10}s are for growth rate of the algae *Pseudokirchneriella subcapitata* (Halling-Sørensen *et al.*, 1996) and mortality in an ELS test with the largemouth bass *Micropterus salmoides* (Black *et al.*, 1983). For *Pseudokirchneriella subcapitata* the EC_{10} of 10 µg/L 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 EC_{10} for growth rate of *Pseudokirchneriella subcapitata* was also higher (23 µg/L: Bisson *et al.*, 2000). The EC_{10} for *Micropterus salmoides* is derived from one effect concentration and the EC_{50} 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).

Apart from these two studies the lowest EC_{10} is for reproduction of *Ceriodaphnia dubia* in a 7-d toxicity test. The value of this EC_{10} is 13 µg/L and is based on measured concentrations. All reported concentrations are measured concentrations.

B.7.1.3.2 Marine environment

Acute toxicity data are available for marine bacteria, annelids, crustaceans, molluscs, and fish. For bacteria, annelids, crustaceans and fish also chronic toxicity data are available. 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 LC_{50} for emergent juveniles of 51 µg/L was reported. This test was used to determine the sublethal concentration mentioned above (Emery & Dillon, 1996). All other effect concentrations for marine species are higher than this value of 51 µg/L. All concentrations are based on nominal values. So, at 20 µg/L few or no mortality occurred.

B.7.1.3.3 Calculation of Predicted No Effect Concentration (PNEC)

<u>Fresh water environment</u>

Because acute and chronic toxicity data are available for algae, *Daphnia*, and fish, an assessment factor can be applied to the lowest NOEC or EC_{10}. This is the 7-d EC_{10} for *Ceriodaphnia dubia* of 13 µg/L. The PNEC is 1.3 µg/L.

<u>Marine environment</u>

At 20 µg/L few or no mortality occurred in the test with the marine annelid. Because the set-up of the chronic test was limited, the results of this part must be treated with care. Other effects for marine species were all observed at higher concentrations than the LC_{50} of 51 µg/L for the annelid.

Therefore, the PNEC for fresh water species of 1.3 µg/L derived from a NOEC of 13 µg/L is also suitable for marine species.

B.7.1.4 Fluoranthene

B.7.1.4.1 Fresh water environment

Acute toxicity data for fluoranthene are available for nine taxonomic groups of fresh water species, amongst which are algae, crustaceans, and fish. In addition to these taxonomic groups, chronic studies are available for annelids, cyanobacteria, aquatic plants, insects, and amphibians.

The EC_{10} 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 EC_{10} for the algae *Pseudokirchneriella subcapitata* is 8.6 µg/L (Bisson *et al.*, 2000). For the annelid *Stylaria lacustris* the reported data are from a study with sediment exposure (Suedel & Rodgers, 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 EC_{10} are in between 1.0 and 1.5 µg/L. Bisson *et al.* (2000) found an EC_{10} 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. Wilcoxen *et al.* (2003) reported a 10-d LC_{10} 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/cm^2 UV-B, 102.08 µW/cm^2 UV-A, and 289.24 µW/cm^2 visible. The LC_{10} decreased strongly with UV-intensity. Under gold light (intensity of 0.17 µW/cm^2 UV-B, 0.09 µW/cm^2 UV-A, 167.72 µW/cm^2 visible) and fluorescent light (intensity of 1.32 µW/cm^2 UV-B, 13.65 µW/cm^2 UV-A, 424.69 µW/cm^2 visible) the LC_{10}s were 56 and 8.0 µg/L, respectively. However, these values are comparable with the reported EC_{50}s.

When exposed under laboratory ultraviolet light with 283 µW/cm^2 UV-A and 47 µW/cm^2 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/cm^2 UV-A, 7.5 µW/cm^2 UV-B, and 289 µW/cm^2 visible light and a photoperiod of 16:8 h light:dark, a 10-d LC_{10} for *Hyalella azteca* was found of 1.1 µg/L (Wilcoxen *et al.*, 2003). Under laboratory ultraviolet light with an intensity of 612 µW/cm^2 UV-A and 82 µW/cm^2 UV-B and a photoperiod of 12:12 h light:dark the NOEC for growth of *Pimephales promelas* exposed for 32 days in an ELS test was 1.4 µg/L (Spehar *et al.*, 1999). In all these experiments concentrations were experimentally determined. For the fresh water mollusc *Utterbackia imbecilis* the 24-h LC_{50} was 2.45 µg/L with UV-A radiation (320-400 nm) at an intensity of 70 µW/cm^2 (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 LC_{50}s of fluoranthene for fresh water species exposed under laboratory lighting with UV are comparable or even lower than the chronic NOEC. The 96-h LC_{50}s 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/cm^2 UV-A and 63-80 µW/cm^2 UV-B and a photoperiod of 12:12 h light dark. The 48-h LC_{50} for *Daphnia magna* was 1.6 µg/L, with ultraviolet light with 783-850 µW/cm^2 UV-A and 104 µW/cm^2 UV-B and a photoperiod of 12:12 h light dark (Spehar *et al.*, 1999).

The study with embryos of four species of amphibians exposed to fluoranthene shows that under laboratory lighting with a limited intensity of radiation (visible light:UV-A:UV-B=100:10:1; UV-A intensity 62-68 µW/cm^2 and UV-B intensity 2-5 µW/cm^2) no significant effects occurred at concentrations of 25 µg/L or below (Hatch & Burton, 1998). Even concentrations up to 25 µg/L in

combination with exposure in full sunlight with 200-1650 µW/cm^2 UV-A and 45-320 µW/cm^2 UV-B had no effect on the hatching of the frog *Rana pipiens*. However, just as for other organisms mortality appeared to be severe. At 5, 25, and 125 µg/L all larvae died, while the controls were unaffected. The intensity of the sunlight was 200-1650 µW/cm^2 UV-A and 45-320 µW/cm^2 UV-B. The test was performed early in April at 18-22 °C. An LT_{50} experiment was also performed outdoors with newly hatched larvae of the Spotted Salamander *Ambystoma maculatum* and the African Clawed Frog *Xenopus laevis*. Median lethal times at 1 µg/L were 14-15 hours. However, also controls exposed to full sunlight showed LT_{50}s of 16-19 hours. Temperature was rather high for the organisms in this experiment (23-28 °C).

B.7.1.4.2 Marine environment

Acute toxicity data are available for six taxonomic groups of marine species (*i.e.* bacteria, annelida, crustaceans, mollusca, echinoderma, and fish). Chronic toxicity data are available for two groups of species, which are bacteria and crustaceans. However, the tests with *Vibrio fisheri* (El-Alawi *et al.*, 2002) have reported effect concentrations, which are all above the aqueous solubility of 200 µg/L. The 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/cm^2 UV-A and 68-109 µW/cm^2 UV-B), the NOEC dropped to 0.6 µg/L.

Under the same UV-conditions conditions, also some LC_{50} values were found. The 48-h LC_{50} for the marine mollusc *Mulinia lateralis* was 2.8 µg/L, the 96-h LC_{50} for *Mysidopsis bahia* was 1.4 µg/L, the 48-h LC_{50} for the urchin *Arbacia punctulata* was 3.9 µg/L and the 96-h LC_{50} for *Pleuronectes americanus* was 0.1 µg/L (Spehar *et al.*, 1999). It appeared that when a comparison was possible with exposure in sunlight, *i.e.* in the case of *Mysidopsis bahia*, *Paleomonetes* species, the Atlantic Lobster *Homarus americanus*, the Purple-spined Sea Urchin *Arbacia punctulata*, the Sheepshead Minnow *Cyprinodon variegatus*, and the fish *Menidia beryllina*, the artificial UV-radiation leads to effects that are very similar to those of sunlight with a midday intensity of 1273-2660 µW/cm^2 UV-A and 76-182 µW/cm^2 UV-B.

B.7.1.4.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water and marine environment

Chronic NOECs are available for algae, *Daphnia*, and fish. The lowest chronic effect concentration for fresh water species of 1.1 µg/L is for *Hyalella azteca* (10-d LC_{10}). This value is similar to the 7 days EC_{10} for *Ceriodaphnia dubia*. At similar concentrations also acute toxic effects were found for fresh water species. The lowest chronic NOEC for marine species is 0.6 µg/L for reproduction of *Mysidopsis bahia* in 31-d study. *Mysidopsis bahia* thus seems to be more sensitive than *Hyalella azteca*. However, the endpoint for *Mysidopsis bahia* reproduction instead of mortality, the exposure duration is 31 days instead of 10 days and, probably most important, the UV-intensity is 5 to 8 times as high. Therefore, when exposed to sunlight both crustaceans might be equally sensitive, or the fresh water species might even be more sensitive. The lowest effect concentration, however, is for the marine arctic fish *Pleuronectus americanus*. The 96-h LC_{50} is 0.1 µg/L. Unlike the phototoxicity studies for anthracene the organisms were not exposed to sunlight for a relatively short time, but for the whole test duration with a photoperiod of 16 hours light and 8 hours dark. Further, for anthracene, the lowest LC_{50} differed by less than a factor of two from the lowest NOEC. Here the lowest LC_{50} is six times the lower than the lowest chronic NOEC , while the other LC_{50}s are higher than this value. However, the larvae of this fish species do live in shallow water, where the attenuation of light is limited. The used light intensity in the study was similar to sunlight. Therefore, the acute phototoxicity of fluoranthene is considered important and should be

taken into account in the PNEC. For these reasons, an assessment factor of 10 is considered sufficient and is applied to the 96-h LC_{50} value for *Pleuronectes americanus* of 0.1 µg/L. The general PNEC for the aquatic environment then becomes 0.01 µg/L.

B.7.1.5 Pyrene

B.7.1.5.1 Fresh water environment

Acute toxicity data for fresh water species tested with pyrene are available for algae, molluscs, crustaceans, insects, fish, and amphibians. It should be noted that the toxicity studies with fish and amphibians have an exposure time of only one day (Kagan *et al.*, 1985, 1987). *Pimephales promelas* were irradiated with UV-radiation at 750 µW/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 µg/L. In the study with embryos of *Rana pipiens*, the embryos were exposed to pyrene in the dark and after 1 hour transferred to sunlight for half an hour and then back to darkness. Mortality was recorded the next day and the LC_{50} was 140 µg/L (Kagan *et al.*, 1987). Although the exposure time of the tests is limited, the sensitivity of fish and amphibians for exposure to pyrene seems limited when compared with *Daphnia magna* and the mollusc *Utterbackia imbecilis*. The lowest value 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/cm^2 (295-365 nm; peak 340 nm) for 2 hours and 1 hour of recovery in the test medium. The EC_{50} for immobility was 1.38 µg/L (Wernersson, 2003). In a similar treatment (2 hours of recovery instead of 1, the EC_{50} 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/cm^2 (320-400 nm; peak 350 nm), the LC_{50} was 4 µg/L (Kagan *et al.*, 1985, 1987). When exposed to UV-B radiation only (intensity 64 µW/cm^2) for four times two hours during 48 hours, the EC_{50} for immobility of neonates ranges from 2.7 to 20 µg/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 µg/L with UV-A radiation (320-400 nm) at an intensity of 70 µW/cm^2 (Weinstein & Polk, 2001). The reported concentrations were not analytically verified.

Chronic toxicity data are available for algae, cyanophyta, crustaceans, and aquatic plants. The lowest chronic values are the 72-h EC_{10} of 1.2 µg/L for growth of *Pseudokirchneriella subcapitata* and the 7-d EC_{10} of 2.1 µg/L for reproduction of *Ceriodaphnia dubia* (Bisson *et al.*, 2000). For the macrophyte *Lemna gibba* (Ren *et al.*, 1994; Huang *et al.*, 1995, 1997ab) 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.* (1997ab) were analytically verified.

B.7.1.5.2 Marine environment

Acute toxicity data for marine species are available for annelids, bacteria, crustaceans, molluscs and coelenterata. The lowest effect concentrations are similar to fresh water species in the presence of UV-radiation for embryos/larvae of molluscs and neonates/nauplii of crustaceans. 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 µW/cm^2, the LC_{50} for nauplii of *Artemia salina* was 8 µg/L (Kagan *et al.*, 1985, 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 µW/cm^2, the LC_{50} for nauplii of *Artemia salina* was estimated from the presented figure to be 36 µg/L (Peachy & Crosby, 1996). The same treatment with sunlight (λ>290 nm) at an intensity of 407-1429 µW/cm^2 resulted in an EC_{50} of 3.4 µg/L (Peachy & 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 µW/cm^2 UV-A (365±36 nm) and

134±22.8 µW/cm^2 UV-B (310±34 nm) with a photoperiod of 16:8 hour light:dark the LC_{50} was 0.89 µg/L. Under the same conditions, the LC_{50} for embryos/larvae of *Mulinea lateralis* was 0.23 µg/L, while the LC_{50} for juveniles of 1 to 1.5 mm of the same species was 1.68 µg/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 µW/cm^2 UV-A and 6.3±0.1 µW/cm^2 UV-B with a photoperiod of 12:12 hour light:dark. The NOEC was 0.5 µg/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). Therefore, in the risk assessment this study can be considered as a chronic study. Except from the study by Pelletier *et al.* (1997) concentrations were not analytically verified.

B.7.1.5.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water and marine environment

For pyrene the base set is complete, although the study with *Pimephales promelas* is limited. However, fish appear to be less sensitive than crustaceans. Chronic toxicity data are available only for algae, crustaceans, cyanophyta and macrophyta. Hence, an assessment factor of 50 should be applied to the lowest NOEC or EC_{10}. For fresh water species, this is the EC_{10} of 1.2 µg/L for *Pseudokirchneriella subcapitata*, a value which is also below the lowest acute LC_{50} for fresh water species. The marine and fresh water acute toxicity data for molluscs and crustaceans do not differ significantly. The lowest NOEC for marine species is 0.5 µg/L for shell abnormalities of embryos/larvae of the Japanese oyster *Crassostrea gigas*. With algae and crustaceans for fresh water species and molluscs as additional group for marine species an assessment factor of 50 can be applied to the lowest NOEC for the derivation of the PNEC for marine water as well. However, the most sensitive endpoint was the LC_{50} of 0.23 µg/L for the clam *Mulinea lateralis* under UV enhanced conditions. Similar to anthracene and fluoranthene this endpoint is used to base the PNEC upon. The PNEC for fresh and marine water then becomes 0.0046 µg/L.

B.7.1.6 Fluorene

B.7.1.6.1 Fresh water environment

Acute and chronic toxicity data of 9*H*-fluorene for fresh water species are available for algae, *Daphnia*, and fish. In addition acute toxicity data are also available for bacteria, insects, and molluscs, and chronic toxicity data for cyanophyta, aquatic plants, and insects. The lowest value is an EC_{10} 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 EC_{10} for *Ceriodaphnia dubia* is similar to the EC_{10} presented in a figure for the immobility of *Daphnia pulex* from an acute 48-h study (Smith *et al.*, 1988).

B.7.1.6.2 Marine environment

Toxicity data of 9*H*-fluorene with marine species are available for bacteria, annelids, and crustaceans. The lowest value is an EC_{50} for bioluminescence of *Vibrio fisheri* of 500 µg/L (Johnson & Long, 1998).

B.7.1.6.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

Because chronic data are available for algae, *Daphnia*, and fish an assessment factor of 10 can be applied to the lowest NOEC or EC_{10}. This is the EC_{10} for *Ceriodaphnia dubia*. The resulting PNEC is 2.5 µg/L.

Marine environment

Consequently, the PNEC for marine water is 0.25 µg/L.

B.7.1.7 Acenaphthylene

B.7.1.7.1 Fresh water environment

Acute toxicity data for acenaphthylene are available only for the fresh water crustacean *Daphnia magna*, the freshwater fish *Oryzias latipes* and for the saltwater bacterium *Vibrio fisheri*. Strictly, the base-set is not complete, because an acute toxicity study with algae is missing. However, a 72-h static study with *Pseudokirchneriella subcapitata* was performed but only the EC_{10} value is reported. The EC_{50} must therefore be higher than this value. Therefore, the base-set is considered to be complete. In the acute toxicity study 48-h with *Daphnia magna*, concentrations were measured and the EC_{50} for immobility was 1800 µg/L (Bisson *et al*, 2000). In a 96-h acute toxicity study with the Japanese Medaka (*Oryzias latipes*), the LC_{50} was 6400 µg/L (Yoshioka & Ose, 1993). The short-term bioluminescence test with *Vibrio fischeri* leads to lower values. Leaving the tests with complex medium (El-Alawi *et al.*, 2001) out of consideration, the 5-min EC_{50} of 340 µg/L (Johnson & Long, 1998) and the 15-min EC_{50}s of 330 and 340 µg/L (El-Alawi *et al.*, 2001) are very consistent. Illumination with simulated solar radiation had no effect on the EC_{50} (340 µg/L versus 330 µg/L in the dark) (El-Alawi *et al.*, 2001). Concentrations in these tests were not verified. In both cases dimethylsulfoxide was used as solvent.

Two long term toxicity studies with acenaphthylene are available. In a 72-h static study with *Pseudokirchneriella subcapitata* the EC_{10} was 82 µg/L. In a 7-d renewal reproduction study with *Ceriodaphnia dubia* the EC_{10} was 64 µg/L. In both studies, concentrations were experimentally determined (Bisson *et al*, 2000). For the saltwater bacterium *Vibrio fischeri* also some long-term experiments were performed (El-Alawi *et al.*, 2001). Growth and bioluminescence were examined after 18 hours of exposure. It appeared that the chronic EC_{50}s were slightly higher than the short-term EC_{50}s. From the study it appears that bioluminescence is almost 1:1 correlated with growth of the bacteria.

B.7.1.7.2 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

Two chronic NOECs for two trophic levels are available. These are the EC_{10} for growth of the algae *Pseudokirchneriella subcapitata* and for reproduction of the crustacean *Ceriodaphnia dubia* (Bisson *et al.*, 2000). It can be questioned if algae would be the trophic level showing the lowest LC_{50}/EC_{50}, because the bacterium species *Vibrio fischeri* has an EC_{50} of 340 µg/L, which is only a factor of 4 higher than the EC_{10} for *Pseudokirchneriella subcapitata*. However, chronic data for species of bacteria are not considered in the derivation of the PNEC water in the case assessment factor are used. Therefore, an assessment factor of 50 seems to be justified and can be applied to the lowest EC_{10} of 64 µg/L for *Ceriodaphnia dubia*. The final PNEC then becomes 1.3 µg/L for fresh water.

Marine environment

With no additional NOECs for marine species, an assessment factor of 500 is applied for the marine environment. The final PNEC for marine water thus becomes 0.13 µg/L.

B.7.1.8 Acenaphthene

B.7.1.8.1 Fresh water environment

Test with fresh water species and acenaphthene have been performed with molluscs, crustaceans (*Daphnia*) and fish. The lowest EC_{50}s 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 LC_{50}s are 580 and 670 µg/L, respectively.

No EC_{50} for algae is reported. However, a good toxicity study with *Pseudokirchneriella subcapitata* is available (Bisson *et al.*, 2000) for which only the EC_{10} of 38 µg/L is reported. The EC_{50} must therefore be higher than this value.

Besides algae, chronic toxicity data are available for cyanophyta, crustaceans and fish. The 7-d EC_{10} for reproduction of *Ceriodaphnia dubia* is 42 µg/L (Bisson *et al.*, 2000).

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.

B.7.1.8.2 Marine environment

Acute toxicity data for marine species are available for bacteria, molluscs and fish and for bacteria and fish also chronic studies. Growth and bioluminescence of the bacterium *Vibrio fischeri* were examined after 18 hours of exposure (El-Alawi *et al.*, 2001). It appeared that the chronic EC_{50}s were slightly higher than the short-term EC_{50}s. Leaving the tests with complex medium (El-Alawi *et al.*, 2001) out of consideration, the 5-min EC_{50} of 750 µg/L (Johnson & Long, 1998) and the 15-min EC_{50}s of 810 and 830 µg/L (El-Alawi *et al.*, 2001) are very consistent. Illumination with simulated solar radiation had no effect on the EC_{50} (830 µg/L versus 810 µg/L in the dark) (El-Alawi *et al.*, 2001). Concentrations in these tests were not verified. In both cases dimethylsulfoxide was used as solvent.

In a flow-through ELS study with the marine fish *Cyprinodon variegatus*, the EC_{10} 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).

B.7.1.8.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

Chronic toxicity data are available for algae, crustaceans (*Ceriodaphnia*) and fish. Hence, an assessment factor of 10 can be applied to the lowest NOEC or EC_{10}. This is the EC_{10} for growth of *Pseuodokirchneriella subcapitata* of 38 µg/L. The PNEC then becomes 3.8 µg/L.

Marine environment

No additional chronic toxicity data for typically marine species are available. Therefore, an assessment factor of 100 will be applied to the lowest NOEC or EC_{10} to derive the PNEC for marine water. This PNEC thus is 0.38 µg/L.

B.7.1.9 Chrysene

B.7.1.9.1 Fresh water environment

The aqueous solubility, determined by generator column techniques, is about 1.6 µg/L, with a range between 1.0 and 3.3 µg/L (Mackay *et al.*, 2000). Around or below this value, no significant effects were observed for any species in a regular toxicity experiment, although chronic toxicity data were performed with algae, crustaceans (including *Daphnia*) and fish. 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 µg/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 µW/cm^2 UV-B (310 ± 36 nm), 120 ±

5 µW/cm^2 UV-A (365 ± 36 nm), and 680 ± 10 µW/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 µg/L.

B.7.1.9.2 Marine environment

For marine species acute toxicity studies were performed with bacteria, annelids and crustaceans. No significant effects at or below the aqueous solubility were observed in any of these toxicity studies.

B.7.1.9.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

No acute toxicity data for algae and fish are available. However, for algae the EC_{10} and thus the EC_{50} for growth of *Pseudokirchneriella subcapitata* (Bisson *et al.*, 2000) is higher than 1 µg/L. Due to the limited solubility of chrysene, no acute effects are expected for fish either. Besides that, an ELS study with the zebra fish *Brachydanio rerio* is available (Hooftman & Evers-De Ruiter, 1992). Chronic studies were performed with algae, daphnids (both *Ceriodaphnia dubia* and *Daphnia magna*) and fish. Therefore an assessment factor of 10 to the lowest NOEC or EC_{10} can be applied. However, no effects were observed at all, although in the test with *Ceriodaphnia dubia* the highest tested measured concentration was 0.09 µg/L. The only study with a significant effect below the aqueous solubility was 50% mortality after 48 hours at 0.7 µg/L (Newsted & Giesy, 1987). In this study, toxicity of chrysene was enhanced by irradiation with UV-light, although the intensity was considerably less than natural sunlight. Sunlight or UV-light comparable with sunlight was used in the case of the lowest effect concentrations for anthracene and fluoranthene. Similar to these compounds an assessment factor of 10 is applied to the lowest effect concentration. The resulting PNEC for fresh water is thus 0.07 µg/L.

Marine environment

No additional chronic toxicity data for typically marine species are available. Therefore, an assessment factor of 100 will be applied to the lowest NOEC or EC_{10} to derive the PNEC for marine water. This PNEC thus is 0.007 µg/L.

B.7.1.10 Benz(a)anthracene

B.7.1.10.1 Fresh water environment

For benz(a)anthracene acute toxicity data are available for algae, crustaceans and amphibians. The growth of *Pseudokirchneriella subcapitata*, exposed to 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 96-h LC_{50} of *Daphnia pulex* exposed under a 12:12 h photoperiod to mixed fluorescent and natural light was 10 µg/L (Trucco *et al.*, 1983). The 48-h LC_{50} of *Daphnia magna* from a test in the dark was higher than 9.1 µg/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 µW/cm^2 for 2 hours and 1 hour of recovery in the test medium lead to an LC_{50} of 3.4 µg/L. UV-radiation thus increases the toxicity of benz(a)anthracene.

Larvae of the amphibian *Pleurodeles waltl* irradiated throughout the experiment with UV-A light (320-400 nm with a maximum at 365 nm) at 250 µW/cm^2 survived at 3.1 µg/L and died all at 6.3 µg/L (Fernandez & L'Haridon, 1992).

Of the mentioned studies, only in the study with *Daphnia magna* from Bisson *et al.* (2000) concentrations were determined.

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 µg/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 µW/cm^2 UV-B (290-336 nm), 95 µW/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 µg/L.

Chronic toxicity data are available for algae, cyanophyta, aquatic plants and crustaceans. The 72-h EC_{10} for inhibition of growth of *Pseudokirchneriella subcapitata* from a study with measured concentrations 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 EC_{10} 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/cm^2, although the light intensities are given at single wavelengths (Cody *et al.*, 1984). The light intensity may play an important role in the lower EC_{10} 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, 1992). Therefore, no clear dose-response relationship was observed. The light regime was continuous light at 951-1903 µW/cm^2 (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.

For *Ceriodaphnia dubia* no effects were observed in a 7-d study at concentrations up to 8.7 µg/L (Bisson *et al.*, 2000). In the study with the duckweed *Lemna gibba* only concentrations far above the aqueous solubility are reported (Huang *et al.*, 1997ab). The same applies to the acute toxicity studies with the marine bacterium *Vibrio fischeri* (El-Alawi *et al.*, 2002; Johnson & Long, 1998).

B.7.1.10.2 Marine environment

Beside toxicity on marine bacterium *Vibrio fischeri* no additional data on marine species are available.

B.7.1.10.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

The determination of the lethal time for *Pimephales promelas* can be considered as an acute fish study and hence the base-set can be considered as complete. Chronic toxicity data are available for algae and crustaceans but not for fish. Because 50% of *Pimephales promelas* died in 89 hours, fish might be the most sensitive species of the base-set. Therefore, an assessment factor of 100 should be applied to derive the PNEC for fresh water. The lowest NOEC or EC_{10} is the EC_{10} of 1.2 µg/L for growth of *Pseudokirchneriella subcapitata*. The PNEC for fresh water is 0.012 µg/L.

Marine environment

No chronic toxicity data are available for marine species. The assessment factor for marine water is 1000 in this case. Therefore, the PNEC for marine water is 0.0012 µg/L

B.7.1.11 Benzo(b)fluoranthene

B.7.1.11.1 Fresh water environment

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 ± 20 µW/cm^2 and a recovery period of 2 hours, the EC_{50} 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).

B.7.1.11.2 Marine environment

Beside toxicity on marine bacterium *Vibrio fischeri* no additional data on marine species are available.

B.7.1.11.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water and marine environment

The only value that can be used to derive the PNEC is the LC_{50} for *Daphnia magna*, which is above the aqueous solubility. For this reason, it is proposed to use the same PNEC for benzo(b)fluoranthene as for benzo(k)fluoranthene (see Section B.7.1.13)

B.7.1.12 Benzo(ghi)perylene

B.7.1.12.1 Fresh water environment

Acute toxicity studies for fresh water species are available for *Daphnia magna* (Bisson *et al.*, 2000) and *Pimephales promelas* (Oris & Giesy, 1987). The 48-h LC_{50} for *Daphnia magna* 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). In the study with *Pimephales promelas* 7-d old larvae were exposed to a measured 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/cm^2 UV-B (290-336 nm), 95 µW/cm^2 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. For algae no EC_{50} is presented. However, in the 72-h study with *Pseudokirchneriella subcapitata* the EC_{10} for growth is larger than 0.16 µg/L (Bisson *et al.*, 2000) and hence the EC_{50} must also be higher than this value.

In addition to algae, chronic toxicity data are available for crustaceans, fish and aquatic plants. The EC_{10} 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. This concentration was experimentally verified. In an ELS study with *Brachydanio rerio* no effects were observed up to concentrations of 0.16 µg/L (Hooftman & Evers-de Ruiter, 1992). For *Lemna gibba* the reported effect concentrations are far above the aqueous solubility (Huang *et al.*, 1997ab). This also applies to the study with the marine bacterium *Vibrio fischeri* (El-Alawi *et al.*, 2002).

B.7.1.12.2 Marine environment

Beside the toxicity on marine bacterium *Vibrio fischeri* no additional data on marine species are available.

B.7.1.12.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

The base-set for benzo(ghi)perylene is complete. Chronic toxicity data are available algae, crustaceans and fish. The lowest EC_{10} or NOEC is the EC_{10} of 0.082 µg/L for reproduction of *Ceriodaphnia dubia*. An assessment factor of 10 can applied in this case. The resulting PNEC for fresh water is 0.0082 µg/L.

Marine environment

With acute and chronic toxicity data for algae, crustaceans and fish an assessment factor of 100 can be applied for marine water. The PNEC for marine water is 0.00082 µg/L.

B.7.1.13 Benzo(k)fluoranthene

B.7.1.13.1 Fresh water environment

Acute toxicity data for benzo(k)fluoranthene are only available for *Daphnia magna*. However, in the two available studies (Bisson *et al.*, 2000; Verrhiest *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. For algae no EC_{50} is presented. However, in the 72-h study with *Pseudokirchneriella subcapitata* the EC_{10} for growth is larger than 1 µg/L (Bisson *et al.*, 2000) and hence the EC_{50} must also be higher than this value. In the 7-d reproduction study with *Ceriodaphnia dubia* no effects were observed either (Bisson *et al.*, 2000). 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 LC_{50} estimated from the presented data with a log-logistic relationship was 0.65 µg/L. From the data for weight and length EC_{10} 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.

B.7.1.13.2 Marine environment

No additional data on marine species are available.

B.7.1.13.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

Although the base-set is not complete, because acute toxicity data for fish are missing, an assessment factor of 10 is considered suitable, because chronic toxicity data are available for algae, crustaceans, and fish. The most sensitive endpoint is length of *Brachydanio rerio* in an ELS test. The EC_{10} for this endpoint is 0.17 µg/L. With an assessment factor of 10, the PNEC for fresh water is 0.017 µg/L.

Marine environment

With acute and chronic toxicity data for algae, crustaceans and fish an assessment factor of 100 can be applied for marine water. The PNEC for marine water is 0.0017 µg/L

B.7.1.14 Benzo(a)pyrene

B.7.1.14.1 Fresh water environment

Acute toxicity data for benzo(a)pyrene with fresh water species are available for algae, cyanophyta, bacteria, crustaceans including *Daphnia*, insects and amphibians. The solubility of benzo(a)pyrene

is 1.2-1.8 µg/L (Mackay *et al.*, 2000). Only acute toxicity data with exposure to UV-light result in effects at concentrations near the aqueous solubility. The lowest acute value is the EC_{50} 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/cm^2 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 EC_{50} was 8.6 µg/L (Wernersson & Dave, 1997).

The lowest EC_{10} for algae *Pseudokirchneriella subcapitata* is 0.78 µg/L (Bisson *et al.*, 2000). This test was performed with a light intensity of 6000 to 8000 lux (~ 2000 µW/cm^2 with cool white fluorescent lamps). Concentrations were measured. For the same species, the EC_{10} can be estimated from the data presented by Cody *et al.* (1984). The EC_{10} 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/cm^2 in all cases. Therefore, the light intensity may play an important role in the lower EC_{10} from the study by Bisson *et al.* (2000).

The 7-d EC_{10} for reproduction of *Ceriodaphnia dubia* is 0.5 µg/L (Bisson *et al*, 2000). Also in this case concentrations were measured. The EC_{10}, 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.

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 µg/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 µg/L (Hannah *et al.*, 1982). Only at 2.4 µg/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 µg/L significantly more abnormalities were observed. However, at intermediate concentrations of 0.37 µg/L and 1.5 µg/L no significant effects were observed. Therefore, the NOEC for abnormalities is 1.5 µg/L. If the presented data are evaluated with a log-logistic relationship, an EC_{10} of 2.9 µg/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*.

B.7.1.14.2 Marine environment

Acute studies for marine species are limited. No significant effects were observed at concentrations near the aqueous solubility. Studies into chronic effects, although some of them have limited exposure times, are available for bacteria, molluscs, and fish. 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 EC_{10} from the presented data is 1.1 µg/L. When UV irradiation with an intensity of 456.2 ± 55 µW/cm^2 UV-A and 6.3 ± 0.1 µW/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 EC_{10} 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.

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 µg/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 µg/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 µg/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.

B.7.1.14.3 Calculation of Predicted No Effect Concentration (PNEC)

Although the base-set is not complete, because an acute study with fish is missing, enough data are available for chronic studies with fish to compensate for this acute study. Chronic studies for fresh water species are available for aquatic plants, algae, crustaceans, and fish. In addition, chronic studies for marine species are available for bacteria, echinoderms, molluscs, and fish. The EC_{10} of 0.22 µg/L for shell development of the marine mollusc *Crassostrea gigas* is used as endpoint for the PNEC. Because additional chronic toxicity data are available for two groups of typical marine species, the used assessment factor for both freshwater and marine water is 10. The PNEC for both fresh and marine water thus becomes 0.022 µg/L.

B.7.1.15 Dibenzo(a,h)anthracene

B.7.1.15.1 Fresh water environment

For algae no EC_{50} is presented. However, in the 72-h study with *Pseudokirchneriella subcapitata* the EC_{10} for growth is 0.14 µg/L (Bisson *et al.*, 2000) and hence the EC_{50} must be higher than this value.

The lowest acute value for immobility of *Daphnia magna* is the EC_{50} 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/cm^2 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 EC_{50} was 4.6 µg/L (Wernersson & Dave, 1997).

For fish, no standard acute toxicity data are available. 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 dibenzo(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/cm^2 UV-B (290-336 nm), 95 µW/cm^2 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.

Chronic toxicity studies with fresh water species are available for crustaceans, aquatic plants, and algae. For *Lemna gibba* no effects at concentrations near the aqueous solubility were observed (Huang *et al.*, 1997ab). No effect was observed at concentrations up to 0.032 µg/L in a 7-d study with *Ceriodaphnia dubia*. The 72-h EC_{10} for the growth rate of *Pseudokirchneriella subcapitata* was 0.14 µg/L (Bisson *et al.*, 2000). In both the test with *C. dubia* and *P. subcapitata* concentrations were measured.

B.7.1.15.2 Marine environment

In the studies with the marine bacterium *Vibrio fischeri* (El-Alawi *et al.*, 2002) and annelid *Neanthes arenaceodentata* (Rossi & Neff, 1978) no effects at concentrations near the aqueous solubility were observed.

B.7.1.15.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

The determination of the lethal time for *Pimephales promelas* can be considered as an acute fish study and hence the base-set can be considered as complete. Chronic toxicity data are available for algae and crustaceans but not for fish. Because the concentration that was tested with *Pimephales promelas* at which no mortality occurred is as low as the EC_{10} for algae, fish might be the most sensitive species of the base-set. Further, the highest tested concentration for *Ceriodaphnia dubia* was four times lower than the EC_{10} for *Pseudokirchneriella subcapitata*. Therefore, an assessment factor of 100 should be applied to derive the PNEC for fresh water. The lowest NOEC or EC_{10} is the EC_{10} of 0.14 µg/L for growth of *Pseudokirchneriella subcapitata*. The PNEC for fresh water is 0.0014 µg/L.

Marine environment

No suitable chronic toxicity data are available for marine species. The assessment factor for marine water is 1000 in this case. Therefore, the PNEC for marine water is 0.00014 µg/L

B.7.1.16 Indeno(123-cd)pyrene

B.7.1.16.1 Fresh water environment

For indeno(123-cd)pyrene, three aquatic toxicity studies are available. Indeno(123-cd)pyrene was not acutely toxic to *Daphnia magna* at concentrations up to 357 µg/L. The 72-h EC_{10} for the growth rate of *Pseudokirchneriella subcapitata* was 1.5 µg/L. The 7-d EC_{10} for reproduction of *Ceriodaphnia dubia* was 0.27 µg/L. In all three cases concentrations were measured (Bisson *et al.*, 2000).

B.7.1.16.2 Marine environment

No additional data on marine species are available.

B.7.1.16.3 Calculation of Predicted No Effect Concentration (PNEC)

Fresh water environment

The base-set is not complete for indeno(123-cd)pyrene. However, due to the hydrophobicity acute toxic effects are not expected to occur. With the EC_{10} for reproduction of *Ceriodaphnia dubia* and growth rate of *Pseudokirchneriella subcapitata* two sensitive endpoints are covered that are the lowest effect concentrations for six other PAHs. Therefore, an assessment factor of 100 is applied to the lowest EC_{10}, despite the fact that the base-set is not complete. The resulting PNEC for fresh water is 0.0027 µg/L.

Marine environment

No suitable chronic toxicity data are available for marine species. The assessment factor for the PNEC for marine water is 1000 in this case. The PNEC for marine water is 0.00027 µg/L.

B.7.1.17 Evaluation of the applied assessment factors

For the majority of the PAHs sufficient toxicity data are available in order to apply an assessment factor of 10 on the most sensitive endpoint found for that particular PAH. For pyrene, benz(a)anthracene, benzo(b)fluoranthene, dibenzo(a,h)anthracene and indeno(123-cd)pyrene less toxicity data are available and consequently higher assessment factors are applied following the general recommendation of the EU TGD (EC, 2003b). By read across with data available for the other PAHs, the possibility of applying lower assessment factors are investigated below.

B.7.1.17.1 Pyrene

For pyrene an assessment factor of 50 has been applied to the lowest endpoint. A factor of 10 was not used because data for chronic data for fish are missing. The EC_{50} for survival and development of *Mulinea lateralis* (Pelletier *et al*, 1997) is the lowest endpoint for pyrene. For anthracene and fluoranthene, this endpoint is also a very sensitive one. A comparison with fluoranthene is made, because this is also a four-ring PAH with a slightly higher log K_{OW} value, and with anthracene, which is a three-ring PAH with a somewhat lower log K_{OW} value.

In the most sensitive phototoxicity studies with *Utterbackia imbecilis* (Weinstein & Polk, 2001), *Daphnia magna* (Wernersson, 2003), the yellow fever mosquito *Aedes aegypti* (Borovsky *et al.*, 1987), and the shrimps *Artemis salina* (Kagan *et al.*, 1985;1987; Peachy & Crosby, 1997) and *Mysidopsis bahia* (Pelletier *et al.*, 1997) pyrene has EC_{50} values that are not smaller than 0.24 times the EC_{50} values of anthracene. The only exception to this is the lowest EC_{50} for pyrene for larvae/embryos of the clam *Mulinea lateralis*. This EC_{50} is 3.5% of that for anthracene. For juveniles this percentage is 2.4%. From a comparison with anthracene it may be concluded that the EC_{50} for *Mulinea lateralis* is a sensitive endpoint for pyrene to base the PNEC upon.

When compared with fluoranthene the EC_{50}s for the most sensitive phototoxicity tests with *Daphnia magna* (Wernersson & Dave, 1997; Wernersson, 2003), *Artemis salina* (Kagan *et al.*, 1985;1987), *Mysidopsis bahia* (Pelletier *et al.*, 1997), and *Mulinea lateralis* (Pelletier *et al.*, 1997), the EC_{50}s of pyrene are 16 to 28% of the EC_{50}s of fluoranthene. Only in another study with *Daphnia magna* (Kagan *et al.*, 1985;1987) the EC_{50}s for both compounds are equal. Thus, despite of the lower hydrophobicity of pyrene, the EC_{50} is lower in all phototoxicity tests by a relatively constant factor. For fluoranthene the PNEC is based on one EC_{50} for the marine fish species *Pleuronectus americanus*, which is more than a factor of 10 lower than the rest of the EC_{50}s. On general fish marine and fresh water fish species appear not to be especially sensitive for fluoranthene (Spehar *et al.*, 1999). Leaving this outlier out of consideration, it can again be concluded that the EC_{50} for *Mulinea lateralis* is a sensitive endpoint for pyrene to base the PNEC upon.

A further comparison can be made with the five-ring PAH benzo(a)pyrene. The PNEC for benzo(a)pyrene is based on the shell development of the marine mollusc *Crassostrea gigas*. This study was also performed with pyrene. Although this is one of the most sensitive endpoints for pyrene as well, the EC_{50} for *Mulinea lateralis* is a factor of 4 lower. Again, the EC_{50} for *Mulinea lateralis* appears to be a very sensitive endpoint.

An assessment factor of 10 has been applied for the derivation of the PNEC of anthracene, fluoranthene, and benzo(a)pyrene. For pyrene an assessment factor of 50 has been applied. In a direct comparison with the compounds mentioned above, it appears that the lowest endpoint for pyrene is sensitive enough to justify the use of an assessment factor of 10 for pyrene as well. Therefore, the final PNEC for pyrene becomes 0.023 µg/L for both fresh and marine water.

B.7.1.17.2 Benz(a)anthracene

For benz(a)anthracene an assessment factor of 100 has been applied because chronic NOECs are not available for fish and it can not be excluded that vertebrates are the most sensitive group of species. Moreover, from the studies with *Daphnia magna* (Wernersson, 2003) and the amphibian *Pleurodeles waltl* (Fernandez & L'Haridon, 1992) it is likely that benz(a)anthracene is rather acutely phototoxic. Therefore, the assessment factors for the PNECs for benz(a)anthracene remain 100 and 1000 for fresh water and marine water, respectively.

B.7.1.17.3 Benzo(b)fluoranthene

An assessment factor of 1000 to the lowest acute EC_{50} has been used for benzo(b)fluoranthene, because the base-set is not complete for this compound. Two chronic NOECs for two trophic levels are available. These are the EC_{10} for growth of the algae *Pseudokirchneriella subcapitata* and for reproduction of the crustacean *Ceriodaphnia dubia* (Bisson *et al.*, 2000). However, no EC_{10} could be established in these studies. On the other hand, it appeared that benzo(b)fluoranthene is phototoxic to *Daphnia magna* (Wernersson & Dave, 1997). In other cases where enough data are available but still phototoxicity appears to be the most sensitive endpoint an assessment factor of 10 has been applied to the lowest EC_{50} (*e.g.* for anthracene and fluoranthene). However, in several cases the EC_{50} for phototoxicity to *Daphnia magna* (Wernersson & Dave, 1997) appears to be more than a factor of 10 higher than the lowest endpoint for those compounds, *e.g.* for fluoranthene a factor of 350, for benzo(a)pyrene a factor of 39, for dibenzo(a,h)anthracene a factor of 33, and for pyrene a factor of 25. In this case, a comparison can better be made with its isomer benzo(k)fluoranthene. A toxicity test with *Daphnia* magna showed that this compound is also not very phototoxic, because no effects were observed up to the limit of solubility after irradiation with UV-A radiation (Verrhiest *et al.*, 2001). For benzo(k)fluoranthene toxicity tests are available for algae, daphnids and fish. The lowest was for fish, the trophic level for which no data are available for benzo(b)fluoranthene. Benzo(k)fluoranthene and benzo(b)fluoranthene are mostly reported

together. Therefore, it is proposed to use the same PNEC for benzo(b)fluoranthene as for benzo(k)fluoranthene. This PNEC is 0.017 µg/L for fresh water and 0.0017 µg/L for marine water.

B.7.1.17.4 Dibenzo(a,h)anthracene

No supporting information from other PAHs is available to lower the assessment factor for dibenzo(a,h)anthracene. Therefore, the assessment factors for the PNECs for dibenzo(a,h)anthracene remain 100 and 1000 for fresh water and marine water, respectively.

B.7.1.17.5 Indeno(123-cd)pyrene

No supporting information from other PAHs is available to lower the assessment factor for indeno(123-cd)pyrene. Therefore, the assessment factors for the PNECs for indeno(123-cd)pyrene remain 100 and 1000 for fresh water and marine water, respectively.

B.7.1.17.6 Comparison between Log K_{OW} and derived PNEC.

From the evaluation above it appears that for some PAHs the applied assessment factor is still higher than 10. In order to investigate whether these assessment factors are too conservative a comparison between the derived PNEC and the Log K_{OW} is made (see Figure B.7.2), suggesting that the mode of action is based on non-polar narcosis. A clear correlation between derived PNEC and Log K_{OW} was found. For those PAHs that exhibit phototoxicity the PNECs generally deviate the most from this relationship (shown in red triangles).

From this figure it is shown that the PNECs which are determined with an assessment factor > 10 do not deviate strongly from this relationship. Therefore, at present we do not see a reason to lower the assessment factors applied.

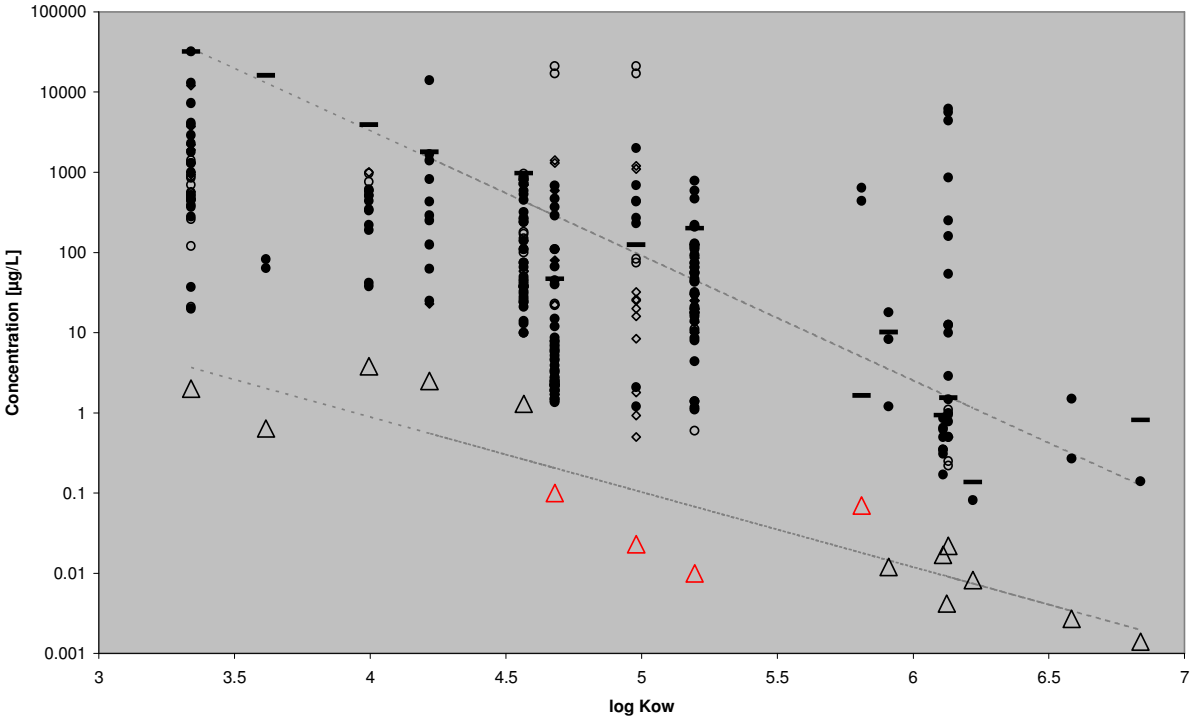


Figure B.7.2. Comparison between derived PNECs (open triangles) and log K_{OW}. Toxicity data are presented in small symbols.

B.7.1.18 PNEC summary for fresh and marine water

Table B.7.1 summarises the PNEC for the different PAHs together with the assessment factor and species used.

Table B.7.1 The PNEC for the various PAHs for fresh and marine water organisms.

Compound	PNEC fresh water (µg/L)	AF [a]	Species [b]	PNEC marine water (µg/L)	AF [a]
Naphthalene	2	10	*Oncorhynchus mykiss*	2	10
Anthracene	0.1	10	*Daphnia pulex acute*	0.1	10
Phenanthrene	1.3	10	*Ceriodaphnia dubia*	1.3	10
Fluoranthene	0.01	10	*Pleuronectes americanus acute*	0.01	10
Pyrene	0.023	10	*Mulinea lateralis acute*	0.023	10
9H-Fluorene	2.5	10	*Ceriodaphnia dubia*	0.25	100
Acenaphthylene	1.3	50	*Ceriodaphnia dubia*	0.13	500
Acenaphthene	3.8	10	*Pseudokirchneriella subcapitata*	0.38	100
Chrysene	0.07	10	*Daphnia magna acute*	0.007	100
Benz(a)anthracene	0.012	100	*Pseudokirchneriella subcapitata*	0.0012	1000
Benzo(b)fluoranthene	0.017	[c]	*Brachydanio rerio*	0.0017	100
Benzo(ghi)perylene	0.0082	10	*Ceriodaphnia dubia*	0.00082	100
Benzo(k)fluoranthene	0.017	10	*Brachydanio rerio*	0.0017	100
Benzo(a)pyrene	0.022	10	*Crassostrea gigas*	0.022	10
Dibenzo(a,h)anthracene	0.0014	100	*Pseudokirchneriella subcapitata*	0.00014	1000
Indeno(123cd)pyrene	0.0027	100	*Ceriodaphnia dubia*	0.00027	1000

[a] AF: assessment factor; [b] The same species is used for both marine and fresh water. [c] For benzo(b)fluoranthene the PNEC is the same as for benzo(k)fluoranthene after read-across with this compound.

B.7.1.19 Toxicity test results for sediment organisms

B.7.1.19.1 Equilibrium partitioning

For a number of PAHs, the PNEC has been calculated by equilibrium partitioning in the absence of sufficient toxicity data for benthic species, by using the following equation:

$$\mathrm{PNEC}_{\mathrm{sed}} = \frac{K_{\mathrm{susp-water}}}{\rho_{\mathrm{sed}}} \times \mathrm{PNEC}_{\mathrm{water}} \times 1000$$

where

$K_{\mathrm{susp\text{-}water}}$ = the suspended matter:water partitioning coefficient

ρ_{sed} = the bulk of wet sediment (1150 kg/m^3)

In Table B.7.2 the partitioning coefficient of each is listed.

B.7.1.19.2 Naphthalene

For sediment only effect concentration are available that relate to 50% effect. The EC_{50} for reburial of *Rhepoxynius abronius* after 10 days of exposure (Boese *et al.*, 1998) is 2900 mg/kg$_{dwt}$, recalculated to a sediment with an organic carbon content of 10%. Irradiation of the crustaceans with UV had no effect on this parameter. It should be noted that although this value is an EC_{50}, the endpoint (10-d reburial) is rather chronic than acute. In the EU-RAR for naphthalene (Munn *et al.*, 2003) usable studies for benthic species were not found.

Calculation of Predicted No Effect Concentration (PNEC)

The only effect concentrations are EC_{50}s and LC_{50}s. With an assessment factor of 1000, a PNEC of 2.9 mg/kg$_{dwt}$ is derived. Because this is the only value, this PNEC has to be compared with one derived by equilibrium partitioning, which was higher than 2.9 mg/kg$_{dwt}$.

For marine sediment, an assessment factor of 10000 should be applied. Therefore, the PNEC for marine sediment is 0.29 mg/kg_{dwt}.

Table B.7.2. The $K_{suspended\ matter}$ for the 16 EPA PAHs.

Compound	$K_{suspended\ matter}$
Naphthalene	34.7
Acenaphthene	155
Acenaphthylene	65.2
Fluorene	256
Anthracene	738
Phenanthrene	573
Fluoranthene	2.44 10^3
Pyrene	1.47 10^3
Benz(a)anthracene	1.25 10^4
Chrysene	9.95 10^4
Benzo(a)pyrene	2.08 10^4
Benzo(b)fluoranthene	2.03 10^4
Benzo(k)fluoranthene	1.99 10^4
Benzo(ghi)perylene	2.55 10^4
Dibenzo(a,h)anthracene	4.88 10^4
Indeno(123-cd)pyrene	5.85 10y

B.7.1.19.3 Anthracene

Two sediment studies are available. The 28-d LC_{50} for *Chironomus riparius* (Bleeker *et al.*, 2003) is 26 mg/kg_{dwt}, recalculated to a sediment with an organic carbon content of 10%. It should be noted that although this value is an LC_{50}, the exposure duration is rather chronic than acute. From the raw data, the LC_{10} is estimated to be 14 mg/kg_{dwt}, recalculated to a sediment with an organic carbon content of 10%. At the lowest tested concentration significant effects on emergence time were still observed, however, only in females. For sediment with 10% organic carbon this NOEC will be lower than 21 mg/kg_{dwt}. Because emergence ratio and survival are directly coupled and the effects on the emergence time are much less at 21 than at 29 mg/kg_{dwt}, the EC_{10} of 14 mg/kg_{dwt} can be considered as a chronic endpoint. A10-d LC_{50} was found for *Hyalella azteca* (Hatch & Burton, 1999). The used sediment has an organic carbon content of 0.39%. The LC_{50} recalculated to a sediment with 10% organic carbon is 85 mg/kg_{dwt}.

Calculation of Predicted No Effect Concentration (PNEC)

For the risk assessment two (semi)chronic LC_{50}s and one chronic LC_{10} are available. With an assessment factor of 100 for the lowest NOEC, a PNEC of 0.14 mg/kg_{dwt} is derived.

For marine sediment species no data are available. The PNEC for marine sediment is derived by applying an assessment factor of 1000 to the NOEC, which results in a PNEC of 0.014 mg/kg_{dwt}.

B.7.1.19.4 Phenanthrene

For sediment data are available for both fresh water sediment and marine sediment. The data for fresh water sediment include chronic tests with annelids, crustaceans and insects. The lowest NOEC is 50 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon, for mortality and growth of both *Hyalella azteca* exposed for 14 days and *Chironomus riparius*, exposed for 10 days (Verrhiest *et al*, 2001). Effect concentrations are based on measured concentrations. In another 28-d study with *Chironomus riparius*, emergence appeared to be somewhat less sensitive (Bleeker *et al*, 2003).

For marine sediment toxicity data are available for two species of crustaceans. Effect concentrations for the amphipod *Rhepoxynius abronius* (Swartz *et al.*, 1997; Boese *et al.*, 1998) appeared to be all above 200 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon. The NOEC for reproduction of the sediment-dwelling copepod *Schizopera knabeni* (Lotufo, 1997) appeared to be lower than 370 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon. From the presented data an EC_{10} might be derived but due to the large extrapolation this value is very uncertain. The value of 370 mg/kg_{dwt} is well above the lowest NOEC for fresh water sediment.

Calculation of Predicted No Effect Concentration (PNEC)

NOECs for fresh water species are available for three different types of benthic species. Therefore, an assessment factor of 10 can be applied to the lowest NOEC. This NOEC is 50 mg/kg_{dwt} for *Hyalella azteca* and *Chironomus riparius*. The resulting PNEC if 5.0 mg/kg_{dwt}.

Marine sediment species are not more sensitive than fresh water sediment species. In addition to the fresh water sediment tests, two marine sediment species were tested in a chronic study. Therefore, the PNEC may apply to both fresh water and marine sediment.

B.7.1.19.5 Fluoranthene

For the annelid *Stylaria lacustris* a NOEC for mortality was reported (Suedel & Rodgers, 1996). Recalculated to sediment with 10% organic carbon this NOEC is 38 mg/kg_{dwt}. With *Daphnia magna* some sediment tests were carried out. Although also some 10-d tests were performed (Suedel *et al.*, 1993; Suedel & Rodgers, 1996), the lowest EC_{50} comes from a 48-h study (Verrhiest *et al.*, 2001). For a sediment with 10% organic carbon this EC_{50} for immobility is 48 mg/kg_{dwt}. Some 30-d test with *Diporeia* species were carried out (Kane Driscoll *et al.*, 1997a; Kane Driscoll & Landrum, 1997). At trace concentrations (0.6-0.9 mg/kg_{dwt}) no effect was observed, at higher concentrations mortality was significantly different from controls (740-900 mg/kg_{dwt}) (Kane Driscoll *et al.*, 1997a). These sediments had low organic matter content (0.37 and 0.53%). In a sediment with higher organic carbon (1.9%) (Kane Driscoll & Landrum, 1997), the concentrations normalised to a sediment with 10% organic carbon up to 570 mg/kg_{dwt} showed no significant mortality. From the studies it can be estimated that the LC_{10} must lie in this range of concentrations.

Several tests with *Hyalella azteca* are available. Results of these studies vary widely. When recalculated to a sediment with 10% organic carbon, LC_{50} (Kane Driscoll & Landrum, 1997; Hatch & Burton, 1999; Suedel *et al.*, 1993; Verrhiest *et al.*, 2001; Wilcoxen *et al.*, 2003) varied from 26 to 6400 mg/kg_{dwt} en NOECs for mortality (Kane Driscoll & Landrum, 1997; Kane Driscoll *et al.*, 1997; Verrhiest *et al.*, 2001; Suedel & Rodgers, 1996) varied from 15 to 2500 mg/kg_{dwt}. Studies are performed with a different percentage organic matter of the sediment ranging from 0.37 to 3.4% as well as with different exposure times ranging from 10 to 30 days. However, both parameters could not explain the large differences in toxicity.

Two chironomid species were tested. 10-d LC_{50} values for *Chironomus tentans* (Suedel *et al.*, 1993) ranged from 68 to 170 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon. For *Chironomus riparius*, exposed for 10 or 11 days to fluoranthene in sediment (Stewart & Thompson, 1995; Verrhiest *et al.*, 2001), these values ranged from 74 to 780 mg/kg_{dwt}. For this species total emergence, emergence time and onset of emergence from a 28-d study were not more sensitive as endpoint as mortality (Stewart & Thompson, 1995). The NOEC for mortality and growth during a 10-d study (Verrhiest *et al.*, 2001) was lower than 15 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon. Fluoranthene was also tested in a mixture together with phenanthrene and benzo(k)fluoranthene, with each compound comprising one third of the total PAH concentration. For this mixture, data are presented from which a clear dose-response relationship can be deduced. If the LC_{10} is determined with a log-logistic relationship, this value is 9.6 mg/kg_{dwt}, on basis of the fluoranthene concentrations in this mixture, and normalised to a sediment with 10% organic carbon.

Because of the additivity of the PAHs in this mixture (Verrhiest *et al.*, 2001), the LC_{10} for fluoranthene alone will be lower than this value. Therefore, 9.6 mg/kg_{dwt} is the lowest effect concentration that was retrieved for fluoranthene in fresh water sediments. All sediment concentrations were verified. Because the measured concentrations were higher than 77% of the nominal concentrations, the data by Verrhiest *et al.* (2001) are based on nominal concentrations.

For marine sediment toxicity studies have been performed with one species of annelids (*Arenicola marina*), five species of crustaceans (*Corophium volutator*, *Corophium spinicorne*, *Rhepoxynius abronius*, *Schizopera knabeni*, *Coullana* spec.), one mollusc (*Abra alba*), and one echinodermata species (*Echinocardium cordata*). Most species are tested with exposure times of 10 days. The test endpoints are often mortality. For *Corophium volutator* (Bowmer, 1994) a geometric mean of 10-d LC_{50} of 370 mg/kg_{dwt} was calculated for sediment with 10% organic carbon. For *Corophium spinicorne* (Swartz *et al.*, 1990) the 10-d LC_{50} was 280 mg/kg_{dwt} or higher. The LC_{10} for this species was 180 mg/kg_{dwt}. For the studies with *Rhepoxynius abronius* (Swartz *et al.*, 1988, 1990, 1997; Boese *et al.*, 1998; Cole *et al.*, 2000; DeWitt *et al.*, 1999) a geometric mean for the 10-d LC_{50} of about 500 mg/kg_{dwt} can be derived when calculated for a sediment with 10% organic carbon. For the studies, in which case a 10-d LC_{10} could be derived from the presented data (Swartz *et al.*, 1988, 1990, 1997; Cole *et al.*, 2000; DeWitt *et al.*, 1999) a geometric mean of 230 mg/kg_{dwt} was derived. Boese *et al.* (1998) reported an EC_{50} for reburial which, transferred to a sediment with 10% organic carbon, is smaller than 67 mg/kg_{dwt}. Reburial is considered to be a better endpoint for chronic toxicity than mortality. In this case, after 10 days of exposure, the organisms were irradiated with UV-radiation (UV-A: 321-400 nm, 315±36 µW/cm^2; UV-B: 280-320 nm, 128±12 µW/cm^2; visible light: 401-700 nm, 3400±278 µW/cm^2) before reburial was monitored. However, in contrast to several other crustaceans, *Rhepoxynius abronius* is a subsurface burrower that typically does not extend body parts in overlying water (Swartz, *et al.*, 1990; Boese *et al.*, 1997). Therefore, this EC_{50} is not very useful in the risk assessment. In contrast, the EC_{10} for mortality for this species is very useful, because from a comparison with data for reburial (Boese *et al.*, 1997), it appears that mortality is almost equally sensitive as reburial.

For the *Schizopera knabeni* the LC_{50} decreases with increasing exposure times. Recalculated to a sediment with 10% organic carbon, the 4-d LC_{50} (Lotufo, 1997) is larger than 14000 mg/kg_{dwt} and the 10-d LC_{50} (Lotufo, 1998) is 1400 mg/kg_{dwt}. For reproduction, the same effect was observed, only this parameter is more sensitive than mortality: the 10-d EC_{50} (Lotufo, 1998) is 360 mg/kg_{dwt}, the 14-d EC_{50} is 250 mg/kg_{dwt}. The EC_{10}s are 98 and 67 mg/kg_{dwt} respectively. Another sensitive endpoint is the grazing rate of algae cells by this organism. From a 6-h study (Lotufo, 1997), an EC_{10} for grazing rate can be derived with a log-logistic model. Recalculated to a sediment with 10% organic carbon, this EC_{10} is 320 mg/kg_{dwt}. When *Schizopera knabeni* were first exposed to fluoranthene for 24 h and the grazing rate was monitored subsequently for 3 hours Lotufo (1998), the calculated EC_{10} is 16 mg/kg_{dwt}. For a *Coullana* species the grazing rate was determined in the same way (Lotufo, 1998). The EC_{10} calculated from the results for this species was 67 mg/kg_{dwt}. Reproduction and mortality from a 10-d study appeared to be less sensitive endpoints.

Tests were also performed with the marine mollusc *Abra alba* and the echinoderm *Echinocardium cordatum*. The EC_{50} for defecation varied from 16.3 to >625 mg/kg_{dwt} in organic rich muddy sediment (Bowmer, 1994). For *Echicardium cordatum* the 10-d LC_{50}s varied from 33 to 116 mg/kg_{dwt} in muddy fine sand (Bowmer, 1994). When possible, recalculation to a sediment with 10% organic carbon lead to LC_{50}s of 2800 to 4100 mg/kg_{dwt}.

Calculation of Predicted No Effect Concentration (PNEC)

The lowest effect concentration for fluoranthene in sediment was found for mortality and growth of *Chironomus riparius*. Because this was a LOEC and not a NOEC and no effect percentage was given, the mixture toxicity study, in which fluoranthene was also used, was considered to derive an

LC_{10} for fluoranthene. The LC_{10} based on the concentrations of fluoranthene in the mixture was 9.6 mg/kg_{dwt}. The LC_{10} for fluoranthene is not expected to be lower than this value, because the other PAHs in the mixture contribute to the toxicity as well, due to the additive toxic action of the PAHs. The range difference between the LOEC and the derived LC_{10} is quite small: 15 versus 9.6 mg/kg_{dwt}. Because data are available for annelids, crustaceans, and insects, an assessment factor of 10 can be applied to this value. The PNEC for fluoranthene in sediment then becomes 0.96 mg/kg_{dwt}.

For marine sediment, chronic studies are available for annelids, crustaceans and echinoderms. It appears that the tested marine species are not more sensitive than *Chironomus riparius*. Therefore, the PNEC for sediment can also be applied to marine sediment.

B.7.1.19.6 Pyrene

Toxicity studies with two fresh water oligochaetes are available. The lowest value for *Limnodrilus hoffmeisteri* is the EC_{10} from a 28-d reproduction study (Lotufo & Fleeger, 1996). However, this value was extrapolated from concentrations showing more than 40% effect. Therefore, the EC_{10} of 54 mg/kg_{dwt} in standard sediment with 10% organic carbon has a large uncertainty. Further, the reproduction falls rapidly with concentration up to 210 mg/kg_{dwt}, but remains almost constant from 210 to 841 mg/kg_{dwt}. It is plausible that the bioavailability of pyrene in the sediment is limited at the higher concentrations by the solubility in the pore water of sediment. If the two highest concentrations are omitted from the determination of EC_{10}, the resulting value of 370 mg/kg_{dwt} in standard sediment is much higher. Further, the reported EC_{25} values determined by bootstrapping, are not in accordance with the log-logistic fit by which the EC_{10} is derived. The EC_{25} values reported by Lotufo & Fleeger (1996) for sediment egestion from a 10-d and a 5-d study and for reproduction from a 28-d study are 51.6, 58.9, and 59.1, respectively, for a sediment with 1.2% organic carbon. With the derived EC_{50}, which is rather certain because it is not an extrapolated value and the reported EC_{25}, EC_{10}s can be derived with a log-logistic model. For the endpoints mentioned above, these EC_{10} values recalculated to a sediment with 10% organic carbon are 370, 370, and 430 mg/kg_{dwt}, respectively. These values probably are more realistic.

The lowest reported endpoint for *Lumbriculus variegatus*, which is wet weight based (Kukkonen & Landrum, 1994), has a much higher EC_{10} value.

For marine sediment only the species *Rhepoxynius abronius* was tested. The EC_{50} for reburial was lower than 42 mg/kg_{dwt} in standard sediment. Similar to fluoranthene, the organisms were irradiated with UV-radiation before reburial was monitored. Because *Rhepoxynius abronius* is a subsurface burrower that typically does not extend body parts in overlying water (Swartz, *et al.*, 1990; Boese *et al.*, 1997), this EC_{50} is not very useful in the risk assessment. Swartz *et al.* (1997) performed some 10-d experiments with *Rhepoxynius abronius*. The LC_{10}s derived from the presented data are 77 and 260 mg/kg_{dwt} standard sediment, the confidence limits of these values are rather small. The geometric mean of these data is 140 mg/kg_{dwt}.

Calculation of Predicted No Effect Concentration (PNEC)

For fresh water sediment two NOECs/EC_{10}s are available for oligochaetes. For marine species one additional EC_{10} for a crustacean is available. This is the lowest value. Because chronic data for two different trophic levels are available, the assessment factor to derive the PNEC for fresh water sediment is 50. Applying an assessment factor of 50 to the value of 140 mg/kg_{dwt} results in a PNEC of 2.8 mg/kg_{dwt}. Because one test is with a fresh water sediment species and one with a marine sediment species, the assessment factor to be applied to derive the PNEC for marine sediment is 100 instead of 500. This results in a PNEC of 1.4 mg/kg_{dwt}.

B.7.1.19.7 Fluorene

No data toxicity for benthic species are available. Therefore, the PNEC for sediment is calculated by equilibrium partitioning, resulting in a value of 2.56 mg/kg_{dwt} for the fresh water environment and 0.26 mg/kg_{dwt} for the marine environment.

B.7.1.19.8 Acenaphthylene

No data toxicity for benthic species are available. Therefore, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 0.17 mg/kg_{dwt} for the fresh water environment and 0.02 mg/kg_{dwt} for the marine environment.

B.7.1.19.9 Acenaphthene

The marine crustacean *Rhepoxynius abronius* is the only benthic species tested with acenaphthene (Swartz *et al.*, 1997; Boese *et al.*, 1998). In this case reburial was not strongly influenced by irradiation with UV. This is similar to the results obtained for naphthalene and phenanthrene but different from the results for fluoranthene and pyrene, for which a significant photoactivation by UV-radiation was found (Boese *et al.*, 1997). The lowest endpoint is therefore the EC_{10} for mortality, derived from the data presented by Swartz *et al.* (1997). From two experiments, the EC_{10}s are 162 and 168 mg/kg_{dwt}, recalculated to standard sediment with 10% organic carbon.

Calculation of Predicted No Effect Concentration (PNEC)

One chronic toxicity study is available for benthic species. In this case, the PNEC for fresh water sediment will be derived with an assessment factor of 100. After application of this factor, the PNEC becomes 1.6 mg/kg_{dwt}.

For marine sediment an assessment factor of 1000 should be applied. The PNEC for marine sediment therefore is 0.16 mg/kg_{dwt}.

B.7.1.19.10 Chrysene

No toxicity data for benthic species are available. Therefore, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 2.79 mg/kg_{dwt} for the fresh water environment and 0.28 mg/kg_{dwt} for the marine environment.

B.7.1.19.11 Benz(a)anthracene

The only benthic species that was tested was the marine crustacean *Rhepoxynius abronius* (Boese *et al.*, 1998). Up to concentrations of 110 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon, no effects were observed. The PNEC for sediment has to be derived by equilibrium partitioning, resulting in a value of 0.6 mg/kg_{dwt} for the fresh water environment and 0.06 mg/kg_{dwt} for the marine environment.

B.7.1.19.12 Benzo(b)fluoranthene

The only benthic species that was tested was the marine crustacean *Rhepoxynius abronius* (Boese *et al.*, 1998). Up to concentrations of 180 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon, no effects were observed. The PNEC for sediment has to be derived by equilibrium partitioning, resulting in a value of 1.38 mg/kg_{dwt} for the fresh water environment and 0.14 mg/kg_{dwt} for the marine environment.

B.7.1.19.13 Benzo(ghi)perylene

No toxicity data for benthic species are available. Therefore, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 0.84 mg/kg_{dwt} for the fresh water environment and 0.08 mg/kg_{dwt} for the marine environment.

B.7.1.19.14Benzo(k)fluoranthene

Benzo(k)fluoranthene in fresh water sediment was tested with three species. For *Hyalella azteca* and larvae of *Chironomus riparius* no toxicity was observed up to concentration of 300 mg/kg_{dwt}, recalculated to a sediment with 10% organic carbon. At this concentration 45% effect was reached in a 48-h toxicity test with *Daphnia magna* (Verrhiest *et al.*, 2001). However, *Daphnia magna* is rather an aquatic organism than a benthic organism. With a sediment concentration of 1500 mg/kg_{dwt} recalculated to sediment with 10% organic carbon, the overlying water is probably saturated. Therefore, this value should be considered as 45% mortality at the aqueous solubility.

Calculation of Predicted No Effect Concentration (PNEC)

Because no toxicity data can be used for deriving the PNEC, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 1.38 mg/kg_{dwt} for the fresh water environment and 0.14 mg/kg_{dwt} for the marine environment.

B.7.1.19.15Benzo(a)pyrene

Because no toxicity data are available for deriving the PNEC, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 1.83 mg/kg_{dwt} for the fresh water environment and 1.83 mg/kg_{dwt} for the marine environment.

B.7.1.19.16Dibenzo(a,h)anthracene

No toxicity data for benthic species are available. Therefore, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 0.27 mg/kg_{dwt} for the fresh water environment and 0.03 mg/kg_{dwt} for the marine environment.

B.7.1.19.17Indeno(123-cd)pyrene

No toxicity data for benthic species are available. Therefore, the PNEC for sediment must be calculated by equilibrium partitioning, resulting in a value of 0.63 mg/kg_{dwt} for the fresh water environment and 0.06 mg/kg_{dwt} for the marine environment.

B.7.1.19.18PNEC summary for marine and fresh water sediments

Table B.7.3 summarises the PNEC for sediment dwelling organisms for the different PAHs together with the assessment factor and species used.

B.7.2 Terrestrial compartment

B.7.2.1 Equilibrium partitioning

For a number of PAHs, the PNEC has been calculated by equilibrium partitioning in the absence of sufficient toxicity data for soil species, by using the following equation:

$$\mathrm{PNEC_{soil}} = \frac{K_{\text{soil-water}}}{\rho_{\text{soil}}} \times \mathrm{PNEC_{water}} \times 1000$$

where

$K_{\text{soil-water}}$ = the soil-water partitioning coefficient, and ρ_{soil} = bulk density of wet soil (1700 kg/m^3)

In Table B.7.4 the partitioning coefficient of each PAH is listed.

Table B.7.3. The PNEC for the various PAHs for fresh and marine sediment organisms.

Compound	PNEC fresh water sediment (mg/kg_{dwt})	Assessment factor [a)]	Species [b)]	PNEC marine sediment (mg/kg_{dwt})	Assessment factor [a)]
Naphthalene	2.9	1000 vs. EqP	*Rhepoxynius abronius*	0.29	10000 vs. EqP
Anthracene	0.14	100	*Chironomus riparius*	0.014	1000
Phenanthrene	5	10	*Hyalella azteca / Chironomus riparius*	5	10
Fluoranthene	0.96	10	*Chironomus riparius*	0.96	10
Pyrene	2.8	50	*Rhepoxynius abronius*	1.4	100
9H-Fluorene	2.56	EqP		0.26	EqP
Acenaphthylene	0.34	EqP		0.03	EqP
Acenaphthene	1.6	100	*Rhepoxynius abronius*	0.16	1000
Chrysene	2.79	EqP		0.28	EqP
Benz(a)anthracene	0.60	EqP		0.06	EqP
Benzo(b)fluoranthene	1.38	EqP		0.14	EqP
Benzo(ghi)perylene	0.84	EqP		0.084	EqP
Benzo(k)fluoranthene	1.38	EqP		0.14	EqP
Benzo(a)pyrene	1.83	EqP		1.83	EqP
Dibenzo(a,h)anthracene	0.27	EqP		0.027	EqP
Indeno(123cd)pyrene	0.63	EqP		0.063	EqP

[a)] EqP: PNEC calculated with equilibrium partitioning (see Section B.7.1.19.1); [b)] The same species is used for both marine and fresh water sediments.

Table B.7.4. The soil-water partitioning coefficient for the 16 EPA PAHs.

Compound	$K_{soil\text{-}water}$
Naphthalene	40.7
Acenaphthene	185
Acenaphthylene	77.3
Fluorene	306
Anthracene	885
Phenanthrene	687
Fluoranthene	$2.93\ 10^3$
Pyrene	$1.77\ 10^3$
Benz(a)anthracene	$1.5\ 10^4$
Chrysene	$1.19\ 10^4$
Benzo(a)pyrene	$2.5\ 10^4$
Benzo(b)fluoranthene	$2.44\ 10^4$
Benzo(k)fluoranthene	$2.38\ 10^4$
Benzo(ghi)perylene	$3.06\ 10^4$
Dibenzo(a,h)anthracene	$5.85\ 10^4$
Indeno(123-cd)pyrene	$7.02\ 10^4$

B.7.2.2 Naphthalene

For soil toxicity studies with macrophyta, collembola, and annelida are available. In addition, one study for terrestrial processes was found. Effect concentrations from terrestrial studies are first transferred to values for standard soil containing 2% organic carbon (3.4% organic matter) by

correcting for the organic carbon content. The lowest usable effect concentration is the NOEC of 10 mg/kg_{dwt} for reproduction of the springtail *Folsomia candida* from a 28-d study (Bleeker *et al.*, 2003). Values for the other three species (the annelid *Enchytraeus crypticus*, the lettuce *Lactuca sativa*, and *Folsomia fimetaria*) were all within one order of magnitude. At the highest test concentration of 24 mg/kg_{dwt} no effects were observed on terrestrial processes (Kirchmann *et al.*, 1991). In the EU-RAR for naphthalene (Munn *et al.*, 2003) no usable studies for terrestrial species were found.

Calculation of Predicted No Effect Concentration (PNEC)

NOECs or EC_{10}s are available for three trophic levels (primary producers, consumers, and decomposers). Therefore, an assessment factor of 10 can be applied to the lowest NOEC. This results in a PNEC of 1.0 mg/kg_{dwt}.

B.7.2.3 Anthracene

For soil data are available for annelida, macrophyta and collembola. The lowest usable endpoint is for reproduction of *Folsomia fimetaria*. The EC_{10} recalculated to a soil with 2% organic carbon is 6.3 mg/kg_{dwt} (Sverdrup *et al.*, 2002e). Unlike the results for naphthalene a similar effect concentration for the related species *Folsomia candida* was not found. Effect were not observed in the highest tested concentration (Bleeker *et al.*, 2003 , Droge *et al.* 2006).

In most tests with macrophyta no effects on growth of seedlings are observed (Mitchell *et al.*, 1988). Growth of seedlings of *Avena sativa* has a 14-d EC_{50} of 51 mg/kg_{dwt}, growth of seedlings of *Cucumis sativus* a 14-d EC_{50} of 1200 mg/kg_{dwt} (Mitchell *et al.*, 1988) and the NOEC for shoot and root growth of *Lolium perenne* exposed for 40 days (Leyval & Binet, 1998) appeared to be smaller than 133 mg/kg_{dwt} (all values recalculated to a soil with 2% organic carbon). The latter study (Leyval & Binet, 1998) was performed with moderate visible light (PAR 400-700 nm at 130 µmol/m^2/s). At the lowest concentration 22 to 41% reduction in growth was observed. From the presented data at the three tested concentrations a reliable EC_{10} could not be derived. In the study by Mitchell *et al.*, (1988), it can be deduced from the figure for percentage emergence of seeds that the LC_{10} for *Avena sativa* should be significantly lower than 170 mg/kg_{dwt}. For the more sensitive effect of growth only the EC_{50} is presented. From the figure for time of emergence it also obvious that for four plant species the NOEC lies below 17 mg/kg_{dwt}.

For the tested annelid *Enchytraeus crypticus*, no effects were observed in the highest concentration (Bleeker *et al.*, 2003 , Droge *et al.*, 2006), *i.e.* NOEC ≥780 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon.

Calculation of Predicted No Effect Concentration (PNEC)

The effect concentrations from chronic studies with macrophyta are one order of magnitude or even less higher than the EC_{10} for *Folsomia fimetaria*. Because it can not be excluded that the NOEC or EC_{10} for some plant species is lower than that of *Folsomia fimetaria*, an assessment factor of 50 will be applied to the lowest effect concentration of the two remaining trophic levels, which is the EC_{10} for *Folsomia fimetaria*. The PNEC for soil then becomes 0.13 mg/kg_{dwt}.

B.7.2.4 Phenanthrene

Chronic toxicity data for phenanthrene in soil are available for annelids, collembola, plants, crustaceans, and microbial processes. Again for phenanthrene, the EC_{10} for reproduction of *Folsomia fimetaria* is the lowest EC_{10} or NOEC (Sverdrup *et al.*, 2001, 2002ce). Four EC_{10} values for *Folsomia* in sandy loam soil are available. The difference between the four values is the ageing of phenanthrene in the soil. Soils were spiked and toxicity testing started after 0, 10, 40, or 120 days after spiking. The EC_{10}s for these cases were 29, 18, 18, and 12 mg/kg_{dwt}, recalculated to a soil with

2% organic carbon. The EC_{10}s are based on measured concentrations. The geometric mean of these EC_{10}s is 18 mg/kg_{dwt}. This value is based on measured concentrations.

Calculation of Predicted No Effect Concentration (PNEC)

Because of the large number of terrestrial toxicity data, an assessment factor of 10 is sufficient. The resulting PNEC is 1.8 mg/kg_{dwt}.

B.7.2.5 Fluoranthene

The lowest NOEC or EC_{10} is the EC_{10} for nitrification in a sandy loam with a test duration of 28 days (Sverdrup *et al.*, 2002a). The EC_{10} recalculated to a soil with 2% organic carbon is 15 mg/kg_{dwt}. The EC_{10} for reproduction of *Enchytraeus crypticus* exposed for 21 days to fluoranthene (Sverdrup *et al.*, 2002b) is only slightly higher, 19 mg/kg_{dwt} recalculated to a soil with 2% organic carbon. In this case the EC_{10} for *Folsomia fimetaria* exposed for 21 days is higher (Sverdrup *et al.*, 2001, 2002c). Recalculated to a soil with 2% organic carbon this EC_{10} is 47 mg/kg_{dwt}.

Calculation of Predicted No Effect Concentration (PNEC)

The lowest NOEC or EC_{10} is the EC_{10} for nitrification of 15 mg/kg_{dwt}. Because chronic data are available for collembola, annelids, terrestrial plants, crustaceans and microbial processes, an assessment factor of 10 can be applied. With this assessment factor the PNEC for soil is 1.5 mg/kg_{dwt}.

B.7.2.6 Pyrene

Chronic toxicity data are available for two species of *Folsomia* (Collembola), two annelid species, three terrestrial plants and the terrestrial process nitrification. The lowest value is the 28-d NOEC for reproduction of *Folsomia candida* (Bleeker *et al.*, 2003). The NOEC recalculated to soil with a organic carbon content of 2% is 10 mg/kg_{dwt}.

The EC_{10}/NOEC for reproduction *Folsomia fimetaria* and *F. candida* are very similar to this value, varying from 10 to 21 mg/kg_{dwt} recalculated to a soil with 2% organic matter. Ageing of the compound for a period 120 days had little effect on the toxicity (Sverdrup *et al.*, 2002c).

The annelid *Enchytraeus crypticus* was tested three times. From the 21-d reproduction study from Sverdrup *et al.* (2002b) a NOEC of 23 mg/kg_{dwt} was derived for standard soil (EC_{10} of 14 mg/kg_{dwt}). A similar 28-d reproduction study by Bleeker *et al.* (2002) results in a NOEC of 160 mg/kg_{dwt} for standard soil. The NOEC for growth of *Eisenia veneta* from a 28-d study (Sverdrup *et al.*, 2002d) was 37 mg/kg_{dwt}, recalculated to 2% organic carbon (EC_{10} of 48 mg/kg_{dwt}). Droge *et al.* (2006) reported an EC_{50} value of 52 $mg/kg_{dwt.}$

For the three terrestrial plant species *Sinapsis alba*, *Trifolium pratense*, and *Lolium perenne* fresh weight is the most sensitive endpoint in the study with an exposure time of 19 to 21 days (Sverdrup *et al.*, 2003). From the reported EC_{50} and EC_{20} values, EC_{10}s can be derived with a log-logistic relationship. These EC_{10} values for the three terrestrial plants are 34, 19, and 546 mg/kg_{dwt} for a soil with 2% organic carbon. The NOEC and EC_{10} for nitrification (Sverdrup *et al.*, 2002a) are 99 and 160 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon.

Calculation of Predicted No Effect Concentration (PNEC)

Because chronic toxicity studies are available for collembola, annelids, terrestrial plants and nitrification an assessment factor of 10 can be applied to the lowest NOEC or EC_{10}. This is the NOEC for reproduction of *Folsomia candida* of 10 mg/kg_{dwt}. The resulting PNEC for soil thus becomes 1.0 mg/kg_{dwt}.

B.7.2.7 Fluorene

The lowest NOEC or EC_{10} recalculated to a soil with 2% organic carbon with a useful endpoint for risk assessment is the 47-w NOEC for growth of the isopod *Oniscus asellus* (Van Brummelen *et al.*, 1996a). In this test the food was refreshed once a week and the concentrations were expressed as nominal concentrations. The initial concentrations appeared to be higher than nominal concentrations. The concentrations after 3 days were estimated to be 41% of nominal concentrations. However, according to the authors this might be an underestimation due to the volatility of 9*H*-fluorene in comparison with the internal standard. This test was actually not conducted in soil but with contaminated food with a organic matter content of more than 90%. Therefore, the extrapolation to a soil with 3.4% organic matter (2% organic carbon) is rather large, introducing some uncertainty. However, in view of the long exposure time, this study is relevant. The EC_{10} that is determined by the authors with a log-logistic model is 14 mg/kg_{dwt}, extrapolated to 2% organic carbon, which is almost a factor of 6 higher than the NOEC from this study.

The EC_{10} for reproduction of *Folsomia fimetaria* in a 21-d study (Sverdrup *et al.*, 2001, 2002e) was 10 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon. The concentrations are expressed as initial measured concentrations. The lowest EC_{10}s for annelids, terrestrial plants and nitrification are in the range of 20-50 mg/kg_{dwt}.

Calculation of Predicted No Effect Concentration (PNEC)

Chronic toxicity data are available for collembola, annelids, terrestrial plants, crustaceans and nitrification. An assessment factor of 10 can therefore be applied to the lowest NOEC or EC_{10}. Because of the uncertainty in the values for the isopod *Oniscus asellus* and the fact that the EC_{10} for this species is higher than the EC_{10} for *Folsomia fimetaria*, the EC_{10} for the latter species is chosen as lowest endpoint for the PNEC, instead of the NOEC for *Oniscus asellus*. With an assessment factor of 10, the PNEC is 1.0 mg/kg_{dwt}.

B.7.2.8 Acenaphthylene

The only terrestrial study with acenaphthylene is a 21-d reproduction study with *Folsomia fimetaria* (Sverdrup *et al.*, 2002e). The EC_{10} from this study was 29 mg/kg_{dwt}, recalculated to a soil with 2% organic matter. The concentrations are expressed as initial measured concentrations.

Calculation of Predicted No Effect Concentration (PNEC)

Only one EC_{10} is available for acenaphthylene. Therefore, an assessment factor of 100 should be applied to this EC_{10}. The PNEC then becomes 0.29 mg/kg_{dwt}. It should be noted that this endpoint is most often the most sensitive for the other PAHs, for which an assessment factor of 10 is applied.

B.7.2.9 Acenaphthene

Two studies with terrestrial species are available for acenaphthene. The first study is a 14-d study for germination and shoot growth of *Lactuca sativa* (Hulzebos *et al*, 1993). The EC_{50} for acenaphthene is 47 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon. No dose-response data or NOEC or EC_{10} are given in the publication. However, in an unpublished underlying report the NOEC is stated to be 1.9 mg/kg_{dwt} recalculated to a soil with 2% organic carbon. The concentrations of acenaphthene were not experimentally determined.

The EC_{10} from a 21-d reproduction study with *Folsomia fimetaria* (Sverdrup *et al.*, 2002e) was 39 mg/kg_{dwt}, recalculated to a soil with 2% organic matter. The concentrations are expressed as initial measured concentrations.

Calculation of Predicted No Effect Concentration (PNEC)

Chronic toxicity data are available for terrestrial plants and collembola. Therefore, an assessment factor of 50 can be applied to the lowest NOEC. This is the NOEC for germination and shoot growth of *Lactuca sativa* of 1.9 mg/kg_{dwt}. The PNEC is 0.038 mg/kg_{dwt}.

B.7.2.10 Chrysene

Chronic toxicity studies were performed with *Folsomia candida* (Sverdrup *et al.*, 2002e), *Folsomia candida* (Bowmer *et al.*, 1993), and the annelid *Eisenia fetida* (Bowmer *et al.*, 1993). No effects were observed in these studies. Therefore, the PNEC for soil has to be derived by equilibrium partitioning, resulting in a value of 0.55 mg/kg_{dwt}.

B.7.2.11 Benz(a)anthracene

Terrestrial toxicity studies for benz(a)anthracene have been performed with annelids, crustaceans, and collembola. The annelid *Enchytraeus crypticus* (Bleeker *et al.*, 2003 , Droge *et al.*, 2006) and the collembola species *Folsomia candida* (Bleeker *et al.*, 2003 , Droge *et al.*, 2006) and *Folsomia fimetaria* (Sverdrup *et al.*, 2002e) showed no signs of toxicity. The same was observed for the crustacean *Porcellio scaber*. For growth of the crustacean *Oniscus asellus* effects were observed (Van Brummelen *et al.*, 1996a). This test was conducted with contaminated food with an organic matter content of more than 90%, which introduces a large extrapolation to a soil with 3.4% organic matter (2% organic carbon). However, in view of the long exposure time, this study is relevant. The food was refreshed once a week and the concentrations were expressed as nominal concentrations. The concentrations after 3 days were estimated to be 83% of nominal concentrations. The EC_{10} that is determined by the authors with a log-logistic model is 21 mg/kg_{dwt}, which corresponds to 0.79 mg/kg_{dwt}, extrapolated to 2% organic carbon.

Calculation of Predicted No Effect Concentration (PNEC)

Because chronic toxicity data are available for annelids, crustaceans and collembola, an assessment factor of 10 can be applied to the lowest NOEC or EC_{10}. In this case, the lowest EC_{10} is 0.79 mg/kg_{dwt} for growth of *Oniscus asellus*. The resulting PNEC for soil is 0.079 mg/kg_{dwt}. In view of the high extrapolation factor, this PNEC might be over-conservative.

B.7.2.12 Benzo(b)fluoranthene

The only toxicity study with terrestrial species for benzo(b)fluoranthene is a 21-d study with *Folsomia fimetaria* (Sverdrup *et al.*, 2002e). Up to concentrations of 450 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon, no effects were observed. The PNEC for soil has to be derived by equilibrium partitioning, resulting in a value of 0.28 mg/kg_{dwt}.

B.7.2.13 Benzo(ghi)perylene

The only toxicity study with terrestrial species for benzo(ghi)perylene is a 21-d study with *Folsomia candida* (Bowmer *et al.*, 1993). Up to concentrations of 61 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon, no effects were observed. The PNEC for soil has to be derived by equilibrium partitioning, resulting in a value of 0.17 mg/kg_{dwt}.

B.7.2.14 Benzo(k)fluoranthene

The only toxicity studies with terrestrial species for benzo(k)fluoranthene are a 21-d study with *Folsomia candida* (Bowmer *et al.*, 1993) and a 21-d study with *Folsomia fimetaria* (Sverdrup *et al.*, 2002e). Up to concentrations of 61 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon, no effects were observed for *F. candida*. For *F. fimetaria* this concentration was 710 mg/kg_{dwt}. The PNEC for soil has to be derived by equilibrium partitioning, resulting in a value of 0.27 mg/kg_{dwt}.

B.7.2.15 Benzo(a)pyrene

Chronic toxicity studies with benzo(a)pyrene are available for 2 species each of terrestrial annelids, crustaceans, and collembola. No effects were found for the studies with *Folsomia* species (Sverdrup *et al.*, 2002e; Bleeker *et al.*, 2003; Droge *et al.*, 2006). For annelids, effects were observed in one study only, with *Eisenia fetida* being slightly more sensitive than *Enchytraeus crypticus* (Achazi *et al.*, 1995). The NOECs for reproduction are 2.6 and 8.7 mg/kg$_{dwt}$ respectively, recalculated to a soil with 2% organic carbon.

For the crustaceans *Oniscus asellus* and *Porcellio scaber* effects were observed in tests that were conducted in contaminated food with an organic matter content of more than 90% (Van Straalen & Verweij, 1991; Van Brummelen & Stuijfzand, 1992; Van Brummelen *et al.*, 1996). This way of exposure introduces a large extrapolation to a soil with 3.4% organic matter (2% organic carbon). No significant effects on growth of *Oniscus asellus* were observed in a 47-w study up to concentrations of 12 mg/kg$_{dwt}$, recalculated to a soil with 2% organic carbon (Van Brummelen *et al.*, 1996). In a 9-w study, however, effects were observed (Van Brummelen & Stuijfzand, 1992). The NOEC for weight and length is 1.2 mg/kg$_{dwt}$ and for mortality 3.8 mg/kg$_{dwt}$ in standard soil. The EC_{10}s derived from the presented data for wet and dry weight and for length vary from 2.8 to 4.2 mg/kg$_{dwt}$.

Also for *Porcellio scaber* no significant effects on growth were observed in a 47-w study up to concentrations of 12 mg/kg$_{dwt}$, recalculated to a soil with 2% organic carbon (Van Brummelen *et al.*, 1996). Also in this case in the 9-w study effects were observed (Van Brummelen & Stuijfzand, 1992). The NOEC for weight is 1.2 mg/kg$_{dwt}$ but for length no significant effects were observed in concentrations of up to 12 mg/kg$_{dwt}$, recalculated to a soil with 2% organic carbon. Although in this case no firm dose-response relationships could be derived from the presented data, a decrease in fresh and dry weight and in length of more than 10% appears from these data at concentrations higher than 0.12 mg/kg$_{dwt}$, recalculated to a soil with 2% organic carbon. At higher concentrations these parameters remain more or less constant. The absence of a clear dose-response curve might be the result of the limited solubility of benzo(a)pyrene. If it is assumed that uptake of the compounds occurs via the aqueous phase, the further uptake and hence more effect can be limited by the aqueous solubility of benzo(a)pyrene of 1.2-1.8 µg/L (Mackay *et al.*, 2000). In a third study, the growth efficiency was studied during 4 weeks (Van Straalen & Verweij, 1991). In this study the food was refreshed once a week and initial concentrations were more than 90% of the nominal concentrations. The concentrations were therefore expressed as nominal concentrations. Growth efficiency was expressed as increase of dry weight divided by the difference between the consumption of food and defecation. Because the consumption of food and the assimilation efficiency were more or less constant for both male and female isopods, the growth efficiency mainly represents the increase in dry weight. The NOEC for growth efficiency of male isopods is 0.94 mg/kg$_{dwt}$, extrapolated to 2% organic carbon. A clear dose-response curve is presented. From the presented figure an EC_{10} of 0.53 mg/kg$_{dwt}$ in standard soil is estimated with a log-logistic model. For female isopods no significant effects were observed at the highest concentration of 4.7 mg/kg$_{dwt}$ recalculated to standard soil. However, for females a strong increase is observed for the growth efficiency at the two lowest concentrations. In comparison with these concentrations the growth efficiency declines strongly after 0.2 mg/kg$_{dwt}$ in standard soil. Due to the initial increase in growth efficiency the fit of the log-logistic dose-response curve is not as good as for male isopods. However, similar values are obtained for the EC_{50} and EC_{10}. The EC_{10} for growth efficiency of male isopods of 0.53 mg/kg$_{dwt}$ is comparable with the concentrations at which 10% effect or more was observed in the study by Van Brummelen & Stuijfzand (1992).

Calculation of Predicted No Effect Concentration (PNEC)

The EC_{10} of 0.53 mg/kg_{dwt} for growth efficiency of the isopod *Porcellio scaber* is used as endpoint for the PNEC. Because chronic toxicity data are available for annelids, crustaceans, and collembola, an assessment factor of 10 can be applied. The PNEC thus becomes 0.053 mg/kg_{dwt}. In view of the high extrapolation factor, this PNEC might be over-conservative.

B.7.2.16 Dibenzo(a,h)anthracene

The only toxicity study with terrestrial species for benzo(ghi)perylene is a 21-d study with *Folsomia fimetaria* (Sverdrup *et al.*, 2002e). Up to concentrations of 980 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon, no effects were observed. The PNEC for soil has to be derived by equilibrium partitioning, resulting in a value of 0.054 mg/kg_{dwt}.

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B.7.2.17 Indeno(123-cd)pyrene

The only toxicity study with terrestrial species for benzo(ghi)perylene is a 21-d study with *Folsomia fimetaria* (Sverdrup *et al.*, 2002e). Up to concentrations of 1100 mg/kg_{dwt}, recalculated to a soil with 2% organic carbon, no effects were observed. The PNEC for soil has to be derived by equilibrium partitioning, resulting in a value of 0.13 mg/kg_{dwt}.

B.7.2.18 PNEC summary for the terrestrial compartment

Table B.7.5 summarises the derived PNEC together with the assessment factor and species used.

Table B.7.5. The PNEC for the various PAHs for soil organisms.

Compound	PNEC soil (mg/kg_{dwt})	Assessment factor [1)]	Species
Naphthalene	1.0	10	*Folsomia candida*
Anthracene	0.13	50	*Folsomia fimetaria*
Phenanthrene	1.8	10	*Folsomia fimetaria*
Fluoranthene	1.5	10	Nitrification
Pyrene	1.0	10	*Folsomia candida*
9H-Fluorene	1.0	10	*Folsomia fimetaria*
Acenaphthylene	0.29	100	*Folsomia fimetaria*
Acenaphthene	0.038	50	*Lactuca sativa*
Chrysene	0.55	EqP	
Benz(a)anthracene	0.079	10	*Oniscus asellus*
Benzo(b)fluoranthene	0.28	EqP	
Benzo(ghi)perylene	0.17	EqP	
Benzo(k)fluoranthene	0.27	EqP	
Benzo(a)pyrene	0.053	10	*Porcellio scaber*
Dibenzo(a,h)anthracene	0.054	EqP	
Indeno(123cd)pyrene	0.13	EqP	

[1)] EqP: PNEC calculated with equilibrium partitioning (see Section B.7.1.19.1).

B.7.3 Atmospheric compartment

Due to the lack of data, no PNEC has been established for the atmospheric compartment. In the risk assessment for man indirectly exposed to the environment the exposure to air concentrations in the vicinity of the different plants is considered. It is to be expected that any precautions necessary to limit that risk will also be protective for wild life.

B.7.4 Microbiological activity in sewage treatment systems

The toxicity of CTPHT (electrode binder BX 90) to *Pseudomonas putida* has been tested in a cell multiplication inhibition test according to a draft guideline (DIN 38412; Hillman, 1991). For preparing the test concentrations, suspensions from 625 to 10000 mg CTPHT/L were treated ultrasonically and mixed carefully in a water bath equipped with a magnetic stirrer overnight. The suspensions were then centrifuged for 10 min at 3000 rpm and the supernatants were used. In order to augment the suspension 0.1 g/L Tween was added (control included). In this test solutions CTPHT were inoculated with a suspension of *P. putida* of defined turbidity. Nutritive medium was added and the mixture incubated for 16 hours at 21 °C. Cell mass was monitored by measuring the turbidity of the bacterial suspension. Over the whole test range no inhibition was observed.

In an additional test in the same study, CTPHT (24 mg) was dissolved in the highest permissible concentration of a solvent toluol (0.1 g/L). Subsequently, after addition of water the solution was ultrasonificated and mixed carefully. From this solution 5 test concentrations from 1.5 to 20 mg/L were prepared. Within this test range no inhibition was observed. No analysis of the test solution was performed.

Although this study is sufficient for the base set of CTPHT, it does not provide data to derive exact $PNEC_{microorganisms}$ values for the individual PAHs in a sewage treatment plant. Based on the solubility data given in Section B.1.3, it can however be assumed that the PNEC values will be in the range of µg/L or higher.

An additional study is available in which creosote was tested for toxicity towards activated sludge according to OECD 209 (OECD, 1984; Lebertz, 1984). Creosote was directly introduced into the test solution, containing the prepared inoculum, synthetic sewage feed and tap water and incubated over a period of 3 hours. The final test concentrations ranged from 125 to 2000 mg/L. No analysis of the solution was preformed. The EC_{50} was determined at 670 mg/L, which suggest that the EC_{50} values for the individual PAHs are not below the µg/L range.

Although toxicity data on *Vibrio fischeri* cannot be used for the risk assessment of a sewage treatment plant, supporting evidence for the last conclusion is found in toxicity studies with this species for the different PAHs (Loibner *et al.*, 2004). The EC_{10} values for the two and three ring PAHs range from 0.13 mg/L for phenanthrene to 0.39 mg/L for naphthalene. For PAHs with four rings or more no toxicity is observed up to the saturated aqueous solution. It was argued that the toxicity of PAHs towards *V. fischeri* seems therefore to be related to the maximum water solubility rather than the toxicity of the individual PAH.

B.7.5 Non compartment specific effects relevant for the food chain (secondary poisoning)

The Technical Guidance Document recommends that the NOAEL from dietary toxicity tests with fish-eating birds or mammals are used to determine the $PNEC_{oral}$. However, data on the PAH toxicity to birds are scarce. Some pertinent data from the literature are reviewed by Albers & Loughlin (2003): Patton & Dieter (1980) exposed mallards (*Anas platyrhynchos*) to a diet containing 10 PAHs for 7 months and observed greater hepatic stress responses and higher testis weights than male mallards fed a mixture of 10 alkanes. Retardation of nestling weight gain and increased adrenal and nasal gland weights was attributed to the PAHs with four or more rings. Immune function and MFO activity of European starlings (*Sturnus vulgaris*) were altered by oral or subcutaneous doses of 7,12-dimethylbenz(a)anthracene, a four-ring PAH (Trust *et al.*, 1994). From these data it is not possible to derive a NOAEL for birds for either of the PAHs.

Also relevant toxicity data to mammals is limited. Almost all of the long term studies reported were designed to assess carcinogenic potency of PAH and are not considered appropriate for the environmental risk assessment. Only for benzo(a)pyrene reprotoxicity data are available. Most

severe effect were observed after administration of 10 mg/kg to CD-1 mice by gavage during gestation which produced decreased gonadal weights and reduced fertility and reproductive capacity in the offspring. Higher doses (40 mg/kg) caused almost complete sterility in both sexes of offspring (Mackenzie & Angevine, 1981). As no lower concentrations are tested a NOAEL can not be determined and consequently no $PNEC_{oral}$ can be derived.

Other mammalian toxicity data for acenaphthene, anthracene, benzo(a)pyrene, fluorene, fluoranthene and pyrene derived from 90-d studies with mice resulted in higher NOAELs. Whilst a PNEC could be derived from these data in the usual way, the reprotoxicity data for benzo(a)pyrene suggest that such a PNEC might not be adequately protective. The ecological relevance of the adverse effect on which some of the NOAELs are based, might also be questionable, *e.g.* heamatological effect and increased ALAT activity (see Table B.7.6).

Based on the available information PNEC oral values for the individual PAHs can not be derived.

To illustrate the potential risk a preliminary assessment for benzo(a)pyrene is made in Section B.10.2.5 for which the PNEC oral is derived as follows:

Based on a LOEC of 10 mg/kg for mice using an extrapolation factor of 2 to obtain a NOEC value, a conversion factor of 8.3 and an assessment factor of 30, a $PNEC_{oral}$ for benzo(a)pyrene can be derived of 1.4 mg/kg food.

Table B.7.6. NOAELs for PAHs found in a 90 subchronic toxicity study with mice.

Compound	NOAEL ($mg/kg_{bw}/d$)	Lowest Toxicity endpoint
Acenaphthene	175	Hepatoxicity
Anthracene	1000	No toxicity observed
Benzo(a)pyrene	< 1100	Growth
Fluorene	125	Heamatological effect
Fluoranthene	125	Increased ALAT activity, pathological effect in the kidney and liver and clinical and haematological changes
Pyrene	125	Nephropathy and decreased kidney weight

Values taken from IPCS report (WHO, 1998).

B.8 PBT and vPvB assessment

B.8.1 Assessment of PBT/vPvB properties-Comparison with criteria of Annex XIII

As described in Section B.4.1 most of the PAHs in CTPHT have a DT_{50} value both in soil and sediment > 125 days, which means that CTPHT meets the P (Persistent) and vP (very persistent) criteria.

In several studies conducted with different fish species BCF values for fluorene, anthracene, phenanthrene, fluoranthene and pyrene were measured > 2000. For anthracene, phenanthrene and fluoranthene the BCF values were even > 5000 (Linder *et al.*, 1985; De Voogt *et al.*, 1991; De Maagd, 1996; Weinstein & Oris, 1999). This means that CTPHT meets the B (bioaccumulative) and vB (very bioaccumulative) criteria.

For all the EPA 16 PAHs the aquatic NOEC values are < 0.01 mg/L, which means that CTPHT also meets the T criterion.

In view of the fact that most of the (higher molecular) PAHs are present in > 0.1% (see Section B.1.2) it can be concluded that CTPHT meets the vP, vB and T criteria and hence is considered as a PBT and vPvB substance.

B.8.2 Emission characterisation

It is well realised that many sources for PAH emissions to the environment exist, mostly unintentional sources due to various combustion processes (traffic, etc.). Although these will not be a formal part of this risk assessment, an overview of the available information of PAH emissions in Europe will be given to put the emissions of PAHs from the use of coal tar pitch, high temperature in perspective.

PAH may enter the environment from both natural (forest fires, volcanoes) and anthropogenic sources. The latter includes production and use of coal tar pitch itself, but PAH is also formed as a by-product during other industrial processes (*e.g.* coke plants). The emissions of PAH from other sources are mainly characterised by combustion processes and by particular industrial processes using PAH-containing compounds such as coal, crude oil, creosote, coal-tar or bitumen. Important non-industrial sources of PAH emissions are the combustion of solid fuels, like wood, peat and coal and the use of all automotive fuels but in particular diesel oil. Natural sources of PAH include the accidental burning of forests, woodland, heath etc. Another natural PAH source is volcanic activity, but no data is available regarding these emissions. In this chapter the different sources are described including the PAH or benzo(a)pyrene emissions to air and water.

B.8.2.1 Sources of PAH emission

B.8.2.1.1 Industrial

The most important industrial emission sources include coke production, primary aluminium production and creosote and wood preservation. The PAH emissions at the various processes of the primary aluminium production and the production and use of electrodes for the aluminium industry are explained in Section B.2.2.2 and B.2.2.4. CTHT is produced at coke plants as such and as a by-product of primary steel production (see Section B.2.1). The main source of PAH emissions in the iron and steel industry is the coke ovens, used to make coke for the steel production. Primarily, the coke is needed to chemically reduce iron oxide to iron metal. Other sources of PAH emissions in coke production are the heating systems of the coke ovens, the plants for coke extinguishing and the loss of coke gas at all stages of the technological cycle (Tsibulsky, 2001). The coke industries improved their PAH emissions markedly by applying modern technology. Nevertheless, old installations still have high PAH emissions, leading to local high ambient air concentrations (EC, 2001b). PAH emissions at steel production using electric arc furnaces originate from the presence of tar in the used refractory material. Creosote is a distillation product of coal tar, a by-product of bituminous coal coking. Emissions of PAH take place at all stages of the wood preservation process: impregnation, storage, transport and use. In the creosote and wood preservation industry, wood is mainly impregnated under pressure in vessels, but can also be sprayed or dipped. Since 2003 creosoted wood is only to be used for certain applications by professionals when treated in vacuum/pressure installations. Creosoted wood, which is treated through spraying, brushing or dipping is banned in the European Union. Creosoted wood is completely banned for certain applications like playgrounds, garden and garden furniture according to the EU Directive 2001/90/EC (EC, 2001d). Consequently wood preservation through spraying and dipping has been phased out in the European Union. Therefore emission from this source is expected to reduce considerably. PAH emissions to air from solvent use, which includes wood impregnation, in the United Kingdom clearly decreased over the period 1990 till 2002 from 104 tonnes to 69 tonnes with no clear decrease in the period 2000 till 2002. From this information it might be concluded that there is no clear direct effect on emissions from PAH resulting from the enforcement of the EU Directive at least in the United Kingdom. Other industrial sources include petrochemical and related industries (refineries), bitumen and asphalt industries (production and use), waste incineration, power plants, rubber tyre production, cement production (combustion of fossil fuels) and motor test rigs.

B.8.2.1.2 Domestic

PAH-emissions from domestic sources are predominantly associated with the combustion of solid fuels as wood and coal for heating and cooking purposes. These sources contribute significantly to the total PAH emission. In Europe there is a large geographic variation in these domestic emissions due to climatic differences and to the heating systems in use. In addition to heating purposes, wood, coal or peat are also burned for the decorative effect in open fireplaces.

B.8.2.1.3 Mobile

Mobile sources include all modes of transport using a combustion engine. PAH emissions from these sources depend on engine type, fuel type, emission control, outdoor temperature, load of vehicle, age of the car/engine and driving habits. Diesel fuelled vehicles have higher particulate emissions and the emission control equipment is less developed than gasoline vehicles. Therefore, diesel fuelled vehicles are responsible for more PAH emissions on the road. The wear and tear of tyres is also an important source of PAH emissions. Due to the extensive use of catalytic converters and improved diesel quality, the PAH emissions from tyres could even be larger than those from the exhaust of vehicles (Edlund, 2001). Non road transport includes all PAH emissions from combustion engines used by shipping activities, railways and aircrafts.

B.8.2.1.4 Agricultural

Agricultural sources involve the burning of organic materials under less optimum combustion activities and therefore produce significant amounts of PAH. These activities include stubble burning, open burning of land for regeneration purposes or the open burning of brushwood, trimmings, straw etc. In some EU countries there are regulations in place regulating these emissions (EC, 2001b).

B.8.2.2 PAH emissions from the different sources

B.8.2.2.1 Emissions to air

Emission data of PAH to air are scarce. The data for PAH (16 PAH, 6 Borneff) are available for the 15 OSPAR member countries, the Netherlands and the UK and benzo(a)pyrene data are available for Germany (see Table B.8.1-Table B.8.5). The UK data presented in Table B.8.4 and Table B.8.5 are actually based on the same reference, but the published figures are dissimilar. The difference is mainly caused by the traffic emissions for naphthalene, which are 1,153,360 kg/y and 34,100 kg/y according to the UK National Atmospheric Emissions Inventory (NAEI) and Department for Environment, Food and Rural Affairs (DEFRA), respectively. Most likely, the largest emission of the NAEI is correct (Munne *et al.*, 2003).

Large non-industrial emission sources are the domestic combustion of solid fuels, the use of coal tar-based products (creosote) for wood preservation and road transport. The emissions of these three sources together can amount 54%-89% of the total PAH emission to air, dependent on the references (see Table B.8.1-Table B.8.5). PAH emissions from wood preservation will reduce due to the entry into force of EU directive 2001/90/EC in 2003. The Directive prohibits the use coal tar based products for wood preservation through spraying and dipping, which consequently is expected to be phased out in the European Union. Therefore emission from this source is also expected to reduce considerably. The contribution of the industrial emissions of the aluminium and steel industry to the total PAH emissions are not unimportant with values up to 22%. For the Netherlands these industrial emissions are very low, mainly because probably a large part is grouped under ‘other processes’. In 2001, in the Netherlands about 500 tonnes have been emitted to air for the total PAH 10 (Duyzer *et al.*, 2002). In the Netherlands consumers (35-64%), traffic and transport (20-35%) and agriculture (12-21%) are the main PAH sources to air (Duyzer *et al.*, 2002).

The importance of agriculture is not confirmed by the emission data presented in Table B.8.2 for 1998.

Table B.8.1. PAH emissions to air (6 Borneff) in 15 OSPAR member countries for the year 1990.

No	Source	PAH (tonnes/year)	(%)
1	Industrial processes		
	- iron and steel production	131	2
	- non-ferro metal industry (primary aluminium and anode baking)	378	5
	- asphalt industry	112	1
	- other processes	16	0.2
2	Industrial combustion	78	1
3	Power generation	14.6	0.2
4	Commercial, institutional and domestic combustion	4,220	54
5	Solvent use wood preservation (coal tar-based products) [a)]	1,820	23
6	Traffic emissions	955	12
7	Other		
	- waste incineration	5.69	0.1
	Total	7,730	

The OSPAR member countries are Belgium, Denmark, Finland, France, Germany, Iceland, Ireland, Luxembourg, the Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and the UK. The data are taken from OSPAR (2001), but originate from Berdowski *et al.* (1997); [a)] The use of carbolineum in wood preservation is being phased out in the EU. There are also restrictions on the use of creosoted wood according to EU directive 2001/90/EC (EC, 2001d).

Table B.8.2. PAH emissions to air (6 Borneff) in The Netherlands for 1998.

No	Source	PAH (kg/y)	(%)
1	Industrial processes		
	- iron and steel production	258	0.2
	- non-ferro metal industry	98.5	0.1
	- petroleum industry	66.3	0.1
	- inorganic and organic chemicals	3.7	0
	- other processes (mainly metal-electro)	35,800	33.8
2	Industrial combustion	169	0.2
3	Power generation	86.2	0.1
4	Domestic combustion	8,270	7.8
5	Commercial, institutional combustion	16.6	0
6	Solvent use wood preservation (coal tar-based products)	27,400	25.9
7	Road transport		
	- combustion	4,402	4.2
	- brake and tyre wear	28,600	27.0
8	Non-road transport	714	0.7
9	Other		
	- waste treatment, agricultural combustion	16.2	0
	Total	105,900	

Data are taken from CCDM (2000).

Table B.8.3. Benzo(a)pyrene emissions to air in UK (1995) and Germany (1994).

No	Source	Germany [a] (kg/y)	%	UK [b] (kg/y)	%
1	Industrial processes				
	- iron and steel production: sinter plants	52	0.4	-	-
	- iron and steel production: coke production	1,090	8	1,100	2.5
	- iron and steel production: electric arc furnaces	257	2	-	-
	- non-ferro metal industry (primary aluminium / anode baking)	2,578	19	16,200	36.4
2	Industrial combustion	27.8	0.2	5,000	11.2
3	Power generation	5.5	0	-	-
4	Domestic combustion				
	- coal	3,992	29	2,200	4.9
	- oil	3,383	25	-	-
	- wood	1,940	14	1,200	2.7
5	Solvent use wood preservation (coal tar-based products)	157	1.2	460	1
6	Traffic emissions	266	1.9	7,700	17.3
7	Other				
	- natural fire	-	-	2,900	6.5
	Total	13,751		44,460	

[a] Figures derived from Grandrass & Salomons (2001); [b] Figures derived from EC (2001b).

Table B.8.4. PAH emissions to air in the UK for 1999.

No	Source	PAH (kg/y) [a]	(%)
1	Industrial processes		
	- iron and steel production (coke, sinter and combustion)	100,618	3.8
	- non-ferro metal industry (aluminium)	277,349	10.4
	- refineries / petroleum industry	4,502	0.2
	- other processes (chemical industry, cement, collieries, etc.)	8,792	0.3
2	Industrial combustion (others)	154,792	5.8
3	Power generation	3,164	0.1
4	Domestic combustion	522,754	19.6
5	Commercial, institutional combustion	588	0.02
6	Solvent use wood preservation (coal tar-based products)	72,765	2.7
7	Road transport	1,293,513	48.6
8	Non-road transport	4,764	0.2
9	Other:		
	- waste treatment (incineration)	123,425	4.6
	- natural fires	94,920	3.6
	- agriculture (combustion)	640	0.02
	Total	2,663,035	

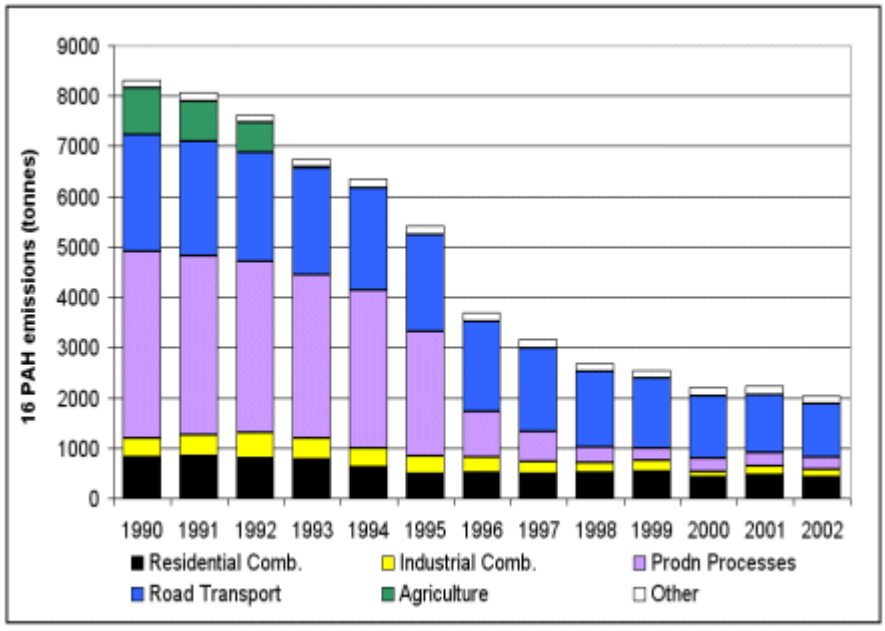
[a] Sum of the 16 EPA PAHs, data taken from the UK National Atmospheric Emissions Inventory (NAEI, 2001).

Table B.8.5. PAH emissions to air in the UK for 1999.

No	Source	PAH (kg/y) [a]	(%)
1	Industrial processes		
	- iron and steel production	22,303	1.6
	- non-ferro metal industry (aluminium)	277,349	19.6
	- petroleum industry	4,593	0.3
	- other processes	80,855	5.7
2	Industrial combustion (others)	166,756	12.4
3	Power generation	3,162	2.2
4	Domestic combustion	540,123	38.2
5	Commercial, institutional combustion	2,692	0.2
6	Solvent use wood preservation (coal tar-based products)	102,564	7.3
7	Road transport		
	- combustion	114,490	8.1
	- brake and tyre wear	48.5	0
8	Non-road transport	4,146	0.3
9	Other		
	- waste treatment (incineration)	298	0
	- natural fires	94,920	6.8
	Total	1,414,300	

[a] Sum of the 16 EPA PAHs, data taken from the UK Department for Environment, Food and Rural Affairs.

According to the EU Working group on PAHs (EC, 2001b), the UK seems to be quite representative of the majority of the European countries and as an example, the trend of the sum of the 16 EPA PAH in the UK between 1990 and 2002 is shown in Figure B.8.1.



Source: http://www.naei.org.uk/pollutantdetail.php.

Figure B.8.1. Atmospheric emission of the sum of the 16 EPA PAHs in the UK between 1990 and 2002.

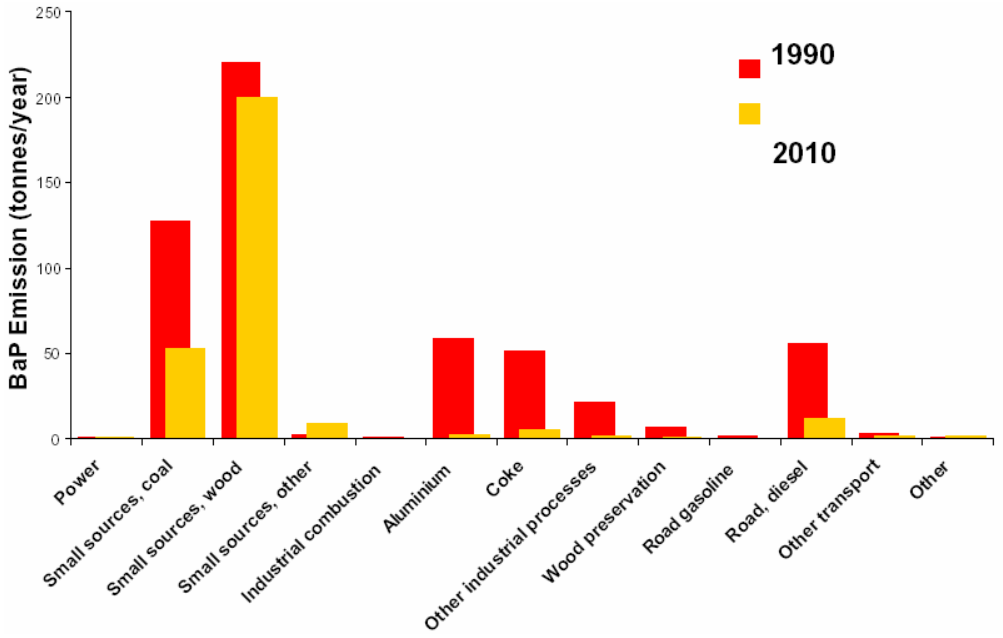
Similarly Table B.8.6 illustrates, again using UK data, the generally downward trend currently being observed within the European Union as a whole. The estimated benzo(a)pyrene emissions for 1990 and 1995, and the forecast emissions for 2010, represent a 'business as usual' scenario[4]. PAHs emissions have decreased significantly since 1990. Between 1990 and 1995, the estimated total emissions of benzo(a)pyrene had decreased by over 50 %. The main reduction was in the emission from natural fires and open agricultural burning which decreased by 90% from 1990 levels because of the ban on stubble burning in England and Wales. During 2002, the largest source of PAH was road transport combustion, contributing 52% to the total emissions. Other major sources include domestic combustion and non-ferrous metal production. The UK benzo(a)pyrene emission is forecast to further decrease by 2010 to 16.4 tonnes (see Table B.8.6). The emission from vehicles is forecast to decrease under the 'business as usual' scenario, due mainly to stricter emission regulations which require *e.g.* the use of catalytic converters, and improved maintenance and vehicle condition. The emissions from anode baking (within the process of primary aluminium production) are predicted to decrease sharply as a result of improved abatement equipment which was brought on-stream during the last 10 years and the implementation of the IPPC Directive with introduction of BAT effective from 2007. The emission from domestic coal combustion is forecast to decrease between 1990 and 2010 due to a decrease in the quantity of coal burned (Figure B.8.2). However, these sources are still likely to be responsible for a significant proportion of the forecast 2010 emission, which is spread across several sectors: vehicles (24 %), industrial combustion (24 %), domestic combustion (18 %), and natural fires (18 %).

Table B.8.6. Summary of benzo(a)pyrene emissions in the UK measured in 1990 and 1995 and estimated for 2010.

Benzo(a)pyrene emissions [a]	1990		1995		2010	
	(tonnes)	(%) [b]	(tonnes)	(%) [b]	(tonnes)	(%) [b]
Vehicles-Diesel	1.4	1.7	2.0	5.4	3.3	20.0
Vehicles-Gasoline	8.5	10.6	5.7	15.5	0.7	4.0
Natural fires / open agricultural burning	31	38.7	2.9	7.8	2.9	17.5
Creosote use	0.06	0.07	0.06	0.16	0.06	0.4
Aluminium production	1.9	2.4	1.4	3.9	0.03	0.2
Anode baking	22.7	28.3	14.8	40.3	1.0	5.9
Coke production	1.3	1.	1.1	2.9	1.1	6.7
Domestic wood combustion	1.2	1.5	1.2	3.2	1.2	7.1
Industrial wood combustion	0.1	0.2	0.1	0.4	0.2	1.0
Domestic coal combustion	5.3	6.6	2.2	6.1	1.9	11.3
Industrial coal combustion	6.3	7.8	4.9	13.3	3.8	23.3
Other sources	0.4	0.5	0.4	1.0	0.4	2.4
Total	80.2	100	36.8	100	16.4	100

[a] Data taken from EC (2001b); [b] % contribution to total benzo(a)pyrene emissions from sources in this table.

[4] In respect of emissions: Business as usual should be interpreted as:
(1) Human activity (industry, transport, domestic consumption, etc.) continue forecasted growth,
(2) There is no new legislation introduced that would affect emissions,
(3) Existing legislation is fully implemented.



Taken from Holland *et al.* (2001)

Figure B.8.2. Current and projected emissions of benzo(a)pyrene for the EU15+6 accession countries.

B.8.2.2.2 Emissions to water

PAH can be emitted to surface water directly or indirectly via a sewage treatment plant by (industrial) point sources and via atmospheric deposition. Information on PAH emission to surface water for the EU is limited to the EPER database (EPER, 2004). Based on the emission estimates for 1998 in the Netherlands, road transport is considered to be by far the largest emission source to water, followed by emissions from agriculture and consumers. The emission from industry is relatively small (see Table B.8.7).

Table B.8.7. PAH emissions to water (tonnes/year) in the Netherlands for 1998.

	agriculture	refineries	industry	waste treatment	road transport	consumers	public service	effluent sewage treatment plant	emission via air and soil	total
PAH (Borneff 6)	0.623	0.001	0.051	0.000	15.3	0.001	0.001	1.51	3.20	21.1

Data taken from Van Harmelen *et al.* (1999).

The European Pollutant Emission Register (EPER, 2004) reports PAH emission of the different point sources for 2001 (see Table B.8.8). The largest industrial emission sources to water are the pre-treatment of fibres or textiles, based on the EPER data.

Based on both measurements and model calculation Duyzer & Vonk (2003) determined the total burden of surface waters in the Netherlands (excluding the North Sea) by atmospheric deposition of three PAH (anthracene, phenanthrene and benz(a)anthracene). The total emission in the Netherlands to surface water via atmospheric deposition of these three PAH was more than 25,000 kg/y (see Table B.8.9).

Table B.8.8. PAH emissions to water in the EU for 2001.

Source	Direct (kg)	Indirect via sewage treatment plant (kg)
Industrial processes		
- iron and steel production	10,271	381
- petroleum industry	558	151
- basic organic chemicals	1,519	16
- pharmaceutical products	0	36
- pre-treatment fibres or textiles	0	12,284
Industrial combustion	1,022	6
- Installations for the production of carbon or graphite	21	0
- Slaughterhouses, plants for the production of milk other animal or vegetable raw materials	267	77
- Industrial plants for pulp from timber or other paper or board production	6	0
Waste disposal	259	80
	6	0
Total	13,923	13,031

Data taken from EPER (2004).

Table B.8.9. Total burden of surface waters in the Netherlands (excluding the North Sea) by atmospheric deposition of anthracene, phenanthrene and benz(a)anthracene for 2002.

PAH	Burden (kg)
Anthracene	1,570
Phenanthrene	22,220
Benz(a)anthracene	1,470
Total	25,260

Data taken from Duyzer & Vonk (2003).

B.8.2.2.3 Summary

Overall, it can be concluded that a consistent description of the emissions and emission sources of PAH to air is not available. The reasons for this are caused for instance by the different ways to express total PAH emissions (6 Borneff, 16 PAH, etc.), the different classifications into categories (*e.g.* does anode baking belong to other processes or to non-ferrous metals) and other striking differences (*e.g.* the emissions of brake and tyre wear). A recent overview of the PAH emissions to air in the EU is not available and the data available is only based on a few EU countries. Nevertheless, it seems that the largest emission sources to air are non-industrial, like domestic combustion, the use of coal tar-based products and road transport. For the emission to surface water even less data is available. Some industrial point sources can be large emission sources of PAH. Compared to (industrial) point source data, the emission via atmospheric deposition seems more important.

B.9 Exposure assessment

B.9.1 General discussion on releases and exposure

CTPHT is a complex hydrocarbon mixture consisting primarily 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 (see Section B.1). The relative proportions of the components in the mixture of coal tar pitch (CTPHT) are complex and variable and dependent on whether low temperature or high temperature processes were involved in the production of the tar. Of interest here is exposure to CTPHT, the solid fraction produced during

the distillation of high temperature coal tar formed with the high-temperature (> 700 °C) carbonisation of coal (IARC, 1985). 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. CTPV are released when coal tar, CTPHT, or their products, are heated (HSDB 2004).

Production and use are described in Sections B.2.1 and B.2.2. Briefly, high temperature coal tar pitch is produced by distillation of high temperature coal tar. Coal tar pitch is mainly used as a binding agent in the production of carbon electrodes, anodes and Søderberg 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 B.2.2.

In Sections B.9.2 to B.9.5 the exposure to CTPHT is assessed for workers (Section B.9.2), for consumers (Section B.9.3), for humans exposed via the environment (Section B.9.4), and for the environment itself (Section B.9.5).

B.9.1.1 Summary of existing legal requirements

Occupational Exposure Limits

Table B.9.1 presents the occupational limit values for CTPV that are used in a number of European countries and the USA.

Environment

EU directive 96/61 (EC, 1996) concerning integrated pollution prevention and control, which regulates pollution emission from industrial point sources, will lead to reduced emission and discharge particularly through application of best available techniques (BAT).

The 4th Daughter Directive (Council Directive 2004/107/EC; EC 2004) provides a target value of 1.0 ng/m^3 and lower (0.4 ng/m^3) and upper assessment (0.6 ng/m^3) thresholds for benzo(a)pyrene that are used to determine the extent of monitoring required in the agglomerations and regional zones in order to avoid, prevent or reduce harmful effects of polycyclic aromatic hydrocarbons on human health and the environment as a whole. To assess the contribution of benzo(a)pyrene in ambient air, each Member State shall monitor other relevant polycyclic aromatic hydrocarbons at a limited number of measurement sites. These compounds shall include at least: benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, indeno(123-cd)pyrene, and dibenz(a,h)anthracene.

EU directive 2000/76 (EC, 2000b) on waste incineration and the EU directive 1999/31 (EC, 1999a) on landfill of waste make provision for low PAH emission from point sources. EU directive 2003/33 (EC, 2003a) establish criteria and procedures for the acceptance of waste at landfills pursuant to article 16 of annex II to EU directive 1999/31. For PAH the limit values has to be set by the Member States themselves.

Under the Water Framework Directive (EC, 2000a) all PAHs has been identified as priority hazardous substances with the exception of fluoranthene and naphthalene which are identified as hazardous substances. Benzo(a)pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)-fluoranthene, fluoranthene, indeno(123-cd)pyrene have been selected as guidance substances.

PAHs have been incorporated in work plans for AMAP, EMEP, EUROTRAC, HELCOM, OSPARCOM, UNEP WMO and others (WMO, 1999).

Crude and refined coal tars are not permitted to be used as substance or preparation in cosmetics according to EU directive 76/768 (EC, 1976a) and 97/45 (EC, 1997).

Table B.9.1. Occupational limit values for CTPV.

Country/ organization	8-hr time weighted average (mg/m³)	Remarks	References
USA			
ACGIH	0.2	coal tar pitch volatiles as benzene soluble aerosol classified as A1 carcinogen, *i.e.* confirmed human carcinogen: the compound is carcinogenic to humans based on the weight of evidence from epidemiological studies.	ACGIH (2003)
OSHA	0.2	coal tar pitch volatiles as benzene soluble fraction	OSHA (2007)
NIOSH	0.1	coal tar pitch volatiles as cyclohexane extractable fraction	NIOSH (2007)
The Netherlands	-	An occupational exposure limit for benzo(a)pyrene and PAHs (from coal tar derived sources) is currently being derived using the meta-analyses of Armstrong et al. (2003; 2004).	Dutch Expert Committee on Occupational Standards (2006); Social Economic Council (2007)
UK	-	Listed among compounds which are currently on the work programme.	HSE (2002)
Germany	-	Listed as pyrolysis products from organic materials. They are classified among compounds which are capable of inducing malignant tumours as shown by experience with humans.	DFG (2006)
		Workplace exposure limits until 2005: 0.002 mg benzo(a)pyrene/m³ (8 h long term value) Remark: the technical based TRGS (Technische Regeln für Gefahrstoffe, Technical Rules for Dangerous Substances) were cancelled with the implementation of the new German Hazardous Substances Ordinance, but as long as there is no replacement the TRGS act as a guideline. It should be noted that these values are not health-based.	CCSG (2007)
Sweden	not listed	The occupational exposure limit for benzo(a)pyrene is 0.002 mg/m³.	Swedish Work Environment Authority (2007)
Denmark	0.2	polycyclic aromatic hydrocarbons as benzene-soluble fraction Coal- and oil-derived substances, as well as coal tar products, including coal tar pitch distillates with boiling point > 200 °C are listed among substances which are considered to be carcinogenic.	Arbejdstilsynet (2005)
Norway	-	Reference is made to polycyclic aromatic hydrocarbons for which an exposure limit of 0.04 mg/m³ has been established.	Direktoratet for arbeidstilsynet (1996)
France	0.2	coal tar pitch volatiles as benzene soluble fraction	Institut National de Recherche et de Sécurité (2006)
European Union	not listed		SCOEL (2007)

B.9.1.2 Summary of effectiveness of the implemented risk management measures

In the smaller applications of CTPHT (*i.e.* refractories, road construction, active carbon, heavy duty corrosion protection, roofing, clay pigeons, and coal briquetting; see Section B.2) no risks were identified (see Section B.10), suggesting that for these applications the existing measures are

sufficient. Especially for CTPHT production, primary aluminium production, and electrode and anode production risks of PAHs are still identified (see Section B.10), which suggests that implemented risk management measures and operational conditions are not sufficient to control the risks.

B.9.2 Exposure assessment for workers

B.9.2.1 Introduction

Because of the complexity and variability of CTPHT, great difficulties have been encountered in assessing exposure. Generally, the presence of coal tars and derived products is detected by the presence of their specific constituents, especially CTPV and Polycyclic Aromatic Hydrocarbons (PAHs), in the air (IARC, 1985). The need to establish a reliable marker compound that could be used in the assessment of exposure to CTPHT has been recognised. Previous exposure limits for CTPV at the workplace have used as a measure the concentration of solvent-extracted particulate matter in the atmosphere. In 1997 the Working group on Assessment of Toxic Chemicals (WATCH) recommended that UK HSE should investigate alternative methods, based on work carried out at the Health and Safety Laboratories (HSL), which showed that the methods that were available to measure airborne CTPV were unreliable and could not be improved upon using the conventional approach. Since PAHs are among the major components of CTPHT, and some individual PAHs are proven animal carcinogens, PAH levels are considered as a measure of exposure to CTPV (WATCH, 2000). The HSE evaluated three ways to assess exposure to PAHs:

1) the summed carcinogenic potency weighting approach
2) the determination of PAH exposure based on the sum of 11 PAHs identified by HSE as having the greatest carcinogenicity potential of the PAH family of compounds, and
3) the use of a single marker compound from which an appropriate measure of total PAH (mixture of all PAH) exposure could be obtained.

With respect to the summed carcinogenic potency weighting approach, evidence from animal studies showed that the carcinogenic potential for individual PAHs did not correlate with the carcinogenic potential of PAH mixtures. Therefore, summing the potential contribution of each PAH would give a misleading impression of the total carcinogenic potential of the mixture. Summing the 11 PAHs identified by the HSE as having the greatest carcinogenic potential, takes the industry-specific exposure profiles into account, but was seen as a complex and costly approach. Existing exposure information suggested that the airborne concentration of benzo(a)pyrene correlates well with the concentration of total PAHs for most workplaces. Based on these findings and the availability of exposure data, WATCH has pinpointed benzo(a)pyrene as the most suitable marker for assessing exposure to PAHs[5] for CTPV industries (WATCH, 2000). In addition, the industry indicated that the fingerprint of the PAH mixture (the proportion of the individual PAHs in the mixture) within each exposure scenario (see Sections B.9.2.2 to B.9.2.9) are fairly stable, which makes it possible to use the concentration of benzo(a)pyrene as an estimate of the whole fingerprint. As such, in conducting the occupational exposure assessment to CTPHT, exposure to benzo(a)pyrene has been adopted as the primary indicator for every exposure scenario.

Indeed, with regard to different scenarios, a fairly wide range of differences in benzo(a)pyrene content of CTPHT PAHs is found: varying from a benzo(a)pyrene content of 0.71-2% of the total PAHs in the coke industry (Buchet *et al.*, 1992; Larson, 1978; WHO, 2000) to 6% of the particulate PAHs in an electrode paste plant (Øvrebø *et al.*, 1994). In 1997 Angerer reported a 3.6% benzo(a)pyrene content in 8 PAH concentrations from a graphite electrode (Angerer et al., 1997). In the Søderberg potroom the benzo(a)pyrene:benzene soluble material ratio was estimated to be

[5] These are 11 PAHs identified by HSE as having the greatest carcinogenic potential of the PAH family of compounds.

around 10 µg:1 mg (Armstrong, 1986). Data provided by industry show that benzo(a)pyrene content in CTPHT is between 0.1% and 1.5%.

In those cases for which direct benzo(a)pyrene measurements are not available, it will be assumed in this document that the concentration of benzo(a)pyrene in CTPHT-PAHs is 10%. This will represent a concentration of benzo(a)pyrene in the CTPHT of 1%. This represents a reasonable worst case assessment of exposure to benzo(a)pyrene and, due to the proportionality of benzo(a)pyrene in CTPHT, extends to that of CTPHT.

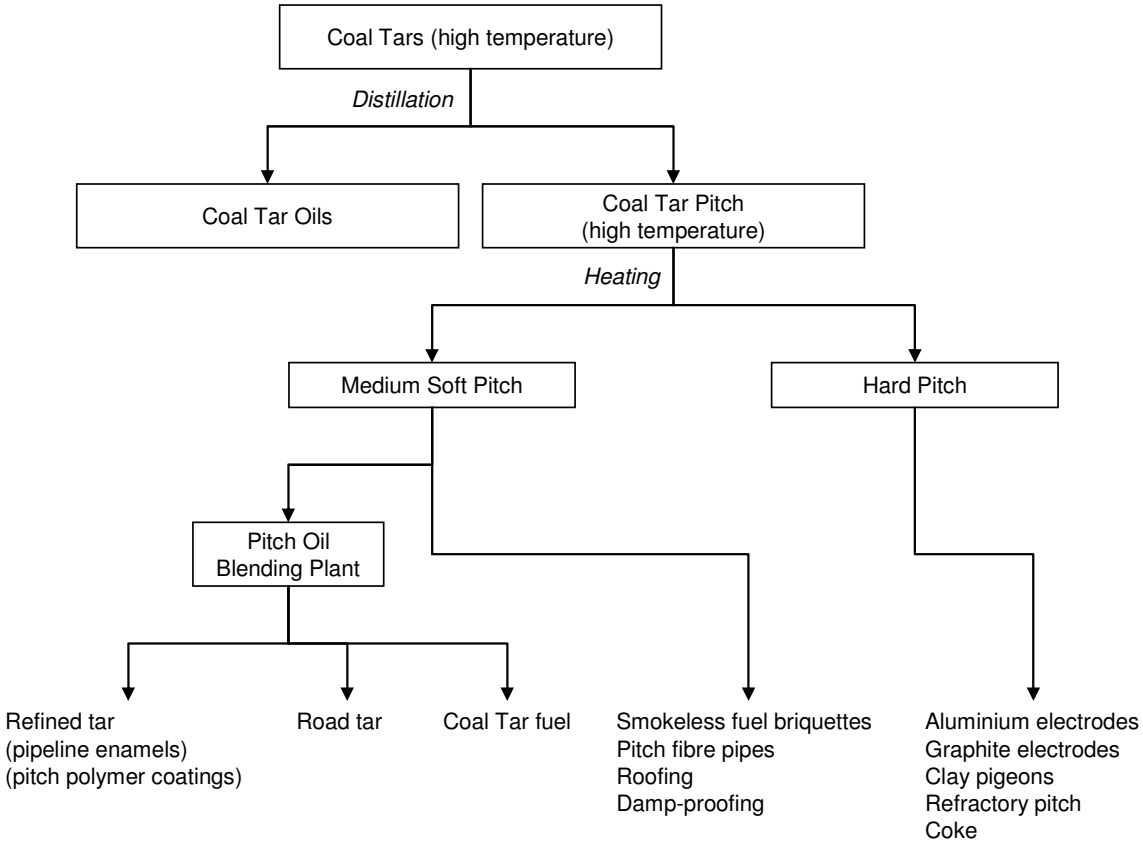


Figure B.9.1. Uses of CTPHT which constitute potential occupational exposure.

Occupational exposure to CTPHT can occur during both the production and use of the CTPHT. The primary source of CTPHT is from coal tar distillation with the subsequent release of CTPV. Also, the coal tar distillation plant may be affiliated with a plant in which the CTPHT produced is used to extract metals from their ores or in some other metal production process *e.g.* iron foundries. Figure B.9.1 shows the different areas under which occupational exposures are possible during use.

The occupational exposures during use of CTPHT arise primarily during its use as a binding agent in several different industries, most notably during its use as a binder in electrodes, *e.g.* aluminium smelting electrodes and graphite electrodes (IARC, 1984). It is also used in roofing materials, surface coatings, black varnishes, and pipe-coating enamels. CTPHT is used to impregnate and strengthen the walls of brick refractories. Hard pitch is used as a binder for foundry cores. Coal tar pitch is used as a binder for carbon brushes and carbon and graphite articles which is then carbonised at high temperatures. The tar oils and solid fractions produced by the distillation of high-temperature coal tars are used in the production of naphthalene, recovery of benzene, production of anthracene paste, briquetting of smokeless solid fuel, impregnation of electrodes and fibres and manufacture of electrodes and graphite and for road paving and construction (IARC, 1985). Potential occupational exposure to CTPHT can occur for workers producing or using pavement tar, CTPHT, coal tar paint, coal tar coatings, coal tar enamels and refractory bricks, electrodes

(including anodes for the primary aluminium production), active carbon and alloys (see Figure B.1.1 in Section B.1 and Section B.2).

The occupational exposure to CTPHT differs according to the processes involved which decide the physical state of the contaminant. Inhalatory and dermal exposure to the CTPHT may be to its gas, vapour or particulate bound form. The vapour mainly contains 3- and 4-ringed PAHs. The higher molecular PAHs are usually particulate bound and CTPVHT comprise these higher molecular weight PAHs and among them our exposure indicator benzo(a)pyrene. High molecular weight gaseous PAHs may also be found adsorbed on particulate materials (Buchet et al., 1992).

The exposure is assessed using the available information on the substance, processes and work tasks. More detailed information on these parameters may lead to a more accurate exposure assessment.

In this part of the assessment, external (potential) exposure is assessed using relevant models and other available methods in accordance with the Technical Guidance Documents and agreements made at official Meetings of Competent Authorities. Internal dose depends on external exposure and the percentage of the substance that is absorbed (either through the skin or through the respiratory system).

The exposure is assessed without taking into account the possible influence of personal protective equipment (PPE). If the assessment as based on potential exposure indicates that risks are to be expected, the use of personal protective equipment may be one of the methods to decrease actual risks, although other methods (technical and organisational) are to be preferred. This is in fact obligatory following harmonised European legislation.

Knowledge of effectiveness of PPE in practical situations is very limited. Furthermore, the effectiveness is largely dependent on site-specific aspects of management, procedures and training of workers. A reasonably effective use of proper PPE for skin exposure may reduce the external exposure by 85%. For respiratory protection the efficiency depends largely on the type of protection used. Without specific information, a tentative reduction efficiency of 90% may be assumed, equivalent to the assigned protection factors for supplied-air respirators with a half mask in negative pressure mode (NIOSH, 1987). Better protection devices will lead to higher protection. Imperfect use of the respiratory protection will lower the practical protection factor compared to the assigned factor. These estimations of reduction are not generally applicable to "reasonable worst case" estimations, but indicative of values based on very limited data. They will not be used directly in the exposure and risk assessment. Furthermore, the reduction of external exposure does not necessarily reflect the reduction of absorbed dose. It has to be noted that the use of PPE can result in a relatively increased absorption through the skin (effect of occlusion), even if the skin exposure is decreased. This effect is very substance-specific. Therefore, in risk assessment it is not possible to use default factors for reduction of exposure as a result of the use of PPE.

In some specific situations the model estimates with normal assumptions for input parameters in the assessed exposure scenarios are expected not to lead to a reasonable assessment of exposure. There exist situations where there is a high risk of direct acute effects, such as manual handling of corrosive substances and hot materials, or possible inhalation exposure of substances with severe acute effects on the respiratory tract. In such cases the total level of containment given by all exposure control measures is assumed to be higher than for similar scenarios with other substances. For estimating a single day exposure, extra protection is assumed, reducing exposure by 90%. The extra protection can be reached by a combination of technical and organisational control measures and personal protective equipment. It is not acceptable that extra protection is reached (mainly) by use of personal protective equipment, and this situation should be changed by further technical and organisational control measures.

The main result of the estimations is the so-called reasonable worst case estimate. This value intends to estimate the exposure level in a reasonable worst case situation, *i.e.* in a situation with exposures in the higher ranges of the full distribution of exposure levels, but below the extremes reached. If a large number of data are available, a 90th percentile is generally used as an estimator of the reasonable worst case value. If limited data sets are available (*e.g.* only measurements from one site or only small numbers of measurements or measurements with very little detail on tasks, working conditions, etc.) often the highest measured value is taken or the results of modelling are preferred. Alternatively, a conservative intermediate value is chosen to account for the weaknesses in the different data sets.

There are a considerable number of possible situations, which may lead to the occupational exposure to CTPHT. Exposure assessments have been conducted for a subset of these. These scenarios given priority were chosen based on information from the International Tar Association (2002) on the market share of use of CTPHT in the EU. Based on this information and research into other scenarios not included herein, it is believed that this document covers the most important sources of occupational exposure to CTPHT. Among these excluded scenarios was possible exposure during coal gasification.

Occupational exposure assessment has been conducted for eight scenarios. The first scenario, production of CTPHT in a coal tar distillation facility, is the most prevalent source of exposure. This is followed by its use as a binder and impregnation of electrodes. The asphalt industry and use as a binder in refractories form the next more relevant exposure scenarios. The other scenarios listed represent a small part of overall use of CTPHT. Unless otherwise stated, the estimated proportion of the indicator, benzo(a)pyrene, in CTPHT is 1%. Its proportion in CTPHT-PAHs is 10%.

With respect to the main applications of CTPHT, the following scenarios are considered to be relevant for occupational exposure assessment:

- Occupational Scenario 1: Production of CTPHT in coal tar distillation plants (see Section B.9.2.2)
- Occupational Scenario 2: Use as a binding agent for electrodes (see Section B.9.2.3)
- Sub-scenarios: (i) Use as a binding agent in electrodes in the aluminium industry (see Section B.9.2.3.1)
- (ii) Use as a binding agent in graphite electrode production and impregnation of electrodes (see Section B.9.2.3.2)
- Occupational Scenario 3: Use as a binding agent in the asphalt industry (see Section B.9.2.4)
- Sub-scenarios: (i) Use as a binding agent in road construction (see Section B.9.2.4.1)
- (ii) Use as a binding agent in roofing and waterproofing (see Section B.9.2.4.2)
- Occupational Scenario 4: Use as a binding agent for refractories (see Section B.9.2.5)
- Occupational Scenario 5: Use as a binding agent for active carbon (see Section B.9.2.6)
- Occupational Scenario 6: Use in heavy duty corrosion protection (see Section B.9.2.7)
- Occupational Scenario 7: Use as a binding agent in coal briquetting (see Section B.9.2.8)
- Occupational Scenario 8: Use as a binding agent for clay pigeons (see Section B.9.2.9)

In this report for each occupational exposure scenario the general description of the exposure will be followed by measured data (if available), and results from similar substances in comparable exposure scenarios if necessary. The methods of estimation for inhalation exposure will be assessed using expert judgement and a choice for the best applicable estimates will be made. Dermal exposure will be described and assessed using measured data (if available) and by means of EASE where applicable.

The following parameters of exposure are assessed for each (sub)scenario:

- *full shift reasonable worst-case inhalation exposure level:* the inhalation exposure considered representative for a high percentile (90 to 95 percentile) of the distribution of full shift exposure levels;
- *full shift typical inhalation exposure level:* the inhalation exposure level considered representative for a median percentile (50 percentile) of the distribution of full shift exposure levels;
- *short-term inhalation exposure level:* the inhalation exposure level considered representative for a high percentile (90 to 95 percentile) of the distribution of short term exposure levels; short term exposure for this purpose is considered to be exposure for up to one hour, with typical duration of approximately 15 minutes;
- *dermal exposure level:* the dermal exposure level considered representative for a high percentile (90 to 95%) of the full shift dermal exposure levels.

B.9.2.2 Occupational Scenario 1: Production of CTPHT in coal tar distillation plants

This section covers all activities relevant to exposure to CTPHT in coal tar distillation plants. During distillation coal tar is boiled between 100 °C and over 315 °C resulting in coal tar oils and coal tar pitch. After distillation the pitch is pumped into storage tanks and maintained at 200 °C. Some pitch is delivered from other works via tanker or exported as a molten liquid to either road tankers or ships.

To solidify the pitch into "pencils" the molten pitch is pumped to the pencilling plant where it is extruded into a pie containing water at 40 °C. Using enclosed conveyor belts the pencils are transported to the pitch storage warehouse. To transport the pencils to a ship a lorry is loaded with the pencils using a mechanical shovel or the pencils are transported via another conveyor belt (site 4).

Based on the information provided by industry occupational groups are derived and presented in Table B.9.2.

The tasks presented in Table B.9.2 cover the work on a day-to-day basis. At one site four times a year all parts of the production equipment are cleaned. These tasks are not included in the risk assessment due to their occasional nature.

Table B.9.2. Occupational groups at coal tar distillation plants.

Group	Tasks leading to exposure	Approximate working hours	% time spent on tasks (site 4)	Number of workers [a]	Technical control measures
Operators	Process and plant control, general plant vents, sampling	8-12	10-20%	6-45	Closed transfer system with air pollutant burning, closed transfer in pipes, scrubbing systems
Cleaners	Cleaning equipment and spillages	8	5-25%	5-15 (50) [b]	Washing pipes with solvents before cleaning, LEV [c]
Drivers	Loading and unloading trucks, warehouses and tankers	8	10%-15%	4-8	Pumps for transfer of liquid pitch, pressure cabins, LEV [c]
Quality control analysts	Analysis	8	-	3-10	General exhaust ventilation, LEV [c]

[a] Number of workers per occupational group; [b] Up to 50 workers during plant shutdown (site 10); [c] LEV: Local Exhaust Ventilation.

The coal tar pitch contains a mixture of PAHs and high-molecular aromatic substances. It is used as a viscous liquid of high temperature (> 200 °C) or as a granulated dusty powder. One site presented a percentage of benzo(a)pyrene in CTPHT with a value of 1.15%. Another site presented a benzo(a)pyrene content between 0.1 and 1.5%. Other sites measured a range of PAH of which the

content in CTPHT is below 5%. According to one site the sum of EPA PAHs in coal tar is about 30% whereas the content of EPA PAHs in pitches ranges usually between 5 and 20%.

All-round technical control measures used are general and local exhaust ventilation and scrubbing systems. Other measures are closed transfer systems with vapour return system, sampling with reduced volume, reduced dead storage capacities, closed transfer in pipes, the use of pumps for transfer of liquid pitch and pressurized cabins for solid pitch and air pollutant burning. One site (site 4) indicated that solid pitch is sprayed with a liquid containing a dust inhibiting agent.

All sites indicate that personal protective equipment (PPE) is used. Equipment used are: protective overalls, full face mask with gas and dust filter, respirator with dust filter, face shield, leather and rubber gloves (according to EN 420) and boots. Other PPE used are helmet and light eye protection (site 4 only). One site (site 4) provided more detailed information on PPE used based on the physical form of the pitch. The latter site also indicated that UV sunscreen is provided to minimize skin irritation caused by contact with pitch dust. The information on PPE of site 4 is presented in the following sections.

General control measures for pitch

Normal maintenance activities are controlled by the Permit To Work system containing a risk assessment which defines the residual hazards and PPE appropriate to the task. The purpose of the PTW is to decontaminate the plant before maintenance work and to minimise exposure using the correct safety measures. Depending on the exposure potential full or half face masks with P3 particle filters or combined organic vapour and particulate filters or compressed air breathing apparatus is used. Operators follow operating procedures and wear Respiratory Protective Equipment (RPE) as required and when appropriate to the task.

Control measures in case of liquid (molten) pitch

During normal plant operations the liquid pitch is contained within the process equipment. However, during maintenance (break-ins) and some process operations like draining circulation pumps inhalation exposure may occur. Inhalation exposure may also occur when a tanker hose is disconnected after off loading and some residual material is drained into a slops tank. RPE and full face visor is required if a tanker has to be “dipped” during loading of a pitch tanker. If entry into an enclosed pitch vessel is unavoidable the Permit To Work and Entry Permit procedures become effective. The entry permit involves a permit issuer to perform a risk assessment. The RPE used for this kind of vessel entries is compressed air breathing apparatus. If pitch circulation pumps are drained by operators the area is restricted and appropriate RPE is required when the operators are in close proximity.

Control measures in case of solid pencil pitch and pitch dust.

During normal plant operations the pencil pitch is contained within the process equipment. The conveyors are designed to shed dust which is collected in a hopper at the bottom of the inclined conveyor. From the inclined conveyor the pencil pitch is transported to the enclosed pitch storage shed. Operators are occasionally entering the pitch shed and are exposed to pitch dust. The pencil pitch is loaded on a lorry for export using an earthmover. When pencil pitch is exported by ship anyone on the wharf or operators in the control cabin can be exposed to pitch dust when the conveying system is used to load the ship.

Pencil pitch that is spilled from the side walls is cleared up by cleaners wearing (dust) overalls. Drivers use sealed shovels to load the lorries with pencil pitch. To prevent exposure the air in the cabin of the shovel is controlled using a filtered air conditioning system. During loading the ventilation in the cabin is switched off.

Inhalation Exposure

Measured inhalation exposure data for manufacturing

Industry provided data on benzo(a)pyrene exposure in Coal Tar Distillation plants. One site presented benzo(a)pyrene estimates based on pitch dust measurements where benzo(a)pyrene content was assumed to be 0.7 %. These data have not been taken into account as these are not benzo(a)pyrene concentration measurements. All data relevant are summarized in Table B.9.3.

Table B.9.3. Inhalation exposure concentrations of benzo(a)pyrene in coal tar distillation plants.

Site No / reference	Year of measurement	Occupation or site	Number of measurements	Duration of measurements	Range of Results (µg/m³)	Arithmic mean (µg/m³)	St. dev. (µg/m³)	Median (µg/m³)	90th percentile (µg/m³)
1	2000	Distillation plant; processing of liquid pitch; tar processing	15	7.5 hrs	< LOD-12.30 a)	0.11 4)		0.11 b)	0.17 b)
		Distillation plant; handling of solid pitch	3	7.5 hrs	1.33-3.90	2.61		2.61	3.64
X c)	1999-2001	Tar-distillation plant	18	8 hrs				0.14	4.74
7		Production Operator	1	8 hrs	1.367	1.367			
		Filling Operators	1	8 hrs	0.135	0.135			
		Quality people	1	8 hrs	0.125	0.125			
		Safety people	1	8 hrs	0.011	0.011			
9		Driver	10 d)	5 hrs	0.1-0.5				
		QC analyst	6 d)	5 hrs	0.12-0.34				
10		Operator	33	6-8 hrs	0.001-0.50	0.06	0.1	0.02	0.2
		Driver	10	6-8 hrs	0.0-0.59	0.11	0.21	0	0.43
		Cleaner	20	6-8 hrs	0.01-0.35	0.1	0.17	0.02	0.3
		QC Analyst	15	6-8 hrs	0.02-0.16	0.07	0.05	0.05	0.14

a) The company stated that the extreme upper value was probably an outlier (see text) ; b) Several values were below the limit of detection, but the limit of detection was not presented. Therefore, these parameters were calculated using half of the lowest detected value for calculation; c) From Preuss *et al.* (2003). The data reported for site 1 and Preuss *et al.* (2003) are actually the same data set. Several parameters for site 1 were estimated from the original data by the rapporteur; d) Assuming 2 measurements per company.

Three sites provided data where workers were exposed to benzo(a)pyrene between 0 and 12.279 µg/m^3. According to additional comments of industry on the highest value, the worker's exposure profile contrasted significantly with the other profiles. Also the excretion of hydroxyphenanthrenes and hydroxypyrene into the urine, which was measured in parallel, did not indicate that the workers' exposure to pitch volatiles was significantly higher than the other co-workers' exposure. Therefore, this value does not appear to reflect the real exposure situation. Without this value, the upper benzo(a)pyrene value would be 3.9 µg/m^3 (Industry, 2006; CCSG, 2006b). Preuss, *et al.* (2003) presented part of the same data. A new analysis of the data from company 1 shows that the higher exposure levels are the result of handling solid pitch, while tar processing and liquid pitch handling leads to substantially lower values (CCSG, 2006b).

As explained in Section B.9.2.1 the concentration of benzo(a)pyrene is used as an indicator of exposure to CTPHT. Although two sites also provided data on other PAHs and another site on total inhalable and respirable dust only data on benzo(a)pyrene concentrations are summarized in Table

B.9.3. Two sites also provided data on hydroxyl-phenanthrene in urine and one of them on 1-hydroxy-pyrene in urine. However this assessment involves inhalatory and dermal exposure, only.

Inhalation exposure conclusions

The reliability of the measured data precludes the necessity of modelling exposure data. Furthermore, the complexity of the exposures in this scenario-vapour, particulate and gaseous forms of the contaminant-adds even greater inaccuracy than normal to the estimates derived via EASE modelling. The measured data are considered to be representative for both the substance and the processes assessed. For the majority of exposure data sources it was possible to establish pattern of use, pattern of control and relevant process parameters. It could be ascertained that the majority of data were collected following good occupational hygiene practice using standardised procedures with respect to sampling strategy and measurement methods (see EC, 2003b). Also, the measured data are considered as representative for Europe.

All sites reported by industry and partly Preuss *et al.* (2003) have considerable overlap in the exposure data. Site 1 performed their measurements in the year 2000 and includes handling of solid pitch, that clearly leads to the highest values. Site 9 and 10 omitted to indicate the year of measurement. According to company 1, the value of 12.30 µg/m^3 for handling of liquid pitch and tar processing was not the result of the normal exposure situation and should be considered to be an outlier. Based on the data from company 1, in comparison with the other data, it is assumed that only for company 1 handling of solid pitch is included in the measurements. This is apparently a high exposure task that does not occur very often. Two sub-scenarios will be used for this scenario: handling of liquid pitch and tar processing on the one hand and handling of solid pitch on the other hand.

The reasonable worst case values are estimated from the 90th percentiles that could be calculated, disregarding the calculations for combined data of company 1 from Preuss *et al.* (2003). The result for tar processing and handling of liquid pitch is taken slightly lower than the highest 90th percentile from company 10. The result for handling of solid pitch is taken from company 1:

- Tar processing and handling of liquid pitch 0.40 µg benzo(a)pyrene/m^3
- Handling of solid pitch 3.60 µg benzo(a)pyrene/m^3

Reasonable worst case (RWC) short-term exposure levels are expected to be up to the higher full-shift exposure levels or may be taken as twice that obtained for RWC full-shift. All data provided by industry present full-shift exposure data only. Using a similar approach as for long-term RWC and taking twice the long-term RWC as a short-term estimate the exposure to benzo(a)pyrene as an indicator for CTPHT exposure is:

- Tar processing and handling of liquid pitch 0.80 µg benzo(a)pyrene/m^3
- Handling of solid pitch 7.20 µg benzo(a)pyrene/m^3

Typical exposure levels to benzo(a)pyrene by workers are based on the available medians. To determine the typical exposure levels a similar approach as for determining RWC levels is used. Estimates are limited to site 1 and site 10 only as site 9 did not provide medians. The typical full-shift exposure level is determined at:

- Tar processing and handling of liquid pitch 0.1 µg benzo(a)pyrene/m^3
- Handling of solid pitch 2.6 µg benzo(a)pyrene/m^3

Dermal exposure

In a study by Ariese *et al.* (1994), (as quoted in Gundel *et al.*, 2000), to investigate occupational exposure to PAHs, it was found that despite the use of protective breathing hoods ventilated with air, there was considerable uptake of CTPV as indicated by biological monitoring. They concluded that the primary uptake of PAH was via the skin. Several researchers assessing PAH exposures in

similar working environments have supported this view (Van Rooij *et al,* 1992; Boogaard & Van Sittert, 1994; Malkin *et al*, 1996; Bentsen-Farmen *et al,* 1999). Van Rooij *et al.* (1993) concluded that the dermal uptake of benzo(a)pyrene was about 51% of the total absorbed amount (geometric mean range 8%-92%, n=12).

Measured dermal exposure data in coal tar distillery plants were not available.

Modelled dermal exposure data for production of CTPHT in coal tar distillation plants

Due to the high temperatures in which tar distillery labourers work it is not expected that there will be direct contact with the CTPHT and exposures will most likely be due to deposition onto the skin and indirectly through contact with contaminated work surfaces. Information regarding dermal exposure to CTPHT is scarce. Using EASE and assuming no direct handling the estimated dermal exposure to CTPHT is negligible.

New information on the measured inhalation exposure data (CCSG, 2006b) has shown that some handling of solid pitch occurs occasionally. This may lead to dermal exposure to dust, including benzo(a)pyrene. EASE is used to model dermal exposure while handling solid pitch, using the following assumptions: wide dispersive use, direct handling, incidental contact level. This gives an exposure concentration to the paste containing the CTP binder of 0.1-1 mg/cm^2/day. Assuming that half of both hands are exposed (area = 420 cm^2) and taking the upper end of the range the exposure will be 420 mg/day. The CTPHT content in the paste is assumed to be the same in both Søderberg potrooms and in the anode paste plant. With a 13% content of CTPHT in the paste and a 1% benzo(a)pyrene content in CTPHT, the exposure to benzo(a)pyrene will be 0.5 mg/day (=420×0.13×0.01).

Conclusions for production of CTPHT in coal tar distillation plants

The following exposure levels will be used for further risk assessment for manufacturing.

Inhalation exposure, reasonable worst case, full-shift:

- Tar processing and handling of liquid pitch 0.4 µg benzo(a)pyrene/m^3
- Handling of solid pitch 3.6 µg benzo(a)pyrene/m^3

Inhalation exposure, reasonable worst case, short-term:

- Tar processing and handling of liquid pitch 0.8 µg benzo(a)pyrene/m^3
- Handling of solid pitch 7.2 µg benzo(a)pyrene/m^3

Inhalation exposure, typical, full-shift:

- Tar processing and handling of liquid pitch 0.1 µg benzo(a)pyrene/m^3
- Handling of solid pitch 2.6 µg benzo(a)pyrene/m^3

Dermal exposure, reasonable worst case, daily dose:

- Tar processing and handling of liquid pitch negligible
- Handling of solid pitch 0.5 mg benzo(a)pyrene/day

B.9.2.3 Occupational Scenario 2: Use as a binding agent for electrodes

B.9.2.3.1 Sub-scenario (i): Use as a binding agent for electrodes in the aluminium industry

Aluminium is manufactured in an electrolytic process at a temperature of about 970 °C. There are two types of anodes used in this process: the Søderberg anode and the prebaked anode. The latter is preferred because of lower emissions (Sim & Benke, 2003). In the Søderberg process the green anode is baked *in situ* in the pot during electrolysis. In the pre-bake process anodes are baked before electrolytic use.

Paste plant

As horizontal stud Søderberg anodes are no longer in practice in Europe only the vertical stud Søderberg technique will be discussed. Anode briquettes are used in the aluminium industry in the Søderberg process. The anode briquette (paste) is manufactured at a separate paste plant.

In the paste plant, ground petroleum coke is blended with a binder of liquid coal tar pitch to form a semi-solid paste, which is then pressed in the forming section into a green anode. If the anode is used in the pre-bake process, cleaned spent anode butts may be added before blending. This production process of the anode is largely automated and run from control rooms; the heating and mixing takes place in an enclosed system with suction hoods at transfer points. At these transfer points workers may be exposed to coke particles or paste. The paste is added directly to the anode casings and baked in the Søderberg reduction cell.

Pre-bake Anode Plant

Pre-bake anodes are used to produce aluminium in the pre-bake process. The pre-bake anodes are manufactured in a separate pre-bake plant.

The starting material for the pre-bake anode is green anode paste which might have been mixed with cleaned spent anode butts. In the oven area, the green anodes are surrounded by packing cokes and baked in ovens for several days at a temperature of approximately 1400 °C. The volatiles and particulates are either kept in the ovens, through under pressure compared to outside the ovens, where they are burned as fuel, the remainder being removed and passed through a filter system. After baking, the carbon anode blocks are fitted onto rods in another section. In the furnace and the anode rodding section, exposure to PAHs is expected to be less than in the paste plant (Petry *et al*., 1996a).

Aluminium Production

During electrolysis, the anode for the Søderberg plant, is baked in its lower, hotter part, and finally consumed by electrolytic oxidation to carbon oxides. The green anode paste is continuously replenished at the top of the cell. In case of the pre-bake process, the anodes are replaced at regular intervals as they are consumed during the electrolytic process. The left-over anode butts are recycled at the anode plant.

As the electrolytic process to produce aluminium is in principle the same for the Søderberg technique and the pre-bake technique many tasks and jobs are similar. The pre-bake plants are more automated. Due to the high demand for energy and the higher emissions rates Søderberg plants are diminishing and may be modernised or replaced by pre-bake anode plants. An estimation of industry is that within a few years more than 90% of EU production will be pre-bake or updated Søderberg plants (Industry, 2006).

Exposure concentrations are dependent on the technology used and the age of the plant.

In many different tasks in an aluminium smelter such as stud-pulling, rack-raising, mounting of flaints and adding of anode paste, the exposure can be considerable. Nowadays the use of cold ramming paste is free of pitch and no exposure is expected from the preparations (ramming) of cathodes. The cathode is converted to carbon in the potroom by putting it into the circuit, resulting in the emission of some volatiles. There is also potential for exposure to particulate bound PAH during removal of the cathode crusts using pneumatic hammers in preparation for the re-lining of the cathode. When pre-baked carbon blocks are used, they are joined together with a warm, liquid pitch paste, resulting in some exposure of workers to pitch volatiles (IARC, 1984). According to industry comments, the information about ramming pastes is largely outdated and most plants have changed to pitch free ramming pastes.

Typical benzo(a)pyrene levels in Søderberg potrooms are expected within a higher range in the Søderberg electrolysis department and in the lower range in the other main type of electrolysis, prebake, and in other departments such as the paste and carbon plant in which the anodes are manufactured (IARC (1984); Armstrong *et al.* (1986) as cited in Boffeta *et al.* (1997).

Potmen, rod raisers and stud pullers are subjected to exposures whilst performing their duties in this area of the plant. Some of these tasks include breaking of the crust with an air hammer (crust breaker), siphoning the molten metal, and removal of the studs embedded in the anode (rod raisers and stud pullers). If the Søderberg pot has vertically placed studs embedded in the anode, fresh anode paste is added from above. To keep the electrode running continuously workers (stud-setters) must stand on a catwalk on top of the anode to renew the anode paste and to make the necessary adjustments to the studs. From this position there is great potential for exposure to pitch volatiles. According to industry workers no-longer have to stand on a catwalk on top of the anode (Industry, 2006). Crane cabins and enclosed vehicles are used for these operations.

CTPV are released continuously from baking anodes in the Søderberg process. During potroom maintenance the maintenance employees are potentially exposed to CTPHT and CTPV and they may be exposed at higher concentrations than daily potroom workers. Employees who clean ducts may be exposed to pitch volatiles. Other relevant personnel are crane operators who transport large crucibles containing the siphoned aluminium to the casting area.

Depending on the technical process and age of the plant different mechanical control measures may be in use. For example, during anode changing a modern pre-bake plant may use overhead cranes with an air-conditioned and filtered cabin. However, if the plant is older the anode changing may be done by two or three setters. Other mechanical control measures may be local exhaust ventilation in potrooms or in case of Søderberg potrooms a skirt hood or complete hood over the cell. Secondary emission control may be in place as well like natural ventilation. Crane drivers in modern potrooms work in enclosed and air filtered cabins. According to industry, all Søderberg potrooms in operation today have either air conditioned and filtered crane cabins or vehicles, from which operations like stud pulling, rack raising, anode briquette filling and metal tapping are performed (Industry, 2005). Based on literature data and information provided by industry potroom workers are required to use respiratory protection, overalls, gloves, safety boots and gaiters, helmet and eye protection. Respirators used, as indicated by industry, are P3 masks. In sections where liquid pitch is used or exposure to high temperatures is expected double layer clothing is required. To reduce dermal exposure to pitch underwear may be given out to the workers.

Inhalation exposure

Measured inhalation exposure data for use as a binding agent for electrodes in the aluminium industry

The range of results for exposures in the aluminium production is wide (<0.02-36 µg/m^3). Table B.9.4 and Table B.9.5 provide (older) data for Søderberg plants and Anode plants respectively, mostly from literature. Table B.9.6 presents recent industry data (EAA, 2006). It is possible to define three distinct exposure groups within this industry-Søderberg potroom workers, anode plant workers and paste plant workers, with the Søderberg potroom workers, according older data sets, falling into a higher exposure range. While there is some overlap between the two groups identified, the upper end of the ranges for the Søderberg potroom workers were generally higher-three datasets presented had an upper range above 20 µg/m^3. For the anode plant workers the upper end of the range of exposure was generally less than 5 µg/m^3. This was also true of data from industry collected during the period 1983-1996. The 90th percentile of the log-normally distributed data calculated for one dataset representing exposures in the Søderberg potroom was 8.29 µg/m^3. A 90th percentile was presented by industry for exposures in anode plants. This was within the range of 0.50-1.50 µg/m^3. Over the period of 2000-2003 data from three Søderberg plants were presented by industry (Table B.9.3). The overall average range was 0.12-16.3 µg/m^3. One of the plants (plant A)

was recently modernised and the measurements done in 2003 represent the situation in modernised plants. The data from plant B and C are from before the modernisation that is presently ongoing. It is expected that this modernisation leads to levels comparable with those of plant A. All Søderberg plants in Europe are presently in a process of modernisation or are expected to be closed within the next two years (personal communication Dr. Nordheim, European Aluminium Association).

Differences in exposures according to work task and work area in a carbon anode plant can be seen in a study by Petry *et al.* (1996ab). They determined the time weighted averages for 6 workers monitored on 5 consecutive shifts. Two workers, one who worked in the anode forming section and the other in the paste plant, showed the highest exposure to benzo(a)pyrene. Their exposure range was 0.89 to 4.88 µg benzo(a)pyrene/m^3. Crane operators had the lowest relative exposure with benzo(a)pyrene exposure range of 0.16-2.03 µg/m^3. One worker with multiple tasks-overseeing, maintenance and operations-had the greatest variation in exposure with a variation or 0.27-3.66 µg benzo(a)pyrene/m^3; arithmetic mean 1.38 µg benzo(a)pyrene/m^3. The overall exposure range for this group of 5 workers was 0.17-4.88 µg/m^3 with an arithmetic mean of 1.16 µg/m^3. In more recent data sets the differences between workers and locations are not very clear and they are therefore not specifically presented in Table B.9.6. Only the spikes operators in the companies with not yet modified cabs show higher values. The use of modern cabs (air conditioned and filtered air) should lead to substantially lower values.

Inhalation exposure conclusions for use as a binding agent for electrodes in the aluminium industry

The reliability of the measured data precludes the necessity of modelling exposure data. Furthermore, the available models are not applicable to this occupational scenario. The measured data is representative for both the substance and the processes assessed. For the majority of exposure-data sources it was possible to establish pattern of use, pattern of control and other relevant process parameters. The data was collected following good occupational hygiene practice using standardised procedures with respect to sampling strategy and measurement methods (see TGD, 2003b). Also, the measured data are representative for Europe. The industry data are the most recent data.

From the data available there appear to be two distinct exposure groups as discussed above. For Søderberg potroom workers, it was possible to calculate the 90^{th} or 95^{th} percentile for one dataset (Becker *et al.*, 1999): 8.3 µg/m^3 (range 0.02-23.5) and from the industry data for plant B and C (between 0.8 and 16.3 µg/m^3), where it is noted that these data are for situations that are being improved. Modern Søderberg plants, according to industry, should have values below 1 µg/m^3 as seen for plant A in Table B.9.6. The exposure range over all the datasets of exposure for potroom workers was 0.02-36 µg benzo(a)pyrene/m^3. Because not all plants are modernised, a conservative estimate for the reasonable worst-case full-shift inhalation exposure to benzo(a)pyrene in Søderberg potrooms in the aluminium production industry has been estimated at 8 µg/m^3, being the approximate 90^{th} percentile of the data from (Becker *et al.*, 1999) and the approximate arithmetic mean for the spikes operators from the industry data of not modernised facilities. For modern plants, the reasonable worst case exposure values are based on the 90^{th} percentile of the 2003 data from plant A (taken after the first modernisation was finished): 0.35 µg/m^3 (see Table B.9.6).

For anode (bake) plant workers and paste plant workers both the variations in exposures and the upper range of exposure is considerably less, with the exception of the data presented by Van Schooten *et al.* (1995) (Table B.9.5). However the geometric means of these data were low (0.35 and 1.51 µg benzo(a)pyrene/m^3) and the year of measurements was relatively old, some 13 years back. The data sent by industry (EAA, 2005) are considered sufficient for estimating reasonable worst case exposure levels for anode bake plants and paste plants together. The (approximated) 95^{th} percentiles are between 0.14 and 1.83 µg/m^3. Based on the combined data the reasonable worst case full shift inhalation exposure levels are estimated to be 1 µg/m^3.

Table B.9.4. Inhalation exposure concentrations of benzo(a)pyrene in aluminium production works (Søderberg Potroom).

Reference	Year of measure-ments	Occupation or site	Number of measure-ments	Duration of measure-ments	Range of results (µg/m³)	Average (µg/m³)			St. dev.	Geometric st. dev.	90th percentile LgN [a]
						Arithmic mean	Geometric mean	Median			
Alexandrie *et al.* (2000)	1995	Vertical stud Søderberg potroom	93	full-shift	0.02-23.5			0.97			
Levin *et al.* (1995)	1990	Pot-anode	4	6 hours	2.3-36			2.8			
		Cathode	3	6 hours	1.9-2.1						
		Crane	2	6 hours	2.8-4.4						
Tremblay *et al.* (1995)	1985-1989	Potman (Søderberg)		TWA [b]		1.04					
Becker *et al.* (1999)		Aluminium melting facility	16	8	0.4-23.5	4.2[f]	2.7[f]	4.2		2.4	8.29

[a] LgN 90%: 90th percentile of the lognormal distribution calculated from presented data; [b] TWA: Time weighted average.

Table B.9.5. Inhalation exposure concentrations of benzo(a)pyrene in alumnium production works (Anode plants).

Reference	Year of measure-ments	Occupation or site	Number of measure-ments	Duration of measure-ments	Range of results (µg/m³)	Average(µg/m³)			St. dev.	Geometric st. dev.	90th percentile LgN [a]
						Arithmic mean	Geometric mean	Median			
Petry *et al.* (1996a) [b]		Paste Plant	5	8	1.37-4.43	2.6	2.39				
	[c]	Anode Forming Section	10	8	0.89-4.88	2.44	2.14				
		Crane Operator (coke store)	5	8	0.17-2.03	0.99	0.72				
		Crane Operator (anode store)	5	8	0.16-0.79	0.39	0.33				
		Overseer, maintenance, operating	5	8	0.27-3.66	1.38	0.92				
Petry *et al.* (1996a) [b]		Carbon anode production	30	8 [1)]	0.17-4.88		1.16				
Industry A	1983-1996	Handling green paste			2.0-5.0						
		Handling green paste			0.5-2.0						0.5-1.5
		Handling green paste			<0.5						

Reference	Year of measure-ments	Occupation or site	Number of measure-ments	Duration of measure-ments	Range of results (µg/m³)	Average(µg/m³)			St. dev.	Geometric st. dev.	90th percentile LgN [a]
						Arithmic mean	Geometric mean	Median			
Van Schooten *et al.* (1995)	1989	Pre-bake anode process									
		Bake oven	22	Full-shift	0.1-14.4		0.35				
		Anode factory	40	Full-shift	0.1-11.6		1.51				
		Pot-relining	41	Full-shift	<0.02-9		1.05				
		Electrolysis	23	Full-shift	<0.02-0.2		0.03				
		Foundry	16	Full-shift	<0.02-0.06		0.02				
Bentsen-Farmen *et al.* (1999)		Electrode Paste Plant	17	Full-shift?		0.30		0.2	0.3		
Van Rooij *et al.* (1992)	1989	Electrode production department:									
		Paste Plant	8	8				1.3 [d] (1.2-3.0)			
		Bake Oven	5	8				0.3 [d] (0.1-0.4)			
		Pot relining	7	8				1.2 [d] (1.0-2.2)			

[a] 90th percentile LgN: 90th percentile of the lognormal distribution of the measured data supplied by Industry; [b] Petry et al. (1996ab). Time weighted averages for 6 carbon anode workers monitored on 5 consecutive shifts. In Petry (1996b), a summary of the exposure data presented in Petry (1996a) was given and is reproduced here; [c] The year the measurements were taken was not always indicated in the research article. In these cases it was assumed that the relevant year was that of the year of the publication, however, it should be noted that this is not always the case and the difference between the year of measurements and year of publication could sometimes be quite large. This has relevance as a factor that adds to the uncertainty of the estimates derived using these data; [d] Each worker was monitored during 5 consecutive shifts. The median given is the median value of 5 days. The range of medians is given in parentheses.

Table B.9.6. Inhalation exposure concentration ranges of benzo(a)pyrene in aluminium production works.

Company [a]	Location/year	Number of measurements	Arithmetic Mean (µg/m³)	Geometric Mean (µg/m³)	Range (µg/m³)	90th percentile (µg/m³)
Søderberg plants						
A [b]	Pot tender potroom 3	61	0.36	0.34		0.73
Modernised plant	Data 2003 (after first modernisation)	61	0.20	0.17	0.04-0.77	0.35
B and C [c]	Pot line; Potroom operator	7	1.13	0.93	0.7-2.10	2.07
Not modernised plants	Pot line; shift foreman / supervisor	6	0.63	0.47	<0.22-1.40	1.26
	Pot line; tapping operator	6	1.06	0.97	0.55-1.70	1.63
	Pot line; spikes operator (before modification cabs)	6	7.99	6.44	3.40-17.0	16.3
	Pot line; spikes operator (partial modification of cabs)	1	4.2			
	Pot line; Metal driver	6	0.74	0.67	0.31-1.20	1.16
	Pot line; Measurement operator	6	0.96	0.87	0.53-1.60	1.55
	Pot line; Crane operator	6	0.74	0.70	0.50-1.40	1.22
	Pot line; Maintenance foreman	6	0.44	0.34	<0.14-0.88	0.79
Anode bake plants						
D, E and H [d]	Date from three companies combined	30	0.16	0.04	<0.10-1.37	0.47
F [e]	Data from 1999; before upgrading	22	0.22±0.14 [f]		<1.35	0.45
Paste plants						
A, D and G [g]	Date from three companies combined/ 2000-2001	24	0.07-0.14	0.06 [h]	0.01-0.28	
	Date from three companies combined/ 2003	23	0.08	0.06	0.14-0.28	0.15
F	Data from 1999; before upgrading	12	0.68±0.7 [f]		-2.2	1.83

[a] Coding by the rapporteur, industry data, assuming full shift measurement duration; [b] Measurements in 2000/2001 before modernisation. In 2003 after modernisation (further modernisation ongoing). Some values estimated from the raw data, while 90th percentile of 2000/2001 data was estimated from the data assuming a similar GSD as in the 2003 data; [c] Approximation from original data by rapporteur; [d] Data from three companies that for 2003 were presented combined. Parameters partly estimated from original data. Industry reported that the highest value should be considered an outlier. However, no explanation was given why this was supposed to be an outlier. The values for this data set without the outlier would be: n = 30; arithmetic mean = 0.12; geometric mean = 0.04; maximum = 0.61; 90th percentile = 0.38; [e] According to industry, the values from this company are outdated; 90th percentile estimated from the data by the rapporteur; [f] Value ± standard deviation; [g] Data from three companies that for 2003 were presented combined. Parameters partly estimated from original data; [h] Value for company D.

Reasonable worst case (RWC) short-term exposure levels are expected to be up to the higher full-shift exposure levels from the industry data in Table B.9.6. The RWA full shift estimates are derived from the 90th percentiles of the data from industry presented in, disregarding the older data from Company F. Although there are no clear reasons presented for the outlier in the data for the anode plants, it is assumed that this is indeed a relatively high value with too much influence on the estimate if it is fully taken into account. For the anode plants the RWC for full shift exposure is estimated as 0.40 µg/m^3 and for the paste plants as 0.15 µg/m^3.

The arithmetic means of exposure from the industry data in Table B.9.6 are used as the basis for deriving the typical, full-shift exposure estimates to benzo(a)pyrene in Søderberg potrooms and anode and paste plants in the aluminium production industry. For not modernised Søderberg potrooms the average arithmetic mean is determined at approximately 1 µg/m^3 (giving limited weight to the high values for spikes operators, before modernisation). For modernised Søderberg potrooms the average arithmetic mean is determined at approximately 0.20 µg/m^3 (based on the arithmetic mean of the 2003 data of plant A). For anode plants the typical value is based on the data in Table B.9.6 excluding the data from company F: 0.15 µg/m^3 and for paste plants the estimate of typical exposure is also derived from the data in Table B.9.6, again excluding the data from company F: 0.08 µg/m^3.

Dermal exposure

According to a publication of Van Rooij *et al.* (1992) the main emission sources of PAHs are from mixing, shaping and baking of the anode in which CTPHT is an ingredient. In this paper it was concluded that the primary uptake of PAH was via the skin. Based on information from industry (2005), only liquid pitch is used nowadays and this is used in closed systems without direct contact. Apart from this specific comment, there is no supportive information to show whether or not solid pitch is still in use. Workers who handle liquid pitch wear double clothing. It is also indicated that underwear is given out to workers with instructions to change these regularly. Working clothes are not allowed to take home.

The production of anodes involves CTPHT dust and therefore wide dispersive use assumed. As hot liquid pitch is added to make the anode paste the pitch, direct handling is assumed with incidental contact level.

Table B.9.7 shows the dermal exposure concentrations measured by Van Rooij *et al.* (1992) in an anode manufacturing plant.

Measured dermal exposure data for use as a binding agent for electrodes in the aluminium industry

Van Rooij *et al.* (1992) measured dermal exposure to benzo(a)pyrene in different work areas of aluminium production plant using solid pitch. The different work areas of interest are mixing and shaping of the anode and baking of the anode. The data presented by Van Rooij were median values and hence cannot be used to estimate the reasonable full-shift worst case exposure to benzo(a)pyrene. These data are used to estimate a typical, full-shift, dermal exposure.

Table B.9.7 gives approximate average exposures derived from the data of Van Rooij *et al.* (1992).

The exposure for the whole body (except hands) is estimated from the values obtained for the jaw/neck and groin area (Table B.9.7). The overall whole body exposure level is taken as the average of the median dermal exposure levels for all work areas: (4+3+4)/3. This gives a value of approximately 4 ng/cm^2. Similarly, the exposure values obtained for the wrists are used to estimate average hand exposure: (15.2+9.6+18.5)/3 = 14 ng/cm^2.

For whole body exposure excluding hands the exposed area is 19,160 cm^2 (20,000-840) and with an exposure level of 4 ng/cm^2, the exposure will be 76,640 ng/day (0.077 mg/day). For hand exposure,

assuming that both hands are exposed (area 840 cm^2) and using an exposure level of 14 ng/cm^2, the exposure will be 11,760 ng/day (0.012 mg/day).

Table B.9.7. Estimated dermal exposure to benzo(a)pyrene in aluminium production plants.

Occupation or site	Number of measurements	Duration of measurements	Range of medians (wrists) [a] (ng/cm^2)	Median (wrists) [b] (ng/cm^2)	Median (jaw/neck & groin) [c] (ng/cm^2)
Paste plant	8	Full-shift	11.5-28.4	15.2	4
Bake oven	5	Full-shift	7.6-34.2	9.6	3
Pot relining	7	Full-shift	16.9-25.2	18.5	4

Values extrapolated from data presented by Van Rooij *et al.* (1992); [a] Each median value is the median value of five days of measurements. The range of medians presented here are the ranges of medians presented by Van Rooij *et al.*, (1992); [b] The median for wrists represents the median value of each range of medians presented for the wrist; [c] The medians here represent whole body exposure and were derived from median ranges representing exposure to the jaw/neck and groin area.

Measured dermal exposure data in Søderberg potrooms were not available.

Modelled dermal exposure data for use as a binding agent for electrodes in the aluminium industry

EASE is used to model dermal exposure in the Søderberg potrooms and anode paste plants using the following assumptions: wide dispersive use, direct handling, incidental contact level. The assumption here is the use of solid pitch. This gives an exposure concentration to the paste containing the CTP binder of 0.1-1 mg/cm^2/day. Assuming that half of both hands are exposed (area = 420cm^2) and taking the upper end of the range the exposure will be 420 mg/day. The CTPHT content in the paste is assumed to be the same in both Søderberg potrooms and in the Anode paste plant. With a 13% content of CTPHT in the paste and a 1% benzo(a)pyrene content in CTPHT, the exposure to benzo(a)pyrene will be 0.5 mg/day (= 420×0.13×0.01).

When liquid pitch is used, the temperature of the pitch is so high that daily direct dermal contact is not to be assumed. Only accidental contact or exposure through contact with contaminated surfaces is possible. The latter can not be estimated with the EASE model.

There are high uncertainties in the estimates derived using the data of Van Rooij *et al.* (1992). This is mainly due to the extrapolation to derive the average exposure from median values. Hence the values derived from modelled data will be used as the estimate of dermal exposure in anode paste plants. In the absence of measured dermal exposure data in Søderberg potrooms, this dermal estimate is also used for dermal exposures in Søderberg potrooms given that there are expected similarities in paste handling and also in the CTPHT content of the paste.

The estimate of dermal exposure depends to a very large degree on the question whether solid or liquid pitch is used. If only (hot) liquid pitch is used, direct contact will be avoided because of the severe direct effect and dermal exposure will be low.

Conclusions for use as a binding agent for electrodes in the aluminium industry

The following exposure levels will be used for further risk assessment for use as a binding agent for electrodes in the aluminium industry.

Inhalation exposure, reasonable worst case, full-shift:

- Søderberg potrooms; not modernised 8 µg benzo(a)pyrene/m^3
- Søderberg potrooms; modernised 0.35 µg benzo(a)pyrene/m^3
- Anode bake plants 0.40 µg benzo(a)pyrene/m^3
- Paste plants 0.15 µg benzo(a)pyrene/m^3

Inhalation exposure, reasonable worst case, short-term

- Søderberg potrooms; not modernised	17	µg benzo(a)pyrene/m^3
- Søderberg potrooms; modernised	0.75	µg benzo(a)pyrene/m^3
- Anode bake plants	1.40	µg benzo(a)pyrene/m^3
- Paste plants	0.30	µg benzo(a)pyrene/m^3

Inhalation exposure, typical, full-shift

- Søderberg potrooms; not modernised	1	µg benzo(a)pyrene/m^3
- Søderberg potrooms; modernised	0.20	µg benzo(a)pyrene/m^3
- Anode bake plants	0.15	µg benzo(a)pyrene/m^3
- Paste plants	0.08	µg benzo(a)pyrene/m^3

Dermal exposure, reasonable worst case, full-shift

- Søderberg potrooms, only for handling solid pitch	0.5	mg/day benzo(a)pyrene
- Anode bake plants, only for handling solid pitch	0.5	mg/day benzo(a)pyrene
- Paste plants, only for handling solid pitch	0.5	mg/day benzo(a)pyrene

B.9.2.3.2 Sub-scenario (ii):Use as a binding agent in the graphite electrode production and impregnation of electrodes

There are six main steps corresponding to six main work areas identified in electrode paste plants: crushing and mixing, pressure moulding of the crude paste, baking, graphitisation, impregnation and conditioning. The coke is crushed and mixed with hot CTPHT. This crude pasted is then pressure moulded. In the baking area the mixed and pressed crude materials are baked up to 1000 °C over several weeks. The electrodes are then impregnated with liquid pitch. During graphitisation the coke is converted to graphite at 2800 °C within two weeks (Angerer *et al.*, 1997; Buchet *et al.*, 1992). There is potential for inhalation and dermal exposure to CTPHT particulates and vapour during this process.

Inhalation exposure

Measured inhalation exposure data for use as a binding agent in the graphite electrode production and impregnation of electrodes

Table B.9.8 represents measured inhalation exposure data to benzo(a)pyrene for the different work areas in electrode paste plants. The exposure ranges differed considerably among the different datasets. The data of Buchet *et al.* (1992) showed exposures within the wide range of 0.02-72.9 µg benzo(a)pyrene/m^3 while the range of Angerer *et al.* (1997) was 0.003-3.39 µg/m^3.

Angerer *et al.* (1997) reported that the exposures found in the baking and impregnation area was significantly higher than that in the crushing, graphitisation and conditioning area. This is not reflected in the data of Buchet *et al.* (1992) where the higher exposures were observed for the workers involved in mixing and grinding and for electrode impregnation. In both datasets the workers involved in finishing/conditioning and graphitisation had significantly lowered exposure levels. Øvrebø *et al.* (1994) and Petry *et al.* (1996a) reported average exposures in electrode paste plants of 0.8 and 0.08 µg/m^3, respectively, however these average values represent all workers in all different work areas and the more detailed data suggests that this is not a uniformly exposed group. Consequently, this workgroup will be divided into two different workgroups based on their levels of exposure. Group A consists of the higher exposed group: Workers in mixing/grinding, baking and impregnation and maintenance and repair staff. Group B, the lower exposed group, consists of workers involved in finishing/conditioning and graphitisation.

Table B.9.8. Inhalation exposure to benzo(a)pyrene in electrode paste plants.

Reference	Occupation or site	Group [a]	Number of measurements	Duration of measurements (hrs)	Range of results (µg/m³)	Average (µg/m³)		90th percentile
						Arithmetic mean	Geometric mean	
Angerer *et al.* (1997)	Crushing	A	2	≥ 2.5	0.01-0.17	0.09		
	Baking	A	5	≥ 2.5	0.14-3.39	1.15		
	Impregnation	A	3	≥ 2.5	0.47-1.46	1.09		
	Graphitisation	B	4	≥ 2.5	0.007-0.01	0.01		
	Conditioning	B	2	≥ 2.5	0.003-0.02	0.01		
Øvrebø *et al.* (1994)	Electrode production		34	6-8		0.8		
Buchet *et al.* (1992)	Mixing and grinding of coke and CTPHT	A	14	6	0.57-25.1		5.4	
	Baking	A	6	6	0.002-1.88		0.04	
	Electrode impregnation (dipping in liquid tar at 280 °C)	A	8	6	0.83-72.9		6.22	
	Graphitisation	B	25	6	0.002-0.5		0.03	
	Finishing	B	10	6	0.002-0.4		0.03	
	Maintenance and repair staff (mainly electricians and mechanics)	B	17	6	0.002-7.53		0.21	
Petry *et al.* (1996a)	Graphite production		16				0.08	
Preuss *et al.* (2003)	Graphite electrode production		68	8			0.49 [b]	4.08
ECGA (2005) [c]	Mixing	A	70	6-8	0.116-6.4	2.207	0.961	4.98
	Baking	A	53	4-8	0.009-14.1	2.553	0.181	7.44
	Impregnation	A	123	5-8	0.02-2.5	1.343	0.841	2.42
ECGA (2006) [d]	Green department (mixing)	A	39	5-6	0.029-16.391	2.262		11.1
	Impregnation	A	28	5-6	0.211-3.487	0.992		2.9
	Baking	A	32	5-6	0.014-1.652	0.266		0.5

[a] Group A: Workers in mixing/grinding, baking and impregnation and maintenance and repair staff, Group B: Workers involved in finishing/conditioning and graphitisation; [b] Median; [c] Data sets are combined for three companies; [d] Data from several companies.

Most data sets in literature are rather old (about 10 years or more) and rather small. The data sets presented by ECGA (2005, 2006ab) and Preuss *et al.* (2003) are more comprehensive and reflect the present situation. Therefore, these data will be used as the basis for the estimates. The most recent data from ECGA (2006ab) suggest that there are differences between departments in exposure, even in the higher exposed Group A with the highest values for the Green department. However, one value will be used for the group A. Approximately the 90th percentile of the 2005 data for the Green department, the next to highest 90th percentile, will be used for the reasonable worst case full shift exposure level. The highest full shift values will be used as the basis for the reasonable worst case short term exposure level. The arithmetic means will be used as the basis for the typical full shift exposure level.

Inhalation exposure, reasonable worst case, full-shift:

- *Group A* 7.5 µg benzo(a)pyrene/m^3

Inhalation exposure, reasonable worst case, short-term

- *Group A* 16 µg benzo(a)pyrene/m^3

Inhalation exposure, typical, full-shift

- *Group A* 2 µg benzo(a)pyrene/m^3

Modelled dermal exposure data for use as a binding agent in the graphite electrode production and impregnation of electrodes

No measured data for dermal exposure in use as a binding agent in the graphite electrode production and impregnation of electrodes are available. Therefore, EASE is used to model dermal exposure in the electrode paste plants. It is assumed that the task leading to the highest exposure is mixing and crushing. Due to the high temperature of the liquid CTP, direct dermal contact is expected to occur only occasionally, because workers will wear protective gloves to protect them from contact with the hot material. Dermal exposure can occur due to contamination of the surfaces in the area and contact of workers with these surfaces (including the outside of protective gloves). This exposure level cannot be estimated. In accordance with the revised TGD (EC, 2003b), daily dermal exposure due to handling hot material is not estimated.

<u>Conclusions for use as a binding agent in the graphite electrode production and impregnation of electrodes</u>

The following conclusions are drawn for the higher exposed group: workers in mixing/grinding, baking and impregnation and maintenance and repair staff.

- Inhalation exposure, reasonable worst case, full-shift: 7.5 µg benzo(a)pyrene/m^3
- Inhalation exposure, reasonable worst case, short-term 16 µg benzo(a)pyrene/m^3
- Inhalation exposure, typical, full-shift 2 µg benzo(a)pyrene/m^3
- Dermal exposure, reasonable worst case (daily): negligible

Other workers will have lower exposures.

B.9.2.4 Occupational Scenario 3:Use as a binding agent in the asphalt industry

This occupational scenario comprises the two sub-scenarios roofing and road construction. These have been considered under one heading due to the similarity in the mixture of exposures-CTPHT and bitumen. There are indications that these scenarios are no longer relevant. However, until better information has been received the scenarios will remain in this document. Within the following

scenarios bitumen [6] is a source of the exposure markers benzo(a)pyrene and PAHs, hence personal monitoring data presented herein will generally be for exposure to benzo(a)pyrene or PAHs arising from both sources, asphalt and CTPHT. Studies suggest that the contribution from CTPHT is by far the greater. Toraason *et al*, (2001) attempted to determine the contribution to exposure to the benzene soluble fraction of total particulates arising from CTPHT in a group of roofers. Their overall results indicated that full-shift breathing zone measurements for total particulates, benzene-solubles and PAHs were significantly higher for coal tar exposed roofers than for roofers not exposed to coal tar (Table B.9.9). Toraason *et al.* (2001) approximated the contribution from CTPHT as 73% of the total. Burstyn *et al.* (2000) noted that in the road construction industry in Europe using bitumen as the binder, the levels of benzo(a)pyrene were generally low, with older European studies being more likely to find detectable levels. In a tar-free environment, re-paving was associated with benzo(a)pyrene exposure that was less that 20 ng/m^3 (n=14) (Burstyn *et al.*, 2000). Similarly low levels of exposure was obtained in a study by Levin *et al.* (1995): exposure was <0.05 µg/m^3 during paving with a bitumen emulsion without added coal tar.

B.9.2.4.1 Sub-scenario (i): Use as a binding agent in road construction

Workers in the road construction industry include asphalt plant workers, ground construction workers and road paving workers (Burstyn *et al.*, 2000). Together they carry out road paving and recycling/resurfacing activities. These individuals can be exposed to a wide range of potentially hazardous substances, among them CTPV, when CTPHT is used as a binder. For observations in which coal tar was used in the binder, the benzo(a)pyrene content of the coal tar used varied from 1670 to 7900 mg/kg. The amount of coal tar added to the asphalt binder was up to 50% in "tar-bitumen" (Burstyn & Kromhout, 2000a). While the use of CTPHT as a binder in the road construction industry in Western Europe has been discontinued, milling of old road surfaces may still result in exposure to coal tar containing materials (Burstyn & Kromhout, 2000a). Despite the discontinuation of CTPHT in the paving of road surfaces it is thought necessary to present exposure estimates for all areas of road construction-the laying down of a new road alongside that of the removal of an old road and the use of recycled asphalt materials. This is to represent cases in which the directive to discontinue CTPHT usage in road paving works has not yet been fully observed.

Road Paving

Small crews of workers (5-9 individuals) perform road paving. They can usually be separated into the following job titles: paver operator, screedman, rakerman, roller driver, transport truck driver and supervisor. Transport truck drivers deliver hot application mix (140 to 200 °C) from an asphalt plant to the paving site. The application mixture is transferred into a paving machine that applies it to the road surface. The paver operator is seated on top of the paving machine, between the hopper that receives hot mix from the transport truck and the screed that discharges the hot mix onto the surface being paved. The screed man controls the discharge of hot mix from the screed, and is normally located immediately above the freshly spread mix. The raker helps to spread the hot mixture discharged from the screed by using a hand rake or a shovel. Rollers are used to compress the application mixture once it has been applied to the paved surface. Roller drivers, who, in modern Europe, are typically seated in the cabin, operate them. However, older rollers that may still be used are commonly not equipped with cabins (Burstyn *et al.*, 2000).

Recycling/resurfacing

Recycling/resurfacing operations are often combined with road paving. Recycling of asphalt road surfaces started in Western Europe in 1980. The old layer of asphalt is stripped and mixed with new

[6] By bitumen it is meant petroleum binder, which when mixed with inorganic materials, yields asphalt. In North America bitumen is synonymous with asphalt (Burstyn *et al.*, 2000).

asphalt at the asphalt plant or at the paving site and re-applied to the road surface. Heating of the old asphalt with propane burners can facilitate resurfacing (hot re-paving) (Burstyn *et al.*, 2000).

During surface dressing operations, a thin film of binder (140-180 °C) is sprayed onto the road surface and is covered with chippings (chip sealing) and then rolled. The binder is sprayed either from a gang bar fitted at the back of a tank truck or is sprayed with a hand-held lance. The spraying operator, who is working at the back of the truck, controls the width of the gang bar. At spots difficult to reach, the spraying is done manually. In such case, a tube handler may assist the spraying operator. Directly after the spraying, stone chips are spread from a truck onto the surface. The fantail operator controls deposition of stone chips onto the binder. He adjusts the spreading mechanism and helps by spreading manually. One or two raker men control the thickness of the chip layer with a broom. Next, the surface is rolled.

The usage of CTPHT in the binder during road works increases the potential of exposure to PAH fumes and particulate bound PAHs. There is potential for both inhalation and dermal exposure to CTPHT for these workers.

Inhalation exposure

Measured inhalation exposure data for use as a binding agent in road construction

The measured data is limited for this scenario in that it comes primarily from one source, however, it is considered a very reliable analysis of data from a rather extensive database of exposure-The Asphalt Worker Exposure (AWE) database. These measurements were collected primarily during asphalt paving operations in Scandinavia (Norway and Sweden). Their representativeness for the whole of Western Europe is assumed. The inclusion of exposure data collected in the given time period (as far back as 1978) should cover countries within the EU that may presently be using older methods of road construction that are consistent with higher exposures. Such data should represent the worst case. The data comprised repeat measurements collected on consecutive days and it was reported that in most surveys all members of a crew that spent most of their time on-site were monitored. Hence, it is believed to be representative of the most important exposure situations among pavers. The nature of the distribution (normal or lognormal) was not stated. However, as a (approximately) lognormal distribution is very common in occupational exposure, the 90th percentile was calculated assuming the lognormal distribution of the different datasets (Table B.9.9).

Table B.9.9. Inhalation exposure to benzo(a)pyrene during road construction: re-paving, re-cycling and other road construction works.

Reference	Year of measure-ments	Occupation or site	N [a]	Duration of measure-ments (hours)	Range of results (µg/m^3)	Average (µg/m^3)			90th percen tile
						Arithmetic mean (st. dev.)	Geometric mean (st. dev.)	Median (geometric st. dev.)	
Burstyn *et al.*, 2000	1985-1986	Re-paving of coal tar containing material	42	2hr-time weighted average				3.0 (3.1)	
Burstyn *et al.*, 2000	1988-1993	Recycling of tar containing asphalt	36	Full-shift			0.12 (3.3)		0.55
Burstyn & Kromhout (2000a) [b]	1978-1996	Road construction workers	26	Full-shift (assumed)	0.006-4.91	0.55 (1.09)	0.16 (0.005)		1.2

[a] N: number of measurements; [b] Data compiled from the AWE database. Burstyn & Kromhout extracted and analysed 34 observations from this database for which binder content was known. For observations in which coal tar was used in the binder, the benzo(a)pyrene content of the coal tar used varied from 1670 to 7900 mg/kg. The amount of coal tar added to the asphalt binder was up to 50% in "tar-bitumen".

During re-paving with coal tar containing road materials in 1985-1986, the median 2-hour average of benzo(a)pyrene exposure of 3.0 µg/m^3 (n=42, geometric standard deviation=3.1) was observed.

In studies conducted in the Netherlands between 1988 and 1993, recycling of tar containing asphalt was associated with geometric mean full-shift benzo(a)pyrene exposures of the order of 0.12 µg/m^3 (n=36, geometric standard deviation =3.3) (Burstyn *et al.*, 2000) (Table B.9.9).

A crew of workers is treated as a uniformly exposed group hence the data is thought to be representative of all jobs within a crew, those paving as well as those under recycling/resurfacing.

Other exposure measurements collected during road construction were found in the literature of Watts *et al.* (1998) and Petry *et al.* (1996ab), however, these sources did not state explicitly than CTPHTs mixed with the bitumen and so they were not used for this exposure assessment.

Inhalation exposure conclusions for use as a binding agent in road construction

The 90th percentiles of 0.55 µg benzo(a)pyrene/m^3 and 1.2 µg benzo(a)pyrene/m^3 are obtained. The data are all from the same data set, but (apparently) from different subsets. The highest 90th percentile will be used as the reasonable worst case value: 1.2 µg benzo(a)pyrene/m^3.

Reasonable worst case short term exposure levels are expected to be up to the higher full shift exposure levels. A value of 5 µg benzo(a)pyrene/m^3 is used to represent reasonable, worst-case, short term inhalation exposure. Typical inhalation exposure level is taken as 0.55 µg benzo(a)pyrene/m^3, based on the arithmetic mean obtained by Burstyn & Kromhout (2000a) for road construction workers.

Table B.9.10. Dermal exposure to benzo(a)pyrene in asphalt paving.

Year of measure-ments	Occupation (Surface dressing workers)	Number of measure-ments	Duration of measurements (hours)	Range of results (mg/cm²/day)	Average (mg/cm²/day)		
					Arithmetic mean	Geometric mean	Median
1986	Rakerman 1	3	10	0.005-0.005	0.005		
	Rakerman 2	3	10	0.026-0.026	0.026		
	Fantail operator	1	10	0.021-0.031	0.027		
	All samples	7	10	0.005-0.031	0.017	0.013	0.021

Data extracted from data that had been graphed in the article by Jongeneelen *et al.* (1988). Skin contamination had been determined using two methods: with exposure pads and hand washing. The data presented here is the combined result of hand washing and that of the exposure pad placed at the wrist. The benzo(a)pyrene content was determined using the PAH profiles presented by Jongeneelen *et al.* (1988).

Dermal exposure

Measured dermal exposure data for use as a binding agent in road construction

There can be considerable skin exposure during road paving via contaminated clothing (Lindstedt & Sollenberg (1982) as quoted by Burstyn & Kromhout (2000a)). Dermal exposure data are rather scarce and though quite old, the exposure data during road construction presented herein was the sole source of measured data available (Table B.9.10). The benzo(a)pyrene concentrations were determined from data that had been graphed in the article by Jongeneelen *et al.* (1988). Jongeneelen *et al.* (1988) surveyed exposure to PAH among surfacing workers using refined coal tar and blended bitumens during chip sealing on road and dyke surfaces (Jongeneelen *et al.* 1988). Skin contamination had been determined using two methods: with exposure pads and hand washing. The data presented here is the combined result of hand washing and that of the exposure pad placed at the wrist. The skin contamination at the wrist was reported to be generally the highest of 5 areas where contamination was assessed. The benzo(a)pyrene content of the PAH was determined from PAH profiles presented by Jongeneelen *et al.* (1988).

At this worksite represented in Table B.9.10, the binder was sprayed on with a hand-held lance. The workers involved in the monitoring program were active in chip handling and spreading (rakermen and fantail operator). The workers involved in the actual spraying of the binder onto the surface and the tank truck driver were not monitored, however, one would expect their exposure would be at

least as great as or greater than those for which exposure measurements were available. An approximate estimate of dermal exposure to benzo(a)pyrene has been calculated from the data of Jongeneelen to be 0.005-0.031 mg/cm^2/day. Assuming both hands and part of the forearms are exposed (area of 1300 cm^2), and using the upper end of this range the exposure to benzo(a)pyrene will be 40 mg/day.

Modelled dermal exposure data for use as a binding agent in road construction

There is a paucity of dermal exposure data, however, the dermal exposure data presented here represents a reliable source of high quality data. Unfortunately the exclusion of workers actually conducting the spraying limits the usefulness of this data. While the exposures of the spraying operator during automatic spraying may be comparable to that of the workers listed in Table B.9.10, exposures during manual spraying are expected to be higher. Likewise, exposure during recycling (milling) of road surfaces. Exposures during manual spraying of the tar-bitumen binder and to particulate bound PAH during milling of road surfaces can be considered as worst case scenarios. EASE is used to assist in estimating exposure to these scenarios, assuming wide dispersive use, direct handling, and extensive contact. This gives an exposure range of 5-15 mg/cm^2/day to the tar-bitumen binder. Assuming both hands and part of the forearms are exposed (area of 1300 cm^2), and using the upper end of the range estimated by EASE the exposure to the binder will be 19,500 mg/day. Assuming a proportion of 50% CTPHT in the tar-bitumen binder, the exposure to CTPHT will be 9,750 mg/day. With a percentage of 1% benzo(a)pyrene in CTPHT, the exposure to benzo(a)pyrene as an indicator of exposure to CTPHT is estimated at 97.5 mg/day.

Dermal exposure conclusions for use as a binding agent in road construction

The exposure estimate obtained using EASE is greater than that obtained in actual field measurements. The EASE value is used to obtain a conservative estimate of the full-shift, reasonable, worst-case estimate of exposure to benzo(a)pyrene as an indicator of exposure to CTPHT, *i.e.* 100 mg/day.

Conclusions for use as a binding agent in road construction

The following exposure levels will be used for further risk assessment for use as a binding agent in road construction:

- Inhalation exposure, reasonable worst case, full-shift 1.2 µg benzo(a)pyrene/m^3
- Inhalation exposure, reasonable worst case, short-term 5.0 µg benzo(a)pyrene/m^3
- Inhalation exposure, typical, full-shift 0.55 µg benzo(a)pyrene/m^3
- Dermal exposure (hands), reasonable worst case, daily dose 100 mg/day benzo(a)pyrene

Uncertainties in derived exposure estimates for use as a binding agent in road construction

Some of the factors contributing to the uncertainties in these derived estimates include the following:

- The estimates for inhalation exposure are based on one source only. This raises questions as to its representiveness for all EU member states.
- The use of old data to estimate recent exposure. It is expected that improved methods and control measures will, in more recent years, reduce exposures. That said, it is believed that the inhalation exposure data do indeed represent the reasonable worst case situations where such measures have not been implemented.
- Extraction of data from a graph as was done with the data of Jongeneelen *et al.,* (1998), does include some degree of error as opposed to using stated values or having access to the raw data.
- For the dermal exposure estimate the errors inherent in the EASE model in deriving exposure values will add to the uncertainty of the value of the estimate.

B.9.2.4.2 Sub-scenario (ii): Use as a binding agent in roofing

During the laying down of a new roof CTPHT is added to provide a seal and adhesion between and above insulation layers. The CTPHT is heated and applied at approximately 191 °C to 204 °C. Volatile matter emanates from the heated asphalt and CTPHT during the various roofing operations. There is potential for inhalation and dermal exposure to particulate bound components of CTPHT and CTPV during roofing operations.

Removal, or "tear-off," of an old roof is often conducted down to the felt or insulation barrier. Following the initial tear off and power brooming with machines, the gravel pitch is shovelled by hand into small waste carts. The roof is also scraped with shovels and other hand tools in preparation for the laying down of a new roof (Emmett, 1986).

Roofers typically cover exposed skin during tear-off procedures to avoid the accompanying skin irritation. This may include wearing hats, long-sleeved shirts, gloves, and scarves over mouth and nose (Emmett, 1986).

Following removal of an old roof, new layers are installed. Flashing around the roof will be prepared and minor areas of damage to the roof may be patched using hot CTPHT. Roof removal is often a very dusty job with each task contributing to the general dustiness and there is potential for inhalatory and dermal exposure to CTPHT particulates (Emmett, 1986; Toraason et al., 2001). This is often exacerbated by windy conditions. A crew of about 20 men may remove about 10,000 square feet of old roof per day (Emmett, 1986).

Inhalation exposure

Measured inhalation exposure data for use as a binding agent in roofing

Table B.9.11. Exposure to PAHs and a rough estimate of the corresponding exposure levels to benzo(a)pyrene for use as a binding agent in roofing.

Reference	Occupation or site	Number of measure-ments	Duration of measurements (hours)	Time weighted average (st. dev.) (µg/m³)		Arithmetic mean (µg/m³)[b]	
				PAH [a]	CTPHT-PAH [b]	CTPHT-PAH	BaP [c]
Toraason et al. (2001)	Roofing removal and laying (1)	8	Full-shift	520 (600)	380 (438)		38
(USA)	Roofing removal and laying (2)	5	Full-shift	730 (380) [c]	533 (277)		53
	Roofing removal and laying (3)	6	Full-shift	190 (100) [c]	139 (73)		14
	Roofing removal and laying-All sites (1)(2)(3)	19	Full-shift	NA		350	35
Emmett E (1986)	Roof laying	45	8	20-530		73	7
	Roof removal	16	8	10-1880		343	34

[a] Exposure to the benzene soluble airborne particulate matter from all sources of exposure during the roofing process; [b] An approximate average exposure estimate to the benzene soluble fraction of total particulates from coal tar. These values were derived using Toraason *et al.*'s estimate that the contribution to the benzene soluble fraction from CTPHT is approximately 73%; [c] These values were derived using a percentage concentration of benzo(a)pyrene in CTPHT-PAH of 10% (see Section B.9.1).

The measured data of Toraason *et al.* (2001) represents data of three asphalt roofing crews (Table B.9.11). The processes taking place during monitoring were tear-off and laying down of a new roof. The exposure values given (ranging from 190 to 730 µg/m^3) are PAH exposure (the benzene soluble fraction of total particulates) arising from all sources of exposure as well as an estimation presented by Toraason *et al.* (2001) for exposure to that arising from coal tar (350 µg/m^3). The contribution to PAH exposure from coal tar pitch was approximated by Toraason *et al.* (2001) to be 73% of the total. Converting all inhalation data for roofing (including that of Emmett, 1986) using this estimated 73% contribution, gives an exposure range over all sites monitored of 8-1372 µg/m^3

CTPHT-PAH (0.73 times the PAH exposure range 10-1880). Using 10% as the proportion of benzo(a)pyrene in CTPHT-PAH gives an exposure range to benzo(a)pyrene of 0.8-137 µg/m^3 (See Section B.9.1).

The data of Emmett *et al.* (1986) indicate the possibility of very high exposure levels to particulate CTPHT-PAHs for roof removal with an upper limit of the exposure range being 1372 µg/m^3. Since these data are old and it is expected that processes of roof laying and removal would have changed in such a way that exposures are reduced, greater weight is placed on the data of Toraason et al. (2001). Since the activities of roof laying and removal were estimated as combined tasks during monitoring, the exposure estimate derived herein will also represent the exposures arising while conducting these combined tasks.

Modelled inhalation exposure data for use as a binding agent in roofing

There is not sufficient information to derive exposure estimates from modelled data.

Inhalation exposure conclusions for use as a binding agent in roofing

Deriving a rough estimate of the standard deviation of the distribution of the benzo(a)pyrene concentrations it is possible to obtain an estimated 90th percentile of exposure to benzo(a)pyrene. For estimating the reasonable worst case value the data of Toraason *et al.* (2001) is used. Because only the arithmetic means and standard deviations are available, the calculation is done assuming that the exposure is normally distributed with the estimated arithmetic mean of 35 µg/m^3 and an estimated standard deviation of 20. This gives a 90th percentile of approximately 60 µg/m^3 and is used as an estimate of the reasonable, worst case, full-shift estimate of benzo(a)pyrene as an indicator of exposure to CTPHT during roof removal and laying.

The short term estimate of exposure to benzo(a)pyrene is estimated to be 120 µg/m^3, *i.e.* twice the value obtained for the reasonable, full-shift worst-case scenario. The arithmetic average obtained by Toraason *et al.* (2001) for roof removal and laying is used to derive the typical full shift exposure estimate of exposure to benzo(a)pyrene, resulting in 35 µg/m^3.

Dermal exposure

Modelled dermal exposure data for use as a binding agent in roofing

Measured dermal exposure data during roofing are not available. EASE is used to assist in estimating exposure during roofing, assuming wide dispersive use, direct handling and extensive contact. This gives an exposure range of 5-15 mg/m^2/day to the tar-bitumen binder. Assuming both hands and part of the forearms are exposed (area of 1300 cm^2), and using the upper end of the range estimated by EASE the exposure will be 19,500 mg/day. Assuming a proportion of 50% CTPHT in the tar-bitumen binder, the exposure to CTPHT will be 9,750 mg/day. With a percentage of 1% benzo(a)pyrene in CTPHT, the exposure to benzo(a)pyrene as an indicator of exposure to CTPHT will be approximately 100 mg/day.

Dermal exposure conclusions for use as a binding agent in roofing

In the absence of measured data the estimate obtained by EASE will be used as the estimate of dermal exposure to benzo(a)pyrene during roofing operations. The full shift, reasonable worst case estimate of dermal exposure to benzo(a)pyrene as an indicator of exposure to CTPHT is 100 mg/day.

Conclusions for use as a binding agent in roofing

The following exposure levels will be used for further risk assessment for use as a binding agent in roofing:

- Inhalation exposure, reasonable worst case, full-shift 60 µg benzo(a)pyrene/m^3

- Inhalation exposure, reasonable worst case, short-term 120 µg benzo(a)pyrene/m^3
- Inhalation exposure, typical, full-shift 35 µg benzo(a)pyrene/m^3
- Dermal exposure, reasonable worst case, daily dose 100 mg/day benzo(a)pyrene

B.9.2.5 Occupational Scenario 4: Use as a binding agent for refractories

Refractories are materials that can retain their physical shape and chemical identity when subjected to high temperatures. They are found in use in many industries for lining boilers, kilns and furnaces of all kinds, but the largest percentage are used in manufacture of metals. Much smaller quantities are used in the following industries: gas, coke and by-products; power-generating plants; chemicals, bake ovens and stoves; cement and lime; ceramics; glass; enamels and glazes; locomotives and ships; nuclear reactors; oil refineries; refuse disposal (incinerators) (Stellman, 1998a).

The raw material of refractories includes the refractory material, which may be mineral-based material, synthetic material or a combination of these two. In the manufacture of some refractories a binder is used. When CTPHT is used as the binder there is potential for inhalation and dermal exposure to CTPHT volatiles and particulates during manufacture.

There also exist potential for exposure during usage of refractory material. During some of the uses listed above handling CTPHT-containing refractory material may be cause for inhalation and dermal exposure to CTPHT particulates. In constructions using refractory material (*e.g.* kilns and furnaces) high temperatures (generally > 700 °C) are used. This could result in the inhalation and dermal exposure to CTPVHT.

Inhalation exposure

Measured inhalation exposure data for use as a binding agent for refractories

One source of inhalation exposure data due to the production and use of refractories is available (Preuss *et al.*, 2003; summarized in Table B.9.12). It does not present information on how the material is produced and or used, but it is a recent source with a relatively large number of data points. Full shift measurements were done in 1999-2001.

Table B.9.12. Exposure to benzo(a)pyrene due to the production and use of refractories, in 1999-2001.

Industry	Number of measurements	Median (µg/m^3)	90th percentile (µg/m^3)
Production of refractories	62	0.17	3.4
Use of refractories	23	0.63	23.3

Data taken from Preuss *et al.* (2003).

Conclusions on inhalation exposure for use as a binding agent for refractories

There is only one source of inhalation exposure data for use as a binding agent for refractories. It does not contain a lot of context related to the processes, tasks of workers and distribution of exposure levels. It is however rather recent and consists of a sufficient number of data points. The (rounded) 90th percentiles of this publication (Preuss *et al.*, 2003) will be used as estimator of the reasonable worst case full-shift inhalation exposure levels. Twice this value will be used for estimating short term exposure levels.

Production of refractories:

- Reasonable worst case full shift inhalation exposure: 3.5 µg benzo(a)pyrene/m^3
- Short term inhalation exposure: 7 µg benzo(a)pyrene/m^3
- Typical full shift inhalation exposure: 0.17 µg benzo(a)pyrene/m^3

Use of refractories:

- Reasonable worst case full shift inhalation exposure: 23 µg benzo(a)pyrene/m^3

- Short term inhalation exposure: 64 µg benzo(a)pyrene/m^3
- Typical full shift inhalation exposure: 0.63 µg benzo(a)pyrene/m^3

Dermal exposure

No information is available on the processes for production and use of refractories that can be used to estimate dermal exposure to benzo(a)pyrene in these industries. Due to the high temperatures in the use of refractories, dermal exposure is expected not to occur repeatedly in this part of the scenario.

B.9.2.6 Occupational Scenario 5: Use as a binding agent for active carbon

No measured information is available on the use of CTPHT as a binding agent for active carbon. At present, there is insufficient information with regard to process details and proportion of CTPHT used in the binder to allow for the derivation of exposure estimates using EASE modelling.

B.9.2.7 Occupational Scenario 6: Use in heavy-duty corrosion protection

Hot-applied coal tar enamel coatings are used in heavy-duty corrosion protection. Coal tar enamels are formulated from refined CTPHT. Among the uses of coal tar enamel coatings is that as a pipeline protective coating.

The operations involved in coating and wrapping pipe with hot-applied coal tar enamel include abrasive blasting and priming, pipe coating and wrapping, and pipe finishing and inspection. Inhalation exposure to CTPVHT is expected with the coating operator, paper latcher (operates kraft paper), breakout man (removes connecting plug for between adjacent joints of pipe at the outbound site of the coating hood), holiday patcher (repairs gaps in the coating using open buckets of molten enamel), end finisher (removes enamel coating to allow field welding) and the kettle tender. During most stages of the process dermal exposure is expected to be low due to the high operating temperature of the process and the subsequent use of protective gloves and other personal protective equipment. Dermal exposure is expected to be incidental and to arise primarily from contact with surfaces where the coal tar enamel may have spilled, cooled and hardened on equipment and other work surfaces.

Inhalation exposure

Measured inhalation exposure data for use in heavy-duty corrosion protection

Table B.9.13. Summarized time weighted averaged benzo(a)pyrene exposure samples among 7 jobs at 8 coating plants.

Job	Number of measurements	Range (µg/m^3)	Mean (µg/m^3)	Median (µg/m^3)	90th-percentile (µg/m^3)
Coating operator	13	5.50-118.50	38.12	23.20	90.40
Paper latcher	11	1.50-41.45	10.55	6	14.60
Breakout man	9	0.70-9.00	4.22	2.80	9.00
Holiday patcher	3	1.15-8.50	3.98	2.30	7.26
End finisher	12	0.50-3.50	1.91	1.50	3.10
Kettle Tender	12	1.00-50.00	11.00	6.25	18.45

Data taken from Larson (1987).

Larson (1987) studied CTPVHT personal exposures among 7 different jobs and 8 coating plants. benzo(a)pyrene was used as the indicator for PAH exposure and hence can be used to assess exposure to CTPHT. The study concluded that the benzo(a)pyrene content in CTPVHT in the pipe coating industry is 0.5%. The results of the personal sampling are presented in Table B.9.13. It was possible to calculate 90th percentile for the different jobs for the data presented, using the arithmetic

mean and the standard deviation and assuming a normal distribution for the calculations. Two distinctive exposure groups can be defined with the coating operators forming one group (worker group A: 5.5-118.5 µg/m^3) and the other group comprising of workers in all other areas (worker group B: 0.5-50 µg/m^3). The estimated 90th percentiles for the groups are 90 µg/m^3 for group A and 31 for group B.

Inhalation exposure conclusions for use in heavy-duty corrosion protection

The reliability of the measured data precludes the necessity of modelling exposure data. It is representative for both the substance and the processes assessed and its reliability is strengthened by the inclusion of several enterprises in the measurement set. This data were collected following good occupational hygiene practice using standardised procedures with respect to sampling strategy and measurement methods (see EC, 2003b). The nature of the process limits the variation in the work procedures from one workplace to the next. It can therefore be assumed that the details of the processes described herein and the subsequent exposures arising from these processes are representative for similar workplaces throughout Europe.

The (rounded) estimates of the 90th percentiles of the normal distributed data for the respective groups are taken as the full-shift, reasonable worst case estimate of exposure:

- Worker group A (coating operators): 90 µg benzo(a)pyrene/m^3
- Worker group B (others): 30 µg benzo(a)pyrene/m^3

The reasonable worst case short-term exposure is estimated to be up to the highest value of exposure obtained for these groups. For worker group A, this is 119 µg/m^3 and for worker group B it is 50 µg/m^3. The typical exposure levels for these groups are estimated from the medians of the exposure data. For worker group A, this is 23 µg/m^3 and for group B the median is 6 µg/m^3.

Dermal exposure

Measured dermal exposure data for use in heavy-duty corrosion protection

No measured dermal exposure data is available.

Modelled dermal exposure data for use in heavy-duty corrosion protection

In the absence of measured dermal exposure data EASE is used to estimate exposure to CTPHT assuming the following: non-dispersive use, direct handling, incidental contact level. This gives an exposure estimate of 0-0.1 mg/cm^2/day. Assuming that half of the two hands may be exposed (area 420 cm^2) and using the upper end of the range the exposure to CTPHT will be 42 mg/day. With a benzo(a)pyrene content of 1% in CTPHT this gives a reasonable worst case full-shift exposure to benzo(a)pyrene of 0.4 mg/day.

One can assume a similar process with similar operating temperatures when CTPHT is used as a heavy-duty corrosion protective coating in other applications. Hence the dermal exposure estimate derived for this scenario will be adopted for similar applications where CTPHT has been used as a protective coating. Similar to this scenario, it is expected that comparable operating temperatures in other applications (<700 °C) will exclude inhalation exposure to high temperature CTPV.

Conclusions for use in heavy-duty corrosion protection

The following (rounded) exposure levels will be used for further risk assessment use in heavy-duty corrosion protection.

Inhalation exposure, reasonable worst case, full-shift:

- *(Group A)* 90 µg benzo(a)pyrene/m^3
- *(Group B)* 30 µg benzo(a)pyrene/m^3

Inhalation exposure, reasonable worst case, short-term

- *(Group A)* 120 µg benzo(a)pyrene/m^3
- *(Group B)* 50 µg benzo(a)pyrene/m^3

Inhalation exposure, typical, full-shift

- *(Group A)* 23 µg benzo(a)pyrene/m^3
- *(Group B)* 6 µg benzo(a)pyrene/m^3

Dermal exposure, reasonable worst case, full-shift

0.4 mg benzo(a)pyrene/day

B.9.2.8 Occupational Scenario 7: Use as a binding agent in coal briquetting

During coal briquetting relatively worthless fine coal is compressed to form a 'patent fuel' or briquette. In order to form a stable briquette a binder is necessary. Coal tars and pitches are used as the binder for this purpose. Following drying, devolatizing and crushing of the coal, it is mixed with the CTPHT binder. The binder addition rate is 5 to 15% by weight. The fine coal and binder are mixed in a pug mill or paddle mixer at an elevated temperature. The coal-binder mixture is then fed to a double roll press (Stellman, 1998b). The process may also involve a heat treatment in the range of 300 °C to harden the briquettes. The heat treatment oven is an enclosed conveyor and heated with hot gases. During the various steps involved there is potential for inhalation and dermal exposure to CTPHT, high temperature particulates.

Inhalation exposure

Measured inhalation exposure data for use as a binding agent in coal briquetting

Lafontaine *et al.* (1987) studied the PAH exposures at different workstations in six different coal briquetting enterprises. Both stationary and personal air monitoring was conducted. Benzo(a)pyrene was used as the indicator for PAH exposure and hence can be used to assess exposure to CTPHT. The results of the personal monitoring are presented in Table B.9.14.

Full-shift personal air monitoring was performed for workers in every stage of the process and hence covers all possible occupational exposure scenarios. The range of exposure was wide (0.07-2200 µg benzo(a)pyrene/m^3). However, it is possible to define two distinctive exposure groups with the cleaning operators forming one group (Worker group A: 30-2200 µg benzo(a)pyrene/m^3) and the other group comprising workers in all other areas (Worker group B: 0.07-80 µg benzo(a)pyrene/m^3). The 90^{th} percentiles of the data of these respective groups were 1760 µg/m^3 for group A and 42 µg/m^3 for Group B, assuming that the exposures were log-normally distributed.

Inhalation exposure conclusions for use as a binding agent in coal briquetting

The reliability of the measured data precludes the necessity of modelling exposure data. It is representative for both the substance and the processes assessed and its reliability is strengthened by the inclusion of several enterprises in the measurement set. This data were collected following good occupational hygiene practice using standardised procedures with respect to sampling strategy and measurement methods (see EC, 2003b). The simple nature of the process limits the variation in the work procedures from one workplace to the next. It can therefore be assumed that the details of the processes described herein and the subsequent exposures arising from these processes are representative for similar workplaces throughout Europe.

The (rounded) 90^{th} percentile of the log-normally distributed data for the respective groups are taken as the full-shift, reasonable worst case estimate of exposure:

- Worker group A (cleaners): 1760 µg benzo(a)pyrene/m^3
- Worker group B(others): 40 µg benzo(a)pyrene/m^3

The reasonable worst case short-term exposure is estimated to be up to the highest value of exposure obtained for these groups. For worker group A, this is 2200 µg/m^3 and for worker group B it is 80 µg/m^3. The typical exposure levels for these groups are estimated from the arithmetic means of the exposure data. For worker group A, this is 670 µg/m^3 and for group B the arithmetic mean is 14 µg/m^3.

Table B.9.14. Airborne personal full-shift exposures to benzo(a)pyrene during briquetting of coal using CTPHT as a binder.

Occupation or site	Number of measure-ments	Range of results (µg/m^3)	Arithmetic mean (µg/m^3)	Median (µg/m^3)	St. dev. (µg/m^3)	Geometric mean (µg/m^3)	Geometric st. dev. (µg/m^3)	90th Percentile [a)] (µg/m^3)
CTPHT supply and grinding	5	5.1-38	18.8	20.8	13.1	14.9	2.3	42.3
Drying	8	2.2-26	12.2	10.5	10.0	8.1	2.8	30.6
Mixing CTPHT	6	2.4-19	8.3	7.5	6.3	6.4	2.3	18.1
Workshop with presses	21	0.65-80	18.3	13.0	19.6	10.5	3.4	50.5
Defumage	12	1.2-30	11.4	9.9	9.2	7.9	2.6	27.5
Cleaning	13	30-2200	669.2	550	566	464	2.8	1760.0
Miscellaneous	3	0.07-20	6.8	0.4	11.4	0.8	18.22	33.4
All Data	68	0.07-2200	139.5	14.6	353	17.53	7.6	235.8
All minus cleaning	55	0.07-80	14.3	10.5	14.5	8.1	3.6	42.3

Data taken from Lafontaine *et al.* (1987). [a)] calculated assuming a lognormal distribution.

Dermal exposure

Modelled dermal exposure data for use as a binding agent in coal briquetting

In the absence of measured dermal exposure data EASE will be used to estimate dermal exposure to benzo(a)pyrene for this scenario. During cleaning operations the following assumptions are made: wide dispersive use, direct handling and intermittent contact level. This gives an exposure estimate of 1-5 mg/cm^2/day to the product that contains 5-15% of the coal tar binder. Using the upper end of the range of exposure, adjusting for a concentration of 15% of the coal tar binder and assuming both hands and part of the forearms are exposed (area of 1300 cm^2), exposure estimate will be 975 mg/day. (5×1300×0.15). With a percentage of 1% benzo(a)pyrene in the CTPHT binder, the exposure to benzo(a)pyrene as an indicator of exposure to CTPHT is estimated at 10 mg/day for Work Group A (cleaning operators).

In estimating exposure, using EASE for the other activities listed in Table B.9.14 the following assumptions are made: non-dispersive use, direct handling and an intermittent contact level. This gives an exposure estimate of 0.1-1.0 mg/cm^2/day to the product that contains 5-15% of the coal tar binder. Using the upper end of the range of exposure, adjusting for a concentration of 15% of the coal tar binder and assuming half of both hands are exposed (area of 420 cm^2), will give an exposure estimate of 63 mg/day (1.0×420×0.15). With a percentage of 1% benzo(a)pyrene in the CTPHT binder, the exposure to benzo(a)pyrene as an indicator of exposure to CTPHT is estimated to be 0.6 mg/day for Work Group B (all except cleaning operations).

Dermal exposure conclusions for use as a binding agent in coal briquetting

These EASE estimates are adopted as the reasonable full-shift worst case estimate of dermal exposure to benzo(a)pyrene as an indicator of exposure to CTPHT coal briquetting enterprises. For worker group A this value is 10 mg benzo(a)pyrene/day and for worker group B it is 0.6 mg benzo(a)pyrene/day.

Conclusions for use as a binding agent in coal briquetting

The following exposure levels will be used for further risk assessment for use as a binding agent in coal briquetting

Inhalation exposure, reasonable worst case, full-shift:

- Worker group A 1760 µg benzo(a)pyrene/m^3 benzo(a)pyrene
- Worker group B 40 µg benzo(a)pyrene/m^3 benzo(a)pyrene

Inhalation exposure, reasonable worst case, short-term:

- Worker group A 2200 µg benzo(a)pyrene/m^3
- Worker group B 80 µg benzo(a)pyrene/m^3

Inhalation exposure, typical, full-shift:

- Worker group A 670 µg benzo(a)pyrene/m^3
- Worker group B 14 µg benzo(a)pyrene/m^3

Dermal exposure, reasonable worst case, daily dose:

- Worker group A 10 mg benzo(a)pyrene/day
- Worker group B 0.6 mg benzo(a)pyrene/day

B.9.2.9 Occupational Scenario 8: Use as a binding agent for clay pigeons

In an artificial shooting target factory, targets (clay pigeons) are made of chalk (70-75%) and a basic binder (23-30%). The binder may be petroleum pitch or CTPHT. The chalk and binder are mixed and the resulting paste is moulded in non-closed automatic presses at 190 °C. The targets are then removed from the mould (the hardening time is about 20 seconds) and placed on a covered but non-closed conveyor belt which leads to the manual packing station (Lafontaine *et al.*, 2000). Indications show that this scenario is a minor market.

The workers involved in this process include packers, maintenance workers and a foreman. The packers are responsible for packing, monitoring the line and minor interventions on the presses. The maintenance workers are responsible for maintenance of the presses and conveyor belts and the foreman is responsible for truck driving and more administrative tasks. Packers may, on occasion, handle nude targets or painted targets without gloves resulting in direct dermal exposure to the clay/coal tar pitch product.

Within this working environment there is potential for exposure to inhalation and dermal exposure to particulate bound PAHs.

Inhalation exposure

Measured inhalation exposure data for use as a binding agent for clay pigeons

LaFontaine *et al.* (2000), monitored exposure to benzo(a)pyrene in an artificial shooting target factory where the basic binder, CTPHT had been substituted by a petroleum binder. However, it was stated that when CTPHT was used as the binder, the values for benzo(a)pyrene concentration were approximately 10 times higher. Using the exposure information for benzo(a)pyrene reported and adjusting upwards by a factor of 10, it was possible to obtain exposure estimates to benzo(a)pyrene during production of clay pigeons when CTPHT is used as the binder (Table B.9.15).

The data suggest that this should be viewed as a uniformly exposed group with exposures to benzo(a)pyrene in the range of 0.4-2.7 µg/m^3, with the highest exposure value being that of the maintenance worker (range: 0.9-2.7; arithmetic mean: 1.6 µg benzo(a)pyrene/m^3). The lowest

values were obtained for 'observers' who were not actually involved in the activities and had low physical activity.

Table B.9.15. Estimated exposure data to benzo(a)pyrene during manufacture of artificial shooting targets (clay pigeons).

Reference	Occupation or site	Number of measurements	Duration of measurements (hours)	Range of results [a] (µg/m³) (BaP)	Arithmic mean
La Fontaine (2000) [a]	Packing [b]	8	4	1.0-1.6	1.2
	Maintenance	8	4	0.9-2.7	1.6
	Packing [b]	7	4	1.0-1.9	1.3
	Packing [b]	8	4	1.1-1.7	1.4
	Foreman	8	4	0.9-1.6	1.2
	Observer	8	4	0.4-1.2	0.8
	Observer	8	4	0.6-1.4	0.9
	All Tasks	55		0.4-2.7	1.2

[a] In the actual monitoring programme conducted by La Fontaine *et al.* (2000), the CTPHT binder had been substituted by petroleum pitch. La Fontaine et al. reported that had CTPHT been used values would be greater by a factor of about 10. The results presented here are the values expected had CTPHT been used as the basic binder (BaP: benzo(a)pyrene); [b] Packers were also responsible for monitoring the line and minor interventions on the presses.

Inhalation exposure conclusions for use as a binding agent for clay pigeons

The extrapolation from the measured data is believed to give a fairly realistic view of exposure to benzo(a)pyrene, therefore these data are used to derive inhalation exposure conclusions for this scenario where possible.

The full-shift, reasonable, worst-case estimate of exposure to benzo(a)pyrene is estimated using the highest exposure levels derived among the packers, maintenance workers and the foreman- approximately 3 µg benzo(a)pyrene/m^3. The short-term reasonable worst case exposure is taken as twice that of the value estimated for full-shift reasonable worst case exposure, *i.e.* 6 µg benzo(a)pyrene/m^3. The typical full-shift estimate of exposure is estimated using the arithmetic mean over all tasks, *i.e.* 1 µg/m^3.

Dermal exposure

Modelled dermal exposure data for use as a binding agent for clay pigeons

In the absence of measured dermal exposure data, EASE is used to estimate dermal exposure to the clay/coal tar pitch product assuming non-dispersive use, direct handling, and intermittent contact level. This gives an exposure estimate of 0.1-1 mg/cm^2/day. Using the upper end of this exposure range, assuming that contact is half of both hands (area 420 cm^2) and adjusting for a concentration of 30 % CTPHT in the clay/coal tar pitch product gives an exposure estimate of 126 mg/day (1×420×0.3). With a 1% benzo(a)pyrene content in the CTPHT, the dermal exposure estimate to benzo(a)pyrene is 1 mg/day.

Conclusions for use as a binding agent for clay pigeons

The following exposure levels will be used for further risk assessment for use as a binding agent for clay pigeons;

- Inhalation exposure, reasonable worst case, full-shift 3 µg benzo(a)pyrene/m^3
- Inhalation exposure, reasonable worst case, short-term 6 µg benzo(a)pyrene/m^3
- Inhalation exposure, typical, full-shift 1 µg benzo(a)pyrene/m^3
- Dermal exposure, reasonable worst case, daily dose 1 mg benzo(a)pyrene/day

Table B.9.16. Conclusion of occupational exposure assessment to CTPHT.

Scenario/subscenario [a)]	Estimated inhalation exposure level (µg/m³) [b)]						Dermal exposure (mg/day) [b,c)]	
	Full-shift (8 hour time weighted average)				Short term		Full-Shift	
	Typical	Method	RWC [d)]	Method	RWC [d)]	Method	RWC [d)]	Method
1. Production of CTPHT in tar distillation plants								
a. Tar processing and handling of liquid pitch	0.1	measured	0.4	measured	0.8	measured	negligible	modelled
b. Handling of solid pitch	2.6	measured	3.6	measured	7.2	measured	0.5	modelled
2. Use-Binder for electrodes								
i. aluminium industry								
Søderberg potrooms (not modernised)	1	measured	8	measured	17	measured	0.5	modelled
Søderberg potrooms (modernised)	0.20	measured	0.35	measured	0.75	measured	0.5	modelled
Anode bake plants	0.15	measured	0.40	measured	1.40	measured	0.5	modelled
Paste plants	0.08	measured	0.15	measured	0.30	measured	0.5	modelled
ii. Graphite electrode paste plants								
a. Mixing and grinding; Baking; Maintenance	2	measured	7.5	measured	16	measured	negligible	modelled
3. Use-Binder in the Asphalt Industry								
i. Road construction	0.55	measured	1.2	measured	5	measured	100	modelled
ii. Roofing	35	measured	60	measured	120	expert judgement	100	modelled
4. Use-Binder for refractories								
a. Production of refractories	0.17	measured	3.5	measured	7	expert judgement	na	-
b. Use of refractories	0.63	measured	23	measured	64	expert judgement	na	
5. Use-Binder for active carbon	na		na		na		na	-
6. Use-Heavy duty corrosion protection								
a. Coating operators	23	measured	90	measured	120	measured	0.4	modelled
b. other workers	6	measured	30	measured	50	measured	0.4	modelled
7. Use-Binder in coal briquetting								
a. Cleaning	670	measured	1760	measured	2200	measured	10	modelled
b. Production	14	measured	40	measured	80	measured	0.6	modelled
8. Use-Binder for clay pigeons operators/packers; foremen	1	measured	3	measured	6	measured	1	modelled

[a)] The eight different occupational scenarios upon which exposure assessments were done are labelled 1, 2, 3 etc., the sub-scenarios are numbered i , ii, etc., and the different workgroups under the scenarios or sub-scenarios, which have different levels of exposure are listed a, b etc.; [b)] The exposure figures presented are for the exposure indicator benzo(a)pyrene; [c)] In the assessment of dermal exposure, supervisors were actually found to be in an intermediately exposed group on their own, with an exposure level between that of workgroup A and B. However, since their upper range was so close to that of the higher exposed group they were placed together with workgroup A for dermal exposure only; [d)] RWC: reasonable worst case.

B.9.2.10 Summary of occupational exposure

Table B.9.16 gives a summary of the results of all the exposure assessments discussed in this document.

B.9.3 Exposure assessment for consumers

Literature was searched with TOXLINE and Current Contents from 1985- April 2003 and Internet (with Google), with search string, CAS No.65996-93-2 and exposure/use.

Some safety data sheets were found in which CTPHT is used in road construction. This use is dealt with in Section B.9.4.

In the US some use in creosote solution is indicated. This use was also known in Europe. However, as creosote use for consumers is not allowed anymore (maximum level 0.2 ppm, accepted 11-03-03, according to EU legislation, http://meta.fgov.be/pdf/pk/nlkfg03.pdf) this use no longer needs to be considered. According to the Scandinavian product registers CTPHT is found in a number of industrial uses (NACE) such as intermediates, adhesives, paint, fillers and process regulators under use category 62. However, consumer use is not identified in 'total use' (SPIN database, 2003).

Based on the use as adhesive/binder CTPHT is put into clay pigeons and barbecue briquettes. The dermal exposure to clay pigeons is considered negligible during unpacking and loading of the catapult. No exposure is expected as a result of their destruction by shooting. The exposure during the use as binding agent in the barbecue briquettes is irrelevant as coal tar pitch will be totally consumed or volatised in the briquettes' production process.

B.9.3.1 Summary of exposure assessment for consumers

Consumer use was not identified by industry, not in literature nor on the Internet. Therefore the exposure to consumers to CTPHT can be considered negligible.

B.9.4 Exposure assessment for humans exposed via the environment

B.9.4.1 Introduction

Like in the environmental risk assessment (see Section B.9.5), the exposure to humans exposed via the environment will focus on the emission of PAHs on a local scale for production of coal tar pitch and the main applications (*e.g.* anode, aluminium, graphite electrode and ferro-alloy production), primarily because lower emissions for the other sources are expected. The emission of PAHs at coke ovens are not considered because coal tar is produced at this process (see Section B.2). In Western Europe the use of coal tar pitch as use of a binder in road construction and in roofing will be discontinued. Milling of old road surfaces may still result in exposure to coal tar containing material (Bursteyn & Kromhout, 2000b).

However, people may be shortly exposed (1 day) as a result of road tarring or roofing with a very low frequency (once every 10 years), and to concentrations in air much, much lower than workers as the distance to the source is larger and the concentration will consequently be reduced due to dilution. It is therefore assumed that an exposure assessment for man exposed via the outdoor air as a result of these activities is not necessary.

It is to be noted that with respect to the use of CTPHT in clay pigeons and coal briquetting no information was provided to justify the claim that in the near future it will significantly be reduced. For this reason, a conclusion i is drawn in Section B.10.2.1.2, where industry is requested to provide information on the release of PAHs from the production and use of these products in order to assess the risk for the environment. Depending on the data submitted, and in concomitant the risk for humans directly exposed via the environment as a consequence of these uses may have to be characterized in future.

B.9.4.2 Human exposure from industrial sources via the environment

Coal tar pitch (CTP) is a complex mixture of constituents of variable and partly unknown composition. The different constituents of CTP will show a different behaviour (fate) in the environment resulting in exposure of man through the environment to several constituents of CTP in a ratio which may be different from the ratio of these constituents in CTP itself.

The environmental exposure assessment was limited to 16 selected PAHs. In view of their differences in physical-chemical parameters, especially log K_{OW}, the distribution of these different PAHs from the point sources will be different. The exposure to the different PAHs for humans exposed via the environment will thus occur via different routes, meaning that in principle the risk characterisation should be based on the effects of each individual component. However, as the composition of CTP is variable and unknown and the human health effects of the known individual components are mostly unknown, this is practically impossible. Therefore, as a practical solution benzo(a)pyrene is chosen as the 'leading' PAH in establishing exposure for humans via the environment, because for this compound the largest amount of effects data is available and benzo(a)pyrene can be considered one of the most toxic PAHs. For this reason the risk assessment will be focussed on the exposure to benzo(a)pyrene. In case a risk is identified already for this one PAH, the other 15 PAHs will not be considered further.

As presented in Section B.9.5.1, the exposure of PAHs to the environment from the production and use of CTPHT is via the emission to air and water. Sludge application is not a relevant exposure route. Consequently, the main exposure route for humans exposed via the environment is through intake of leaf crops, meat, milk and the consumption of water organisms as a result of atmospheric deposition. Based on EUSES calculations, an air concentration of 1 ng benzo(a)pyrene/m^3 will result in 2.4 µg benzo(a)pyrene/kg leaf crop/grass, 5.6 µg benzo(a)pyrene/kg meat and 1.8 µg benzo(a)pyrene/kg milk, corresponding to a daily intake of 41, 24 and 14 ng/kg_{bw}, respectively. In comparison to these exposure routes, the intake of benzo(a)pyrene via air and root crops is only minor, given that an air concentration of 1 ng benzo(a)pyrene/m^3 will result in a total daily intake (including air and root crops) of 80 ng benzo(a)pyrene/kg_{bw}/day. For the calculations the following parameters are used: $K_{leaf\text{-}air}$: $4.68 \cdot 10^8$ m^3/m^3; TSCF: 0.0378; BAF_{meat}: 0.0339; BAF_{milk}: 0.0107.

In the current prediction the fraction benzo(a)pyrene bound to aerosol is set equal to 0.723 (standard calculation in EUSES).This means that 28% is available for gaseous exchange between air and leaves. However, for most sites the fraction benzo(a)pyrene bound to aerosol will probably be higher than 0.723. Consequently, the uptake by plants via gaseous diffusion will be lower than predicted. On the other hand, particles can also be deposited from air to leaves, which is at present not accounted for in EUSES. As the value for conductance is equal to the deposition velocity, the concentration in leaf crops will still be significant when most of the substance is bound to particles and might even be higher than those shown in Table B.9.19.

It should also be noted that the predicted concentrations in meat and milk do not account for the specific kinetics of benzo(a)pyrene in cattle. The limited information in literature available indicates that the transfer of benzo(a)pyrene and most of the other PAHs to milk is limited (Kan *et al.*, 2003), which might be due to metabolism in the cow. This, however, does not exclude the possibility that (carcinogenic) metabolites will be present in meat and transferred to milk. Some evidence for this is provided by Lutz *et al.* (2006), who found increased levels of 2-hydroxyfluorene, 3-hydroxy-phenanthrene and 1-hydroxypyrene (but not of 3-hydroxybenzo(a)pyrene) in milk after exposure to soil-bound PAHs. Rather than dealing with the metabolites at this stage, we prefer to deal with benzo(a)pyrene as a sort of measure for these metabolites.

The estimated concentrations of B(a)P in air and food and the resulting estimated human daily intake are given in Table B.9.17 and Table B.9.18, respectively.

Table B.9.17 Estimated concentrations of benzo(a)pyrene in air and food for humans.

source	Air (ng/m³)	Root crops (µg/kg)	Leaf crops (µg/kg)	Meat (µg/kg)	Milk (µg/kg)	Drinking water (ng/L) [a]
Production sites						
1	7.7	0.81	19	43	14	0.09
3	5.5	0.58	13	31	9.7	0.06
4	2.0	0.21	4.8	11	3.5	**1.9**
5	4.9	0.52	12	27	8.6	0.06
6	4.1	0.43	9.9	23	7.2	0.05
7	1.6	0.17	3.9	8.9	2.8	0.02
8	6.1	0.64	15	34	11	0.07
9	4.6	0.49	11	26	8.1	**0.23**
Downstream users						
Ferro-alloy	56	5.9	140	310	99	0.65
Graphite	13	1.4	31	72	23	0.15
Primary aluminium production and anode baking facilities						
S1	36	3.8	87	200	63	0.42
S3 [b]	92	9.7	220	510	160	1.1
S4 [b]	98	10	240	540	170	1.1
P7	2.9	0.31	7.0	16	5.1	0.0
S5	100	11	240	560	180	1.2
S6	98	10	240	540	170	1.1
PA1+S2	27	2.9	65	150	48	0.31
PA2	11	1.2	27	61	19	0.13
PA3	0.01	0.0	0.0	0.1	0.0	0.0
PA4	1.2	0.1	2.9	6.7	2.1	0.01
PA5	7.3	0.8	18	41	13	0.08
PA6	70	7.4	170	390	120	0.8
PA7	1.1	0.1	2.7	6.1	1.9	0.01
PA8	0.26	0.0	0.6	1.4	0.5	0.00
PA9	610	64	1500	3400	1100	7.0
PA10	6.8	0.7	16	38	12	0.08
PA11	26	2.7	63	140	46	0.30
PA12	0.73	0.1	1.8	4.1	1.3	**0.12**
PA13	94	9.9	230	520	170	**10**
PA14	70	7.4	170	390	120	0.81
PA15	0.031	0.0	0.1	0.2	0.1	0.0
A1	380	40	920	2100	670	**115**

[a] Drinking water concentrations given in bold are based on PEC surface water, taking into account a purification factor of 0.25. The other drinking water concentrations are based on estimated groundwater concentrations. [b] For site S3 and S4 measurements in the vicinity of the plants showed that the air concentrations were more than 10 times lower than predicted, provided that the measurements are reliable and representative.

Table B.9.18. Estimated human daily intake of benzo(a)pyrene via environmental routes.

Source	Air	Root crops	Leaf crops	Meat	Milk	Drinking water	Total
Production sites							
1	2.2	4.5	320	180	110	0.00	620
3	1.6	3.2	230	130	77	0.02	440
4	0.6	1.2	83	48	28	2.2	160
5	1.4	2.8	200	120	69	0.02	390
6	1.2	2.4	170	98	58	0.01	330
7	0.5	0.9	66	38	22	0.01	130
8	1.7	3.5	250	150	86	0.02	490
9	1.3	2.7	190	110	65	0.26	370
Downstream users							
Ferro-alloy	16	32	2300	1300	790	0.18	4500
Graphite	3.7	7.5	540	310	180	0.04	1000
Primary aluminium production and anode baking facilities							
S1	10	21	1500	860	510	0.12	2900
S3 [a)]	26	53	3800	2200	1300	0.30	7400
S4 [a)]	28	57	4000	2300	1400	0.32	7900
P7	0.8	1.7	120	69	41	0.01	230
S5	29	58	4100	2400	1400	0.33	8000
S6	28	57	400	2300	1400	0.32	7900
PA1+S2	7.7	16	1100	650	380	0.09	2200
PA2	3.1	6.4	450	260	150	0.04	880
PA3	0.00	0.01	0.41	0.24	0.14	0.00	0.80
PA4	0.34	0.70	50	29	17	0.00	96
PA5	2.1	4.2	300	170	100	0.02	590
PA6	20	41	2900	1700	980	0.23	5600
PA7	0.31	0.64	45	26	15	0.00	88
PA8	0.07	0.15	11	6.2	3.6	0.00	21
PA9	170	350	25000	15000	8600	2.01	49000
PA10	1.9	3.9	280	160	95	0.02	550
PA11	7.4	15	1100	620	370	0.09	2100
PA12	0.21	0.42	30	17	10	0.13	58
PA13	27	55	3900	2200	1300	11	7500
PA14	20	41	2900	1700	980	0.23	5600
PA15	0.01	0.02	1.3	0.74	0.43	0.00	2.5
A1	110	220	16000	9100	5300	130	31000

Values expressed as ng/kg_{bw}/d. Estimated human daily intake of: drinking water 2 L/day, fish 0.115 kg/day, leaf crops 1.2 kg/day, root crops 0.384 kg/day, meat 0.301 kg/day, dairy products 0.561 kg/day. Inhalation rate: 20 m^3/day. [a)] Provided that the measured air concentrations for site S3 and S4 are reliable and representative (see Table B.9.17).

For estimating the concentration in fish no reliable BCF values for benzo(a)pyrene are available. On the other hand, a BCF value of 600 could be used as a upper limit for benzo(a)pyrene (see Section B.4.3). For molluscs reliable BCF vales are available, with a mean value of 95,400. It is open for discussion whether the consumption of molluscs from contaminated sites is considered a relevant exposure route. For illustrative purposes only, in Table B.9.19 the benzo(a)pyrene concentration in fish and mollusc tissue is calculated for those sites which discharge their waste water to open sea.

Based on a water concentration of 1 ng/L, the concentration in fish and molluscs will be 0.6 and 95.4 µg benzo(a)pyrene/kg, respectively, corresponding to an intake of 1 and 160 ng benzo(a)pyrene/kg_{bw}/day when using the EUSES human fish consumption figure of 0.115 kg/day for both fish and molluscs.

Table B.9.19 Estimated concentration in molluscs and fish and the resulting human daily intake of benzo(a)pyrene.

Sites	Sea water concentration (ng/L)	Concentration in molluscs (µg/kg)	Daily intake via molluscs (µg/kg_{bw}/d)	Concentration in fish (µg/kg)	Daily intake via fish [a] (µg/kg_{bw}/d)
Production site 4	0.1	10	0.02	0.06	0.0001
Ferro-alloy	1	95	0.16	0.6	0.001
S1	8.5	811	1.3	5.1	0.01
S3	2.5	239	0.39	1.5	0.003
S4	260	24804	40.8	160	0.26
PA1+S2	15	1431	2.4	9.0	0.015
PA2	24	2290	3.8	14	0.024
PA5	99	9445	15.5	59	0.10
PA7	6.7	639	1.1	4.0	0.007
PA10	1.6	153	0.25	0.96	0.002
PA11	0.59	56	0.093	0.35	0.001
PA14	360	34344	56.5	220	0.36
PA15	0.012	1	0.002	0.0072	0.00001

[a] Human daily intake of 0.115 kg fish/day

B.9.4.3 Regional exposure via the environment

Since many unintentional sources contribute to the total emission of PAHs into the environment (see Section B.8.2.2), which by extension are not related to production and use of CTPHT, the risk characterisation will only be focussed on the PAHs emitted by producers and downstream users of CTPHT on a local scale. To put this risk characterisation into perspective, the daily dose is also calculated for the regional background using monitoring data available for air (EC, 2001b) and fresh water environment (COMMPS database). No formal conclusions will be derived for the regional background.

The estimated regional concentrations of benzo(a)pyrene in air and food and the resulting estimated human daily intake are given in Table B.9.20 and Table B.9.21, respectively.

Table B.9.20. Estimated concentrations of benzo(a)pyrene in air and food for humans based on regional background concentrations in air.

sites	Air (ng/m^3)	Root crops (µg/kg)	Leaf crops (µg/kg)	Meat (µg/kg)	Milk (µg/kg)	Ground (drinking) water (ng/L)
Remote	0.02	0	0	0.1	0	0
Rural	0.02-1.6	0-0.2	0-3.9	0.1-8.9	0-2.8	0
Urban	0.4-2	0-0.2	1.0-4.8	2.2-11	0.7-3.5	0
Traffic	0.7-3.1	0.1-0.3	1.7-7.5	3.9-17	1.2-5.5	0
Industrial	0.5-39	0.1-4.1	1.2-94	2.8-217	0.9-69	0-0.4

B.9.4.4 PAHs in cigarette smoke

PAHs are formed as by-products during the incomplete combustion of organic matter from various sources, including tobacco. Cigarette smoke therefore presents an additional, albeit unintentional, source of PAH inhalation exposure for both smokers (via mainstream and sidestream smoke) and non-smokers (via sidestream smoke). For illustrative purposes, the contribution of cigarette

smoking to the inhalation exposure of smokers was assessed (see below). Exposure by passive smoking was not considered, because hardly any data were available on sidestream smoke.

Table B.9.21. Estimated human daily intake of benzo(a)pyrene via environmental routes based on regional background concentrations in air.

sites	Air	Root crops	Leaf crops	Meat	Milk	Ground (drinking) water [a)]	Total
Remote	0.01	0.01	0.83	0.48	0.28	0	1.6
Rural	0.01-0.46	0.01-0.93	0.83-66	0.48-38	0.28-22	0	1.6-130
Urban	0.11-0.57	0.23-1.2	17-83	9.6-48	5.6-28	0	32-160
Traffic	0.20-0.88	0.41-1.8	29-130	17-74	9.8-43	0	56-250
Industrial	0.14-11	0.29-23	21-1610	12-930	7.0-550	0-0.01	40-3100

Values expressed as ng/kg_{bw}/d. [a)] When taking the mean benzo(a)pyrene concentration in European surface waters of 12.3 ng/L into account, the estimated human daily intake via drinking water is 0.35 ng/kg_{bw}.

Table B.9.22. Concentrations of PAHs and benzo(a)pyrene in mainstream smoke.

Reference	Cigarette brands-Origin and number	Average total PAH concentration (ng/cigarette) [range] (# of PAHs measured)	Average benzo(a)pyrene concentration (ng/cigarette) [range]
Chepiga *et al.* (2000)	US market; - Ultra low tar (n=8) - Full flavour low tar (n=11) - Full flavour tar (n=10)	NA	2.50 [0.79-5.49] 6.30 [5.15-7.50] 9.10 [7.96-9.97] study average: 6.0
Counts *et al.* (2004)	Worldwide market; 48 Philip Morris brands in 15 categories	NA	7.3 [1.0-13.9]
Lodovic *et al.* (2004)	Italian market; 14 brands	260.6 [117.8-373.5] (9)	3.7 [1.9-5.1] [a)]
Ding *et al.* (2006)	U.S. and non-U.S. market; Transnational U.S. brand (Marlboro) and 1 local brand per country; 14 countries	1240.5 [801-2673] (14)	10.2 [5.5-20.1]
Kalaitzoglou & Samara (2006)	Greek market; 59 brands in 15 categories	613.5 [105-1369] (16)	5.6 [0.9-10.0]
Ding *et al.* (2007)	US market; 9 full flavour king size brands (n=10)	117.8 [88.4-144.4] (10)	13.5 [10-15.8] [b)]
Melikian *et al.* (2007)	US market; - Low-yield nicotine (n=87) - Medium-yield nicotine (n=109) - High-yield nicotine (n=61)	NA	15.7 (14.3-17.2) [c)] 20.1 (18.8-21.6) [c)] 23.4 (21.1-25.7) [c)] study average: 19.7

[a)] In this study also concentrations in sidestream smoke, as generated by a home-made smoking machine, were measured. The average benzo(a)pyrene concentration was 47.7 ng/cigarette (range 15 to 78 ng/cigarette); [b)] When smoked under a more intensive puffing regimen (55-ml puff volume, 2-s puff duration, 30-s puff interval), the concentration of benzo(a)pyrene increased on average by 2-fold, demonstrating that smoking conditions greatly influence benzo(a)pyrene concentration in mainstream smoke; [c)] Geometric means (95% confidence interval); machine smoking under conditions mimicking smoking behaviour of participants.

A search was conducted on recent literature reporting concentrations of benzo(a)pyrene in cigarette smoke. In most of the studies found, concentrations were measured in mainstream smoke, following machine smoking of commercially available cigarettes under standard smoking conditions (35-ml puff volume, 2-s puff duration, 60-s puff interval). The results are presented in Table B.9.22.

The considerable variability in benzo(a)pyrene concentration between the reports are likely to be due to differences in cigarette composition, smoking conditions, sample preparation and analytical methodology used to determine benzo(a)pyrene concentrations. Despite the variability in the results and limitations in comparing the different studies, the data presented above were used to determine

an average benzo(a)pyrene concentration in mainstream cigarette smoke. The average benzo(a)pyrene concentration from all studies is 9.4 ng/cigarette.

There is also considerable variability in smoking prevalence and the average quantity of cigarettes consumed per smoker among the European countries (EEIG, 2003). A literature survey on cigarette consumption in the EU was recently carried out by HERAG (2006), reporting also the average cigarette consumption in the EU of 16.35 cigarettes per day as found by EEIG (2003). Further analyses of the EEIG data by HERAG resulted in a median cigarette consumption per smoker of 16.9 cigarettes per day, with the consumption ranging between 11.8 (minimum) and 23.6 cigarettes (maximum) per day.

Using the average benzo(a)pyrene concentration in mainstream smoke and the median cigarette consumption figure of 16.9/day, the inhalation exposure due to cigarette smoking is calculated to be 2.3 ng benzo(a)pyrene/kg_{bw}/day for a smoker weighing 70 kg. When comparing this dose to the doses via ambient air it can be concluded that for smokers cigarette smoke attributes considerably to the inhalation exposure to benzo(a)pyrene. No formal risk characterization will however be performed, because PAH emission during smoking is considered to be an unintentional source.

B.9.5 Exposure assessment for the environment

B.9.5.1 Scenarios

The exposure assessment for the environment will be focussed on the emission of PAHs on a local scale for production of coal tar pitch and the main applications, primarily because lower emissions for the other sources are expected[7]. Moreover, the amounts of coal tar pitch used for roofing and road paving decrease as it is replaced by petroleum pitch on account of the lower PAH content (worker hygiene). Some manufacturers claim to produce “environmentally” friendly clay pigeons by applying petroleum pitch in order to meet the EEC environmental protection directives, or apply no binder at all. The emission of PAHs at coke ovens are not considered because coal tar is produced at this process. Coal tar is used as a feedstock for the production of coal tar pitch and therefore the coke ovens are not part of the life cycle of coal tar pitch which actually starts at the production stage of coal tar pitch.

With respect to the main applications of coal tar pitch, the following point sources will be considered in the exposure assessment for the environment:

- Anode production
- Aluminium production applying prebakes (with and without) anode baking.
- Aluminium production using Søderberg technology
- Graphite electrode production
- Production of steel, silicon, etc., applying electric arc furnaces with Søderberg electrodes.

B.9.5.2 Environmental releases

Any application of pitch and pitch containing products results into environmental release of PAHs. The release to the environment is influenced by the type of (production) process used and the degree to which emission abatement equipment is applied. The focus will be on the largest application of coal tar pitch high temperature *e.g.* its use in the production of carbon and graphite electrodes and the application of these electrodes for instance in the production of primary aluminium and ferro-alloys.

[7] At present information on the emission of PAHs from the use of coal tar pitch as binder for refractories is very limited. However, it can not be excluded that the PAH emission from the use of this application can be significant (comparable to the main applications, depending on the abatement techniques used).

Site specific data was available for the CTPHT producing companies, anode production and primary aluminium production applying Søderberg and prebake anodes. The risk assessment for the other applications of CTPHT is based on generic (realistic worst case) scenarios. For most of the production sites complete emission profiles for all 16 EPA PAHs were provided for both water and air. If absent, the emission rates were related to sites with comparable operational management. With respect to the application of CTPHT, emission rates were either provided by industry (production of anodes and electrodes, aluminium production) or obtained from literature. For all applications the information was limited to only a number of PAHs, not specified PAH totals or to benzo(a)pyrene only. Therefore, emission rates for the rest of the 16 EPA PAHs were determined using typical profile of the 16 EPA PAHs for the process of concern. For the anode and graphite production and aluminium production based on Søderberg technology these profiles were provided by industry, if needed completed with information found in literature. For production of ferro-alloys profiles were obtained from open literature.

B.9.5.2.1 Release from coal tar pitch production

As explained above coal tar pitch is produced from coal tar at coal tar processing facilities. At these facilities many other products, essentially different kinds of oils are produced. All these different production steps contributed to the total release of PAHs by the facility. As coal tar pitch is the final product, which remains after several distillation steps it is difficult to consider it separate from all the other production steps in coal tar processing. Therefore it should be noted that the reported figures do not concern the production of coal tar pitch per se, but the whole process of coal tar processing.

Emission to air

Recent information on the release of 16 EPA PAHs by the coal tar pitch producing companies was provided for eight production sites within the European Union. One production site (no. 2) has recently been closed. The production sites represent a total production volume of 817,800 tonnes per year in 2004 (Table B.2.1).

For site 1 and 7 no information on the PAH emissions to air was reported. For site 3, 4 and site 6 complete emission profiles were reported. Site 5 reports emissions for seven PAHs. The mutual proportion between the PAHs emitted at site 3 and site 5 were comparable, with a difference of less than a factor of 3 with the exception of naphthalene. It is therefore assumed that the emission rates for the missing PAHs at site 5 were the same as those measured at site 3. Site 8 reported emissions for only two PAHs and site 9 reported only total PAH emission from storage. All these sites indicated that gaseous emissions from process point sources are collected to some extent and treated either through incineration or via scrubbers. Only site 6 reported estimated emissions from diffuse sources like valves, pressure-relief valves, flanges, pump and compressor seals and sampling connections. Additionally for site 6 it was indicated that air emissions from process point sources after treatment are negligible compared to diffuse emissions. Therefore for all sites emissions from diffuse sources are estimated from the emission profile of site 6 for diffuse sources and added to the reported air emissions from point sources (site 3 and 4). The emissions from diffuse sources for a specific site have been calculated from the ratio of the production volumes of the relevant site relative to that of site 6. The ratio is applied as a scaling factor to calculate the diffuse emission. With this information the emission of all the 16 EPA PAHs are composed for all the other sites, the release data are presented in Table B.9.23. Site 1 and 7 are comparable in the way that all tanks are connected to a vapour collection system and the collected vapours are incinerated. Site 1 and 6 indicated that emissions of PAHs were negligible after incineration compared to diffuse emissions. Therefore emissions after incineration are also considered negligible for site 7. For both site 8 and 9 process emissions are treated. Emissions from storage are estimated for site 9 to be about 24 kg PAH per year and are 6% of the estimated diffuse emissions and are therefore not considered. For

site 8 emissions from pitch storage are not measured and emissions from tar oil storage are about 20-30% of the diffuse emissions for the two substances considered. In the absence of sufficient information only diffuse emissions have been considered for site 8 as well.

Table B.9.23. Release to air of PAHs from coal tar processing (including coal tar pitch production).

Site	1 [a]	3 [a]	4 [a]	5 [a]	6 [a]	7 [a]	8 [a]	9 [a]
Production volume (kg/year) [b]	Conf.	Conf.	Conf.	Conf.	Conf.	Conf.	Conf.	Conf.
Number of days (d)	350	325	320	130	340	330	333	358
Naphthalene	**730**	7,472	3,700	8,700	410	**170**	**480**	**430**
Acenaphthylene	**210**	171	62	**460** [d]	120	**49**	**140**	**120**
Acenaphthene	**82**	228	81	1,300	46	**19**	**54**	**48**
Fluorene	**43**	153	44	**2,700** [d]	24	**10**	**28**	**25**
Phenanthrene	**110**	483	58	**8,800** [d]	61	**25**	**71**	**64**
Anthracene	**37**	80	16	1,400	21	**9**	**24**	**22**
Fluoranthene	**75**	189	32	**3,000** [d]	42	**17**	**49**	**44**
Pyrene	**63**	118	23	590	35	**15**	**41**	**37**
Benz(a)anthracene	**19**	18	5.6	**95** [d]	11	**4.4**	**12**	**11**
Chrysene	**17**	29	4.9	140	9.4	**3.9**	**11**	**10**
Benzo(b)fluoranthene	**240**	**169** [c]	66	**46** [c]	130	**54**	**150**	**140**
Benzo(k)fluoranthene	**45**	42	13	71	25	**10**	**30**	**27**
Benzo(a)pyrene	**29**	22	8.0	49	16	**6.5**	**24**	**17**
Indeno(123-cd)pyrene	**16**	13	4.5	**35** [d]	8.8	**3.6**	**16**	**9.3**
Dibenzo(a,h)anthracene	**36**	27	10	44	20	**8.2**	**28**	**21**
Benzo(ghi)perylene	**10**	8.3	2.7	**35** [d]	5.3	**2.2**	**6.2**	**5.6**

[a] Values in g/d. Figures presented in bold are values estimated by the rapporteur based on diffuse emissions only; [b] The production rate of the individual sites is not reported for the reason of confidentiality; [c] No emission data are reported for benzo(b)fluoranthene, only diffuse emissions have been estimated by the rapporteur; [d] No emission data from point sources are reported for these substances. Emissions from point sources have been estimated based on the profile of site 3. Additional emissions from diffuse sources have also been included.

Emissions to water

Waste water from sites 1, 3, and 7 is treated in an off-site municipal sewage treatment plant. Waste water from site 5 is treated in an off-site industrial waste water treatment plant. The waste water from sites 4, 6, 8 and 9 is treated on-site and emitted to the local receiving water. For all sites, except site 3 emission factors of all 16 EPA PAHs are available. Emissions for the missing PAHs of site 3 have been estimated based on the derived maximum emission factors of the other sites (Table B.9.24).

B.9.5.2.2 Release from industrial use of CTPHT as a binding agent

Release at this stage of the life cycle includes the production of carbon and graphite electrodes, including the production of Søderberg and prebake anodes for the aluminium industry and Søderberg electrodes for the ferro-alloy industry. The use of electrodes in primary aluminium production and ferro-alloy production is also considered in this section. The production of 'green paste' for the production of Søderberg and prebake anodes and graphite electrodes is generally performed at the same site where the anodes and electrodes are produced and is therefore considered as an integral part of this use stage. As already indicated the anode production often occurs at the same site where primary aluminium is produced. In those cases the release from both the anode production and the primary aluminium production should be aggregated.

Table B.9.24. Release to (waste) water from coal tar processing (including coal tar pitch production).

Site	1 [a]	3 [a]	4 [a]	5 [a]	6 [a]	7 [a]	8 [a]	9 [a]
Production volume (kg/year) [b]	Conf.	Conf.	Conf.	Conf.	Conf.	Conf.	Conf.	Conf.
Number of days (d)	350	325	320	130	340	330	333	358
WWTP/STP flow (m^3/s) [c]	16 [d]	0.10 [d]	0.0027 [e]	0.027 [d]	0.012 [e]	4.0 [d]	0.0028 [e]	0.0070 [e]
River flow (m^3/s)	2,500	[f]	15	- [g]	1.2	123	20	56
Dilution factor	157	100 [f]	1,000 [h]	10	101	32	1,000 [h]	1,000 [h]
Naphthalene	2.14	0.58 [i]	0.17	0.30	0.08	0.98	<0.77	<0.602
Acenaphthylene	n.d.	0.11 [i]	0.41	0.39	0.01	0.20	<0.602	<0.301
Acenaphthene	0.41	0.09 [i]	0.10	0.10	0.04	0.21	<0.664	<0.301
Fluorene	0.31	0.08 [i]	0.10	0.11	0.02	0.46	<0.084	<0.301
Phenanthrene	0.82	0.10 [i]	0.29	<0.0037	0.07	2.95	<0.12	<0.301
Anthracene	0.10	0.24 [i]	0.20	0.22	0.03	0.23	<0.026	<0.301
Fluoranthene	0.61	0.20 [i]	1.52	0.13	0.06	2.13	<0.143	<0.301
Pyrene	2.55	0.52 [i]	1.12	0.089	0.04	1.81	<0.172	<0.301
Benz(a)anthracene	<0.1	0.08 [i]	1.17	0.030	0.01	0.98	<0.052	<0.602
Chrysene	<0.1	0.08 [i]	1.02	<0.0037	0.01	0.98	<0.044	<0.602
Benzo(b)fluoranthene	0.10	0.07 [i]	6.76	0.0037	0.01	1.48	<0.019	<1.205 [i]
Benzo(k)fluoranthene	<0.1	0.07 [i]	2.20	0.0037	0.01	0.49	<0.019	[j]
Benzo(a)pyrene	<0.1	0.08 [i]	4.52	<0.0037	0.01	0.98	<0.018	<1.205
Indeno(123-cd)pyrene	<0.1	0.08 [i]	3.08	<0.0037	0.01	0.98	<0.008	<1.205
Dibenzo(a,h)anthracene	<0.1	0.08 [i]	0.77	<0.0037	0.01	0.21	<0.007	<1.205
Benzo(ghi)perylene	<0.1	0.08 [i]	2.76	<0.0037	0.01	0.82	<0.012	<1.205

[a] Values in g/d. [b] The production rates of the individual sites is not reported for the reason of confidentiality; [c] WWTP/STP: Waste Water Treatment Plant/Sewage Treatment Plant; [d] Discharge to off-site (public owned sewage) treatment plant, mean of three measurements in 2005; [e] Discharge to water from on-site (biological) waste water treatment plant; [f] Discharge to open sea; [g] River flow is unknown. Either the default river flow rate of 0.208 m^3/s or the default dilution factor of 10 is used; [h] Site specific dilution exceeds the maximum dilution factor of 1000. Therefore the maximum value for the dilution factor is used; [i] Based on the mean of three measurements in waste water treatment plant outlet in 2005; [j] For benzo(b)fluoranthene and benzo(k)fluoranthene together.

Release from primary aluminium production (including anode production)

Primary aluminium production plants, which use the Søderberg technology usually, have their own paste preparation plant or production hall. In this plant the green paste for the Søderberg electrodes is prepared as an integral part of the primary aluminium production process. Prebaked electrodes may also be produced in a production facility adjacent to the primary aluminium production plant.

The primary aluminium production process applying horizontal stud Søderberg anodes (HSS) is not further described as this production process is no longer applied in Europe.

For the use of coal tar pitch in the production of prebaked anodes for primary aluminium production (on and off-site) as well as the vertical stud Søderberg anodes (VSS) plants, the risk assessment is based on site specific information. Site specific data has been provided by the aluminium industry (EAA, 2006). The information includes emissions to air of either total PAH and/or benzo(a)pyrene. The emission to water is also based on benzo(a)pyrene and/or the total of the six PAHs of Borneff. Additionally information on waste water effluent flows, production capacity and applied electrolysis technology of primary aluminium production and the location of the facilities has been provided (see Table B.9.25).

The information of EAA (2006) indicates that all anode plants and all VSS plants have dry scrubbing systems for anode gas or pot gases, using either alumina or coke. It is stated that water emissions from anode baking units come from cooling water used to cool the green anodes.

For the VSS plants, it was indicated that these sites (S1, S3 and S4 in Table B.9.25) use seawater to scrub the ventilation air from the potroom. The VSS plant at site S2 is closing in 2007 and is therefore not included. The emissions from these sites are to sea. As shown in Table B.9.25 for most sites the effluents go to the marine environment: only two sites are located near fresh surface water (A1 and PA12).

Table B.9.25. Site specific release information for primary aluminium production and anode baking plants.

Site [a]	Cell type [b]	PAH emission air		PAH emission water		Effluent flow [c]	
		PAH (tonne/year)	benzo(a)pyrene (kg/year)	benzo(a)pyrene [d] (kg/year)	Borneff (kg/year)	Fresh (10^3 m^3/year)	Marine
S1	VSS	11.8	560	115	2300		60000
P1	PB	NR [e]		NR [e]			
PA1	PB	5.2	35.5 [g]	1.6	20	2365.2	
P2	PB	NR [e]		NR [e]			
S3	VSS	42	1438	37.1	742		66000
PA2	PB	2.2	15 [g]	6.8	171	1285	
P3	PB	NR [e]		NR [e]			
P4	PB	NR [e]		NR [e]			
PA3	PB	0.002	0.01 [f]		0		
PA4	PB		1.56		0		
P5	PB	NR [e]		NR [e]			
PA5	PB	0.74	9.55	0.2	5	9	
PA6	PB	1.948	34		0		
PA7	PB	0.04	1.4 [f]	<0.2	< 5	133.5	
P6	PB	NR [e]		NR [e]			
S4	VSS	18.2	1529	315	6300		54000
PA8	PB	0.05	0.34 [f]		0		
PA9	PB	147	799		0		
PA10	PB	1.3	8.9 [f]	0.008	0.2	219.0	
PA11 [g]	PB	3.73	33.9 [h]	0.0024	0.06	18	
PA12 [g]	PB	0.105	1.0 [h]	0.0052	0.13	500 [i]	
PA13 [j]	PB		123	1.2		134.0	
S5 [j]	VSS		1602		0		
S6 [j]	VSS		1548		0		
PA14 [j]	PB		92	0.9		111.7	
PA15	PB	0.006	0.04 [f]	<0.00004	< 0.001	0	
A1 [g]	Anode	55	500 [h]	0.68	16.88	65.5 [i]	

[a] S = Primary aluminium production plant, using the Søderberg technology, P = Primary aluminium production plants using prebaked anodes not produced on-site, PA = Primary aluminium production plant with an on-site anode baking facility, A = anode plant not located at the same site of a primary aluminium production facility; [b] VSS = Vertical Stud Søderberg, PB = Prebake, Anode = anode plant only; [c] All water emissions are to sea, except for PA12 and A1 (see also note [j]); [d] Benzo(a)pyrene emission to water is based on the fraction of benzo(a)pyrene in the Borneff 6, being 5% for VSS and 4% for anode plants; [e] NR-not relevant, no PAH emissions; [f] Benzo(a)pyrene emission to air is based on the fraction of benzo(a)pyrene in the 16 EPA PAH profile, which is 0,68% for anode plants; [g] Emission estimation is based on the reported emission of total PAH; [h] Emission factor for benzo(a)pyrene is based on the process specific emission profiles; PAH emissions at PB smelters is from the anode plant, from VSS smelters mainly from electrolysis and small component from paste plant; [i] Emission to fresh water; [j] Emissions are calculated using the second highest reported emission factors, see also text.

Some primary aluminium production sites have been closed recently or are due to close in 2007 (EAA 2007). These sites, two in Germany, one in France, one in Norway and one in Switzerland, have been left out the risk assessment and have also not been included in Table B.9.25. For prebake

production plants no site specific risk assessment has been conducted, as the emissions where considered to be negligible to cause an environmental risk. The sites with primary aluminium production using the prebake technology and an on-site anode baking facility should be distinguished from the prebaked anode production, which is not located at the same site of a primary aluminium production facility. Of the latter there is only one plant in the European Union, site A1.

For some sites no emissions of benzo(a)pyrene to air and/or water have been reported. In that case the emission of benzo(a)pyrene has been estimated based on a default fraction of benzo(a)pyrene in the PAH emission profiles presented in Section B.9.5.2.3. For the emission to air the fraction of benzo(a)pyrene in the total PAH emission is 0.7% for anode baking (EAA, 2005). For the emission to water the fraction of benzo(a)pyrene in the six PAHs of Borneff is 5% for the VSS-technology and 4% for anode baking (EAA, 2005) (see Section B.9.5.2.3 for the process specific emission profiles).

Furthermore four sites did not provide any information on either emissions to air or water. For these sites the highest reported emission factor to air and/or water has been applied together with the specific emission profiles to estimate the emissions to air and if relevant to water. For anode baking an emission factor for benzo(a)pyrene of 1.0 g/tonne and for the emission of benzo(a)pyrene to water 0.01 g/tonne has been used. For primary aluminium production using dry scrubbers (S5 and S6), the emission factors for benzo(a)pyrene to air is 18 g/tonne. The emission to air and water stem from the reported site specific data. The second highest emission factor was selected as a worst case, regarding the highest emission factor as an outlier. The waste water effluent flow is calculated from the anode capacity and the volume of waste water generated per tonne anode produced, assumed to be 6 m^3 per tonne (Section B.9.5.2.4).

Seven sites, all relating to prebake primary aluminium production with an on-site anode production facility, report zero emission to water, due to closed water circulation systems (EAA, 2007).

The emission of all the 16 EPA PAHs has been calculated by applying the specific emission profiles to the reported or estimated (derived from the reported total PAH emission) emission factor of benzo(a)pyrene. In some cases the calculated total amount of PAHs did not match with the reported PAH emission. This is probably due to the use of average PAH emission profiles for the calculation. In the absence of site-specific PAH profiles the following conservative approach is applied:

In case the calculated emission of the total of the 16 EPA PAHs was lower than the reported emission of total PAH, the reported emission of total PAH was used to estimate the emission of the individual PAHs, using the average emission profiles mentioned in see Section B.9.5.2.3. When the calculated total PAH emission is higher than the reported total PAH emission, the calculated total was used.

Release factors for production of graphite electrodes

Emission to air

For electrode baking one emission factor for benzo(a)pyrene, 10.2 gram per tonne, was found in literature (EC, 2001b) The emission factor is stated to be typical for German (modern European) electrode baking facilities and is well in line with the emission factor reported for anode baking in the United Kingdom. Information from the industry (ECGA, 2006) on the emissions of benzo(a)pyrene at the different stages of the graphite electrode production process indicated emissions in the range of 0.0014-0.33 g/tonne of product using abatement techniques ranging from dust injection technology in combination with bag-house filters to thermal oxidisers (see Table B.9.26). According to the same information, emissions to water are not to be expected at the facilities applying these abatement techniques.

For paste preparation (green anodes) emissions to air are about 7 gram PAH per tonne of product on average. Maximum emissions are in the range of 40 gram PAH per tonne of product. Benzo(a)pyrene emission makes about 1% of the total PAH emissions to air for both anode baking and green paste preparation (EAA, 2005).

Table B.9.26. Emission factors for release of benzo(a)pyrene to air from anode baking and graphite electrode baking facilities.

Process	Emission factor (g benzo(a)pyrene/ tonne) [a)]	Abatement	Remarks	Reference
Green paste preparation	Average : 0.07 Worst case: **0.4**	unknown		EAA (2005)
Green paste preparation	0.02	Dry scrubbing	total PAH emission of 1.8 kg/ tonne products and assuming 1% benzo(a)pyrene	Alcoa (2002)
Graphite Electrode production	10.2	unknown	Typical German (modern European)	EC (2001b)
Graphite Electrode production	0.0014-**0.33**	Dust injection-thermal oxidisers	sum of the emissions from the mixing, baking, impregnation and prebaking stage	ECGA (2006)

[a)] Values in g/tonne. Values in bold are used for the scenario calculations.

Emission to water

Graphite electrode production is generally a dry process. Discharge of process wastewater is usually limited to cooling water, used to cool the green anodes. However, most processes use a sealed (closed) cooling system, depending on the technology (EC, 2001a; ECGA, 2004). Paste does not get cooled down by water at all sites. Three sites report the use of cooling water in the process. These sites either have a closed system or treat the water before it gets discharged. Two sites that cool down the paste by using water have water treatment plants installed on site and the treated water either gets reused in the plant (one site) or is discharged to surface water (one site). For the last site measured concentrations for 10 PAHs in discharged water were below the detection limit of 0.025-0.1 µg/L. Based on the emission profile for paste preparation (see Section B.9.5.2.3) it is expected that the concentration in surface water for most PAHs will be < 0.0025 µg/L. Hence, for this site the emission of PAHs is considered negligible. A third site uses a water tank that gets emptied once or twice a year by an external registered water treatment company to a licensed waste treatment facility for recovery that treats the water in a legally accepted way (ECGA, 2007). Consequently, for the graphite electrode production the emission of PAHs to water is not further considered in this risk assessment.

Ferro-alloy and non-ferrous metal production other than primary aluminium

Emission to air

Ferro-alloy production using electric arc furnaces in combination with Søderberg electrodes is a potential source of PAH. The information found in public literature is limited. EC (2001a) reports emission factors for PAH of 1.5 g/tonne for FeSi production and 3 g/tonne of product for Silicon-metal. The PAHs released from this production process are expected to be related to the use of Søderberg electrodes. Relating the PAH emission per tonne product to the amount of electrode material consumed during the process, *e.g.* 50 and 100 kg of electrode per tonne product, results in an emission factor of 30 g PAH per tonne electrode consumed. No information was provided, on the PAH profile. EEA (2001) reports an emission factor for benzo(a)pyrene of 17 gram per tonne of product for electric arc furnaces. There was no information reported with respect to abatement technique used and the type of product produced. EPA (1998a) listed emission factors for benzo(a)pyrene per mega-Watt of electricity consumed in ferro-alloy production. The emissions

range from 0.0007 g/MWh electricity consumed for electric arc furnaces with bag house equipment to 0.046 g/MWh electricity consumed for electric arc furnaces equipped with wet scrubbers. Additionally an emission factor of 0.00001 g benzo(a)pyrene per tonne for electric steel production was reported. There was no information, which addresses the kind of product produced and the type of electrodes used. With the electricity consumption and electrode consumption per tonne of product the per mega-Watt emission figures can be recalculated into an emission factor per tonne of electrode material consumed or per tonne of product produced, 0.10-6.67 g/tonne electrode consumed giving 0.005-0.317 g benzo(a)pyrene per tonne product respectively based on average specific consumption rates of 6.9 MWh per tonne of product and 6.9 kg electrode per MWh electricity consumed. The average value has been established using the data in Table B.9.27.

Table B.9.27. Emission factors for the release of benzo(a)pyrene to air from ferro-alloy production using electric arc furnaces with Søderberg electrodes.

Process	Emission factor (g benzo(a)pyrene/ tonne) [a)]	Abatement	Remarks	Reference
FeSi	1.5	Unknown	30 gram per tonne electrode. PAH not specified.	EC (2001a)
Si-metal	3.0	Unknown	30 gram per tonne electrode. PAH not specified.	EC (2001a)
Ferro-alloy	17	Unknown	plant in Czechoslovakia	EEA (2001)
Ferro-alloy	0.005	bag house	calculated	
Ferro-alloy	**0.317**	wet scrubbers	calculated, see Section B.9.5.2.4; 0.32 gram per tonne electrode	

[a)] Values in g/tonne. Values in bold are used for the scenario calculations.

Table B.9.28. Release of PAHs to water from ferro-alloy smelters using electric arc furnaces with Søderberg electrodes.

Site	PAH Emission (kg/year)	Production volume (ktonnes/year)	Emission factor (g/tonne)	Remarks
Porsgrunn				
1986	4300	116	37.1	Production is estimated, using 10% growth in 5 years
1990	350	131	2.7	Production is estimated, using 10% growth in 5 years
1995	44	145	0.3	Production volume concerns both FeMn and FeSi production [a)]
2000	80	170	0.5	Production volume is actually total production capacity
Sauda				
1985	8000	140	57	Production is estimated, using 10% growth in 5 years
1990	400	158	2.4	Production is estimated, using 10% growth in 5 years
1996	30	175	0.2	Production volume concerns both FeMn and FeSi production [a)]
2001	20	190	0.1	Production volume is actually total production capacity [b)]

Data taken from Eramet (2003) and Næs (1995). [a)] Production volume taken from Bellona (2004); [b)] Production capacity taken from Eramet (2004).

Emission to water

PAH emissions from ferromanganese production plants have been reported by Næs *et al.* (1995) and Eramet (2003). Emissions have only been reported as total PAH. No information was given on the estimation and analytical methods used. Emission factors for the Porsgrunn and Sauda ferromanganese smelter range from 2.7 and 2.4 gram per tonne in 1990 to 0.3 and 0.1 gram per tonne in 2000 and 2001, respectively (Table B.9.28). The share of benzo(a)pyrene in the total PAH emissions to water are about 2% for facilities using Søderberg technology with wet scrubbers (EAA, 2005). Using this value and a realistic worst case emission factor of 2.7 gram of total PAH

per tonne product, an emission factor to water for benzo(a)pyrene of 0.054 gram per tonne of product can be estimated.

B.9.5.2.3 Process-specific PAH profiles

Information on the process-specific PAH profiles was provided by industry or if lacking gained from open literature. Emission factors as well as concentrations in air are used to derive profiles. Emission profiles are defined here as concentrations relative to the concentration of benzo(a)pyrene. By expressing the different PAHs in the profiles as a relative fraction of benzo(a)pyrene, the emission factors can be calculated by multiplying these values with the emission factors of benzo(a)pyrene. Therefore in the profiles presented below the value of benzo(a)pyrene is 1.

From the calculated emission profiles it is clear that the information is to some extent limited in that not all of the 16 EPA PAHs are covered in the reported profiles.

Emission to air

Emission factors for the various processes are generally only available in public literature for benzo(a)pyrene. Some emission factors for other PAHs have been found though (EPA, 1998b; OSPAR, 2002; Tsibulski, 2001). In case insufficient information was provided by the industry, the PAH emission to air for the downstream-users were determined using the benzo(a)pyrene emission factors found for the different processes in combination with the PAH profiles measured (indoor concentrations or derived from emissions). Thus, the information gathered in research on occupational exposure to PAHs is also used to determine PAH-profiles.

It is recognised that there are many variables, which influence the relation between outdoor and indoor concentrations, and which complicate the use of indoor PAH-profiles. On the other hand, there is probably no other information available in public literature. Possibly, the indoor profiles are most related to outdoor profiles when no abatement techniques are used, and can therefore be considered as a worst case scenario. In case there is a limited amount of information available it is assumed that the concentration profile as measured inside a facility will be the same as the emission profile outside a facility.

For the various industries, a range of benzo(a)pyrene emission factors are reported. For several of these factors it is known which abatement techniques were used. For others this was not reported. As mentioned for the Søderberg companies it is essential for all the downstream-users to know which abatement techniques are used. Since this information is as yet not available, the highest emission factor is used.

For primary aluminium production processes using the VSS technology, two emission profiles have been submitted by the industry (EAA, 2005). One profile for VSS technology applying dry scrubbers (VSS I) and a profile for VSS technology applying wet scrubbers as for emission abatement (VSS II). These profiles do not include all 16 EPA PAHs considered in this transitional dossier. For naphthalene, acenaphthene, acenaphthylene and fluorene no information was provided. The emission profiles for the VSS technology have been completed by applying the average values from other VSS profiles found in literature (see Annex J.5, Table J.6.1).

Also for paste preparation and anode baking emission profiles for PAH have been provided. For anode baking information on acenaphthylene was lacking. The average value of two other emission profiles obtained from literature has been used to complete the emission profile. For paste preparation there was no information for acenaphthene and acenaphthylene. For these two substances the relative emissions from anode baking have been applied to complete the PAH emission profile (see Annex J.5, Table J.6.2 and Table J.6.3).

For graphite electrode baking (see Annex J.5, Table J.6.4) a complete emission profile for the 16 EPA PAHs has been submitted by industry. This profile is used for the emission calculation for the graphite electrode production.

For the prebake aluminium production process (see Annex J.5, Table J.6.5) and metal recycling (electric steel) and non-ferrous metal and ferro-alloy industry (silicon carbide) no complete profile could be established. Considering the similarity of the technology used, for ferro-alloy industry the emission profiles from the VSS aluminium production technology have been used as a substitute for the emission calculations.

The estimated profiles for the different use categories are presented in Table B.9.29.

Table B.9.29. Estimated (emission) profiles for the 16 EPA PAHs in air for different production processes.

PAH compound	VSS I	VSS II	Prebake [a)]	Anode baking	Electrode baking	Green paste	Ferro-Alloy [b)]
Naphthalene	16.5	16.5	0.003	29.0	77.3	2.0	16.5
Acenaphthene	2.5	2.5	n.i.a.	3.0	28.3	3.6	2.5
Acenaphthylene	6.3	6.3	0.9	0.1	17.0	0.3	6.3
Fluorene	7.5	7.5	0.2	5.0	10.9	9.0	7.5
Anthracene	1.2	2.0	n.i.a.	2.0	9.6	15.3	2.0
Phenanthrene	7.2	19.5	n.i.a.	24.0	131.5	48.0	19.5
Fluoranthene	3.7	11.0	16.8	10.0	90.1	34.1	11.0
Pyrene	2.9	5.5	7.2	5.0	49.8	21.0	5.5
Benz(a)anthracene	0.9	1.0	2.2	2.0	6.3	4.3	1.0
Chrysene	1.8	2.0	3.1	5.0	17.2	4.6	2.0
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Benzo(b)fluoranthene	4.1	3.0	1.0	8.0	8.9	5.0	3.0
Benzo(k)fluoranthene			1.0		4.6		
Benzo(ghi)perylene	0.6	0.5	0.5	1.0	0.9	0.0	0.5
Dibenzo(a,h)anthracene	0.1	0.2	0.2	1.0	0.5	0.0	0.2
Indeno(123-cd)pyrene	0.5	0.5	0.3	1.0	1.1	0.0	0.5

[a)] n.i.a. = no information available; [b)] Same profile as VSS II.

Emission to water

For several Norwegian primary aluminium producing facilities using Søderberg technology PAH profiles in waste water have been reported by Kadar *et al.*(1980), Olufsen, (1980), Berglind (1982), OSPAR (2002) and EAA (2004). Not all profiles covered all 16 EPA PAHs, especially for the lower PAH compounds the information is very limited (occurs only in the profile reported by Berglind, 1982). The emission profiles used for the risk assessment are based on the average values completed with the single values for the lower PAHs obtained from the profile reported by Berglind (1982) (see Annex J.5, Table J.6.6). The information addressing the type of Søderberg technology used was limited. Only EAA (2005) specified the use of wet-scrubbers. Other abatement techniques to reduce the emission of pot gases are dry scrubbing systems using injected alumina techniques in combination with bag house filter equipment to collect the particulate matter. In some cases spray towers using seawater are used after the dry scrubbing systems for sulphurdioxide scrubbing. Some plants also have scrubbing systems for ventilation air in the pot rooms, typically used in some Søderberg plants, using seawater as scrubbing medium. In CWPB (Centre Worked Prebake) and PFPB (Point Feed Prebake) primary aluminium production plants this is not necessary due to the higher collection efficiency from the pots.

Two comparable emission profiles for paste preparation were reported by the industry (EAA, 2004, 2005), see Table B.9.30. The average profile has been used for calculating the emissions from paste preparation (see Annex J.5, Table J.6.6).

Table B.9.30. Estimated profiles for (the emission to) water of the 16 EPA PAHs for primary aluminium production facilities using the Søderberg technology and anode baking.

PAH compound	Søderberg (VSS) [a]	Paste preparation [b]
Naphthalene	0.3	0.3
Acenaphthene	1.3	1.3
Acenaphthylene	0.3	0.3
Fluorene	0.8	0.8
Anthracene	0.8	10.3
Phenanthrene	8.8	36.9
Fluoranthene	14.8	23.8
Pyrene	9.1	16.9
Benz(a)anthracene	2.5	4.2
Chrysene	4.5	3.3
Benzo(a)pyrene	1.0	1.0
Benzo(b)fluoranthene	3.0	2.1
Benzo(k)fluoranthene [c]	NA	NA
Benzo(ghi)perylene	0.5	0.3
Dibenzo(a,h)anthracene	0.2	0.4
Indeno(123-cd)pyrene	0.5	0.4

[a] See Annex J.5, Table J.6.5; [b] See Annex J.5, Table J.6.6; [c] No data available (indicated by NA).

B.9.5.2.4 Generic release scenarios for industrial use

For the primary aluminium production applying the Søderberg electrolysis cells, anode baking, green paste preparation and the graphite electrode production the release calculations are almost completely based on information provided by industry. The information on emission factors and emission profiles has been presented in the previous sections. For anode baking and all aluminium smelters using Søderberg electrolysis cells or prebaked anodes site specific information has been provided. As little or no site-specific information was available for the graphite electrode production and the ferro-alloy production, for these types of use realistic worst case scenarios were formulated. Representative, realistic worst case emission factors for benzo(a)pyrene were selected and process and technology specific emission profiles were used to estimate the emissions of the 16 EPA PAHs.

Production capacity of the industrial use categories

For graphite electrode production a realistic worst case production volume of 55 ktonnes per year is assumed (GrafTech, 2004). For ferro-alloy production the size of the largest production facilities is in the order of 200 ktonnes per year (Tinfos, 2004). As stated before green paste preparation (green anodes) can be seen as a separate formulation step. The green paste preparation though is considered as an integral part of anode, graphite electrode, ferro-alloy and the VSS primary aluminium production and is therefore integrated in these specific scenarios for coal tar pitch use. At the graphite electrode production the loss in weight during baking is about 15% (EC, 2001a). For the ferro-alloy production the anode consumption is assumed to be about 0.1 tonne anode per tonne product (see Table B.2.4). The metal production sites are also assumed to be self-sufficient with respect to green paste demand.

Effluent flows

The release scenarios can be divided in those applying dry abatement technologies (dry alumina scrubbers or electrostatic precipitation) and those applying wet abatement technologies (mainly wet scrubbers). In the scenarios, which consider the wet abatement technology, effluent flows are relevant. From Søderberg primary aluminium production sites in Norway an average scrubber effluent flow rate of 440 m^3/tonne aluminium was estimated from ventilation air scrubbers. From a combined anode and aluminium production plant in the Netherlands the scrubber effluent volume was calculated to be 210 m^3/tonne of anode produced (Pechiney, 2002). Alcoa (2002) reported a scrubber effluent volume of 188 m^3/tonne of aluminium produced for combined anode and aluminium production site. EC (2001a) reports wastewater effluents from sea water scrubbers in the range of 10-300 m^3/tonne aluminium. The average effluent from the Norwegian Søderberg sites is used for the scenario calculations. For the preparation of green anodes wastewater results from cooling water, amongst used to cool the green anodes. OSPAR (2001) reports 6 m^3 cooling water per tonne paste from Søderberg paste production. Cooling water consumption in anode production was reported to be 3 m^3/tonne anode by US-DOE (2003). Alcoa (2002) gives a cooling water consumption rate of about 1 m^3/tonne anode. The data from OSPAR are used for the scenario calculations. These data are also used for the generic scenarios for the graphite production industry and ferro-alloy industry.

Emission factors

The selected realistic worst case emission factors for the production of graphite electrodes are shown in Table B.9.31.

The realistic worst case emission factor for green paste preparation for air and water are 0.4 and 0.05 g benzo(a)pyrene per tonne paste, respectively.

Table B.9.31. Realistic worst case scenarios for the industrial use of coal tar pitch.

Scenario [a]	Process	Production volume [b] (tonnes/year)	Emission factors benzo(a)pyrene (g/tonne)		Emission (kg/year) benzo(a)pyrene		Effluent (m^3/day)
			air	water	air	water	
	Dry scrubbers/ESP						
Graphite	Graphite electrodes [c]	55,000	0.3		16.5		
	Wet scrubbers						
Ferro-alloy	Ferro-alloy	200,000	0.32	0.054	64	10.8	241,000
	paste preparation	20,000	0.4	0.05	8	1	330

[a] In this column the scenario names are given. These names will be used in the remainder of the document, for instance in the headers of the tables presenting the results of the scenario calculations; [b] Amount of graphite electrodes produced for the graphite and carbon scenarios; [c] Air emissions from paste preparation (mixing) are also treated by dry cleaning systems and are already included in the emission factor to air.

For graphite electrode baking it should be noted that according to the industry emissions to water are not to be expected at facilities applying 'dry' abatement techniques. One scenario presented here for graphite electrode baking reflects facilities applying 'dry' abatement techniques (dust injection in combination with bag filters) with closed cooling water system for the green anodes. The emission factor for air of 0.30 g benzo(a)pyrene per tonne (see Table B.9.26) was taken for this scenario, which also include paste preparation. Based on information provided by industry the emission of PAHs to water is not further considered in this risk assessment.

For ferro-alloy production the risk assessment is focussed on facilities applying wet scrubbers located near to coast which discharge the waste water to the marine environment. An emission factor to air and water of 0.32 and 0.054 g benzo(a)pyrene per tonne of product is used, respectively (see Section B.9.5.2.2).

All processes in the processing scenarios are assumed to be continuous and therefore the number of working days is taken to be 365 days per year for all processes.

Emission profiles are not available for all processes. For air there was no emission profile available for processes which apply electric arc furnaces with Søderberg electrodes. As a substitute the emission profile for VSS technology using wet scrubbers was used. For water there were only emission profiles for the aluminium production using the Søderberg technology and the green paste preparation process. For the scenarios representing electric arc furnaces applying Søderberg electrodes and anode baking process in combination with wet scrubbers, the emission profiles for water from the aluminium production using the Søderberg technology is used as an alternative.

B.9.5.3 Environmental concentrations

B.9.5.3.1 Aquatic compartment (incl. sediment)

In view of the strong contribution of the unintentional sources to the regional background concentration, it was decided to present C_{local} and PEC regional separately to get a better understanding of the additional risk that is caused by the emission sources under investigation. As sufficient monitoring data are available no separate calculation of the regional PECs had been performed. Since the different PAH emission sources are already mapped by several authorities (see Section B.8.2.2) it is not expected that a comparison between calculated regional PECs and monitoring data would elucidate that a significant emission source is overlooked.

Calculation of predicted environmental concentrations

The local concentrations for the selected 16 EPA PAHs in the aquatic compartment have been calculated according to the methods in the Technical Guidance Document (EC, 2003b) for both the CTPHT production sites and generic scenarios. The concentrations shown in the following tables do not yet include the regional background concentration.

Some CTPHT production plants discharge their waste water to a municipal sewage treatment plant or off-site biological waste water treatment plant. For these sites the sewage treatment model has been applied to calculate the fate in the sewage treatment plant. The emission from the production sites and the effluent flow rate of the external waste water treatment facility are required as input. For those production plants with on-site waste water treatment for which effluent concentrations have been reported the emission from these treatment plants has been used as input. Besides emissions from these sites the flow rate of the river is required as input. The model either calculates the emission from the sewage treatment plant to air, the concentration in sewage sludge and the concentration in the effluent. A detailed description of the sewage treatment plant model is given in the Technical Guidance Document (EC, 2003b).

$$C_{local(inf)} = \frac{E_{local(water)}}{effluent}$$

where $C_{local(inf)}$ = the concentration of the substance in untreated waste water (kg/L), $E_{local(water)}$ = local emission rate to (waste) water during an episode (kg/d), and effluent = the effluent discharge rate of the sewage treatment plant (L/d).

$$C_{local(eff)} = C_{local(inf)} \times F_{stp}$$

where $C_{local(eff)}$ = concentration of the substance in the sewage treatment plant effluent (mg/L), F_{stp} = the fraction of emission directed to water by the sewage treatment plant (= % to water in Table B.4.9), and $C_{local(inf)}$ = concentration of the substance in untreated waste water (kg/L).

From the effluent concentration the concentration in surface water and adjoining sediment is estimated according the following equations:

$$C_{local(water)} = \frac{C_{local(eff)}}{(1 + K_{p(susp)} \times Susp_{water} \times 10^{-6}) \times dilution}$$

where $C_{local(water)}$ = the local concentration in surface water during the emission episode (mg/L), $C_{local(eff)}$ = the concentration of the substance in the sewage treatment plant effluent (mg/L), $K_{p(susp)}$ = the solids-water partitioning coefficient of suspended matter (L/kg), $Susp_{water}$ = the concentration of suspended matter in water (mg/L), and dilution = the dilution factor.

The annual average concentration is calculated as follows:

$$C_{local(water,ann)} = C_{local(water)} \times \frac{T_{emission}}{365}$$

where $C_{local(water,ann)}$ = the annual average concentration in surface water (mg/L), $C_{local(water)}$ = the concentration in surface water during an emission episode (mg/L), and $T_{emission}$ = the number of emission days per year.

For the calculation of the concentration in sediment the properties of suspended matter are used. The concentration in bulk sediment can be derived from the corresponding water body concentration, assuming a thermodynamic partitioning equilibrium:

$$C_{local(sediment)} = C_{local(water)} \times \frac{K_{susp\text{-}water}}{\rho_{susp}} \times 1000$$

where $C_{local(sediment)}$ = the local concentration in sediment (mg/kg), $C_{local(water)}$ = the concentration in surface water during an emission episode (mg/L), $K_{susp\text{-}water}$ = the suspended matter-water partition coefficient (m^3/m^3), and ρ_{susp} = the bulk density of suspended matter (kg/m^3).

Table B.9.32. Local concentration during emission episode in surface water for the production sites.

Substance	Site 1 [a]	Site 3 [b]	Site 4 [a]	Site 5 [a]	Site 6 [a]	Site 7 [a]	Site 8 [a]	Site 9 [a]
Naphthalene	0.0045	0.27	0.7	2.1	0.68	0.037	2.9	1.0
Acenaphthene	0.00086	0.03	0.4	0.72	0.35	0.0081	2.5	0.5
Acenaphthylene	n.d.	0.07	1.6	3.7	0.046	0.010	2.3	0.5
Fluorene	0.0006	0.03	0.4	0.69	0.19	0.015	0.3	0.5
Anthracene	0.00011	0.05	0.7	0.81	0.22	0.0045	0.1	0.5
Phenanthrene	0.0010	0.03	1.1	0.016	0.58	0.069	0.4	0.5
Fluoranthene	0.00034	0.02	5.0	0.25	0.49	0.021	0.5	0.4
Pyrene	0.0019	0.08	3.9	0.22	0.31	0.024	0.6	0.5
Benz(a)anthracene	0.000024	0.004	2.6	0.024	0.045	0.0043	0.1	0.6
Chrysene	0.000027	0.005	2.4	0.0034	0.046	0.0048	0.1	0.6
Benzo(a)pyrene	0.000017	0.006	7.6	0.0022	0.041	0.0031	0.0	0.9
Benzo(b)fluoranthene	0.000018	0.003	12.0	0.0023	0.031	0.0048	0.0	0.5
Benzo(k)fluoranthene	0.000018	0.003	3.8	0.0023	0.028	0.0016	0.0	0.5
Benzo(ghi)perylene	0.000015	0.002	4.2	0.0019	0.018	0.0023	0.0	0.8
Dibenzo(a,h)anthracene	0.000009	0.0	0.8	0.0012	0.014	0.00037	0.0	0.5
Indeno(123-cd)pyrene	0.000008	0.001	2.6	0.0010	0.011	0.0015	0.0	0.5

[a] Concentration in fresh water (ng/L); [b] Concentration in marine water (ng/L).

Calculation of C_{local} for production

The local concentration in surface water and sediment for the different production sites are given in Table B.9.32 and Table B.9.33, respectively. For all sites, except site 3 and 5, site specific

information on river flow is available. For these sites the dilution factor is set accordingly. For site 3 and 5 de default dilution factor as recommended in the EU TGD (EC, 2003b) is applied: 10 for fresh water and 100 for marine water. When the reported on-site emissions are discharged to off-site wastewater treatment facilities (sewage treatment plant) the sewage treatment plant-model is used in the calculations with the appropriate effluent flow of the off-site sewage treatment plant (site 1, 3, 5 and 7).

Table B.9.33. Local concentration during emission episode in sediment for the production sites.

Substance	Site 1 [a)]	Site 3 [b)]	Site 4 [a)]	Site 5 [a)]	Site 6 [a)]	Site 7 [a)]	Site 8 [a)]	Site 9 [a)]
Naphthalene	0.00064	0.0003	0.10	0.83	0.10	0.0060	0.45	0.14
Acenaphthene	0.00055	0.022	0.27	1.2	0.23	0.0055	1.7	0.31
Acenaphthylene	n.d.	0.020	0.46	2.7	0.013	0.0029	0.64	0.13
Fluorene	0.00060	0.035	0.44	2.0	0.21	0.017	0.35	0.51
Anthracene	0.00033	0.19	2.4	6.9	0.69	0.015	0.31	1.4
Phenanthrene	0.0024	0.065	2.8	0.10	1.4	0.17	1.1	1.1
Fluoranthene	0.0034	0.26	55	6.9	5.1	0.23	5.1	4.3
Pyrene	0.012	0.53	26	3.7	2.0	0.16	3.9	2.7
Benz(a)anthracene	0.0012	0.23	147	3.5	2.4	0.23	6.4	29
Chrysene	0.0011	0.21	110	0.38	2.0	0.21	4.6	25
Benzo(a)pyrene	0.0015	0.27	736	0.51	3.7	0.29	2.8	78
Benzo(b)fluoranthene	0.0015	0.23	1104	0.51	2.7	0.43	2.9	38
Benzo(k)fluoranthene	0.0015	0.23	345	0.51	2.3	0.14	2.9	38
Benzo(ghi)perylene	0.0016	0.30	506	0.55	2.0	0.26	2.0	83
Dibenzo(a,h)anthracene	0.0019	0.36	166	0.64	2.9	0.078	1.5	101
Indeno(123-cd)pyrene	0.0020	0.37	690	0.69	2.8	0.38	1.7	106

[a)] Concentration in fresh water sediment (µg/kg_{dwt}); [b)] Concentration in marine water sediment (µg/kg_{dwt}).

Calculation of C_{local} for industrial/professional use

With respect to the industrial uses considered the emissions are specified in the fraction dissolved and bound to particles. Based on the considerations given in Section B.4.1.2 and the uncertainties on this topic, it was decided not to include a correction for binding to soot-like materials in the current risk assessment. Therefore, the calculation of the concentration in surface water and sediment will be based on the total concentration in effluent and the partitioning based on the coefficients mentioned in Section B.4.1.2.

Table B.9.34. Local concentrations during emission episode in sea water, marine sediment, fresh water, and fresh water sediment for ferro-alloy producing industry (including paste preparation).

Environmental compartment	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(k)fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)anthracene	Indeno(123-cd)pyrene
Sea water (ng/L)	0.5	2.8	0.5	1.6	33	18.8	27.5	17.4	2.8	5.9	1	1.9	NA	0.4	0.1	0.2
Marine sediment (µg/kg_{dwt})	0.1	1.7	0.1	1.7	10	42	266.5	107.2	140.5	240.5	79.7	153.5	NA	42	21.7	53.6

Table B.9.35. Local concentrations in water (fresh and marine) during the emission episode for primary aluminium production and anode baking facilities.

Use category	Site	Naphthalene (ng/L)	Acenaphthene (ng/L)	Acenaphthylene (ng/L)	Fluorene (ng/L)	Anthracene (ng/L)	Phenanthrene (ng/L)	Fluoranthene (ng/L)	Pyrene (ng/L)	Benz(a)anthracene (ng/L)	Chrysene (ng/L)	Benzo(a)pyrene (ng/L)	Benzo(b)fluoranthene (ng/L)	Benzo(k)fluoranthene (ng/L)	Benzo(ghi)perylene (ng/L)	Dibenzo(a,h)anthracene (ng/L)	Indeno(123-cd)pyrene (ng/L)
VSS II	S1	5.8E+00	2.4E+01	5.8E+00	1.6E+01	2.1E+01	1.9E+02	2.6E+02	1.6E+02	2.8E+01	5.4E+01	8.5E+00	2.6E+01		3.8E+00	1.0E+00	2.2E+00
VSS II	S3	1.7E+00	7.2E+00	1.7E+00	4.5E+00	6.0E+00	5.5E+01	7.6E+01	4.8E+01	8.2E+00	1.6E+01	2.5E+00	7.6E+00		1.1E+00	3.0E-01	6.4E-01
VSS II	S4	1.8E+02	7.4E+02	1.8E+02	4.7E+02	6.2E+02	5.7E+03	7.8E+03	5.0E+03	8.5E+02	1.6E+03	2.6E+02	7.9E+02		1.1E+02	3.1E+01	6.6E+01
PB+Anode I	PA1 [a)]	1.0E+01	4.2E+01	1.0E+01	2.7E+01	3.3E+02	1.2E+03	6.9E+02	5.3E+02	8.1E+01	7.0E+01	1.5E+01	3.2E+01		4.0E+00	3.4E+00	3.0E+00
PB+Anode I	PA2	1.6E+01	6.7E+01	1.6E+01	4.2E+01	5.3E+02	1.9E+03	1.1E+03	8.3E+02	1.3E+02	1.1E+02	2.4E+01	5.0E+01		6.3E+00	5.4E+00	4.7E+00
PB+Anode I	PA5	6.7E+01	2.8E+02	6.7E+01	1.7E+02	2.2E+03	8.0E+03	4.6E+03	3.5E+03	5.3E+02	4.6E+02	9.9E+01	2.1E+02		2.6E+01	2.3E+01	2.0E+01
PB+Anode I	PA7	4.5E+00	1.9E+01	4.5E+00	1.2E+01	1.5E+02	5.4E+02	3.1E+02	2.3E+02	3.6E+01	3.1E+01	6.7E+00	1.4E+01		1.8E+00	1.5E+00	1.3E+00
PB+Anode I	PA10	1.1E+00	4.6E+00	1.1E+00	2.9E+00	3.6E+01	1.3E+02	7.5E+01	5.7E+01	8.8E+00	7.5E+00	1.6E+00	3.5E+00		4.3E-01	3.7E-01	3.2E-01
PB+Anode I	PA11	4.0E-01	1.7E+00	4.0E-01	1.0E+00	1.3E+01	4.8E+01	2.7E+01	2.1E+01	3.2E+00	2.7E+00	5.9E-01	1.3E+00		1.6E-01	1.4E-01	1.2E-01
PB+Anode I	PA12 [a)]	3.2E-01	1.3E+00	3.2E-01	8.2E-01	1.0E+01	3.8E+01	2.1E+01	1.6E+01	2.5E+00	2.1E+00	4.6E-01	9.8E-01		1.2E-01	1.1E-01	9.2E-02
PB+Anode I	PA13	2.7E+01	1.1E+02	2.7E+01	7.1E+01	8.9E+02	3.2E+03	1.8E+03	1.4E+03	2.2E+02	1.9E+02	4.0E+01	8.5E+01		1.1E+01	9.1E+00	7.9E+00
PB+Anode I	PA14	2.5E+02	1.0E+03	2.5E+02	6.3E+02	8.0E+03	2.9E+04	1.7E+04	1.3E+04	1.9E+03	1.7E+03	3.6E+02	7.7E+02		9.6E+01	8.2E+01	7.1E+01
PB+Anode I	PA15	8.1E-03	3.3E-02	8.1E-03	2.1E-02	2.6E-01	9.6E-01	5.5E-01	4.2E-01	6.4E-02	5.5E-02	1.2E-02	2.5E-02		3.2E-03	2.7E-03	2.3E-03
Anode I	A1 [a)]	3.1E+02	1.3E+03	3.1E+02	8.1E+02	1.0E+04	3.7E+04	2.1E+04	1.6E+04	2.5E+03	2.1E+03	4.6E+02	9.8E+02		1.2E+02	1.0E+02	9.1E+01

[a)] Concentration in fresh surface water.

Table B.9.36. Local concentrations in sediment (fresh and marine) during the emission episode for primary aluminium production and anode baking.

Use category	Site	Naphthalene (µg/kg$_{dwt}$)	Acenaphthene (µg/kg$_{dwt}$)	Acenaphthylene (µg/kg$_{dwt}$)	Fluorene (µg/kg$_{dwt}$)	Anthracene (µg/kg$_{dwt}$)	Phenanthrene (µg/kg$_{dwt}$)	Fluoranthene (µg/kg$_{dwt}$)	Pyrene (µg/kg$_{dwt}$)	Benz(a)anthracene (µg/kg$_{dwt}$)	Chrysene (µg/kg$_{dwt}$)	Benzo(a)pyrene (µg/kg$_{dwt}$)	Benzo(b)fluoranthene (µg/kg$_{dwt}$)	Benzo(k)fluoranthene (µg/kg$_{dwt}$)	Benzo(ghi)perylene (µg/kg$_{dwt}$)	Dibenzo(a,h)anthracene (µg/kg$_{dwt}$)	Indeno(123-cd)pyrene (µg/kg$_{dwt}$)
VSS II	S1	8.0E-01	1.5E+01	1.5E+00	1.6E+01	6.1E+01	4.3E+02	2.5E+03	9.6E+02	1.4E+03	2.1E+03	7.1E+02	2.1E+03		3.9E+02	2.0E+02	5.1E+02
VSS II	S3	2.3E-01	4.4E+00	4.4E-01	4.7E+00	1.8E+01	1.3E+02	7.4E+02	2.8E+02	4.1E+02	6.3E+02	2.1E+02	6.2E+02		1.1E+02	5.8E+01	1.5E+02
VSS II	S4	2.4E+01	4.6E+02	4.6E+01	4.8E+02	1.8E+03	1.3E+04	7.7E+04	2.9E+04	4.2E+04	6.5E+04	2.2E+04	6.4E+04		1.2E+04	6.0E+03	1.5E+04
PB+Anode I	PA1 [a)]	1.4E+00	2.6E+01	2.7E+00	2.7E+01	9.9E+02	2.8E+03	6.8E+03	3.1E+03	4.1E+03	2.8E+03	1.3E+03	2.6E+03		4.1E+02	6.7E+02	7.0E+02
PB+Anode I	PA2	2.2E+00	4.1E+01	4.2E+00	4.3E+01	1.6E+03	4.4E+03	1.1E+04	4.9E+03	6.4E+03	4.4E+03	2.0E+03	4.1E+03		6.4E+02	1.1E+03	1.1E+03
PB+Anode I	PA3	7.2E+00	1.6E+00	3.8E+00	9.8E-01	3.4E-01	4.4E-01	1.0E-01	1.7E-01	2.0E-02	2.5E-02	1.2E-02	1.2E-02		9.8E-03	5.1E-03	4.3E-03
PB+Anode I	PA5	9.4E+00	1.7E+02	1.8E+01	1.8E+02	6.5E+03	1.8E+04	4.4E+04	2.0E+04	2.7E+04	1.8E+04	8.2E+03	1.7E+04		2.7E+03	4.4E+03	4.6E+03
PB+Anode I	PA7	6.3E-01	1.2E+01	1.2E+00	1.2E+01	4.4E+02	1.2E+03	3.0E+03	1.4E+03	1.8E+03	1.2E+03	5.6E+02	1.2E+03		1.8E+02	3.0E+02	3.1E+02
PB+Anode I	PA10	1.5E-01	2.8E+00	2.9E-01	2.9E+00	1.1E+02	3.0E+02	7.3E+02	3.4E+02	4.4E+02	3.0E+02	1.4E+02	2.8E+02		4.4E+01	7.2E+01	7.5E+01
PB+Anode I	PA11	5.6E-02	1.0E+00	1.1E-01	1.1E+00	3.9E+01	1.1E+02	2.7E+02	1.2E+02	1.6E+02	1.1E+02	4.9E+01	1.0E+02		1.6E+01	2.6E+01	2.7E+01
PB+Anode I	PA12 [a)]	4.4E-02	8.1E-01	8.2E-02	8.3E-01	3.0E+01	8.6E+01	2.1E+02	9.6E+01	1.2E+02	8.5E+01	3.9E+01	8.0E+01		1.3E+01	2.1E+01	2.1E+01
PB+Anode I	PA13	3.8E+00	7.0E+01	7.1E+00	7.2E+01	2.6E+03	7.4E+03	1.8E+04	8.3E+03	1.1E+04	7.4E+03	3.3E+03	6.9E+03		1.1E+03	1.8E+03	1.9E+03
PB+Anode I	PA14	3.4E+01	6.3E+02	6.4E+01	6.5E+02	2.4E+04	6.7E+04	1.6E+05	7.5E+04	9.7E+04	6.6E+04	3.0E+04	6.2E+04		9.8E+03	1.6E+04	1.7E+04
PB+Anode I	PA15	1.1E-03	2.1E-02	2.1E-03	2.1E-02	7.8E-01	2.2E+00	5.3E+00	2.5E+00	3.2E+00	2.2E+00	9.9E-01	2.0E+00		3.2E-01	5.3E-01	5.5E-01
Anode I	A1 [1)]	4.3E+01	8.0E+02	8.2E+01	8.3E+02	3.0E+04	8.5E+04	2.1E+05	9.5E+04	1.2E+05	8.5E+04	3.8E+04	7.9E+04		1.2E+04	2.0E+04	2.1E+04

[a)] concentration in fresh surface water

The concentration in sea/fresh water and marine/fresh water sediment for the ferro-alloy production plants is given in Table B.9.34. As mentioned in Section B.9.5.2.2 the release of PAHs from graphite is considered negligible. For primary aluminium production and anode baking facilities these are presented in Table B.9.35 and Table B.9.36, respectively.

All primary aluminium production plants, except sites PA12 and PA1, are located near the coastal zone and therefore the calculated concentrations refer to the marine surface water compartment. This exception also holds for the anode plant A1. As was indicated in the EU TGD (EC, 2003b) for the sea water compartment a dilution factor of 100 has to be used. Only where the point of release is more subject to tidal influence a default dilution factor of 500 could be used. Many of the primary aluminium production sites are located either at the coast of the Atlantic Ocean or the North Sea. A dilution factor of 10 is thought to be more representative for the Mediterranean Sea or comparable waters, which have less tidal influence (EC, 2003b). It should be noted though that the two sites in Italy and one site in Greece are located at the coast of the Mediterranean Sea. A dilution factor of 100 will be used for all scenarios especially those using sea water scrubbers with the exception of the scenario and sites with no discharge to water (Graphite) and the sites located at the Mediterranean Sea and Gulf of Bothnia (S4 ,PA10, PA14 and PA15). For the latter a default dilution factor of 10 is assumed.

It should be stressed that applying fixed default dilution factors leads to the situation where high effluents flows will result in lower concentrations than low effluent flows with the same amount of substance discharged. The default river flow as derived from the TGD (EC, 2003b) is 20,000 m^3 per day and for the marine environment this will be 200,000 m^3 per day, using the default effluent flow rate of 2,000 m^3 per day and a dilution factor of 10 for fresh surface water and 100 for the marine environment respectively. For the primary aluminium and ferro-alloy production plants using wet scrubbers the effluent flow is more than 100 times higher than the default flow.

Furthermore it is assumed that the (scrubber) effluent is not treated in a waste water treatment plant; although the rapporteur is familiar with the fact the scrubber effluent is sometimes treated in so called settling ponds.

Measured levels

Water phase - Local

No monitoring data have been provided by industry on water concentrations in the vicinity of the sources under investigation. Naes *et al.* (1995) reported for two Norwegian aluminium production sites at Lista and Vefsnfjord typical PAH concentrations in scrubber waste water measured in the year 1991 (see Table B.9.52).

Table B.9.37. PAH concentrations in scrubber waste water of two Norwegian aluminium production sites measured in 1991.

Site	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(j,k)fluoranthene	Benzo(ghi)perylene	Indeno(123-cd)pyrene	total
Lista	100	500	600	400	300	600	1100	600	600	500	7900
Vefsnfjord	21	70	905	960	215	236	850	-	212	150	4840

Data taken from Naes *et al.* (1995), values in ng/L.

More recently, Axelman *et al.* (1999) measured in 1996 the concentration of the less volatile PAHs (heavier than fluorene) in sea water at a site contaminated by discharged PAHs from an aluminium reduction plant at Lista in southern Norway and at a reference site approximately 200 km from the contaminate site near Arendal. The concentration of PAHs, shown in Table B.9.38, were specified for those in the dissolved, colloidal and particulate fractions.

Table B.9.38. Local PAH concentrations in sea water measured in 1996 near an aluminium production plant and at a reference site in Norway.

Substance	Reference site			Smelter		
	colloids	dissolved	particles	colloids	dissolved	particles
Anthracene	0.06	0.14		11	2.7	4.2
Phenanthrene	1.6	1.2	0.6	780	99	33
Fluoranthene	0.58	0.63	0.86	1000	120	280
Pyrene	16	0.14	0.42	280	33	110
Benz(a)anthracene	0.05	0.03		41	4.1	90
Chrysene	0.2	0.09	0.56	150	17	380
Benzo(a)pyrene	0.1			7.8	0.26	57
Benzo(k)fluoranthene	0.23		0.15	16	1.3	100
Benzo(ghi)perylene				8.0		61
Indeno(123-cd)pyrene				9.1		73

Data taken from Axelman *et al.* (1999), values in ng/L.

Table B.9.39. The range of 90-percentile and mean PAH water concentrations in different Dutch rivers.

Compound	2000		2001		2002	
	90-percentile (µg/L)	Mean (µg/L)	90-percentile (µg/L)	Mean (µg/L)	90-percentile (µg/L)	Mean (µg/L)
Acenaphthene	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Anthracene	< 0.01	< 0.01	< 0.01-0.01	< 0.01-<0.03	< 0.01-0.03	< 0.01-< 0.04
Benz(a)anthracene	0.01-0.02	< 0.02-<0.01	0.02	< 0.02-<0.03	< 0.01-0.07	< 0.01-0.026
Benzo(a)pyrene	0.016-0.04	< 0.05-0.03	0.01-1.63	< 0.0064-1.66	< 0.01-0.07	< 0.01-0.018
Benzo(b)fluoranthene	0.02-0.06	< 0.02-0.047	< 0.005-0.03	< 0.005-0.025	< 0.01-0.04	< 0.01-0.025
Benzo(ghi)perylene	0.0096-0.03	< 0.005-0.025	< 0.02-1.33	< 0.0057-0.99	< 0.01-0.03	< 0.01
Benzo(k)fluoranthene	< 0.01-0.03	< 0.005-0.017	< 0.01-0.93	< 0.005-0.69	< 0.01-0.02	< 0.01-< 0.03
Fluoranthene	0.03-0.13	< 0.02-0.09	0.02-3.11	< 0.012-2.3	0.01-3.11	< 0.01-2.3
Indeno(123-cd)pyrene	< 0.01-0.05	< 0.005-0.0079	< 0.01-1.61	< 0.01-1.16	< 0.01-0.03	< 0.01-< 0.1
Naphthalene	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Phenanthrene	0.02-0.03	< 0.03	0.02	< 0.03	< 0.01- 0.08	< 0.01-0.05
Dibenzo(a,h)anthracene	< 0.01	< 0.01-<0.03	< 0.01	< 0.01-<0.02	< 0.01	< 0.02
Chrysene	< 0.01-0.03	< 0.01-<0.011	0.02	< 0.02-<0.03	< 0.01-0.07	< 0.01-0.024
Pyrene	0.03-0.04	< 0.03-<0.05	0.02	< 0.03	0.02-0.14	< 0.02-0.05

Data from the Dutch Institute for Inland Water Management and Waste Water Treatment (RIZA); n.a. No data are available.

Water phase - Regional-Fresh water

In the Netherlands the concentrations of several PAHs are measured yearly in different rivers (RIZA). In 2001 the concentrations of benzo(a)pyrene, benzo(ghi)perylene, fluoranthene and indeno(123-cd)pyrene were > 100 ng/L. In 2002 only fluoranthene and pyrene concentrations were

> 100 ng/L. Table B.9.39 shows the concentrations measured in 2000, 2001 and 2002. More recent data were not available.

Denzer *et al.* (1999; COMMPS database) collected information on concentrations of a number of PAHs in the surface water throughout the EU. Table B.9.40 shows the years and member states for which monitoring data were collected and the size of the data sets. The 90-percentile concentrations ranged from 1.68 µg/L to 0.025 µg/L, measured for naphthalene and benzo(k)fluoranthene, respectively (see Table B.9.41).

Table B.9.40. Years for which monitoring data were provided and size of the data sets after initial quality and plausibility check.

	Member State															
Year	**AU**	**B**	**D**	**DK**	**E**	**F**	**FIN**	**GR**	**I**	**IRL**	**LUX**	**NL**	**Pt**	**S**	**UK**	**Σ all MS**
unknown								1								1
1994	2,397		36,674				2,700			420	247				178,660	221,098
1995	1,740	2,882	40,963			8,341	2,620		4,940	652	247			9,549	160,180	232,114
1996	3,265	9,418	17,389		1,045	7,612	2,498	1,327	2,704	982	132	6,997		11,179	175,991	240,539
1997	6,999	12,371	5,729	282	809		1,543	3,011	50			6,866	8	11,399	7,031	56,098
1998	472		94					1,628								2,194
total	14,873	24,671	100,849	282	1,854	15,953	9,361	5,966	7,694	2,054	626	13,863	8	32,127	521,862	752,043

Data taken from Denzer *et al.* (1999). Values indicate the number of records available.

Table B.9.41. Results of the aggregation of monitoring data of PAH in surface water.

Compound	90-percentile (µg/L) [a,b]	median (µg/L) [a]	arithmetic mean (µg/L) [a]	st. dev. of mean (µg/L) [a]	number of sampling stations [c]	number of measurements [d]	number of measurements >DL [e]
Naphthalene	1.6825	0.0650	0.2673	0.5004	9	95	32
Acenaphthene	0.4175	0.0103	0.0615	0.1314	10	67	40
Anthracene	0.0834	0.0042	0.0141	0.0257	10	54	45
Fluoranthene	0.0823	0.0160	0.0645	0.1719	49	832	587
Benz(a)anthracene	0.0831	0.0211	0.0278	0.0176	14	116	72
Benzo(a)pyrene	0.0272	0.0070	0.0123	0.0094	38	579	388
Benzo(b)fluoranthene	0.0484	0.0088	0.0175	0.0143	41	768	448
Benzo(k)fluoranthene	0.0248	0.0044	0.0091	0.0070	29	451	294
Benzo(ghi)perylene	0.0470	0.0080	0.0171	0.0108	29	446	286
Indeno(123-cd)pyrene	0.0936	0.0335	0.0355	0.0186	11	70	54

Data taken from Denzer *et al.* (1999). [a] EU-level value; [b] These values are used for exposure scoring; [c] Number of sampling stations from which data were used to calculate the exposure concentrations; [d] Number of measurements used to calculate the exposure concentrations; [e] Number of used measurements with concentrations higher than the corresponding determination limit (DL).

Water phase - Regional-Marine water

As stated by OSPAR in their quality status report (OSPAR, 2000), high variability is typical for total and individual PAH concentrations in sea water. PAH concentrations in Atlantic sea water range from 0.3 ng/L for individual, more water-soluble, lower molecular weight PAHs (two- and three-ringed compounds) to less than 0.001 ng/L for the high molecular weight PAHs (five- or more-ringed compounds) (see Table B.9.42). Higher concentrations were generally found in coastal and estuarine samples with total PAH concentrations ranging from not detectable to 8500 ng/L. Also high concentrations are found in sea surface microlayer. Cincinelli *et al.* (2001) reported a total PAH concentration in Mediterranean Sea surface water and the microlayer of 3.4 and 47.5 µg/L, respectively. The PAH levels in seawater from the German coast varied over one order of

magnitude depending on the sampling site. In open seawater, the concentrations of two- to four-ringed PAHs (naphthalene, fluorene, phenanthrene, fluoranthene, and pyrene) were 0.1-5 ng/L, and those of five- to six-ringed PAHs ranged from < 0.01 to 0.2 ng/L. Near the coast, the concentration of five- to six-ring PAHs increased with the content of particles, to which they have greater affinity than two- to four-ringed PAHs (ICCP, 2000 in German Federal Office for Sea Navigation and Hydrography, 1993). The PAH concentrations measured by Axelman *et al.* (1999) at a reference site in Norway were in the same order of magnitude (see Table B.9.38).

Table B.9.42. Ranges in background/reference concentrations of PAHs in surface water for application in selected regions of the OSPAR area.

PAH	northern North Sea	central and southern North Sea	North-east Atlantic
Benzo(a)pyrene	0.002- 0.005	0.002-0.004	0.001
Fluoranthene	0.073-0.285	0.104-0.264	0.036-0.054
Benzo(b) fluoranthene	0.004-0.017	0.003-0.009	0.001-0.004
Pyrene	0.014-0.053	0.011-0.024	0.02-0.033

Values in ng/L. Data taken from OSPAR (2000).

Sediment - Local

Sediment concentrations in the vicinity of the sources under investigation are scare. Naes *et al.* investigated the occurrence of PAH in marine organisms and sediments from aluminium smelters using Søderberg technology, and manganese-alloy production plants discharge in Norway (Naes *et al.*, 1995, Naes & Oug, 1998). Surface sediment concentrations were in the range of some hundred mg total PAH/kg_{dwt}. An overview of surface (top 1-2 cm) concentrations of total PAHs and benzo(a)pyrene found in sediment from different fjords near five smelters in the period 1983-1989 is given in Table B.9.43.

Table B.9.43. Concentrations of PAH in surface sediment from different aluminium smelter discharges.

Site	Sampling year	Total PAH (mg/kg_{dwt})	Benzo(a)pyrene (mg/kg_{dwt})	Sampling locations (km from source)
Årdalsfjord [a)]	1983/1989	45-790	4-83	0.5-1.5
Sunndalsfjord [a)]	1986	1.1-770	0.07-34	0.05-50
	1980	6.4-66		0.5 -3
Karmsund [a)]	1988	8.8-90	0.6-6.7	0.5-10
Vefsnfjord [a)]	1989	1.1-34	0.005-2.4	0.1-25
Høyangerfjord [a)]	1988	0.3-103	0.008-2.9	0.15-7
Feda [b)]	1984	0.3-144		0.1-19
Sauda [b)]	1986	0.8-87		0.3-20
	1990	8.3-499		0.4-2.5

Data taken from Naes *et al.* (1995) and Naes & Oug (1998). [a)] Aluminium production plant; [b)] Manganese-alloy production plant.

Sediment - Regional-Fresh water

In the Netherlands the concentrations of several PAHs are measured yearly at 12 different lake sediment and river sediment monitoring points (RIZA). The ranges of mean sediment concentrations are presented in Table B.9.44. 90 percentile values were not available. No sediment data was available for the years after 2000. All upper limits of the presented range of mean concentrations belong to one site (Sas van Gent). Without that site the upper limit of mean concentrations would be more than a factor 10 lower and range up to 2.3 mg/kg.

Denzer *et al.* (1999; COMMPS database) collected information on concentrations of a number of PAHs in the sediment throughout the EU. Table B.9.45 shows the years and the member states for which monitoring data were collected and the size of the data sets. The 90-percentile of sample station arithmetic mean concentrations ranged from 468 to 3410 µg/kg measured for anthracene and phenanthrene, respectively (see Table B.9.46). No median or mean values in sediment were available in the COMMPS database.

Table B.9.44. The range of mean PAH sediment concentrations in different Dutch rivers for 2000.

Compound	Mean PAH concentration (mg/kg)
Acenaphthene	n.a.
Anthracene	<0.05-6.87
Benz(a)anthracene	<0.05-25.7
Benzo(a)pyrene	<0.05-29.3
Benzo(b)fluoranthene	<0.05-31.3
Benzo(ghi)perylene	<0.05-18.3
Benzo(k)fluoranthene	<0.05-12.8
Fluoranthene	<0.1-55.1
Indeno(123-cd)pyrene	<0.05-25.3
Naphthalene	n.a.
Phenanthrene	<0.5-25.1
Dibenzo(a,h)anthracene	<0.05-4.16
Chrysene	<0.01-33.8
Pyrene	<0.05-44.9

Data from the Dutch Institute for Inland Water Management and Waste Water Treatment (RIZA).

Table B.9.45. Years for which monitoring data were provided and size of the data sets after initial quality and plausibility check.

	Member State										
Year	AU	B	D	DK	E	F	FIN	NL	S	UK	Σ all MS
unknown		5	18			6		29	24	43	125
1994	159	1,274	16,778							157	18,368
1995	122	2,800	17,993			4,497					25,412
1996	135		8,923		592	4,185		4,141		167	18,143
1997	72		501	583	449		125	5,078			6,808
1998	24										24
total	512	4,079	44,213	583	1,041	8,688	125	9,248	24	367	68,880

Data taken from Denzer *et al.* (1999). Values indicate the number of records available.

Regional-Marine water

As stated by OSPAR in their quality status report (OSPAR, 2000), on a worldwide basis, background values for PAHs in sediments appear to be within the range 0.01 to approximately 1 mg/kg_{dwt}. The highest levels of oil in bottom sediments typically occur in river mouths, estuaries, and bays, as well as in areas of regular shipping, oil production and transportation. From a sediment survey, which described the presence of PAHs in twenty-two estuaries in Western Europe, it appeared that fluoranthene was the most prominent PAH. Total PAH concentrations in the twenty two estuaries were between 200 $µg/kg_{dwt}$ (Wadden Sea) and over 6,000 $µg/kg_{dwt}$ (Scheldt Estuary). The background reference concentrations (BRCs) of a number of PAHs established by OSPAR which are considered to be the typical range of concentrations found in uncontaminated water in the

OSPAR area are presented in Table B.9.47. Measurements in twelve sea lochs on the west coast of Scotland showed that most sediments concentrations were in the range of the BRCs of OSPAR (Webster *et al.*, 2004).

Table B.9.46. Results of the aggregation of monitoring data of PAH in sediment.

Compound	90-percentile (µg/kg) [a,b]	median (µg/L) [a]	arithmetic mean (µg/L) [a]	st. dev. of mean (µg/L) [a]	number of sampling stations [c]	number of measurements [d]	number of measurements >DL [e]
Naphthalene	1,940.45	146.87	556.01	1,064.7	38	291	256
Acenaphthene	678.38	43.68	155.42	155.68	44	292	234
Acenaphthylene	565.50	57.20	137.95	153.11	14	15	15
Fluorene	736.40	228.75	388.70	551.55	41	112	96
Anthracene	468.40	174.14	206.68	131.70	55	580	520
Phenanthrene	3,410.50	610.00	1,249.33	897.37	62	705	638
Fluoranthene	2,562.15	580.00	1,329.16	2,476.5	226	1,722	1,701
Pyrene	3,162.00	550.00	1,482.10	3,273.2	47	120	110
Benz(a)anthracene	1,361.61	299.58	612.83	576.19	62	703	623
Chrysene	4,103.50	506.25	1,666.76	3,660.3	29	93	92
Benzo(a)pyrene	976.00	310.00	516.63	655.24	215	1,825	1,718
Benzo(b)fluoranthene	1,385.68	340.00	742.29	1,649.61	215	1,831	1,755
Benzo(k)fluoranthene	663.16	195.25	381.44	970.76	209	1,820	1,678
Benzo(ghi)perylene	1,000.00	295.00	496.92	512.38	190	1,709	1,639
Dibenzo(a,h)anthracene	nd	nd	nd	nd	nd	nd	nd
Indeno(123-cd)pyrene	1,300.00	310.00	589.07	950.19	163	306	288

Data taken from Denzer *et al.* (1999). [a] EU-level value; [b] These values are used for exposure scoring; [c] Number of sampling stations from which data were used to calculate the exposure concentrations; [d] Number of measurements used to calculate the exposure concentrations; [e] Number of used measurements with concentrations higher than the corresponding determination limit (DL).

Table B.9.47. Ranges in background/reference concentrations of PAHs in surface sediments for application in selected regions of the OSPAR area.

PAH	northern North Sea/ Skagerrak	southern North Sea	Arctic Ocean/Iceland Sea
Benzo(a)pyrene	8.8-112	< 0.2-51	1.0-3.8
Fluoranthene	14-160	0.72-97	1.5-7.5
Benzo(b+k)fluoranthene	46-434	1.1-142	7.4-30
Pyrene	11-128	0.6-78	1.7-6.4

Values in µg/kg_{dwt}. Data taken from OSPAR (2000).

Production

As no monitoring data for the production site are available no comparison between predicted and measured levels can be made. In comparison to the regional concentrations reported in COMMPS database, the water concentration predicted for site 1 and 5 up to 9 were well lower. For site 4 the predicted water concentrations were comparable to the median values. The marine water concentration for site 3 was comparable to the background reference concentrations reported by OSPAR.

Comparison between predicted and measured levels

The calculated local concentrations in sediment for site 1 and 5 up to 9 were well below the 90 percentile regional concentrations reported in COMMPS database. The predicted concentration for site 4 were much closer the 90 percentile. Of the COMMPS database, especially the high molecular PAHs. The predicted marine sediment concentrations for benzo(a)pyrene, fluoranthene,

benzo(b+k)fluoranthene and pyrene at site 3 were comparable or lower than the background reference concentrations used by OSPAR. For the other PAHs no background reference concentrations are given and consequently no comparison can be made.

Industrial use

In comparison to the background levels reported by OSPAR and measured on a reference site by Axelman *et al.* (1999), the local concentrations (C_{local}) in marine water near anode and Vertical Stud Søderberg (VSS) plants and ferro-alloy plants were much higher. Also the calculated local concentrations in fresh water were much higher in comparison to the mean values in EU rivers, whereas the C_{local} in freshwater near ferro-alloy plants is comparable to the mean values in the EU rivers, although the calculated local concentrations for fluoranthene were higher. In the absence of monitoring data, no comparison can be made for the C_{local} near the emission points of anode plants and ferro-alloy plants. The monitoring data available in sea water in the vicinity for aluminium smelters using web scrubbers (Axelman *et al.*, 1999) were comparable to those predicted when the different forms (dissolved, colloids and particles) are added up.

The calculated local concentrations in sediment were for all applications much higher than the regional background concentrations, where the calculated local concentrations near ferro-alloy plants were much closer to these values. Like for the water phase the monitoring data for sediment near emission points are limited to aluminium smelters. The C_{local} for marine sediment near VSS plants is well within the range of benzo(a)pyrene concentrations measured in the vicinity near different smelters.

B.9.5.3.2 Terrestrial compartment

In view of the strong contribution of the unintentional sources to the regional background concentration, it was decided to present C_{local} and $PEC_{regional}$ separately to get a better understanding of the additional risk that is caused by the emission sources under investigation. As sufficient monitoring data are available no separate calculation of the regional PECs had been performed. Since the different PAH emission sources are already mapped by several authorities (see Section B.8.2.2) it is not expected that a comparison between calculated regional PECs and monitoring data would elucidate that a significant emission source is overlooked.

Calculation of predicted environmental concentrations

Local concentrations were calculated for agricultural soil and grassland according the EU TGD (EC, 2003b). These predicted environmental concentrations in soils take into account application of sewage sludge in agriculture and dry and wet deposition from the atmosphere.

With regard to the professional/industrial uses, the indirect releases of PAHs to soils via atmospheric deposition is the main route as release to a sewage treatment plant and consequently application of sewage sludge to agricultural soil, will not occur.

The local concentrations of the selected PAHs in the terrestrial compartment have been calculated according to the methods in the EU TGD (EC, 2003b). The concentrations in agricultural soil and grassland presented in Table B.9.48 up to Table B.9.52 do not include the regional background concentration.

The local concentration in soil results from the application of sewage sludge, if relevant and from dry and wet deposition from the atmosphere. Sludge application rates for agricultural land and grassland are 0.5 kg/m^2 and 0.1 kg/m^2, respectively. Atmospheric deposition is assumed to be a continuous flux throughout the year. Removal processes like degradation, volatilisation and leaching are also taken into account. To take accumulation into account it is assumed that sludge is applied once a year for ten consecutive years. The average concentration in soil is calculated for a period of 30 days for ecosystem assessment. This is done for the top layer of the soil compartment

which is of interest. A depth of 20 cm is taken for agricultural soil and a depth of 5 cm is taken for grassland.

Table B.9.48. Local concentrations in agricultural soil averaged over 30 days for the production sites.

Substance	Site 1	Site 3	Site 4	Site 5	Site 6	Site 7 a)	Site 8	Site 9
Naphthalene	221	2,210	1,053	988	121	51	143	130
Acenaphthene	34	88	31	208	18	9	21	21
Acenaphthylene	86	64	23	69	47	20	53	52
Fluorene	56	182	53	1,287	30	22	35	34
Anthracene	51	101	20	715	27	17	31	31
Phenanthrene	143	611	72	4,420	81	112	91	88
Fluoranthene	273	637	108	4,030	156	208	169	169
Pyrene	208	364	70	728	113	169	130	126
Benz(a)anthracene	442	390	121	819	247	169	273	260
Chrysene	494	780	130	1,560	273	182	312	299
Benzo(a)pyrene	1,690	1,144	416	1,014	884	416	1,287	988
Benzo(b)fluoranthene	8,190	5,460	2,080	598	4,550	1,950	5,200	5,070
Benzo(k)fluoranthene	2,990	2,600	793	1,690	1,560	663	1,950	819
Benzo(ghi)perylene	702	546	182	923	364	208	416	416
Dibenzo(a,h)anthracene	2,730	1,950	702	1,248	1,560	611	2,080	1,690
Indeno(123-cd)pyrene	1,209	910	312	988	650	338	1,170	728

Values expressed in ng/kg_{dwt}. a) Only for site 7 sludge from the municipal sewage treatment plant is spread on agricultural land.

Table B.9.49. Local concentrations in grassland averaged over 180 days for the production sites.

Substance	Site 1	Site 3	Site 4	Site 5	Site 6	Site 7 1)	Site 8	Site 9
Naphthalene	351	3,380	1,690	1,560	195	77	221	208
Acenaphthene	66	169	60	403	36	16	42	40
Acenaphthylene	169	122	44	130	88	36	101	99
Fluorene	109	364	103	2,470	60	27	68	65
Anthracene	100	208	40	1,430	56	25	62	61
Phenanthrene	299	1,196	143	8,710	156	95	182	182
Fluoranthene	546	1,287	221	8,060	299	182	338	325
Pyrene	416	728	143	1,430	221	143	260	247
Benz(a)anthracene	897	780	247	1,690	507	221	533	533
Chrysene	1,001	1,560	260	2,990	533	247	611	598
Benzo(a)pyrene	3,250	2,340	832	2,080	1,820	728	2,600	1,950
Benzo(b)fluoranthene	16,900	11,050	4,290	1,209	9,100	3,640	10,270	10,010
Benzo(k)fluoranthene	5,980	5,200	1,560	3,510	3,250	1,261	3,770	1,690
Benzo(ghi)perylene	1,430	1,105	351	1,820	741	325	845	819
Dibenzo(a,h)anthracene	5,590	3,900	1,430	2,470	2,990	1,196	4,160	3,250
Indeno(123-cd)pyrene	2,470	1,820	624	1,950	1,300	546	2,340	1,430

Values are total concentrations in ng/kg_{dwt}. 1) Only for site 7 sludge from the municipal sewage treatment plant is spread on agricultural land.

Table B.9.50. Local concentrations in agricultural soil over 30 days for primary aluminium production and anode baking.

Use cat.	Site	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(k)fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)-anthracene	Indeno(123-cd)pyrene
VSS II	S1	6.7E+02	1.4E+02	3.3E+02	1.3E+03	4.2E+02	3.6E+03	5.7E+03	2.6E+03	3.4E+03	7.9E+03	7.7E+03	1.5E+04		4.7E+03	2.0E+03	5.0E+03
VSS II	S3	1.7E+03	3.5E+02	8.5E+02	3.4E+03	1.1E+03	9.3E+03	1.5E+04	6.7E+03	8.7E+03	2.0E+04	2.0E+04	3.7E+04		1.2E+04	5.2E+03	1.3E+04
VSS II	S4	1.8E+03	3.7E+02	9.0E+02	3.6E+03	1.1E+03	9.9E+03	1.6E+04	7.1E+03	9.3E+03	2.2E+04	2.1E+04	4.0E+04		1.3E+04	5.5E+03	1.4E+04
SWPB	P7	1.0E-02		3.9E+00	2.9E+00			6.8E+02	2.5E+02	5.5E+02	9.6E+02	6.0E+02	3.7E+02	7.0E+02	3.9E+02	1.7E+02	2.5E+02
VSS I	S5	1.9E+03	4.0E+02	9.4E+02	3.7E+03	8.3E+02	4.2E+03	6.2E+03	4.2E+03	8.8E+03	2.1E+04	2.2E+04	5.6E+04		1.6E+04	2.9E+03	1.4E+04
VSS I	S6	1.8E+03	3.8E+02	9.1E+02	3.5E+03	8.0E+02	4.1E+03	6.0E+03	4.1E+03	8.5E+03	2.0E+04	2.1E+04	5.5E+04		1.5E+04	2.8E+03	1.4E+04
Anode I	PA1	7.0E+02	1.3E+02	6.1E+00	7.9E+02	7.2E+02	4.1E+03	6.0E+03	3.0E+03	6.1E+03	1.4E+04	5.7E+03	2.6E+04		5.5E+03	5.8E+03	5.8E+03
Anode I	PA2	3.0E+02	5.5E+01	2.6E+00	3.4E+02	3.1E+02	1.7E+03	2.5E+03	1.3E+03	2.6E+03	6.1E+03	2.4E+03	1.1E+04		2.3E+03	2.5E+03	2.5E+03
Anode I	PA3	2.7E-01	5.0E-02	2.3E-03	3.1E-01	2.8E-01	1.6E+00	2.3E+00	1.2E+00	2.4E+00	5.6E+00	2.2E+00	1.0E+01		2.1E+00	2.2E+00	2.2E+00
Anode I	PA4	3.1E+01	5.7E+00	2.7E-01	3.5E+01	3.2E+01	1.8E+02	2.6E+02	1.3E+02	2.7E+02	6.3E+02	2.5E+02	1.1E+03		2.4E+02	2.6E+02	2.6E+02
Anode I	PA5	1.9E+02	3.5E+01	1.6E+00	2.1E+02	1.9E+02	1.1E+03	1.6E+03	8.1E+02	1.7E+03	3.9E+03	1.5E+03	7.0E+03		1.5E+03	1.6E+03	1.6E+03
Anode I	PA6	2,8E+01	5,1E+00	2,4E-01	3,1E+01	2,8E+01	1,6E+02	2,4E+02	1,2E+02	2,4E+02	5,7E+02	2,2E+02	1,0E+03		2,2E+02	2,3E+02	2,3E+02
Anode I	PA6	1.8E+03	3.4E+02	1.6E+01	2.1E+03	1.9E+03	1.1E+04	1.6E+04	7.8E+03	1.6E+04	3.7E+04	1.5E+04	6.7E+04		1.4E+04	1.5E+04	1.5E+04
Anode I	PA7	2.8E+01	5.1E+00	2.4E-01	3.1E+01	2.8E+01	1.6E+02	2.4E+02	1.2E+02	2.4E+02	5.7E+02	2.2E+02	1.0E+03		2.2E+02	2.3E+02	2.3E+02
Anode I	PA8	6.7E+00	1.2E+00	5.8E-02	7.6E+00	6.9E+00	4.0E+01	5.8E+01	2.9E+01	5.9E+01	1.4E+02	5.5E+01	2.5E+02		5.3E+01	5.6E+01	5.6E+01
Anode I	PA9	1.6E+04	2.9E+03	1.4E+02	1.8E+04	1.6E+04	9.3E+04	1.3E+05	6.8E+04	1.4E+05	3.3E+05	1.3E+05	5.8E+05		1.2E+05	1.3E+05	1.3E+05
Anode I	PA10	1.7E+02	3.2E+01	1.5E+00	2.0E+02	1.8E+02	1.0E+03	1.5E+03	7.6E+02	1.5E+03	3.6E+03	1.4E+03	6.5E+03		1.4E+03	1.5E+03	1.5E+03
Anode I	PA11	6.7E+02	1.2E+02	5.8E+00	7.6E+02	6.9E+02	3.9E+03	5.7E+03	2.9E+03	5.9E+03	1.4E+04	5.4E+03	2.5E+04		5.2E+03	5.6E+03	5.6E+03
Anode I	PA12	1.9E+01	3.5E+00	1.6E-01	2.1E+01	1.9E+01	1.1E+02	1.6E+02	8.2E+01	1.7E+02	3.9E+02	1.5E+02	7.0E+02		1.5E+02	1.6E+02	1.6E+02
Anode I	PA13	2.4E+03	4.5E+02	2.1E+01	2.7E+03	2.5E+03	1.4E+04	2.1E+04	1.0E+04	2.1E+04	5.0E+04	2.0E+04	9.0E+04		1.9E+04	2.0E+04	2.0E+04
Anode I	PA14	1.8E+03	3.4E+02	1.6E+01	2.1E+03	1.9E+03	1.1E+04	1.6E+04	7.8E+03	1.6E+04	3.7E+04	1.5E+04	6.7E+04		1.4E+04	1.5E+04	1.5E+04
Anode I	PA15	8.1E-01	1.5E-01	7.0E-03	9.2E-01	8.3E-01	4.7E+00	6.9E+00	3.5E+00	7.1E+00	1.7E+01	6.6E+00	3.0E+01		6.3E+00	6.7E+00	6.7E+00
Anode I	A1	9.9E+03	1.8E+03	8.6E+01	1.1E+04	1.0E+04	5.8E+04	8.4E+04	4.3E+04	8.7E+04	2.0E+05	8.0E+04	3.7E+05		7.7E+04	8.2E+04	8.2E+04

Values expressed in ng/kg_{dwt}

Table B.9.51. Local concentrations in grassland over 180 days for primary aluminium production and anode baking.

Use cat.	Site	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)-fluoranthene	Benzo(k)-fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)-anthracene	Indeno(123-cd)-pyrene
VSS II	S1	1.1E+03	2.7E+02	6.2E+02	2.6E+03	8.5E+02	7.4E+03	1.1E+04	5.1E+03	6.6E+03	1.6E+04	1.5E+04	2.8E+04		9.5E+03	4.0E+03	1.0E+04
VSS II	S3	2.7E+03	7.0E+02	1.6E+03	6.6E+03	2.2E+03	1.9E+04	2.9E+04	1.3E+04	1.7E+04	4.1E+04	3.9E+04	7.3E+04		2.5E+04	1.0E+04	2.6E+04
VSS II	S4	2.9E+03	7.5E+02	1.7E+03	7.0E+03	2.3E+03	2.0E+04	3.1E+04	1.4E+04	1.8E+04	4.3E+04	4.2E+04	7.8E+04		2.6E+04	1.1E+04	2.8E+04
SWPB	P7	1.6E-02		7.4E+00	5.7E+00			1.4E+03	5.1E+02	1.1E+03	1.9E+03	1.2E+03	7.4E+02	1.4E+03	7.6E+02	3.3E+02	5.1E+02
VSS I	S5	2.9E+03	7.7E+02	1.8E+03	7.3E+03	1.6E+03	8.5E+03	1.3E+04	8.5E+03	1.8E+04	4.2E+04	4.2E+04	1.1E+05		3.2E+04	5.8E+03	2.8E+04
VSS I	S6	2.8E+03	7.4E+02	1.7E+03	7.1E+03	1.6E+03	8.2E+03	1.2E+04	8.2E+03	1.7E+04	4.1E+04	4.1E+04	1.1E+05		3.1E+04	5.6E+03	2.7E+04
Anode I	PA1	1.1E+03	2.6E+02	1.1E+01	1.5E+03	1.4E+03	7.9E+03	1.2E+04	6.1E+03	1.2E+04	2.9E+04	1.2E+04	5.2E+04		1.1E+04	1.2E+04	1.2E+04
Anode I	PA2	4.6E+02	1.1E+02	4.8E+00	6.4E+02	6.1E+02	3.4E+03	5.2E+03	2.6E+03	5.2E+03	1.2E+04	4.9E+03	2.2E+04		4.6E+03	4.9E+03	4.9E+03
Anode I	PA3	4.2E-01	1.0E-01	4.4E-03	5.8E-01	5.5E-01	3.1E+00	4.7E+00	2.3E+00	4.7E+00	1.1E+01	4.4E+00	2.0E+01		4.2E+00	4.4E+00	4.4E+00
Anode I	PA4	4.8E+01	1.1E+01	5.0E-01	6.7E+01	6.3E+01	3.5E+02	5.4E+02	2.7E+02	5.4E+02	1.3E+03	5.1E+02	2.3E+03		4.8E+02	5.1E+02	5.1E+02
Anode I	PA5	2.9E+02	7.0E+01	3.0E+00	4.1E+02	3.9E+02	2.1E+03	3.3E+03	1.6E+03	3.3E+03	7.8E+03	3.1E+03	1.4E+04		2.9E+03	3.1E+03	3.1E+03
Anode I	PA6	1,0E+03	2,5E+02	1,1E+01	1,5E+03	1,4E+03	7,6E+03	1,2E+04	5,8E+03	1,2E+04	2,8E+04	1,1E+04	5,0E+04		1,0E+04	1,1E+04	1,1E+04
Anode I	PA6	2.8E+03	6.7E+02	2.9E+01	3.9E+03	3.7E+03	2.1E+04	3.2E+04	1.6E+04	3.2E+04	7.5E+04	3.0E+04	1.3E+05		2.8E+04	3.0E+04	3.0E+04
Anode I	PA7	4.3E+01	1.0E+01	4.5E-01	6.0E+01	5.7E+01	3.1E+02	4.8E+02	2.4E+02	4.8E+02	1.1E+03	4.6E+02	2.0E+03		4.3E+02	4.6E+02	4.6E+02
Anode I	PA8	1.0E+01	2.5E+00	1.1E-01	1.5E+01	1.4E+01	7.6E+01	1.2E+02	5.8E+01	1.2E+02	2.8E+02	1.1E+02	5.0E+02		1.0E+02	1.1E+02	1.1E+02
Anode I	PA9	2.4E+04	5.8E+03	2.6E+02	3.4E+04	3.2E+04	1.8E+05	2.8E+05	1.4E+05	2.8E+05	6.5E+05	2.6E+05	1.2E+06		2.4E+05	2.6E+05	2.6E+05
Anode I	PA10	2.7E+02	6.5E+01	2.8E+00	3.8E+02	3.6E+02	2.0E+03	3.1E+03	1.5E+03	3.1E+03	7.2E+03	2.9E+03	1.3E+04		2.7E+03	2.9E+03	2.9E+03
Anode I	PA11	1.0E+03	2.5E+02	1.1E+01	1.4E+03	1.4E+03	7.6E+03	1.2E+04	5.8E+03	1.2E+04	2.8E+04	1.1E+04	5.0E+04		1.0E+04	1.1E+04	1.1E+04
Anode I	PA12	2.9E+01	7.0E+00	3.1E-01	4.1E+01	3.9E+01	2.1E+02	3.3E+02	1.6E+02	3.3E+02	7.8E+02	3.1E+02	1.4E+03		2.9E+02	3.1E+02	3.1E+02
Anode I	PA13	3.7E+03	9.0E+02	3.9E+01	5.2E+03	5.0E+03	2.7E+04	4.2E+04	2.1E+04	4.2E+04	1.0E+05	4.0E+04	1.8E+05		3.7E+04	4.0E+04	4.0E+04
Anode I	PA14	2.8E+03	6.7E+02	2.9E+01	3.9E+03	3.7E+03	2.1E+04	3.2E+04	1.6E+04	3.2E+04	7.5E+04	3.0E+04	1.3E+05		2.8E+04	3.0E+04	3.0E+04
Anode I	PA15	1.2E+00	3.0E-01	1.3E-02	1.7E+00	1.7E+00	9.2E+00	1.4E+01	7.0E+00	1.4E+01	3.3E+01	1.3E+01	6.0E+01		1.2E+01	1.3E+01	1.3E+01
Anode I	A1	1.5E+04	3.7E+03	1.6E+02	2.1E+04	2.0E+04	1.1E+05	1.7E+05	8.5E+04	1.7E+05	4.1E+05	1.6E+05	7.3E+05		1.5E+05	1.6E+05	1.6E+05

Values expressed in ng/kg_{dwt}

Calculation of C_{local} for production

Only for those sites where emissions are directed to a municipal wastewater treatment plant the local concentrations (averaged over 30 days) in grassland and agricultural soil are the result of atmospheric deposition and sludge application. For a number of these sites the sludge is not spread on arable land but incinerated (*i.e.* site 1 and 3). For site 5 wastewater is directed to an onsite industrial sewage treatment plant for which it is assumed that sludge is not used for agricultural purposes. Consequently, sludge-application to arable land is applicable to site 7 only. For the other sites the effluent is treated in on-site wastewater treatment facilities or directly discharged to water. For now it is assumed that for on-site wastewater treatment facilities sludge is treated as chemical waste and sludge is not allowed to be used on agricultural soil.

Calculation of C_{local} for industrial/professional use

The local concentrations (averaged over 30 days) in grassland and agricultural soil are the result of only atmospheric deposition as no waste water treatment of the (scrubber/cooling water) effluent was assumed. The route of waste water treatment sludge to agricultural soil therefore is not relevant for the generic scenarios. The concentrations predicted in agricultural soil and grassland at sites near the different downstream users are given in Table B.9.50, Table B.9.51and Table B.9.52.

Table B.9.52. Local concentrations in agricultural soil and grassland for the ferro-alloy and graphite production industry.

	agricultural soil		grassland	
Substance/Scenario	**Ferro-Alloy [a)]**	**Graphite [b)]**	**Ferro-Alloy [a)]**	**Graphite [b)]**
Naphthalene	0.9	1.1	1.4	1.8
Acenaphthene	0.2	0.6	0.4	1.1
Acenaphthylene	0.5	0.3	0.9	0.6
Fluorene	2.1	0.7	4.0	1.3
Anthracene	1.0	0.6	2.0	1.2
Phenanthrene	6.2	8.3	12.5	16.9
Fluoranthene	10.3	15.6	20.8	31.2
Pyrene	4.9	7.8	9.9	15.6
Benz(a)anthracene	6.6	7.2	13.0	14.3
Chrysene	14.3	23.4	27.3	48.1
Benzo(a)pyrene	11.8	2.6	23.4	5.3
Benzo(b)fluoranthene	23.4	14.3	46.8	29.9
Benzo(k)fluoranthene	- [c)]	18.6	- [c)]	28.6
Benzo(ghi)perylene	6.8	3.0	13.0	6.0
Dibenzo(a,h)anthracene	3.3	1.8	6.4	3.5
Indeno(123-cd)pyrene	7.2	3.8	14.3	7.7

Values expressed in ng/kg_{wwt}. [a)] Ferro-alloy production (including paste preparation); [b)] Graphite: production of graphite electrodes (including paste preparation) using dry scrubbers; [c)] No information available.

Measured levels

Individual PAH concentrations in soils produced by natural processes like vegetation fires and volcanic exhalations have been estimated to be in the range of 1-10 µg/kg. At present, even the lowest measured concentrations in temperate soils are about 10 times higher than the concentrations assumed to have been present prior to industrialization and thus already include an anthropogenic contribution (Edwards, 1983).

PAH profiles were found to be dependent on the depth of soil from which the samples were taken. PAH profiles with lower boiling points, such as fluoranthene, chrysene and pyrene, decreased with depth, whereas concentrations of PAH with higher boiling points, such as indeno(123-cd)pyrene, dibenzo(a,h)anthracene, benzo(ghi)perylene, increased relatively with depth. The opposite would have been expected on the basis of the solubility of these PAH (WHO, 1998). The most abundant individual PAHs include the benzo(bjk)fluoranthenes, chrysene, fluoranthene and pyrene (Wilcke, 2000).

Local

Soil concentrations in the vicinity of the sources under investigation are scarce. PAH levels in soil that mainly result from industrial sources are reported for a site near a former coal gasification plant in the Netherlands and the USA, near a former coking plants in Canada and a dismantled refinery in Alberta (USA). The separate PAH levels of these industrial sites range from 12 to 506 mg/kg (WHO, 1998). As an indication of the PAH concentration in soil in the vicinity of a plant with high PAHs emission, Table B.9.53 shows the PAH concentration in the top 10 cm of the mineral soil of 10 forest sites with increasing distance from a blast furnace plant located in IJmuiden (The Netherlands) processes 7.5 million tons of iron ore per year using 3.7 million tons of coal per year.

Table B.9.53. PAH concentrations in the top 10 cm of mineral soil at ten forest sites with increasing distance from the blast furnace plant at IJmuiden, The Netherlands.

	Concentration in µg/kg$_{dwt}$									
Distance (km)	**0.3**	**0.5**	**1.2**	**1.7**	**2.0**	**2.5**	**2.8**	**3.4**	**4.5**	**6.6**
Fluorene	46.1	70.5	8.4	10.6	4.7	5.9	0	7.2	6.2	7
Phenanthrene	230	329	75	53	21	48	18	29	8.4	19
Anthracene	49	75	8.9	6.9	1.1	6.1	1.9	2.5	0.4	1.7
Fluoranthene	329	470	137	84	34	83	29	52	16	35
Pyrene	237	309	106	64	21	55	18	48	12	33
Benz(a)anthracene	173	234	67	38	14	40	13	20	5.6	14
Chrysene	179	200	74	51	20	48	20	32	5.2	26
Benzo(b)fluoranthene	372	508	138	97	37	81	37	61	22	51
Benzo(k)fluoranthene	105	141	39	24	9.6	24	8.6	15	4.9	12
Benzo(a)pyrene	182	252	73	24	7.2	42	6.8	0	2.5	16

Data are values measured by Van Brummelen *et al.* (1996).

Regional

A summary of the current state of information on the concentrations of PAHs in temperate top soils on a global scale is given by Wilcke (2000). Based on the median and mean values the agricultural top soils typically contain between 200 and 350 µg/kg for the sum of the 16 EPA PAHs, respectively. In forest soil the concentrations are somewhat higher with a mean and median around 900 and 400 µg/kg, respectively (see Table B.9.54).

The concentrations analysed are comparable with those measured in 1997 at cattle, vegetable and bulb farms in the framework of the National Soil Monitoring Network performed by National Institute of Public Health and Environment (RIVM), the Agricultural Economic Research Institute (LEI) and Alterra (see Table B.9.55) (Groot *et al.*, 2003).

Table B.9.54. Range, arithmetic mean, and median of PAH concentrations in temperate soils.

PAH [a]	Arable				Grassland				Forest				Urban			
	Range [b]	Mean	Median [b]	n [c]	Range [b]	Mean	Median [b]	n [c]	Range [b]	Mean	Median [b]	n [c]	Range [b]	Mean	Median [b]	n [c]
Naphthalene	nd-200	5.7	4.0	14	nd-25	4.0	3.0	33	2-156	33	20	42	nd-269	39	17	47
Acenaphthylene	nd-200	17	2.0	14	nd-23	2.3	nd	33	nd-30	3.4	1.6	43	nd-406	16	nd	73
Acenaphthene	nd-51	9.6	nd	14	nd-54	22	20	33	nd-17	2.0	1.2	44	nd-1800	57	1.2	73
Fluorene	nd-5	1.1	nd	10	nd-5.0	1.4	1.0	8	nd-71	6.9	2.8	54	nd-550	23	1.8	63
Phenanthrene	6-67	26	17	14	7-67	19	14	33	3-329	60	29	55	nd-2809	190	37	108
Anthracene	nd-11	2.4	1.6	14	nd-4.3	1.6	1.5	33	nd-75	8.6	3.4	54	nd-1400	58	18	94
Fluoranthene	3-230	62	49	20	8-350	51	34	37	0.1-693	118	59	62	nd-14200	805	273	138
Pyrene	5-134	39	28	15	6-8	25	23	33	0.4-453	72	36	54	nd-11900	593	155	117
Benz(a)anthracene	3-230	39	17	15	3-100	26	23	33	nd-402	43	15	54	nd-7734	437	92	118
Chrysene	5-82	22	10	10	7-82	21	10	8	nd-1123	117	43	54	nd-3556	278	53	106
Benzo(b)fluoranthene	5-100	33	30	19	4-290	34	17	37	nd-692	158	80	38	nd-9700	456	80	91
Benzo(k)fluoranthene	nd-38	10	10	15	2-81	20	11	12	nd-2064	186	56	43	nd-4224	236	30	81
Benzo(a)pyrene	2-85	18	14	20	3-170	19	10	37	nd-252	39	16	61	nd-6461	350	88	138
Indeno(123-cd)pyrene	5-35	15	8.0	11	6-35	14	9.0	8	0.4-825	82	35	48	nd-8000	387	76	90
Dibenzo(a,h)anthracene	nd-73	11	4.5	14	nd-190	10	4.0	33	nd-126	15	5.0	44	nd-822	55	12	94
Benzo(ghi)perylene	5-48	19	18	15	nd-96	28	23	33	nd-523	62	29	48	nd-8370	370	91	130

These data are obtained from a peer review of Wilcke (2000) in which 36 studies were included published in the years 1984-2000. [a] Values are expressed as µg/kg; [b] In some cases the lowest concentration was too low to be detected (indicated by nd: not detected); [c] Number of measurements.

Table B.9.55. Soil PAH concentration in soil at cattle, vegetable and bulb farms in Netherlands measured 1997.

	Cattle farms		Vegetable farms		Bulb farms	
Compound	Minimum (µg/kg)	Maximum (µg/kg)	Minimum (µg/kg)	Maximum (µg/kg)	Minimum (µg/kg)	Maximum (µg/kg)
Naphthalene	< 10	17.3	< 10	53.6	< 10	< 10
Anthracene	< 0.5	12	0.71	12.5	0.6	6.7
Phenanthrene	< 1.5	63.8	4.9	36.7	2.7	25.7
Fluoranthene	15.2	154.6	11.9	168.5	14.2	75.5
Pyrene	15	131.4	13.9	143.3	8	60.2
9H-Fluorene	< 2	5.1	< 2	< 2	< 2	< 2
Acenaphthylene						
Acenaphthene	< 20	< 20	< 20	< 20	< 20	< 20
Chrysene	8.5	72	9.4	64.8	6.5	34.1
Benz(a)anthracene	7.8	64.4	6.6	63.4	5	34.9
Benzo(b)fluoranthene	11.4	103.4	15.7	90.5	9.5	47.4
Benzo(ghi)perylene	11.8	78.1	12.6	59.6	6.4	35.2
Benzo(k)fluoranthene	4.1	39.9	6.1	38.7	3.5	20.2
Benzo(a)pyrene	7.5	76.7	< 2	58.6	4.6	35.3
Dibenzo(a,h)anthracene	< 3	17.1	< 3	14.3	< 3	7.8
Indeno(123cd)pyrene	8.6	80.2	10.9	56.4	5.2	32.3

Data taken from Groot *et al.* (2003).

The sum of 10 PAH concentrations measured in the upper 10 cm of soil in rural areas of the Netherlands, varies from 165-1229 µg/kg (Table B.9.56). The highest value of the western grasslands cannot be seen as typically rural, because this is a result of high atmospheric deposition of PAH and the presence of old cities compost with PAH containing remains of coal (RIVM, milieucompendium). These rural concentrations of the Netherlands are in conformity with the separate PAH concentrations of non-polluted areas of 5-50 µg/kg, which are reported by the WHO (1998). More recent concentrations in rural areas in the upper layer (0-10 cm) and the lower layer (0.5-1 m) in the Netherlands are presented in Table B.9.57. The benzo(a)pyrene concentrations in upper layer were comparable to those reported by Wilcke (2000) and Groot *et al.* (1997).

Table B.9.56. Soil PAH concentrations in rural areas of the Netherlands, monitoring data from 1993-1997.

Area	Benzo(a)pyrene (µg/kg) [a)]	ΣPAH [b)] (µg/kg) [a)]
Northern sea clay area	19	178
Northern peat grasslands	37	424
North-Holland reclaimed land and IJssel lake polders	18	165
Western grasslands	142	1229
Southern sea clay area	43	336
Northern sand area	22	260
Eastern and central sand area	33	296
River clay area	42	397
Southern sand area	22	219

Data taken from Groot *et al.* (2003). [a)] Average value of the soil upper 10 cm; [b)] Sum of the 10 most occurring PAH.

Table B.9.57. PAH concentrations measured in the upper and lower layer in rural soils in the Netherlands.

	Upper layer (0-0.1 m) (mg/kg$_{dwt}$)			Lower layer (0.5-1 m) (mg/kg$_{dwt}$)		
Substance	**mean**	**90-percentile**	**max**	**mean**	**90-percentile**	**max**
Naphthalene	0	0.01	0.88	0	0	0.06
Phenanthrene	0.01	0.07	1.16	0	0.02	0.09
Anthracene	0	0.02	0.54	0	0	0.06
Fluoranthene	0.03	0.14	2.20	0.01	0.03	0.32
Chrysene	0.01	0.06	0.74	0	0.01	0.068
Benz(a)anthracene	0.01	0.06	0.84	0	0.01	0.26
Benzo(a)pyrene	0.01	0.06	0.76	0	0.01	0.08
Benzo(k)fluoranthene	0.01	0.03	0.31	0	0.01	0.04
Indeno(123cd)pyrene	0.02	0.07	0.46	0	0.02	1.01
Benzo(ghi)perylene	0.01	0.06	0.39	0	0.02	0.14

Data from AW (2000).

Comparison between predicted and measured levels

Production

In the absence of local monitoring data, no comparison between the predicted and measured levels can be made. Though, for all sites the predicted concentrations for all 16 EPA PAHs were within the range of the background concentrations reported for arable-and grassland and below those measured in urban areas. The highest concentrations were predicted for site 1 and 3, especially for the PAHs phenanthrene, fluoranthene, pyrene, benzo(bjk)fluoranthene and indeno(123-cd)pyrene.

Industrial use

The C_{local} concentrations for anode and Vertical Stud Søderberg production plants are within the range of urban areas reported by Wilcke (2000) or higher. For ferro-alloy plants the local concentrations are comparable to those given for arable- and grassland. The local concentrations for plants using prebaked anodes were negligible in comparison to background concentrations. As no monitoring data has been provided by the industry, no comparison for the local environmental concentrations can be made.

B.9.5.3.3 Atmosphere

Calculation of predicted environmental concentrations

The local concentrations of the 16 EPA PAHs in the atmosphere have been calculated in according to the Technical Guidance Document (EC, 2003b). The concentrations in air near the production sites and sites near pitch processing plants uses presented in Table B.9.58, Table B.9.59 and Table B.9.60 do not include the regional background concentration.

The local air compartment receives its input from direct emissions to air, and volatilisation from the sewage treatment plant. The concentration in air at a distance of 100 meters from the point source is estimated with a Gaussian plume model. Degradation and wet and dry deposition of both vapour and aerosol particles are taken into account as the most important fate processes. For further details the Technical Guidance Document (EC, 2003b) can be consulted.

Table B.9.58. Local concentrations in air at 100 m from the point source at the production sites.

Substance	Site 1	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9
Naphthalene	190	1900	900	860	110	42	120	120
Acenaphthene	13	56	20	130	12	4.8	14.0	13.0
Acenaphthylene	57	42	15	45	31	12.0	35	34
Fluorene	12	38	11	260	6.2	2.50	7.1	6.8
Anthracene	10	20.0	3.9	140	5.4	2.1	6.1	6.0
Phenanthrene	29	120	14	870	16	6.3	18.0	18.0
Fluoranthene	20	47	7.8	290	11	4.3	12.0	12.0
Pyrene	17	29	5.6	59	9.1	3.8	10.00	10.0
Benz(a)anthracene	5.1	4.5	1.4	9.4	2.9	1.10	3.0	3.0
Chrysene	4.5	7.2	1.2	14	2.4	0.98	2.8	2.70
Benzo(a)pyrene	7.7	5.5	2.0	4.9	4.1	1.60	6.1	4.6
Benzo(b)fluoranthene	63	42	16	4.6	34	14.0	39	38
Benzo(k)fluoranthene	12	10.0	3.2	7.0	6.5	2.50	7.6	3.3
Benzo(ghi)perylene	2.7	2.1	0.66	3.5	1.4	0.55	1.6	1.50
Dibenzo(a,h)anthracene	10	6.7	2.4	4.4	5.2	2.10	7.1	5.7
Indeno(123-cd)pyrene	4.3	3.2	1.1	3.5	2.3	0.91	4.1	2.50

Values are expressed in ng/m^3.

Table B.9.59. Local concentrations in air, at 100 m from point source for ferro-alloy and graphite production industry.

Substance/Scenario	Ferro-Alloy [a)]	Graphite [b)]
Naphthalene	820	970
Acenaphthene	140	360
Acenaphthylene	310	210
Fluorene	420	140
Anthracene	190	120
Phenanthrene	1,200	1,700
Fluoranthene	750	1,100
Pyrene	400	630
Benz(a)anthracene	75	81
Chrysene	130	220
Benzo(a)pyrene	56	13
Benzo(b)fluoranthene	180	110
Benzo(k)fluoranthene		58
Benzo(ghi)perylene	25	11
Dibenzo(a,h)anthracene	11	6.1
Indeno(123-cd)pyrene	25	13

Values expressed as ng/m^3. [a)] Ferro-alloy production (including paste preparation); [b)] Graphite: production of graphite electrodes (including paste preparation) using dry scrubbers.

Table B.9.60. Local concentrations in air for the primary aluminium production and anode baking facilities.

Use category	Site	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)-fluoranthene	Benzo(k)-fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)-anthracene	Indeno(123-cd)-pyrene
VSS II	S1	5.6E+02	8.9E+01	2.2E+02	2.7E+02	8.0E+01	7.3E+02	4.0E+02	2.1E+02	3.8E+01	7.3E+01	3.6E+01	1.1E+02		1.7E+01	6.9E+00	1.7E+01
VSS II	S3	1.4E+03	2.3E+02	5.6E+02	6.9E+02	2.1E+02	1.9E+03	1.0E+03	5.3E+02	9.8E+01	1.9E+02	9.2E+01	2.8E+02		4.4E+01	1.8E+01	4.4E+01
VSS II	S4	1.5E+03	2.4E+02	6.0E+02	7.3E+02	2.2E+02	2.0E+03	1.1E+03	5.7E+02	1.0E+02	2.0E+02	9.8E+01	2.9E+02		4.7E+01	1.9E+01	4.7E+01
SWPB	P7	8.7E-03		2.6E+00	5.9E-01			5.0E+01	2.1E+01	6.3E+00	8.7E+00	2.9E+00	2.9E+00	2.9E+00	1.4E+00	5.9E-01	8.7E-01
VSS I	S5	1.6E+03	2.6E+02	6.4E+02	7.6E+02	1.6E+02	8.3E+02	4.6E+02	3.5E+02	1.0E+02	1.9E+02	1.0E+02	4.1E+02		5.9E+01	1.0E+01	4.9E+01
VSS I	S6	1.6E+03	2.5E+02	6.1E+02	7.3E+02	1.5E+02	8.1E+02	4.4E+02	3.3E+02	9.8E+01	1.9E+02	9.8E+01	4.0E+02		5.7E+01	9.8E+00	4.8E+01
Anode I	PA1	6.1E+02	8.3E+01	4.0E+00	1.6E+02	1.4E+02	8.3E+02	4.3E+02	2.4E+02	7.2E+01	1.3E+02	2.7E+01	1.9E+02		2.1E+01	2.1E+01	2.1E+01
Anode I	PA2	2.6E+02	3.5E+01	1.7E+00	6.8E+01	6.1E+01	3.5E+02	1.8E+02	1.0E+02	3.1E+01	5.6E+01	1.1E+01	8.2E+01		8.7E+00	8.7E+00	8.7E+00
Anode I	PA3	2.3E-01	3.2E-02	1.5E-03	6.2E-02	5.5E-02	3.2E-01	1.7E-01	9.4E-02	2.8E-02	5.1E-02	1.0E-02	7.5E-02		7.9E-03	7.9E-03	7.9E-03
Anode I	PA4	2.7E+01	3.6E+00	1.8E-01	7.1E+00	6.3E+00	3.7E+01	1.9E+01	1.1E+01	3.2E+00	5.9E+00	1.2E+00	8.5E+00		9.0E-01	9.0E-01	9.0E-01
Anode I	PA5	1.6E+02	2.2E+01	1.1E+00	4.3E+01	3.9E+01	2.2E+02	1.2E+02	6.6E+01	1.9E+01	3.6E+01	7.3E+00	5.2E+01		5.5E+00	5.5E+00	5.5E+00
Anode I	PA6	5,8E+02	8,0E+01	3,8E+00	1,5E+02	1,4E+02	8,0E+02	4,1E+02	2,3E+02	6,9E+01	1,3E+02	2,6E+01	1,9E+02		2,0E+01	2,0E+01	2,0E+01
Anode I	PA6	1.6E+03	2.2E+02	1.0E+01	4.2E+02	3.7E+02	2.2E+03	1.1E+03	6.3E+02	1.9E+02	3.5E+02	7.0E+01	5.0E+02		5.3E+01	5.3E+01	5.3E+01
Anode I	PA7	2.4E+01	3.3E+00	1.6E-01	6.3E+00	5.7E+00	3.3E+01	1.7E+01	9.6E+00	2.8E+00	5.3E+00	1.1E+00	7.7E+00		8.1E-01	8.1E-01	8.1E-01
Anode I	PA8	5.9E+00	8.0E-01	3.8E-02	1.5E+00	1.4E+00	8.0E+00	4.2E+00	2.3E+00	6.9E-01	1.3E+00	2.6E-01	1.9E+00		2.0E-01	2.0E-01	2.0E-01
Anode I	PA9	1.4E+04	1.9E+03	9.0E+01	3.6E+03	3.2E+03	1.9E+04	9.7E+03	5.5E+03	1.6E+03	3.0E+03	6.1E+02	4.4E+03		4.6E+02	4.6E+02	4.6E+02
Anode I	PA10	1.5E+02	2.1E+01	1.0E+00	4.0E+01	3.6E+01	2.1E+02	1.1E+02	6.1E+01	1.8E+01	3.3E+01	6.8E+00	4.9E+01		5.1E+00	5.1E+00	5.1E+00
Anode I	PA11	5.8E+02	7.9E+01	3.8E+00	1.5E+02	1.4E+02	8.0E+02	4.1E+02	2.3E+02	6.9E+01	1.3E+02	2.6E+01	1.9E+02		2.0E+01	2.0E+01	2.0E+01
Anode I	PA12	1.6E+01	2.2E+00	1.1E-01	4.3E+00	3.9E+00	2.2E+01	1.2E+01	6.6E+00	1.9E+00	3.6E+00	7.3E-01	5.2E+00		5.5E-01	5.5E-01	5.5E-01
Anode I	PA13	2.1E+03	2.9E+02	1.4E+01	5.6E+02	5.0E+02	2.9E+03	1.5E+03	8.4E+02	2.5E+02	4.6E+02	9.4E+01	6.7E+02		7.1E+01	7.1E+01	7.1E+01
Anode I	PA14	1.6E+03	2.2E+02	1.0E+01	4.2E+02	3.7E+02	2.2E+03	1.1E+03	6.3E+02	1.9E+02	3.5E+02	7.0E+01	5.0E+02		5.3E+01	5.3E+01	5.3E+01
Anode I	PA15	7.0E-01	9.6E-02	4.6E-03	1.9E-01	1.7E-01	9.6E-01	5.0E-01	2.8E-01	8.3E-02	1.5E-01	3.1E-02	2.2E-01		2.4E-02	2.4E-02	2.4E-02
Anode I	A1	8.6E+03	1.2E+03	5.6E+01	2.3E+03	2.0E+03	1.2E+04	6.1E+03	3.4E+03	1.0E+03	1.9E+03	3.8E+02	2.7E+03		2.9E+02	2.9E+02	2.9E+02

Values expressed as ng/m^3.

Calculation of C_{local} for production

Local concentrations 100 m from the point source are presented in the following table. Atmospheric release from the waste water treatment plant does not contribute to the local concentration for those sites with reported on-site waste water treatment; either biological or physical (site 4, 6, 8 and 9). This is caused by the facts that in these cases the direct emissions to water are used as input and the sewage treatment plant calculation procedure is not used in the local assessment. In general it can be stated that for CTPHT production sites the contribution from the waste water treatment is not significant with respect to the local air emissions from the production process.

Calculation of C_{local} for industrial/professional use

Local concentrations 100 m from the point source are presented in the following table. Atmospheric release from the waste water treatment plant does not contribute to the local concentration because it was assumed that the wet scrubber effluent and cooling water effluent is not treated in the local sewage treatment plant. In two reports of the Norwegian Institute for Air Research (NILU) detailed information is given on the emission of fluoride and PAHs at two Norwegian aluminium production sites using Vertical Stud Søderberg between 1990 and 1997. According to the European Aluminium Association (EAA), these sites are representative for other aluminium smelters in Europe. The data showed that roughly 10% of all hydrofluoride and PAH is emitted via chimneys of several gas scrubbers, while the main part (approximately 90%) is emitted to the atmosphere with ventilation air over a roof of an aluminium electrolysis pot room, which can be up to 1,000 meters in length. In addition, hydrofluoride and PAH is emitted at a height (of both roof and chimney) of at least 25 m.

The calculations in EUSES 2.0.3 are based on an included OPS (operational priority substances) model assuming 100 m from one point source at an emission height of 10 m. According to EAA, a more realistic assumption for the aluminium smelters would be an emission from multiple sources at a height higher than 10 m. This has been modelled by NILU for two Norwegian aluminium smelters, where it is shown that the atmospheric fluor and PAH concentrations are significantly lower than those calculated with EUSES 2.0.3.

Table B.9.61. Ratios between hydrofluoride concentrations at 100 and 500 m distance from the emission source.

		Emission height		
Emission type	**Distance to source (m)**	**10 m**	**25 m**	**50 m**
Point source	100	100 [a)]	15	1.6
	500	15	5.3	1.8
Over a surface of 500 by 500 m.	100	29	8.5	2.6
	500	10	2.8	1.3

[a)] Average hydrofluoride concentration conform EUSES default settings is set to 100%, other values are presented as % of this concentration.

In order to demonstrate the impact of the type of emission (point versus multiple source) and emission height, additional OPS-Pro 4.1 calculations were carried out in the framework of the risk assessment of aluminiumfluoride (see risk assessment report on AlF_3) to predict the hydrofluoride concentrations at 100 and 500 m distance from the smelter (see Table B.9.61). These calculations are based on the provided NILU reports, assuming a realistic worst-case emission height of 25 m, as given for plant 1 (the other plant has an emission height of 50 m) and emissions to take place from a surface of 500 by 500 m (representing the emissions from both the roof and gas scrubbers). The hydrofluoride concentration calculated for the emission of a point source at a distance of 100 m with an emission height of 10 m (EUSES defaults) was set to 100%. As input for the model Dutch climatic and environmental parameters are used. The distance from the surface of emission is the distance measured from the edge of the surface. The concentrations given are in fact averages of all wind directions. These calculations demonstrate that the impact of adjusting the main assumptions

results in a reduction of predicted atmospheric concentrations of 91.5%. It should be noted that the old version of OPS (v1.20E) and consequently EUSES, predicts 20 to 25% higher concentrations in comparison to the latest model version (Pro 4.1), which has been left out of the current calculations.

This exercise revealed atmospheric concentrations of hydrofluoride in the same range as the NILU predictions. Applying the ratio to the predicted PAH concentrations, both predictions also within the same order of magnitude.

It can therefore be concluded that by assuming emissions over a surface instead of one point source and a higher emission height will lower the predicted air concentrations significantly. Using a point source and an emission height of 10 m the OPS model (a component of EUSES 2.0.3.) predicts a concentration at 100 m distance, roughly 12 times higher than predicted with emission height 25 m and emissions over a surface of 500 by 500 m. The OPS-Pro 4.1 derived local concentrations are therefore considered to be more realistic for the Vertical Stud Søderberg plants and have been taken forward in the risk assessment.

Measured levels

Local

Monitoring data for the production of CTPHT and the metal-alloy industry are scarce. For the aluminium production using prebaked anodes and Søderberg technology a recent monitoring study was performed during 2 months each in the period of end 2001-beginning 2002 at 5 Norwegian sites (Hagen, 2002). The measured benzo(a)pyrene and total concentration are presented in Table B.9.62.

Table B.9.62. Benzo(a)pyrene and total PAH concentration in air in the vicinity of 5 aluminium production sites measured in the period November 2001 – March 2002.

Site	Plants	Distance from the plant (km)	Benzo(a)pyrene		Total PAH	
			Range	Mean	Range	Mean
Lista	Søderberg	1	0.05-2.27	0.50	31-263	78
Karmøy	Søderberg	0.1-0.2	0.03-0.30	0.10	48-205	100
Høyanger	Søderberg/ prebake	0.5	0.22-2.0	1.1	77-416	197
Øvre Årdal	Søderberg/ prebake (2X)	1	0.5-15.4	4.5	172-2,110	652
Sunndalsøra	Søderberg (2X)	1	0.73-13.6	3.5	104-1,770	449

Data taken from Hagen (2002). Values expressed as ng/m^3.

For Sunndalsøra (site PA2) more recent measurements showed that the benzo(a)pyrene concentration at a point about 500 m from the plant were on average 0.16 ng/m^3 in the summer 2006 (NILU report OR 89/2006). According to the applicant, the summer concentration is the most relevant here, as the winter values are impacted by local wood burning. It should also be noted that the production process of this plant has been changed from Søderberg technology to the use of prebaked anodes.

At site S4 the measured benzo(a)pyrene concentration at a location about 200 m from the plant in 2006, was on average (summer and winter) 2.5 ng/m^3.

Regional

According to the EU working group on PAHs, the PAH concentrations are lower at remote background sites than at rural sites. Higher concentrations are found in urban areas, with peak concentrations measured at urban sites with both traffic and nearby industrial installations. In the 1990s typical annual mean levels of benzo(a)pyrene in rural background areas varied between 0.1 and 1 ng/m^3; for urban areas levels were between 0.5 and 3 ng/m^3 (with traffic sites at the upper boundary of this range); levels up to 30 ng/m^3 have been measured within the immediate vicinity of

a cookery. Few measurement data exist for rural communities burning coal and wood domestically; however, these measurements suggest levels similar to those found in cities. Table B.9.63 gives a summary of recent (not older than 1990) typical European PAH and benzo(a)pyrene concentrations in ng/m^3 as annual mean value.

Table B.9.63. Summary of recent typical European PAH and benzo(a)pyrene concentrations in as annual mean value.

Compound	Remote site	Rural site	Urban	Traffic	Industrial
Acenaphthene	0.01	0.3-2.6			98
Acenaphthylene	0.01				
Anthracene	nd [a)]	0.04-15		0.2-0.6	1.1
Benz(a)anthracene	0.0-0.02	0.01-0.9	0.2-1.3	0.6-4.2	0.37-42
Benzo(a)pyrene	0.02	0.02-1.6	0.4-2	0.7-3.1	0.5-39
Benzo(e)pyrene	0.01-0.02	0.18-1.1	0.2-2.1	0.9-3.7	0.65
Benzo(b)fluoranthene	0.00-0.01	0.04-0.6			0.3-34
Benzo(ghi)perylene	0.01	0.15-1.0	0.5-2.8	1-4.7	0.7-52
Benzo(k)fluoranthene		0.04-0.32	0.2-1		0.3-17
Chrysene		0.02-4.4	0.3-2.2		0.3-37
Coronene	0.00-0.01	0.02-0.5	0.1-0.6	0.4-2.5	0.26-5.2
Dibenzo(a,h)anthracene	nd [b)]	0.02-1.1	0.06-0.3	0.1-0.4	0.05-7.5
Fluoranthene	0.14	0.04-7.4			42
Fluorene	0.2-0.4	0.3-46		9.9-16.7	
Indeno(123-cd)pyrene	0.02-0.04	0.04-0.21	0.3-2.1	1.3-2.6	0.4-37
Phenanthrene	0.1-0.3	0.42-150			16
Pyrene	0.08	0.1-6.1	0.24-1.2	9.2-15	75

Data taken from EU working group (2001). This table summarises recent (not older than 1990) typical European PAH- and benzo(a)pyrene concentrations in ng/m^3 as annual mean value. It has to be emphasised that the data reported within the response to the questionnaire and in the literature are derived from different measurement techniques and are often of unknown quality. In addition, the ranges for different compounds are not always directly comparable, since different data sets were combined. These original data sets partly comprise different single compounds; [a)] nd: not detected.

In support to the discussion on the implementation of the EU daughter directive for PAHs consequential to the "Framework Directive on ambient air quality assessment and management", in 1998/1999 the presence of PAHs and then especially the PAH indicator benzo(a)pyrene in air in the Netherlands has been investigated. Using measurements data, a limited supplemental monitoring effort and the results of modelling calculations, it has been possible to obtain a realistic picture of air quality in the Netherlands with respect to these substances. The background level of benzo(a)pyrene is estimated at 0.05-0.15 ng/m^3 on a yearly average. Increased PAHs and benzo(a)pyrene concentrations are expected in the vicinity of a number of industrial sources and in urban surroundings. benzo(a)pyrene concentrations near the Hoogovens (Steelworks) measure 0.2-0.5 ng/m^3, depending on their position in relation to the source complex. The urban background level comes to 0.2-0.4 ng/m^3 and on roads yearly averages of up to 0.7 ng/m^3 can occur (Buijsman, 1999). The concentration measured for the other PAHs are shown in Table B.9.64

Table B.9.64. Average PAH air concentration measured in the Netherlands at background locations (De Zilt, De Rijp and Vredepeel) in 1998, near industrial sources and urban surroundings.

Compound	background	Near industrial sources	Urban surroundings
Naphthalene	-	-	14
Acenaphthene	-	-	2.4
Acenaphthylene	-	-	6.2
Fluorene	-	-	8.7
Anthracene	-	1.6	1.1-3.2
Phenanthrene	-	-	19
Fluoranthene	0.08-3.5	6.2	4.9-11
Pyrene	0.05-1.5	0.28-3.0	2.5-7.2
Benz(a)anthracene	0.04-1.0	0.33-1.1	0.22-1.5
Chrysene	0.06-0.45	0.40-0.85	0.46-0.89
Benzo(a)pyrene	0.06-0.11	0.11-0.54	0.13-0.36
Benzo(b)fluoranthene	0.16-0.31	0.37-1.2	0.41-0.72
Benzo(k)fluoranthene	0.06-0.11	0.12-0.48	0.14-0.30
Benzo(ghi)perylene	0.15-0.23	0.30-1.1	0.36-0.91
Dibenzo(a,h)anthracene	0.01-0.06	0.03-0.17	0.04-0.17

Data taken from Buijsman (1999). Values expressed as ng/m^3.

Comparison between calculated and measured levels

Production

The calculated air concentrations near all production sites are all close to the upper range or higher than those measured in urban areas. Though most of PAHs concentrations were within the range reported for industrial areas. Only the concentration predicted for site 5 were higher.

Industrial use

The data obtained by Hagen (2002; see Table B.9.62) showed that the benzo(a)pyrene concentrations in air in the vicinity of plants using Søderberg technology are between 0.1 and 10 ng/m^3. The calculated concentration for the plants using this technology are between 30 and 100 ng/m^3, which is one to two orders of magnitude higher. For the plant located at Karmøy (site S3), where the measured and predicted concentrations are related to the same distance (*i.e.* 100 m distance), the predicted air concentrations seem to be two orders of magnitude higher. For other plants the concentration measured at 1 km distance. When it is assumed that roughly the concentration at 100 m are 10 times higher than at 1 km, the concentrations for the other sites shown in Table B.9.62 seems to deviate to much lesser extent from the predicted concentrations than for Karmøy.

For site PA2 (Sunndalsøra), more recent data shows that the concentration benzo(a)pyrene at 500 m distance from the plant is a factor of 70 lower than estimated at 100 m distance (0.16 ng/m^3 versus 11 ng/m^3). For site S4 recent measurements indicate that the benzo(a)pyrene concentration at 200 m distance were a factor of 40 lower than estimated (2.5 ng/m^3 versus 98 ng/m^3). It is unknown to which extent the actual concentrations for the other PAHs deviate from those predicted.

For site PA7, measured hydrofluoride concentration at a point 100 m from the plant is 0.5 µg/m^3. The corresponding modelled value is 9.4 µg/m^3.

Overall, the measured data shows that the modelled air concentration can be considered as a conservative prediction. However, a more accurate measure of air concentration can only be obtained by local measurements at a relevant distance and direction from the emission source.

As stated above no calculation of the regional PEC_{air} is performed because of the high contribution of unintentional sources. The monitoring data were considered sufficient. The contribution of the emission source under investigation to the regional concentration will be based on the emission data given in Section B.8.2.1.

B.9.5.3.4 Calculation of PEC in sewage treatment plant

Some CTPHT production plants discharge their waste water to a municipal sewage treatment plant or off-site biological waste water treatment plant. For these sites the sewage treatment model has been applied to calculate the fate in the sewage treatment plant. The emission from the production site and the effluent flow rate of the external waste water treatment facility are required as input. The model calculates the emission from the sewage treatment plant to air, the concentration in sewage sludge and the concentration in the effluent. A detailed description of the sewage treatment plant model is given in the Technical Guidance Document (EC, 2003b). The highest PEC for total PAH in the effluent which is considered relevant for the risk assessment is 114 µg/L at site 4. The concentrations in the effluent of the other production sites were ≤ 2 µg/L (Table B.9.65).

Table B.9.65. Concentrations in sewage treatment plant effluent for the relevant production sites.

Substance	Site 1 [a]	Site 3 [a]	Site 4 [b]	Site 5 [a]	Site 6 [b]	Site 7 [a]	Site 8 [c]	Site 9 [c]
Naphthalene	0.73	73	750	60	74	1.3	n.r.	n.r.
Acenaphthene	0.14	48	433	20	38	0.3	n.r.	n.r.
Acenaphthylene	n.d. [d]	92	1,783	105	5	0.4	n.r.	n.r.
Fluorene	0.094	152	435	20	21.5	0.6	n.r.	n.r.
Anthracene	0.018	37	867	24	24.5	0.2	n.r.	n.r.
Phenanthrene	0.17	88	1,267	0.46	65.5	2.5	n.r.	n.r.
Fluoranthene	0.06	91	6,592	8.0	61.5	0.9	n.r.	n.r.
Pyrene	0.33	73	4,842	6.9	36.5	0.9	n.r.	n.r.
Benz(a)anthracene	0.0067	30	5,075	1.2	8.5	0.3	n.r.	n.r.
Chrysene	0.0070	31	4,433	0.15 [e]	8	0.3	n.r.	n.r.
Benzo(a)pyrene	0.0064	13	19,608	0.14 [e]	10	0.2	n.r.	n.r.
Benzo(b)fluoranthene	0.0064	156	29,333	0.14 [e]	7.5	0.4	n.r.	n.r.
Benzo(k)fluoranthene	0.0064	52	9,533	0.14 [e]	6.5	0.1	n.r.	n.r.
Benzo(ghi)perylene	0.0063	64	11,992	0.14 [e]	5	0.2	n.r.	n.r.
Dibenzo(a,h)anthracene	0.0060	17	3,325	0.13 [e]	6	0.1	n.r.	n.r.
Indeno(123-cd)pyrene	0.0060	73	13,350	0.13 [e]	5.5	0.2	n.r.	n.r.

[a] Discharge from these sites is directed to off-site (public) sewage treatment plants. Sludge from these sewage treatment plants goes to agricultural soil. These effluent concentrations (ng/L) have been calculated applying the sewage treatment plant model in EUSES (EC, 2003b); [b] On-site waste water treatment, reported effluent concentration (ng/L); [c] These sites have no on-site biological (activated sludge) wastewater treatment, therefore the table does not give effluent data for these sites, indicated with n.r. ; [d] not detected; [e] detection limit is 0.01 µg/L.

B.9.5.3.5 Secondary poisoning

As an indication of the PAH concentration in marine invertebrates in contaminated areas Table B.9.66 gives an overview of PAH concentrations of 272 samples of *Mytilus edulis, Modiolus modiolus, Littorina littorea* and *Patella vulgate* collected over a 20-year period from seven smelter-affected fjords (Naes *et al.* 1998). Table B.9.67 gives an overview made by Meador (2003) of the observed concentrations in marine invertebrates from different sites around the world.

Table B.9.66. Sum PAH concentrations in different marine benthic organism sampled in seven smelter-affected fjords.

Fjord area	Organism	Sampling year	Range (mg/kg_{dwt})	Median (mg/kg_{dwt})	Average (mg/kg_{dwt})	Number of samples
Årdal	*Modiolus modiolus*	1983/1990/1992/1994	0.38- 490	7.7	36	25
Sunndal	*Mytilus edulis*	1987/1991	0.15-26	1.2	3.9	13
	Modiolus modiolus	1987/1991/1992	0.21-120	1.5	18	13
	Littorina littorea	1987/1991	0.90-56	1.1	13	7
Lista	*Littorina littorea*	1985-1990	0.20-270	2.7	56	20
	Patella vulgate	1980-1986	0.026-143	4.1	19	17
Vefsn	*Mytilus edulis*	1978/1984/1985/1989/1990/1991	2.3-268	22	38	38
	Modiolus modiolus	1978/1989/1990	0.67-103	2.8	16	16
Feda	*Mytilus edulis*	1984	1.4-18	7.8	8.3	10
Sauda	*Mytilus edulis*	1976/1980/1981/1986/1990-1994	0.004-1620	0.86	90	36
	Modiolus modiolus	1976/1980/1981/1986/1990-1992	0.006-480	1.1	38	29
Brevik	*Mytilus edulis*	1980/1983-1986/1990-1992	0.051-15	1.5	2.8	48

Data taken from Naes *et al.* (1998).

Table B.9.67. Occurrence of PAHs in marine bivalves and invertebrates from field collections.

Species	Feeding type [a]	Area	Total PAH (ng/g) [b]	Sites/ PAHs [c]	dwt / wwt	Reference [d]	Type of sampling
Mussels and oysters							
Mytilus edulis	FF	Norway	500-12845	11/32	dwt	Knutzen & Sortland (1982)	widespread
Mytilus galloprovincialis	FF	Mediterranean	25-390	23/14	dwt	Baumard *et al.* (1998)	widespread
Mussels and oysters	FF	USA (all coasts)	77-1100 [e]	214/24	dwt	O'Conner (2002)	widespread
Mussels and oysters	FF	USA (all coasts)	192-503 [f]	97-191/44	dwt	NOAA (1998)	widespread, multiyear
Mytilus edulis	FF	Gulf of Naples, Italy	205	6/16	wwt	Cocchieri *et al.* (1990)	local
Mytilus galloprovincialis	FF	Mediterranean, Spain	190-5490	6/NS	wwt	Porte & Albaiges (1993)	widespread
Mytilus edulis	FF	Northern Baltic Sea	440	3/19	dwt	Broman *et al.* (1990)	local
Mytilus edulis	FF	Finland (archipelago Sea)	ND-150	7/7	wwt	Rainio *et al.* (1986, 1990)	widespread
Mytilidae	FF	Gulf of Mexico, USA	36-7530	4/17	dwt	Wade *et al.* (1989)	natural petroleum seepage areas
Mytilus galloprovincialis	FF	Greece	77-110	57/17	wwt	Losifidou *et al.* (1982)	widespread
Crassostrea virginica	FF	Florida, USA	361-11026	14/>25	dwt	Fisher *et al.* (2000)	local
M. edulis, M. galloprovincialis and *C. gigas*	FF	France	ND-300000	110/NS	dwt	Claisse (1989)	widespread, multiyear
Mytilus edulis	FF	Netherlands	45-100	2/6	wwt	Stronkhorst (1992)	local, multiyear
Mytilus edulis	FF	Scotland	54-2803	27/10	wwt	Mackie *et al.* (1980)	widespread
Mytilus edulis	FF	Puget Sound, Washington, USA	40-63600	9/24	dwt	Krishnakumar *et al.* (1994)	local
Mytilus spp.	FF	San Francisco Bay, California, USA	180-4100*	6/34	dwt	Miles & Roster (1999)	local

Species	Feeding type [a)]	Area	Total PAH (ng/g) [b)]	Sites/ PAHs [c)]	dwt / wwt	Reference [d)]	Type of sampling
Benthic invertebrates							
Macoma balthica	DF/FF	Scheldt, Netherlands	947 (449)	2/12	dwt	Stronkhorst *et al.* (1994)	local
Crangon crangon	scav	Scheldt, Netherlands	410 (285)	2/12	dwt	Stronkhorst *et al.* (1994)	local
Nereis diversicolor	omn	Scheldt, Netherlands	785 (409)	2/12	dwt	Stronkhorst *et al.* (1994)	local
Homarus americanus	scav	Nova Scotia, Canada	253-73000	1/10	wwt	Uthe & Musial (1986)	local, multiyear
Littorina littorea	herb	Southern Norway	595-1430	4/27	dwt	Knutzen & Sortland (1982)	widespread
Patella vulgata	herb	Southern Norway	674-15462	2/31	dwt	Knutzen & Sortland (1982)	local
Asterias rubens	pred	Southern Norway	325-458	2/19	dwt	Knutzen & Sortland (1982)	local
Macropipus tuberculatus	omn	Spain	60-930	6/NS	wwt	Porte & Albaiges (1993)	widespread

[a)] Feeding types are: deposit feeder (DF), filter feeder (FF); omnivore (omn); herbivore (herb), and predator (pred); [b)] Values are ranges or mean concentrations, some with standard deviations in parenthesis. Values represent all sites and PAHs measured. All studies examined the soft tissue of whole organisms except *: hepatopancreas. Many studies were surveys and included samples from relatively uncontaminated to heavily polluted locations. ND: not detected; [c)] The number of sites sampled and PAHs analyzed. NS: not stated; [d)] For references, see Meador (2003); [e)] 15th-85th percentile; [f)] annual mean values. Mean for most stations = 3-5ppm.

B.10 Risk characterisation

B.10.1 Human health

B.10.1.1 General aspects

B.10.1.1.1 Introduction

CTP and related substances like CTPV, creosotes and tars are complex and variable compositions. CTP is a complex hydrocarbon mixture in which over 400 compounds have been identified, and probably as many as 10 000 are actually present. The identity and amount of compounds present in coal tar pitches varies dependent on source materials and manufacturing processes used, including different temperatures and times of carbonization; no two coal tars or pitches are chemically identical (see Section B.1).

As illustrated in Section B.5 on Effect assessment, and summarized below, the database on possible health hazards induced by CTPHT is rather limited, implicating that a full risk assessment for all the required endpoints is not possible. 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 striking hazard associated with CTPHT. This is attributed to the presence of the PAHs in CTPHT (see Section B.9.1). Given the uncertainties with respect to the effects of other chemical constituents of CTPHT and related substances also exposed to, 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 effect, quite possibly even to zero. Therefore, in spite of the limited available data on non-carcinogenic properties of CTPHT, it is decided that in this risk characterisation for CTPHT conclusions on risks and further testing for some endpoints will be subordinated to conclusions on risks based on carcinogenic and mutagenic properties, using the best-studied PAH benzo(a)pyrene as a guidance substance (see also Sections B.9.1, B.5, and B.5.8).

Exposure

The human population may be exposed to CTPHT and CTPVHT at the workplace during production and use in various industries. The level of exposure varies considerably among industries (see Table B.9.16). As already outlined in Section B.5.8.2, it is also concluded that this indicator function of benzo(a)pyrene is rather scenario-specific: *i.e.* the amount of total PAHs may correlate well with the airborne concentration of benzo(a)pyrene (in $\mu g/m^3$) in most workplaces, while the PAH profile (the relative distribution of the individual PAHs) may be different for the different workplaces. In addition, the workers studied in the available epidemiological studies are exposed not only to CTPHT and CTPVHT, but also to coal tar and/or other chemicals, which makes it difficult to determine which components of these mixed exposures are the most important causal agents of the observed carcinogenic effects. Ideally, therefore, industry- and scenario-specific hazard estimates should be used. However, the meta-analyses, which analysed and summarised all acceptable epidemiological data relevant for worker exposure to CTPHT, demonstrated that sufficient industry-specific data were only available for the aluminium smelter industry (as indicated in Section B.5.8.2). For the other scenarios, the overall estimate from the meta-analyses is used to derive potency estimations for their exposures to CTPHT.

Consumer exposure can be considered negligible (see Section B.9.2.10).

Human exposure via the environment is difficult to assess, because the exposure assessment is focussing on PAHs and their are many diffuse sources of PAHs. On a local scale, however, some assessments can be done (see Section B.10.1.4).

Hazard assessment

In the data set animal as well as human studies are available. Some of the studies were not performed according to current standards, and were in some cases not suitable to be used in risk assessment.

There were no data available on the toxicokinetics of CTPHT. Some information on the toxicokinetics of selected homocyclic polycyclic aromatic hydrocarbons was available. From these data, it was concluded that PAHs are lipophilic compounds that can be absorbed through the respiratory and gastrointestinal tract and the skin. After absorption, PAHs are widely distributed throughout the organism to almost all organs, especially the lipid-rich ones. They can cross the placenta and reach foetal tissues. The metabolism of PAHs can take place in the liver, respiratory tract, and the skin, and appears very complex leading to a variety of metabolites from a limited number of reaction types. Only a few metabolites are toxicologically relevant. Most metabolic processes result in detoxification products that are excreted in urine and faeces. However, some pathways yield reactive compounds capable of binding to DNA and initiating tumour formation. Generally, the metabolism appears to be qualitatively similar with respect to cell or tissue type. However, large quantitative variations may occur between different cell types, tissues, and species caused by the inducibility and availability of enzyme systems, leading to differences in the susceptibility for the carcinogenic action of PAHs. Based on the calculated dermal absorption of ten different PAHs from dermally applied coal tar to pig-ears a dermal absorption of PAHs from CTPHT of 30% is taken forward to risk assessment. Since no data were available to allow a quantitative estimation of absorption after inhalation and oral exposure, for CTPHT default values of (in this case) 100% may be used for absorption of critical components via inhalation and oral exposure. It is emphasized though that these absorption rates are not used for consumer risk assessment, because of the absent of relevant identified exposures, and not for worker risk assessment, because both hazard- and exposure assessment are based on similar worker scenarios, *i.e.* include the combined specific inhalation and dermal exposure conditions.

From acute oral and dermal toxicity studies in experimental animals conducted according to EU guidelines, it is concluded that the substance does not need classification and labelling according to EC criteria (EC, 2001c) for these exposure routes. No inhalation studies in animals were available. No human data were available on the acute toxicity.

Skin effects were observed in animals and humans after repeated exposure to CTP(V) or combined exposure to CTP(V) and sunlight. However, from the available animal and human data it is not possible to tell if the observed dermal effects are caused by irritation and sensitisation (photosensitisation or sensitisation after repeated exposure), therefore classification of CTPHT for skin irritation is not possible. In view of the human data on occupation exposure to CTP (fumes, volatiles and dust, not further specified) which show eye irritation and, after repeated exposure, chemosis of the conjunctiva, ulceration and infiltration of the cornea, deep staining of the cornea, and conjunctival discolouration and irritation, classification as 'irritant' with 'risk of serious damage to eyes' (Xi, R41) is proposed. Sunlight aggravated irritating effects of CTP(V) on the eyes and skin.

No experimental data on the potential corrosivity and sensitising properties of CTPHT required as specified in Annex VIIA of Directive 67/548/EEC (EC. 1967) were available. Taking the available human and animal data into account, there are no indications that CTP has corrosive properties. According to the Directive 1999/45/EC relating to the classification, packaging and labelling of dangerous preparations (EC, 1999b), preparations containing more than 1% of a skin sensitising substance need to be classified as a skin sensitizer. Since CTPHT may contain up to 1.5% benzo(a)pyrene (a skin sensitizer) it is proposed to classify CTPHT as a skin sensitizer (Xi;R43).

With regard to repeated dose toxicity, apart from one oral study of limited significance in pigs, no repeated dose toxicity animal studies with CTPHT addressing effects other than carcinogenicity were available to the rapporteur. Therefore, the available data set does not meet the basic requirements as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967) and no NOAEL for non-carcinogenic effects could be derived from these studies.

In humans no statistical significant effects on lung function parameters were found in a group of phosphorus rock refinery workers exposed at the time of study to about 0.1 mg/m^3 CTPV in addition to other substances, including phosphorus pentoxide (about 2.2 mg/m^3) and fluorides (about 4.2 mg/m^3).

In addition, animal data was available on high-boiling coal liquid (LOAEC of 30 mg/m^3 in rats regarding semichronic inhalation exposure), and manufactured gas plant residue (a coal-tar like material) (NOAEL for male mice, oral exposure: 462 mg/kg/day; NOAEL for female mice, oral exposure: 344 mg/kg/day). These, however, are not considered representative for establishing a NOAEL value for risk characterisation of CTPHT.

The data set available on the mutagenicity/genotoxicity of CTPHT does not meet the basis requirements as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967). From mutagenicity testing in *S. typhimurium* conducted according to EU 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 of 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.

In addition, numerous genotoxicity studies with coal tar, coal tar waste, coal tar products, and individual PAHs demonstrated the genotoxicity of these substances (ATSDR, 2002, WHO, 1998).

According to the Directive 1999/45/EC relating to the classification, packaging and labelling of dangerous preparations (EC, 1999b), preparations containing more than 0.1% of a category 1 or 2 mutagen need to be classified as a category 1 or 2 mutagen. CTPHT may contain a variable amount of mutagenic PAHs. The mutagenic effect of these individual PAHs may be considered at least additive. Since CTPHT may at least contain up to 1.5% benzo(a)pyrene (a category 2 mutagen), the amount of category 2 mutagens in CTPHT is estimated to be more than 0.1% in nearly if not all circumstances.

Based on the amount of category 2 mutagens in CTPHT and the available genotoxicity data on CTPHT, CTPVHT, coal tar, coal tar waste, coal tar products, and individual PAHs, classification of CTPHT as a category 2 mutagen is proposed (T; R46).

There were no data available on the potential carcinogenicity of CTPHT after oral exposure in experimental animals. However, studies with coal tar resulted in increased tumour incidences in various organs. After oral exposure in mice main target organs appeared to be liver, lung, and forestomach. Studies with benzo(a)pyrene resulted in increased tumour incidences in amongst others the liver, forestomach, and auditory canal in rats and forestomach and upper gastrointestinal tract in mice.

Inhalation of CTPHT caused lung tumours rats and mice, while dermal exposure to CTPHT caused skin tumours in mice. Although most of the available experimental animal studies were not conducted according to EC or OECD guidelines, they clearly indicate that CTPHT is carcinogenic following inhalation and dermal exposure.

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 already in 1985 (IARC, 1985, 1987). Several additional studies have been published since including some attempting to derive quantitative cancer risk estimates. A recent meta-analysis by Armstrong *et al.* (2003; 2004) showed statistically increased overall relative risks for lung and bladder cancer for all CTPV exposure scenarios, and an industry-specific increased relative risk for workers exposed in aluminium smelters. In spite of the inherent uncertainties underlying these relative risk values (see Sections B.1, B.9.1, B.5.8.2 and B.5.8.2.3) these meta-analyses estimates are considered the best estimates of the risk on lung and bladder cancer risk due to exposure of CTPHT. Therefore, the relative risk value found for lung cancer in this meta-analysis is forwarded to the risk characterisation: an overall relative risk estimate of 1.20 (95% confidence interval: 1.11-1.29) per unit of 100 µg/m^3·year cumulative benzo(a)pyrene exposure.

Furthermore, for aluminium smelters, the only industry exposed to CTPVHT for which rather precise estimates could be established in the meta-analysis, the combined relative risk estimate was 1.16 (95% confidence interval: 1.05-1.28) for lung cancer. This value will be taken forward to the risk characterisation for aluminium smelters.

Regarding bladder cancer, for which the association with PAH exposure was less robust than the PAH-lung cancer association, the overall relative risk estimate of 1.33 (95% confidence interval: 1.17-1.51) per unit of 100 µg/m^3·year cumulative benzo(a)pyrene exposure is forwarded to the risk characterisation.

Furthermore, for aluminium smelters, the only industry exposed to CTPVHT for which rather precise estimates could be established in the meta-analysis, the combined relative risk estimate was 1.42 (95% confidence interval: 1.23-1.65) per unit of 100 µg/m^3·year cumulative benzo(a)pyrene exposure for bladder cancer. This value will be taken forward to the risk characterisation for aluminium smelters.

Furthermore, it is noted that lung cancer has a larger impact than bladder cancer on the excess life time risks (see Section B.10.1.2.7) and is therefore the most critical type of cancer with regard to exposure to CTPHT.

Based on the available experimental and epidemiological data on the carcinogenicity of CTPHT and CTPVHT and the evaluation of these data by the IARC, CTPHT and CTPVHT will be classified as a category 1 carcinogen (T; R45).

Based on the genotoxic and carcinogenic properties of CTPHT, for risk characterisation a non-threshold approach will be adopted.

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTPHT. 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/m^3 (NOAEC 30 mg/m^3). At the highest tested concentration (690 mg/m^3) 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$_{bw}$/day, 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) (Hackett *et al.* 1984; Zangar *et al.*, 1989; Springer *et al.* 1982, 1986b, 1987; Creosote Council Europe, 2004).

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 differences in sperm count and sperm morphology were observed in workers exposed to CTPV in an aluminium reduction plant. In a small retrospective study among psoriasis or dermatitis patients, dermal exposure to coal tar did not induce a significant increase in spontaneous abortion.

According to the Directive 1999/45/EC relating to the classification, packaging and labelling of dangerous preparations (EC, 1999b), preparations containing more than 0.5% of a substance classified as toxic for reproduction fertility and development need to be classified as a toxic for reproduction fertility and development. Since CTPHT may contain up to 1.5% benzo(a)pyrene, which is classified for effects on reproduction (category 2; T, R.60/61), it is proposed to classify CTPHT as toxic to reproduction(T; R60/61).

B.10.1.2 Workers

Assuming that oral exposure is prevented by personal hygiene measures, the risk characterisation for workers is limited to the dermal and inhalation routes of exposure.

B.10.1.2.1 Acute toxicity

Given the low toxicity observed in the acute oral and dermal toxicity studies and the anticipated occupational exposure levels it is concluded that CTPHT is of no concern for workers with regard to acute effects (conclusion ii).

B.10.1.2.2 Irritation

Skin

Skin effects were observed in animals and humans after repeated exposure to CTP(V) or combined exposure to CTP(V) and sunlight. However, from the available animal and human data it is not possible to conclude if the observed dermal effects are caused by irritation or sensitisation (photosensitisation or sensitisation after repeated exposure), therefore the data do not allow a conclusive statement on the skin irritating properties of CTPHT.

However, since it is concluded that the carcinogenic activity of CTPHT is the critical effect, the need for more information on local skin effects of CTPHT will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion i on hold).

Eye

Given the effects observed in humans exposed to CTP (fumes, volatiles and dust, not further specified), it is proposed to classify CTPHT as irritant with risk of serious damage to eyes (Xi, R41). Although the data are insufficient for quantitative risk characterisation, it is concluded that CTPHT is of concern for workers. However, if the required protection is strictly adhered to, exposure will occur only incidentally, so conclusion ii is justifiable.

B.10.1.2.3 Corrosivity

No experimental data on the potential corrosivity of CTPHT are available, however taking the available human and animal data into account, there are no indication that CTPHT has corrosive properties, so conclusion ii is justifiable.

B.10.1.2.4 Sensitisation

No experimental data on the sensitisation potential of CTPHT are available. However, since CTPHT may contain up to 1.5% benzo(a)pyrene, which is classified for skin sensitisation, it is proposed to classify CTPHT as a skin sensitizer (R43). The data are insufficient for quantitative risk characterisation. However, as sensitisation is considered as a non-threshold effect, it is concluded that CTPHT is of concern for workers (conclusion iii).

B.10.1.2.5 Repeated dose toxicity

No valid experimental animal studies addressing the potential non-carcinogenic effects of CTPHT were available to the rapporteur. In humans no statistically significant effects on lung function parameters were found in a group of phosphorus rock refinery workers exposed at the time of study to about 0.1 mg/m^3 CTPV in addition to other substances, including phosphorus pentoxide (about 2.2 mg/m^3) and fluorides (about 4.2 mg/m^3), after adjustment for smoking.

However, since it is concluded that the carcinogenic activity of CTPHT is the critical effect, the need for more information on non-carcinogenic effects of CTPHT after repeated exposure will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion i on hold).

B.10.1.2.6 Mutagenicity

Based on the proposal to classify CTPHT as a category 2 mutagen, it is concluded that exposure to CTPHT is associated with a mutagenic risk: conclusion iii.

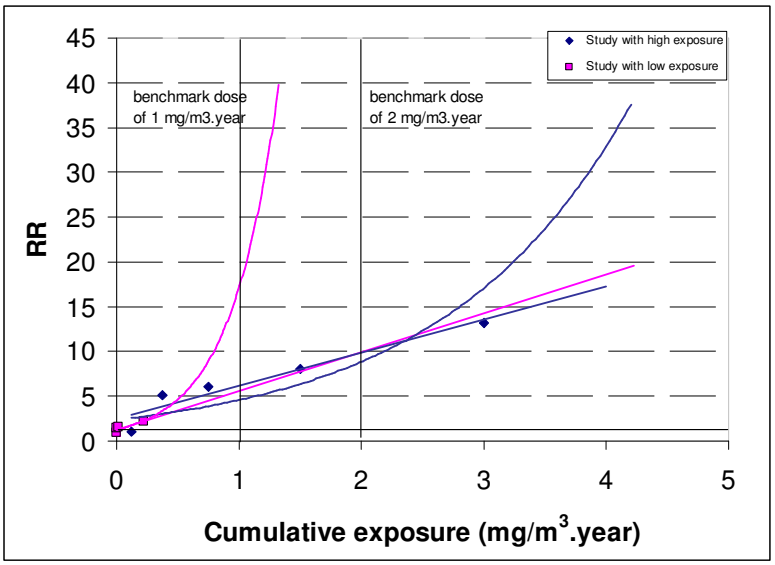
B.10.1.2.7 Carcinogenicity

Based on the available experimental and epidemiological data and the evaluation of these data by the IARC, it is concluded that CTPHT and CTPVHT should be classified as category 1 carcinogens (see B.5.8). Human data are mainly available on lung and bladder cancer risk in occupationally CTPVHT-exposed cohorts. Although a considerable number of epidemiological studies on CTPVHT exposure and risk of cancer is available, many of them have little statistical power (are imprecise), they vary with respect to type of industry and workplace, and in more than half of them no information on exposure is presented. 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 precise risk estimate based on all PAH-exposed cohorts is still considered superior to deriving industry-specific but very uncertain estimates. Although a few larger studies, mainly in the aluminium industry, are available, a better (*i.e.* precise and more realistic) risk estimate can be obtained using a weight-of-the-evidence approach, such as a meta-analysis (Goldbohm *et al.*, 2006). Recently, a meta-analysis on lung and bladder cancer risk after exposure to PAHs has been published by Armstrong *et al.* (2003; 2004) (see Section B.5.8). As exposure to benzo(a)pyrene has been adopted as the primary indicator of exposure to CTPVHT at the workplace (see Section B.9.1) and is also used as indicator of exposure in the meta-analysis, the results of this meta-analysis provide currently the best option for deriving a quantitative risk estimate for exposure to CTPVHT.

In this meta-analysis, unit relative risks for lung and bladder cancer were estimated by fitting a log-linear model to the data (Armstrong *et al.*, 2003; 2004). An overall relative risk per unit of 100 µg/m^3·year cumulative benzo(a)pyrene exposure of 1.20 (95% confidence interval: 1.11-1.29) for lung cancer and 1.33 (95% confidence interval: 1.17-1.51) for bladder cancer was calculated. The

combined unit relative risk estimates in aluminium smelters, the only industry exposed to CTPVHT for which rather precise estimates could be established, were 1.16 (95% confidence interval: 1.05-1.28) and 1.42 (95% confidence interval: 1.23-1.65) per unit of 100 µg/m^3·year cumulative benzo(a)pyrene exposure, for lung and bladder cancer, respectively.

Although a log-linear model is the most logical model to fit relative risks, it is not the best model per se for deriving quantitative risk estimates. In particular when benchmark exposures or exposure scenarios outside the range of data observed in the underlying study or studies are compared with the fitted model, unrealistic estimates may be the result (Goldbohm *et al.*, 2006). A linear relative risk model (relative risk = 1 + *bx*) is often better suited for risk assessment, but there are statistical limitations in conducting a meta-analysis fitting a linear model and results should be viewed more cautiously. However, Armstrong *et al.* (2004) also fitted a linear model, resulting in an overall unit relative risk of 1.19 for lung cancer, very similar to the overall estimate from the log-linear model, although estimates for the major industries differed more. For bladder cancer, no results on the linear model were reported. Comparison between industry-specific unit relative risks derived from the two models revealed that studies in industries with relatively low exposure, for example tar distillation, had very high unit relative risks in the log-linear model, but lower unit relative risks in the linear model. The explanation is that the benchmark exposure of 100 µg/m^3·year cumulative benzo(a)pyrene is much higher than that in the highest exposure category of industries with relatively low exposures (*e.g.* the benchmark of 100 µg/m^3·year is ten times higher than the exposure in the highest exposure category in tar distilleries) and therefore these industry-specific unit relative risks are overestimated using the results from the log-linear model due to extrapolation. See Figure B.10.1 for an illustration with a hypothetical example. Therefore industry-specific unit relative risks estimated with the log-linear model should not be used for industries for which the benchmark exposure (100 µg/m^3·year cumulative benzo(a)pyrene) is far higher than the observed exposure range.



A hypothetical example of fitting log-linear and linear slopes to exposure-risk data points from studies with relatively low exposure (below 1 mg/m^3·yr, red) to studies with relatively high exposure 0-3 mg/m^3·yr, blue). In the log-linear case, using a benchmark dose above the range of exposure data for which the curve was fitted, results in (severe) overestimation of the relative risk.

Figure B.10.1. Influence of the choice of a benchmark dose on predicted relative risk

Considering the results and arguments presented in the meta-analyses by Armstrong *et al.* (2004), the following decisions were taken in deriving risk estimates for each of the exposure scenarios addressed in this report.

1) The overall unit relative risks of 1.20 for lung cancer and 1.33 for bladder cancer, estimated from the log-linear model, are the best estimates for all relevant industry/workplace combinations.

Due to lack of statistical precision and extrapolation problems in studies with low exposures, industry-specific estimates do not provide the best estimate. An exception may be the aluminium smelters, as the statistical precision is sufficient and the benchmark exposure is comprised in the observed exposure range. The unit relative risks for aluminium smelters were 1.16 (95% confidence interval: 1.05-1.28) for lung cancer and 1.42 (95% confidence interval: 1.23-1.65) for bladder cancer.

2) Exposure scenarios resulting in exposures (much) higher than the benchmark exposure (100 µg/m^3·year cumulative benzo(a)pyrene exposure) should not be compared with the unit relative risk from the log-linear model, but with that from the linear model instead. This unit relative risk was 1.19 for lung cancer (no confidence interval and no unit relative risk for bladder cancer were presented). At exposures within the range of the data from which the unit relative risks were estimated, the log-linear and linear models will give similar estimates.

Excess lifetime risk (ELR) was calculated from the relative risk at the reasonable worst case exposure estimated for the specified exposure scenarios (see Table B.9.16) with the formula:

$$ELR = (RR \times P) - P$$

in which RR denotes the relative risk and P denotes the background risk in the exposed target population (*i.e.* the population figuring in the exposure scenario) (see Table B.10.1 and Table B.10.2).

As typical exposure levels may be substantially lower than the reasonable-worst-case exposure levels and it is unlikely that a worker is exposed to worst-case exposure during the whole working life, they might be valuable input to the risk management process (note: both types of exposure levels need to be well-defined in terms of technical and organisational conditions of exposure; see Sections 4.3 and 4.14.2.5 of the final draft TGD Human Health Risk Characterisation (EC, 2005): for this reason typical exposure values are included as well.

The relative risk at the exposure level specified in the exposure scenario, was calculated from the unit relative risk at 100 µg/m^3·year cumulative benzo(a)pyrene derived from the log-linear model as follows:

$$RR_x = URR^{\left(\frac{x}{100}\right)}$$

and from the linear model as follows:

$$RR_x = 1 + \left((URR - 1) \times \left(\frac{x}{100}\right)\right)$$

in which RR_x = relative risk at exposure level x, URR = unit relative risk and x = exposure level.

Background lifetime risks were chosen as 0.08 for lung cancer and 0.018 for bladder cancer, being the 1997 figures for British males, also used in the papers by Armstrong *et al.* (2003; 2004).

For comparison: in Europe in the mid-nineties, the background lifetime risks for male lung cancer up to age 74 varied between 0.10 (Eastern Europe) and 0.03 (Sweden), while bladder cancer risk varied between 0.05 (Italy) and 0.02 (Sweden) (IARC, 2002). As several uncertainties are inherently associated with the data and approach used (see also Section B.5.8.2.4), presentation of a calculated exact figure would be misleading. Therefore, the calculated excess lifetime risks (point estimates) were rounded to the nearest order of magnitude.

Table B.10.1. Occupational lung and bladder cancer risk characterisation workers using reasonable worst case exposure values.

Exposure scenario	Cancer type	Estimated RWC exposure [1] (µg/m³ benzo(a)pyrene)	Estimated RWC cumulative exposure [2] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [3] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [3]	Order of magnitude of estimated excess lifetime risk	Conclusion
1. a. Tar distillation plants-Tar processing and handling of liquid pitch	lung	0.4	16	1.20 (CI: 1.11-1.29)	log-linear	1.03 (CI: 1.02-1.04)	10^{-3}	iii
	bladder	0.4	16	1.33 (CI: 1.17-1.51)	log-linear	1.05 (CI: 1.03-1.07)	10^{-3}	iii
1. b. Tar distillation plants-Handling of solid pitch	lung	3.6	144	1.20 (CI: 1.11-1.29)	log-linear	1.30 (CI: 1.16-1.44)	10^{-2}	iii
	bladder	3.6	144	1.33 (CI: 1.17-1.51)	log-linear	1.51 (CI: 1.25-1.81)	10^{-2}	iii
2. i. a. Søderberg potroom Not modernised	lung	8	320	1.16 (CI: 1.05-1.28)	log-linear	1.61 (CI: 1.17-2.20)	10^{-2}	iii
	bladder	8	320	1.42 (CI: 1.23-1.65)	log-linear	3.07 (CI: 1.94-4.97)	10^{-2}	iii
2. i. b. Søderberg potroom Modernised	lung	0.35	14	1.16 (CI: 1.05-1.28)	log-linear	1.02 (CI: 1.01-1.04)	10^{-3}	iii
	bladder	0.35	14	1.42 (CI: 1.23-1.65)	log-linear	1.05 (CI: 1.03-1.07)	10^{-3}	iii
2. i. c. Anode bake plants	lung	0.40	16	1.16 (CI: 1.05-1.28)	log-linear	1.02 (CI: 1.01-1.04)	10^{-3}	iii
	bladder	0.40	16	1.42 (CI: 1.23-1.65)	log-linear	1.06 (CI: 1.03-1.08)	10^{-3}	iii
2. i. d. Paste plants	lung	0.15	6	1.16 (CI: 1.05-1.28)	log-linear	1.01 (CI: 1.0-1.01)	10^{-3}	iii
	bladder	0.15	6	1.42 (CI: 1.23-1.65)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
2. ii. Graphite electrode past plants	lung	7.5	300	1.20 (CI: 1.11-1.29)	log-linear	1.73 (CI: 1.37-2.15)	10^{-1}	iii
	bladder	7.5	300	1.33 (CI: 1.17-1.51)	log-linear	2.35 (CI: 1.60-3.44)	10^{-2}	iii
3. i. Road construction	lung	1.2	48	1.20 (CI: 1.11-1.29)	log-linear	1.09 (CI: 1.05-1.13)	10^{-2}	iii
	bladder	1.2	48	1.33 (CI: 1.17-1.51)	log-linear	1.15 (CI: 1.08-1.22)	10^{-3}	iii
3. ii. Roofing	lung	60	2400	1.19	linear	5.56	10^{-1}	iii
	bladder	60	2400	n.a.[3]	linear		10^{-2}	iii
4. a. Production of refractories	lung	3.5	140	1.20 (CI: 1.11-1.29)	log-linear	1.29 (CI: 1.16-1.43)	10^{-2}	iii
	bladder	3.5	140	1.33 (CI: 1.17-1.51)	log-linear	1.49 (CI: 1.25-1.78)	10^{-2}	iii
4. b. Use of refractories	lung	23	920	1.19	linear	2.75	10^{-1}	iii
	bladder	23	920	n.a. [4]	linear		10^{-2}	iii
6. a. Use-Heavy duty corrosion protection-coating operators	lung	90	3600	1.19	linear	7.84	$>10^{-1}$	iii
	bladder	90	3600	n.a. [4]	linear		10^{-2}	iii
6. b. Use-Heavy duty corrosion protection-other workers	lung	30	1200	1.19	linear	3.28	10^{-1}	iii
	bladder	30	1200	n.a. [4]	linear		10^{-2}	iii

Exposure scenario	Cancer type	Estimated RWC exposure [1] (µg/m³ benzo(a)pyrene)	Estimated RWC cumulative exposure [2] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [3] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [3]	Order of magnitude of estimated excess lifetime risk	Conclusion
7. a. Use-Binder in coal briquetting-Production	lung	1760	70400	1.19	linear	135	$>10^{-1}$	iii
	bladder	1760	70400	n.a. [4]	linear		10^{-2}	iii
7. b. Use-Binder in coal briquetting-Cleaning	lung	40	1600	1.19	linear	4.04	10^{-1}	iii
	bladder	40	1600	n.a. [4]	linear		10^{-2}	iii
8. Binder for clay pigeons	lung	3	120	1.20 (CI: 1.11-1.29)	log-linear	1.24 (CI: 1.13-1.36)	10^{-2}	iii
	bladder	3	120	1.33 (CI: 1.17-1.51)	log-linear	1.41 (CI: 1.21-1.64)	10^{-2}	iii

[1] Reasonable Worst Case (RWC) exposure (Time Weighted Average over 8 hours of airborne concentration); [2] Reasonable Worst Case (RWC) cumulative exposure (Time Weighted Average over 8 hours x 40 year) ; [3] CI: 95% confidence interval; [4] Linear unit relative risk estimates were not available for bladder cancer (indicated by n.a.). In these cases, the excess lifetime risk for bladder cancer was assumed to be approximately one third of that for lung cancer as the excess lifetime risk values for bladder cancer are about one third of the excess lifetime risk value for lung cancer for each scenario for which the log-linear method was used; [4] CI: 95% confidence interval.

Table B.10.2. Occupational lung and bladder cancer risk characterisation workers using typical exposure values.

Exposure scenario	Cancer type	Estimated RWC exposure [1] (µg/m³ benzo(a)pyrene)	Estimated RWC cumulative exposure [2] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [3] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [3]	Order of magnitude of estimated excess lifetime risk	Conclusion
1. a. Tar distillation plants-Tar processing and handling of liquid pitch	lung	0.1	4	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.0-1.01)	10^{-3}	iii
	bladder	0.1	4	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.01-1.02)	10^{-4}	iii
1. b. Tar distillation plants-Handling of solid pitch	lung	2.6	104	1.20 (CI: 1.11-1.29)	log-linear	1.21 (CI: 1.11-1.30)	10^{-2}	iii
	bladder	2.6	104	1.33 (CI: 1.17-1.51)	log-linear	1.35 (CI: 1.18-1.54)	10^{-2}	iii
2. i. a. Søderberg potroom Not modernised	lung	1	40	1.16 (CI: 1.05-1.28)	log-linear	1.06 (CI: 1.02-1.10)	10^{-2}	iii
	bladder	1	40	1.42 (CI: 1.23-1.65)	log-linear	1.15 (CI: 1.09-1.22)	10^{-3}	iii
2. i. b. Søderberg potroom Modernised	lung	0.20	8	1.16 (CI: 1.05-1.28)	log-linear	1.01 (CI: 1.0-1.02)	10^{-3}	iii
	bladder	0.20	8	1.42 (CI: 1.23-1.65)	log-linear	1.03 (CI: 1.02-1.04)	10^{-3}	iii
2. i. c. Anode bake plants	lung	0.15	6	1.16 (CI: 1.05-1.28)	log-linear	1.01 (CI: 1.0-1.01)	10^{-3}	iii
	bladder	0.15	6	1.42 (CI: 1.23-1.65)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
2. i. d. Paste plants	lung	0.08	3.2	1.16 (CI: 1.05-1.28)	log-linear	1.0 (CI: 1.0-1.01)	10^{-4}	iii
	bladder	0.08	3.2	1.42 (CI: 1.23-1.65)	log-linear	1.01 (CI: 1.01-1.02)	10^{-4}	iii
2. ii. Graphite electrode past plants	lung	2	80	1.20 (CI: 1.11-1.29)	log-linear	1.16 (CI: 1.09-1.23)	10^{-2}	iii
	bladder	2	80	1.33 (CI: 1.17-1.51)	log-linear	1.26 (CI: 1.13-1.39)	10^{-2}	iii

Exposure scenario	Cancer type	Estimated RWC exposure [1] (µg/m³ benzo(a)pyrene)	Estimated RWC cumulative exposure [2] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [3] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [3]	Order of magnitude of estimated excess lifetime risk	Conclusion
3. i. Road construction	lung	0.55	22	1.20 (CI: 1.11-1.29)	log-linear	1.04 (CI: 1.02-1.06)	10^{-3}	iii
	bladder	0.55	22	1.33 (CI: 1.17-1.51)	log-linear	1.06 (CI: 1.04-1.09)	10^{-3}	iii
3. ii. Roofing	lung	35	1400	1.19	linear	3.66	10^{-1}	iii
	bladder	35	1400	n.a.[4]	linear		10^{-2}	iii
4. a. Production of refractories	lung	0.17	6.8	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.02)	10^{-3}	iii
	bladder	0.17	6.8	1.33 (CI: 1.17-1.51)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
4. b. Use of refractories	lung	0.63	25.2	1.20 (CI: 1.11-1.29)	log-linear	1.05 (CI: 1.03-1.07)	10^{-3}	iii
	bladder	0.63	25.2	1.33 (CI: 1.17-1.51)	log-linear	1.07 (CI: 1.04-1.11)	10^{-3}	iii
6. a. Use-Heavy duty corrosion protection-coating operators	lung	23	920	1.19	linear	2.75	10^{-1}	iii
	bladder	23	920	n.a. [4]	linear		10^{-2}	iii
6. b. Use-Heavy duty corrosion protection-other workers	lung	6	240	1.20 (CI: 1.11-1.29)	log-linear	1.55 (CI: 1.28-1.84)	10^{-2}	iii
	bladder	6	240	1.33 (CI: 1.17-1.51)	log-linear	1.98 (CI: 1.46-2.69)	10^{-2}	iii
7. a. Use-Binder in coal briquetting-Production	lung	670	26800	1.19	linear	52	$>10^{-1}$	iii
	bladder	670	26800	n.a. [4]	linear		10^{-2}	iii
7. b. Use-Binder in coal briquetting-Cleaning	lung	14	560	1.19	linear	2.06	$>10^{-1}$	iii
	bladder	40	1600	n.a. [4]	linear		10^{-2}	iii
8. Binder for clay pigeons	lung	1	40	1.20 (CI: 1.11-1.29)	log-linear	1.08 (CI: 1.04-1.11)	10^{-2}	iii
	bladder	1	40	1.33 (CI: 1.17-1.51)	log-linear	1.12 (CI: 1.06-1.18)	10^{-3}	iii

[1] Reasonable Worst Case (RWC) exposure (Time Weighted Average over 8 hours of airborne concentration); [2] Reasonable Worst Case (RWC) cumulative exposure (Time Weighted Average over 8 hours x 40 year) ; [3] CI: 95% confidence interval.; [4] Linear unit relative risk estimates were not available for bladder cancer (indicated by n.a.). In these cases, the excess lifetime risk for bladder cancer was assumed to be approximately one third of that for lung cancer as the excess lifetime risk values for bladder cancer are about one third of the excess lifetime risk value for lung cancer for each scenario for which the log-linear method was used

Table B.10.3. Comparison of occupational lung and bladder cancer risk characterisation for workers using reasonable worst case (RWC) and typical exposure values.

Exposure scenario	Cancer type	Estimated RWC cumulative exposure (µg/m³ benzo(a)pyrene·year)	Order of magnitude of estimated excess lifetime risk	Conclusion	Estimated typical cumulative exposure (µg/m³ benzo(a)pyrene·year)	Order of magnitude of estimated excess lifetime risk	Conclusion
1. a. Tar distillation plants-Tar processing and handling of liquid pitch	lung	16	10^{-3}	iii	4	10^{-3}	iii
	bladder	16	10^{-3}	iii	4	10^{-4}	iii
1. b. Tar distillation plants-Handling of solid pitch	lung	144	10^{-2}	iii	104	10^{-2}	iii
	bladder	144	10^{-2}	iii	104	10^{-2}	iii
2. i. a. Søderberg potroom Not modernised	lung	320	10^{-2}	iii	40	10^{-2}	iii
	bladder	320	10^{-2}	iii	40	10^{-3}	iii
2. i. b. Søderberg potroom Modernised	lung	14	10^{-3}	iii	8	10^{-3}	iii
	bladder	14	10^{-2}	iii	8	10^{-3}	iii
2. i. c. Anode bake plants	lung	16	10^{-3}	iii	6	10^{-3}	iii
	bladder	16	10^{-3}	iii	6	10^{-4}	iii
2. i. d. Paste plants	lung	6	10^{-3}	iii	3.2	10^{-4}	iii
	bladder	6	10^{-4}	iii	3.2	10^{-4}	iii
2. ii. Graphite electrode past plants	lung	300	10^{-1}	iii	80	10^{-2}	iii
	bladder	300	10^{-2}	iii	80	10^{-2}	iii
3. i. Road construction	lung	48	10^{-2}	iii	22	10^{-3}	iii
	bladder	48	10^{-3}	iii	22	10^{-3}	iii
3. ii. Roofing	lung	2400	10^{-1}	iii	1400	10^{-1}	iii
	bladder	2400	10^{-2}	iii	1400	10^{-2}	iii
4. a. Production of refractories	lung	140	10^{-2}	iii	6.8	10^{-3}	iii
	bladder	140	10^{-2}	iii	6.8	10^{-4}	iii
4. b. Use of refractories	lung	920	10^{-1}	iii	25.2	10^{-3}	iii
	bladder	920	10^{-2}	iii	25.2	10^{-3}	iii
6. a. Use-Heavy duty corrosion protection-coating operators	lung	3600	$>10^{-1}$	iii	920	10^{-1}	iii
	bladder	3600	10^{-2}	iii	920	10^{-2}	iii
6. b. Use-Heavy duty corrosion protection-other workers	lung	1200	10^{-1}	iii	240	10^{-2}	iii
	bladder	1200	10^{-2}	iii	240	10^{-2}	iii

Exposure scenario	Cancer type	Estimated RWC cumulative exposure (µg/m³ benzo(a)pyrene·year)	Order of magnitude of estimated excess lifetime risk	Conclusion
7. a. Use-Binder in coal briquetting-Production	lung	70400	$>10^{-1}$	iii
	bladder	70400	10^{-2}	iii
7. b. Use-Binder in coal briquetting-Cleaning	lung	1600	10^{-1}	iii
	bladder	1600	10^{-2}	iii
8. Binder for clay pigeons	lung	120	10^{-2}	iii
	bladder	120	10^{-2}	iii

Estimated typical cumulative exposure (µg/m³ benzo(a)pyrene·year)	Order of magnitude of estimated excess lifetime risk	Conclusion
26800	$>10^{-1}$	iii
26800	10^{-2}	iii
560	$>10^{-1}$	iii
560	10^{-2}	iii
40	10^{-2}	iii
40	10^{-3}	iii

Because only data on airborne concentrations are available from the epidemiological studies, the (8-h Time Weighted Average of the) airborne concentration of benzo(a)pyrene (in $\mu g/m^3$) is used for risk assessment (see Section B.9.1). It is assumed that in the epidemiological studies, the effects of combined exposure (inhalation and dermal) were studied (see Section B.5.8.2) Assuming a constant (linear) relationship between the airborne concentration and the inhalation as well as the dermal exposure, the airborne concentration can be used for risk assessment of combined exposure.

All excess lifetime risk values listed in Table B.10.1 and Table B.10.2 are equal or higher than an additional risk level of 1×10^{-4}. Therefore, not only the reasonable worst case exposure estimates but also the typical exposure estimates for the specified exposure scenarios lead to unacceptable high risks for lung as well as bladder cancer, respectively. Application of other background lifetime risks of lung and bladder cancer as prevailing in Europe (with a maximum threefold variation across the countries), does not alter these conclusions: therefore, conclusion iii is drawn.

There is insufficient information with regard to exposure scenario 5 for the derivation of exposure estimates. However, based on the proposal to classify CTPHT and CTPVHT as category 1 carcinogens and a category 2 mutagen, and the quantitative risk assessment for the other exposure scenarios, conclusion iii is also applicable for scenario 5.

B.10.1.2.8 Toxicity for reproduction

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTPHT. However, animal studies have shown that exposure to high-boiling coal liquid, coal tar derived products, and creosote cause effects on fertility in mice and rats. Although some developmental effects were also observed in these studies, it is not clear that they were directly induced by high-boiling coal liquid, coal tar derived products, or creosote. In humans no adverse effects on sperm characteristics were observed in workers exposed to CTPV in an aluminium reduction plant. In a small retrospective study among psoriasis or dermatitis patients, dermal exposure of to coal tar did not induce a significant increase in spontaneous abortion.

Since CTPHT may contain up to 1.5% benzo(a)pyrene (classified as toxic for effects on reproduction; category 2) it is proposed to classify CTPHT as toxic to reproduction (category 2). Although the data are insufficient for quantitative risk characterisation, it is concluded that CTPHT is of concern for workers. However, since it is concluded that the carcinogenic activity of CTPHT is the critical effect, the need for more information on the reproductive toxicity of CTPHT will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion i on hold).

B.10.1.2.9 Occupational limit values

In Table B.9.1 occupational limit values for CTPV are specified.

B.10.1.3 Consumers

Since there is no consumer exposure, no risk characterization is performed.

B.10.1.4 Humans exposed via the environment

In view of the differences in physical-chemical parameters, the exposure to the different PAHs for humans exposed via the environment will occur via different routes. In principle, this would mean that the risk characterisation should be based on the effects of each individual component. However, as the composition of CTP is variable and unknown and the effects of the known individual components are mostly unknown, this is practically impossible.

From the available database it appears that carcinogenicity is a striking hazard associated with CTPHT, attributable to the presence of PAHs in CTPHT, and that benzo(a)pyrene is the best-studied PAH and one of the most toxic ones. Therefore, as a practical solution benzo(a)pyrene is

chosen as the 'leading' PAH on which the risk characterisation will focus. Although carcinogenicity may not be the only relevant effect of CTPHT, given the quite high carcinogenic potencies of the PAHs it is likely that limitation of the risk for cancer will automatically reduce the risk for any other possible effect, quite possibly even to zero.

B.10.1.4.1 Repeated dose toxicity

No valid experimental animal studies or human data addressing the potential non-carcinogenic effects of CTPHT were available to the rapporteur. However, since it is concluded that the carcinogenic activity of CTPHT is the critical effect, the need for more information on non-carcinogenic effects of CTPHT after repeated exposure will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion i on hold).

B.10.1.4.2 Mutagenicity

Based on the classification of CTPHT as a category 2 mutagen, it is concluded that exposure to CTPHT is associated with a mutagenic risk: conclusion iii.

B.10.1.4.3 Carcinogenicity

CTPHT and CTPVHT are classified as category 1 carcinogens. For quantitative risk assessment, valid human data (mainly in occupationally CTPVHT-exposed cohorts) and experimental animal data are available for inhalation and oral exposure, respectively.

Exposure via air-Local

For the inhalatory route, the risks for humans exposed via the environment to CTPHT can be determined using benzo(a)pyrene as a marker for total PAHs in the same way as for workers because of the low volatility of the carcinogenic PAHs (the more volatile PAHs are less carcinogenic). Aerosol particles with a fixed ratio of PAHs are formed during the different processes described and will either be released from the factory or be removed from the air. It is assumed that the ratio of the carcinogenic PAHs in the released aerosols will be the same as for the worker.

In conformity with the risk characterization for workers (see Section B.10.1.2.7), starting points for the risk characterization for humans exposed inhalatory via the environment are the airborne concentrations of benzo(a)pyrene from Table B.9.17 and the unit relative risks for lung and bladder cancer as estimated by Armstrong *et al.* (2003; 2004) in a recent meta-analysis on lung and bladder cancer risk after occupational exposure to PAHs, using benzo(a)pyrene as indicator of exposure. For lung cancer, the overall unit relative risk per unit of 100 µg/m^3·year cumulative benzo(a)pyrene exposure was 1.20 (95% confidence interval: 1.11-1.29), and for bladder cancer this was 1.33 (95% confidence interval: 1.17-1.51).

First, the exposure estimates for the different sites were multiplied by 70, to account for lifetime (70 years) exposure. Then, the relative risks at the (cumulative) exposure level were calculated from the unit relative risks at 100 µg/m^3·year cumulative benzo(a)pyrene as follows:

$$RR_x = URR^{\left(\frac{x}{100}\right)}$$

in which RR_x = relative risk at exposure level *x*, URR = unit relative risk and *x* = exposure level. Subsequently, excess lifetime risks (ELR) were calculated from the RRs with the formula:

$$ELR = (RR \times P) - P$$

in which RR = relative risk and P is the background lifetime risk in the exposed target population (*i.e.* the population figuring in the exposure scenario). Background lifetime risks were chosen as 0.08 for lung cancer and 0.018 for bladder cancer, being the 1997 figures for British males, also

used by Armstrong *et al.* (2003; 2004). As several uncertainties are inherently associated with the data and approach used, presentation of a calculated exact figure would be misleading. Therefore, the calculated ELRs (point estimates) were rounded to the nearest order of magnitude.

The results are presented in Table B.10.4.

From Table B.10.4 it can be seen that the ELR for lung cancer is related to the concentration of benzo(a)pyrene in air as follows:

ELR lung cancer	Concentration benzo(a)pyrene in air
$\leq 1x10^{-6}$	0-0.1 ng/m^3
between $1x10^{-6}$ and $1x10^{-5}$	0.1-1 ng/m^3
between $1x10^{-5}$ and $1x10^{-4}$	1-10 ng/m^3
between $1x10^{-4}$ and $1x10^{-3}$	10-100 ng/m^3
between $1x10^{-3}$ and $1x10^{-2}$	100-1000 ng/m^3

Similarly, for bladder cancer the relation is as follows:

ELR bladder cancer	Concentration benzo(a)pyrene in air
$\leq 1x10^{-6}$	0 -0.3 ng/m^3
between $1x10^{-6}$ and $1x10^{-5}$	0.3-3 ng/m^3
between $1x10^{-5}$ and $1x10^{-4}$	3-30 ng/m^3
between $1x10^{-4}$ and $1x10^{-3}$	30-300 ng/m^3
between $1x10^{-3}$ and $1x10^{-2}$	300-3000 ng/m^3

With a few exceptions (sites PA3 and PA15), all excess lifetime risk values listed in Table B.10.4 were equal to or higher than an additional risk level of 1×10^{-6}. Therefore, the inhalatory exposure estimates for all but 2 sites lead to unacceptable high risks for lung as well as bladder cancer. Therefore, a conclusion iii is drawn for these sites. For sites PA3 and PA15 also a conclusion iii is drawn, but for these two scenarios the level of concern is low.

Exposure via food and water-Local

For the oral route, the risks for humans exposed via the environment to CTPHT should be determined for the 16 individual PAHs because the ratio of the PAHs in the human intake media will be different. However, as a practical approach in first instance the carcinogenic risk due to benzo(a)pyrene will be determined. If already for this one PAH a risk is identified, the other 15 PAHs will not be considered further, nor the combination of these PAHs.

Starting points for the risk characterization for humans exposed orally via the environment are the intake estimates for benzo(a)pyrene from Table B.9.18 and the overall dose descriptor T_{25} derived for benzo(a)pyrene from the oral carcinogenicity studies in mice and rats described in B.5.8.

In the study by Culp *et al.* (1998), administration of 0, 5, 25, or 100 ppm benzo(a)pyrene in the diet of female B6C3F1 mice for 2 years resulted in a dose-related increase in papillomas and/or carcinomas of the forestomach, oesophagus, tongue and larynx (see Table B.5.5 in B.5.8). The lowest dose at which a statistically significant increased tumour incidence was observed was 25 ppm (equivalent to 3 mg/kg$_{bw}$/d): at this dose the incidence of forestomach neoplasms was 36/46, as compared to 1/48 for the control group, resulting in a net incidence of 76% ((36-1)/46). The T_{25} from this study was calculated to be 1 mg/kg$_{bw}$/d (*i.e.* (25/76)×3 mg/kg$_{bw}$/d).

Table B.10.4. Lung and bladder cancer risk characterisation for humans exposed inhalatory via the environment.

Source	Cancer type	Estimated exposure (ng/m³ benzo(a)pyrene)	Estimated cumulative exposure [1] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [2] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [2]	Order of magnitude of estimated excess lifetime risk	Conclusion
PRODUCTION SITES								
1	lung	7.7	0.54	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	7.7	0.54	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
3	lung	5.5	0.39	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	5.5	0.39	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
4	lung	2.0	0.14	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	2.0	0.14	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
5	lung	4.9	0.34	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	4.9	0.34	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
6	lung	4.1	0.29	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	4.1	0.29	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00))	10^{-5}	iii
7	lung	1.6	0.11	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	1.6	0.11	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
8	lung	6.1	0.43	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	6.1	0.43	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
9	lung	4.6	0.32	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	4.6	0.32	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
DOWNSTREAM USERS								
Ferro-alloy	lung	56	3.9	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.00-1.01)	10^{-3}	iii
	bladder	56	3.9	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.01-1.02)	10^{-4}	iii
Graphite	lung	13	0.91	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	13	0.91	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
PRIMARY ALUMINIUM PRODUCTION AND ANODE BAKING FACILITIES								
S1	lung	36	2.5	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.01)	10^{-4}	iii
	bladder	36	2.5	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.00-1.01)	10^{-4}	iii
S3 [3]	lung	92	6.4	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.02)	10^{-3}	iii
	bladder	92	6.4	1.33 (CI: 1.17-1.51)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii

Source	Cancer type	Estimated exposure (ng/m³ benzo(a)pyrene)	Estimated cumulative exposure [1] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [2] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [2]	Order of magnitude of estimated excess lifetime risk	Conclusion
S4 [3]	lung	98	6.9	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.02)	10^{-3}	iii
	bladder	98	6.9	1.33 (CI: 1.17-1.51)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
P7	lung	2.9	0.20	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	2.9	0.20	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
S5	lung	100	7.0	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.02)	10^{-3}	iii
	bladder	100	7.0	1.33 (CI: 1.17-1.51)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
S6	lung	98	6.9	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.02)	10^{-3}	iii
	bladder	98	6.9	1.33 (CI: 1.17-1.51)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
PA1+S2	lung	27	1.9	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	27	1.9	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.00-1.01)	10^{-4}	iii
PA2	lung	11	0.77	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	11	0.77	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
PA3	lung	0.01	0.0007	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-7}	iii [2]
	bladder	0.01	0.0007	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-8}	iii [2]
PA4	lung	1.2	0.084	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	1.2	0.084	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-6}	iii
PA5	lung	7.3	0.51	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	7.3	0.51	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
PA6	lung	70	4.9	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.01)	10^{-3}	iii
	bladder	70	4.9	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.01-1.02)	10^{-4}	iii
PA7	lung	1.1	0.077	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	1.1	0.077	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-6}	iii
PA8	lung	0.26	0.018	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-6}	iii
	bladder	0.26	0.018	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-6}	iii
PA9	lung	610	42.7	1.20 (CI: 1.11-1.29)	log-linear	1.08 (CI: 1.05-1.11)	10^{-2}	iii
	bladder	610	42.7	1.33 (CI: 1.17-1.51)	log-linear	1.13 (CI: 1.07-1.19)	10^{-3}	iii
PA10	lung	6.8	0.48	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	6.8	0.48	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii

Source	Cancer type	Estimated exposure (ng/m³ benzo(a)pyrene)	Estimated cumulative exposure [1] (µg/m³ benzo(a)pyrene·year)	Estimated unit relative risk [2] (per 100 µg/m³ benzo(a)pyrene·year)	Model	Calculated relative risk at the estimated cumulative exposure level [2]	Order of magnitude of estimated excess lifetime risk	Conclusion
PA11	lung	26	1.8	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-4}	iii
	bladder	26	1.8	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.00-1.01)	10^{-4}	iii
PA12	lung	0.73	0.051	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-5}	iii
	bladder	0.73	0.051	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-6}	iii
PA13	lung	94	6.6	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.02)	10^{-3}	iii
	bladder	94	6.6	1.33 (CI: 1.17-1.51)	log-linear	1.02 (CI: 1.01-1.03)	10^{-4}	iii
PA14	lung	70	4.9	1.20 (CI: 1.11-1.29)	log-linear	1.01 (CI: 1.01-1.01)	10^{-3}	iii
	bladder	70	4.9	1.33 (CI: 1.17-1.51)	log-linear	1.01 (CI: 1.01-1.02)	10^{-4}	iii
PA15	lung	0.031	0.002	1.20 (CI: 1.11-1.29)	log-linear	1.00 (CI: 1.00-1.00)	10^{-7}	iii [4]
	bladder	0.031	0.002	1.33 (CI: 1.17-1.51)	log-linear	1.00 (CI: 1.00-1.00)	10^{-7}	iii [4]
A1	lung	380	26.6	1.20 (CI: 1.11-1.29)	log-linear	1.05 (CI: 1.03-1.07)	10^{-3}	iii
	bladder	380	26.6	1.33 (CI: 1.17-1.51)	log-linear	1.08 (CI: 1.04-1.12)	10^{-3}	iii

[1] Estimated exposure × 70 years; [2] CI: 95% confidence interval; [3] For site S3 and S4, measurements in the vicinity of the plants showed that the air concentrations were more than 10 times lower than predicted. Provided that the measurements are reliable and representative, the excess lifetime risk will be 10 times lower as well; [4] Low concern.

In the study by RIVM (Kroese *et al.*, 2001), gavage administration of 0, 3, 10, or 30 mg benzo(a)pyrene/kg_{bw} in soy-oil, 5 days a week for 104 weeks, to male and female Riv:TOX rats of the Wistar strain resulted in dose-dependent increases in tumour incidence in a variety of organs and/or tissues in both sexes (see Table B.5.6 in B.5.8). The most prominent carcinogenic effects were observed in the liver, forestomach, and epidermal structures (among 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}/d and higher. At 10 mg/kg_{bw}/d, the incidence of hepatocellular adenomas and carcinomas was 38/52 in male rats (as compared to 0/52 in controls; net incidence 73%) and 39/52 in female rats (as compared to 0/52 in controls; net incidence 75%). The T_{25} from this study was calculated to be 2.4 mg/kg_{bw}/d (*i.e.* (25/75)×10 mg/kg_{bw}/d, corrected by 5/7 because the animals were treated only 5 days a week).

The lowest, overall T_{25} of 1 mg/kg_{bw}/d is used for the risk characterisation. From this T_{25} a human T_{25} (HT_{25}) of 0.14 mg/kg_{bw}/d is calculated by applying an overall assessment factor of 7 to the T_{25}. The overall assessment factor of 7 only covers for the allometric scaling part of interspecies differences, which is 7 when extrapolating from mice to humans. Other factors (*e.g.* for intraspecies differences) can be set to 1, because according to the final draft TGD on human health risk characterisation (EC, 2005) the linear model used for high to low dose extrapolation is considered sufficiently conservative to cover also for these factors.

The estimated lifetime risks for the exposures in the different scenarios were calculated from the HT_{25} using the formula:

$$\text{estimated lifetime risk} = \frac{\text{exposure}}{\left(HT_{25}/0.25\right)}$$

The calculated estimated lifetime risks (point estimates) were rounded to the nearest order of magnitude. The results are presented in Table B.10.5.

All estimated lifetime risk values listed in Table B.10.5 were equal to or higher than an additional risk level of $1 \cdot 10^{-6}$. Therefore, for all sites the total oral exposure estimates lead to unacceptable high risks for cancer. Therefore, a conclusion iii is drawn for all sites.

Since already exposure to this one PAH shows a considerable risk for cancer, the carcinogenic risks of the 15 other PAHs will not be determined, nor the carcinogenic risk for the combined PAHs. It is to be noted, though, that if there are carcinogens among these PAHs with higher potency than benzo(a)pyrene, the estimated lifetime risk could be even higher, depending on the exposure estimates for these higher potency PAHs. As to combined exposure to all 16 PAHs, this could also result in even higher lifetime risks than for benzo(a)pyrene alone.

Exposure via air and food and water-Regional

As indicated in Section B.9.4, no formal conclusions will be derived for the regional background exposure because of the many unintentional sources contributing to the total emission of PAHs into the environment. For illustrative purposes, however, the lifetime risks have been calculated for the lowest and highest regional benzo(a)pyrene concentrations found in air (0.02 and 39 ng/m^3, respectively; see Table B.9.20 in Section B.9.4.3) and for the resulting lowest and highest total daily benzo(a)pyrene intake (1.6 and 3100 ng/kg_{bw}/d, respectively; see Table B.9.21 in Section B.9.4.3), in the same way as described above for the local exposures. The results are presented in Table B.10.6.

Table B.10.5. Cancer risk characterisation for humans exposed orally via the environment.

Source	Total daily intake (ng/kg$_{bw}$/day)	estimated lifetime risk	Conclusion
Production sites			
1	620	10^{-3}	iii
3	440	10^{-3}	iii
4	160	10^{-4}	iii
5	390	10^{-3}	iii
6	330	10^{-3}	iii
7	130	10^{-4}	iii
8	490	10^{-3}	iii
9	370	10^{-3}	iii
Downstream users			
Ferro-alloy	4500	10^{-2}	iii
Graphite	1000	10^{-3}	iii
Primary aluminium production and anode baking facilities			
S1	2900	10^{-2}	iii
S3 [1)]	7400	10^{-2}	iii
S4 [1)]	7900	10^{-2}	iii
P7	230	10^{-4}	iii
S5	8000	10^{-2}	iii
S6	7900	10^{-2}	iii
PA1+S2	2200	10^{-3}	iii
PA2	880	10^{-3}	iii
PA3	0.80	10^{-6}	iii
PA4	96	10^{-4}	iii
PA5	590	10^{-3}	iii
PA6	5600	10^{-2}	iii
PA7	88	10^{-4}	iii
PA8	21	10^{-5}	iii
PA9	49000	10^{-1}	iii
PA10	550	10^{-3}	iii
PA11	2100	10^{-3}	iii
PA12	59	10^{-4}	iii
PA13	7500	10^{-2}	iii
PA14	5600	10^{-2}	iii
PA15	2.5	10^{-6}	iii
A1	31000	10^{-1}	iii

[1)] Based on the measured air concentrations in the vicinity of the plants at site S3 and S4 that were more than 10 times lower than predicted, the total daily intake might be 10 times lower, and consequently also the estimated lifetime risk.

Table B.10.6. Cancer risk characterisation for humans exposed via the environment-regional.

Regional	excess lifetime risk	estimated lifetime risk	Conclusion
Air concentration of benzo(a)pyrene			
0.02 ng/m^3	10^{-7} 1)		iii 2)
39 ng/m^3	10^{-4} 1)		iii
Total daily intake of benzo(a)pyrene			
1.6 ng/kg$_{bw}$/d		10^{-6}	iii
3100 ng/kg$_{bw}$/d		10^{-2}	iii

1) The same values hold for both lung and bladder; 2) Low concern.

B.10.1.4.4 Toxicity for reproduction

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTPHT. However, animal studies have shown that exposure to high-boiling coal liquid, coal tar derived products, and creosote cause effects on fertility in mice and rats. Although some developmental effects were also observed in these studies, it is not clear that they were directly induced by high-boiling coal liquid, coal tar derived products, or creosote. In humans no adverse effects on sperm characteristics were observed in workers exposed to CTPV in an aluminium reduction plant. In a small retrospective study among psoriasis or dermatitis patients, dermal exposure of to coal tar did not induce a significant increase in spontaneous abortion.

Since CTPHT may contain up to 1.5% benzo(a)pyrene (classified as toxic for effects on reproduction, category 2), CTPHT is classified as toxic to reproduction (category 2). Although the data are insufficient for quantitative risk characterisation, it is concluded that CTPHT is of concern for humans exposed indirectly via the environment. However, since it is concluded that the carcinogenic activity of CTPHT is the critical effect, the need for more information on the reproductive toxicity of CTPHT will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion i on hold).

B.10.2 Environment

Considering that a range of PAHs are emitted simultaneously, it is obvious to assess the risk for the mixture of PAHs and not for the PAHs individually. A common method to determine the toxicity of a mixture is the toxic unit concept. A toxic unit (TU) is defined as the ratio of the concentration in a medium to the effect concentration in that medium. The toxicity of the mixture is the sum of the individuals TUs. Use of the toxic unit concept requires that the dose-response relationships of the individual compounds have similar shapes, which in general holds for compounds with the same mode of action. The additivity of the toxicity of narcotic chemicals has been demonstrated by a number of investigators and is also considered applicable for PAHs (DiToro *et al.*, 2000; DiToro & McGrath, 2000). As shown in Section B.7, the most sensitive endpoints were not for all PAHs based on the same mode of action. For a limited number of PAHs (anthracene, fluoranthene, pyrene and chrysene) the lowest toxicity is based on phototoxicity and not non-polar narcosis. However, the difference in toxicity is overall small and limited to the aquatic compartment. Therefore, the TU approach is considered feasible for the sum of the 16 EPA PAHs.

For the risk assessment of CTPHT the TU is expressed as a ratio of the C_{local} to the PNEC for each PAH. The toxicity of the combination of PAHs is assessed by adding all the risk quotients (C_{local}/PNEC) together. The exposure to the mixture is considered as a risk in case the sum is higher than 1.

Since many unintentional sources contribute to the total emission of PAHs into the environment (see Section B.8.2.2), which by extension are not related to production and use of CTPHT , the risk characterisation will only be focussed on the PAHs emitted by producers and downstream users of

CTPHT on a local scale. To put the risk ratio's derived for the local scale into perspective risk ratio's for the regional background are calculated using monitoring data available for fresh water environment (COMMPS database-Combined Monitoring-Based and Modelling-Based Priority Setting), the marine environment (OSPAR BRCs-Background/Reference Concentrations) and soil (peer review of Wilcke, 2000) and the PNEC determined for the 16 EPA PAHs. No formal conclusions are derived for the regional background.

B.10.2.1 Aquatic compartment (incl. sediment)

In Table B.9.7 and Table B.9.8 the risk characterisation for surface water and sediment is presented for the CTPHT production. For the industrial use the risk characterisation is listed in Table B.9.9, Table B.9.10 and Table B.9.11. The risk characterisation is based on the PECs listed in Table B.9.30 to Table B.9.21 and PNECs listed in Table B.7.1 and Table B.7.3. In accordance to the EU TGD (EC, 2003b), for all PAHs with a log K_{OW} > 5 an additional factor of 10 is applied to the $PNEC_{sediment}$ in case no experimental data are available and therefore the equilibrium partitioning approach is used.

B.10.2.1.1 Production

Table B.10.7. C_{local}/PNEC for surface water and marine water for the different CTPHT production sites.

Substance/Site	1	3 [1)]	4	5	6	7	8	9
Naphthalene	0.0	0.0	0.0	0.001	0.0	0.0	0.0	0.0
Acenaphthene	0.0	0.0	0.0	0.000	0.0	0.0	0.0	0.0
Acenaphthylene	n.d.	0.0	0.0	0.003	0.0	0.0	0.0	0.0
Fluorene	0.0	0.0	0.0	0.000	0.0	0.0	0.0	0.0
Anthracene	0.0	0.0	0.0	0.008	0.0	0.0	0.0	0.0
Phenanthrene	0.0	0.0	0.0	0.000	0.0	0.0	0.0	0.0
Fluoranthene	0.0	0.0	0.5	0.025	0.0	0.0	0.0	0.0
Pyrene	0.0	0.0	0.2	0.010	0.0	0.0	0.0	0.0
Benz(a)anthracene	0.0	0.0	0.22	0.002	0.0	0.0	0.0	0.0
Chrysene	0.0	0.0	0.0	0.000	0.0	0.0	0.0	0.0
Benzo(a)pyrene	0.0	0.0	0.3	0.000	0.0	0.0	0.0	0.0
Benzo(b)fluoranthene	0.0	0.0	0.7	0.000	0.0	0.0	0.0	0.0
Benzo(k)fluoranthene	0.0	0.0	0.2	0.000	0.0	0.0	0.0	0.0
Benzo(ghi)perylene	0.0	0.0	0.5	0.000	0.0	0.0	0.0	0.1
Dibenzo(a,h)anthracene	0.0	0.0	0.5	0.001	0.0	0.0	0.0	0.4
Indeno(1.2.3-cd)pyrene	0.0	0.0	1.0	0.000	0.0	0.0	0.0	0.2
Sum PAH	0.0	0.0	4	0.05	0.1	0.01	0.1	0.9

[1)] The values for this site are concerning marine water, the other values are concerning fresh water.

B.10.2.1.2 Conclusion

Based on the risk characterisation ratios derived above it can be concluded that a risk to water and sediment could exist for some CTPHT production sites and at sites using CTPHT for anode, electrode baking and in Søderberg anodes.

Table B.10.8. C_{local}/PNEC for sediment for the different CTPHT production sites.

Substance/Site	1	3	4	5	6	7	8	9
Naphthalene	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.0
Acenaphthene	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.0
Acenaphthylene	0.0	0.0	0.0	0.01	0.0	0.00	0.0	0.0
Fluorene	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.0
Anthracene	0.0	0.1	0.0	0.05	0.0	0.00	0.0	0.0
Phenanthrene	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.0
Fluoranthene	0.0	0.0	0.1	0.01	0.0	0.00	0.0	0.0
Pyrene	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.0
Benz(a)anthracene	0.0	0.0	2.5	0.06	0.0	0.00	0.1	0.5
Chrysene	0.0	0.0	0.4	0.00	0.0	0.00	0.0	0.1
Benzo(a)pyrene	0.0	0.0	4.0	0.00	0.0	0.00	0.0	0.4
Benzo(b)fluoranthene	0.0	0.0	8.0	0.00	0.0	0.00	0.0	0.3
Benzo(k)fluoranthene	0.0	0.0	2.5	0.00	0.0	0.00	0.0	0.3
Benzo(ghi)perylene	0.0	0.0	6.0	0.01	0.0	0.00	0.0	1.0
Dibenzo(a,h)anthracene	0.0	0.0	6.1	0.02	0.1	0.00	0.1	3.7
Indeno(1.2.3-cd)pyrene	0.0	0.0	10.9	0.01	0.0	0.01	0.0	1.7
Sum PAH	0.0	0.0	41	0.2	0.3	0.02	0.3	8

B.10.2.1.3 Industrial use/processing

Table B.10.9. C_{local}/PNEC for water and sediment for the ferro-alloy production industry (including paste preparation).

Substance	Sea water	Marine sediment
Naphthalene	0.00028	0.00025
Acenaphthene	0.0072	0.010
Acenaphthylene	0.0084	0.0084
Fluorene	0.0064	0.0065
Anthracene	0.033	0.71
Phenanthrene	0.014	0.0084
Fluoranthene	2.8	0.28
Pyrene	0.75	0.077
Benz(a)anthracene	2.3	23
Chrysene	0.85	8.5
Benzo(a)pyrene	0.043	0.43
Benzo(b)fluoranthene	1.1	11
Benzo(k)fluoranthene	NA	NA
Benzo(ghi)perylene	0.49	4.9
Dibenzo(a,h)anthracene	0.81	8.1
Indeno(123-cd)pyrene	0.85	8.4
Sum of PAH	10	69

Conclusions to the risk assessment for the production stage[8]:

Conclusion (i) **(on hold)** applies for production site 9 as the sum of the PEC/PNEC for all PAHs is > 1 for sediment.

To refine the $PNEC_{sediment}$ there is need for information on the toxicity for sediment dwelling organisms of benz(a)anthracene, chrysene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(ghi)perylene, dibenzo(a,h)anthracene and indeno(123-cd)pyrene. (production site 9).

Conclusion (ii) applies to production site 1, 3, 5, 6, 7 and 8.

Conclusion (iii) applies for production site 4. For site 4 there is a need for limiting the risk beside the risk reduction measures which are already being applied, as for this site the $C_{local}/PNEC_{water}$ ratios are higher than 1 based on PAHs for which the $PNEC_{water}$ is derived from a complete data set and the local concentrations were based on site specific emission data.

Conclusions to the risk assessment for industrial use/processing:

Conclusion (i) applies to industry using CTPHT for the production of binder for coal briquetting, clay pigeons and heavy duty corrosion protection (see Section B.2.2.3). Industry is requested to provide information on the release of PAHs from production and use of these types of use.

Conclusion (ii) applies to the following primary aluminium plants: plants S5 and S6 (using Søderberg anodes) and plants PA3, PA4, PA6, PA8 and PA9 (using prebakes anodes with an anode production on-site), as they do not emit to water. Conclusion (ii) also applies to site PA15 where the PEC/PNEC ratio is below 1 for water and sediment. Furthermore conclusion (ii) applies for all primary aluminium plants using prebaked anodes without an anode plant on site and the graphite industry as the emission of PAHs is negligible. No further information is considered necessary.

Conclusion (iii) applies to the primary aluminium plants S1, S3, S4, PA1, PA2, PA5, PA7, PA10, PA11, PA12, PA13, PA14, and anode production site A1 with respect to surface water and sediment, as here the $C_{local}/PNEC$ ratios are higher than 1, even based on PAHs for which a complete data set is available and the calculated local concentrations are based on measured emission data. More information on the chronic toxicity of the PAHs mentioned above could be considered for further refinement of the PNECs to determine the extent in which the emission to water have to be reduced to exclude a risk for the aquatic environment. There are also indications that PAH in sediments around aluminium smelters might be less bioavailable than the extent calculated by the methods used. More research is needed to elucidate this aspect.

Conclusion (iii) applies to the ferro-alloy industry. This use category has been assessed using emission rates to water obtained from literature and emission profiles based on those used for VSS. Using the available information to estimate the emission to water $C_{local}/PNEC$ ratios are higher than 1 for PAHs for which a complete data set is available for water (fluoranthene) and sediment (benz(a)anthracene).

[8] **Conclusion (i)**: There is a need for further information and/or testing. **Conclusion (ii)**: There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already. **Conclusion (iii)**: There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

Table B.10.10. C_{local}/PNEC in marine and fresh water for primary aluminium production and anode baking.

Use cat.	Site	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)anthracene	Indeno(123-cd)pyrene	Total C_{local}/PNEC
VSS II	S1	2.9E-03	6.4E-02	4.4E-02	6.2E-02	2.1E-01	1.4E-01	2.6E+01	7.1E+00	2.3E+01	7.7E+00	3.9E-01	1.5E+01	4.6E+00	7.3E+00	8.1E+00	100
VSS II	S3	8.5E-04	1.9E-02	1.3E-02	1.8E-02	6.0E-02	4.2E-02	7.6E+00	2.1E+00	6.8E+00	2.2E+00	1.1E-01	4.5E+00	1.4E+00	2.1E+00	2.4E+00	29
VSS II	S4	8.8E-02	2.0E+00	1.4E+00	1.9E+00	6.2E+00	4.4E+00	7.8E+02	2.2E+02	7.1E+02	2.3E+02	1.2E+01	4.6E+02	1.4E+02	2.2E+02	2.5E+02	3038
Anode I	PA1 [1)]	5.1E-03	1.1E-01	7.9E-02	1.1E-01	3.3E+00	9.4E-01	6.9E+01	2.3E+01	6.8E+01	1.0E+01	6.9E-01	1.9E+01	4.9E+00	2.5E+01	1.1E+01	234
Anode I	PA2	8.1E-03	1.8E-01	1.2E-01	1.7E-01	5.3E+00	1.5E+00	1.1E+02	3.6E+01	1.1E+02	1.6E+01	1.1E+00	3.0E+01	7.7E+00	3.9E+01	1.7E+01	369
Anode I	PA5	3.4E-02	7.3E-02	5.2E-02	7.0E-02	2.2E+01	6.2E+00	4.6E+02	1.5E+02	4.4E+01	6.5E+00	4.5E+00	1.2E+01	3.2E+00	1.6E+01	7.2E+00	730
Anode I	PA7	2.3E-03	4.9E-02	3.5E-02	4.7E-02	1.5E+00	4.2E-01	3.1E+01	1.0E+01	3.0E+01	4.4E+00	3.0E-01	8.3E+00	2.2E+00	1.1E+01	4.9E+00	104
Anode I	PA10	5.5E-04	1.2E-02	8.5E-03	1.1E-02	3.6E-01	1.0E-01	7.5E+00	2.5E+00	7.3E+00	1.1E+00	7.4E-02	2.0E+00	5.3E-01	2.6E+00	1.2E+00	25
Anode I	PA11	2.0E-04	4.4E-03	3.1E-03	4.2E-03	1.3E-01	3.7E-02	2.7E+00	9.1E-01	2.7E+00	3.9E-01	2.7E-02	7.4E-01	1.9E-01	9.7E-01	4.3E-01	9
Anode I	PA12 [1)]	1.6E-04	3.4E-04	2.4E-04	3.3E-04	1.0E-01	2.9E-02	2.1E+00	7.1E-01	2.1E-01	3.1E-02	2.1E-02	5.8E-02	1.5E-02	7.5E-02	3.4E-02	3
Anode I	PA13	1.4E-02	3.0E-02	2.1E-02	2.8E-02	8.9E+00	2.5E+00	1.8E+02	6.1E+01	1.8E+01	2.7E+00	1.8E+00	5.0E+00	1.3E+00	6.5E+00	2.9E+00	295
Anode I	PA14	1.2E-01	2.7E-01	1.9E-01	2.5E-01	8.0E+01	2.2E+01	1.7E+03	5.5E+02	1.6E+02	2.4E+01	1.6E+01	4.5E+01	1.2E+01	5.9E+01	2.6E+01	2659
Anode I	PA15	4.0E-06	8.8E-05	6.2E-05	8.4E-05	2.6E-03	7.4E-04	5.5E-02	1.8E-02	5.3E-02	7.9E-03	5.4E-04	1.5E-02	3.9E-03	1.9E-02	8.7E-03	0.2
Anode I	A1 [1)]	1.6E-01	3.4E-01	2.4E-01	3.2E-01	1.0E+02	2.9E+01	2.1E+03	7.0E+02	2.1E+02	3.0E+01	2.1E+01	5.7E+01	1.5E+01	7.5E+01	3.4E+01	3386

[1)] emission to fresh water

Table B.10.11. C_{local}/PNEC for marine and fresh water sediment at primary aluminium production and anode baking sites.

Use cat.	Site	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)anthracene	Indeno(123-cd)pyrene	Total C_{local}/PNEC
VSS II	S1	2.8E-03	9.5E-02	4.6E-02	6.1E-02	4.3E+00	8.5E-02	2.6E+00	6.9E-01	2.3E+01	7.7E+00	3.9E+00	1.5E+02	4.6E+01	7.3E+01	8.1E+01	395
VSS II	S3	8.1E-04	2.8E-02	1.3E-02	1.8E-02	1.3E+00	2.5E-02	7.7E-01	2.0E-01	6.8E+00	2.2E+00	1.1E+00	4.5E+01	1.3E+01	2.2E+01	2.4E+01	116
VSS II	S4	8.4E-02	2.9E+00	1.4E+00	1.9E+00	1.3E+02	2.6E+00	8.0E+01	2.1E+01	7.1E+02	2.3E+02	1.2E+02	4.6E+03	1.4E+03	2.2E+03	2.5E+03	12019
Anode I	PA1 [1)]	4.9E-03	1.6E-01	8.1E-02	1.0E-01	7.1E+01	5.6E-01	7.0E+00	2.2E+00	6.8E+01	9.9E+00	6.9E+00	1.9E+02	4.9E+01	2.5E+02	1.1E+02	761
Anode I	PA2	7.7E-03	2.6E-01	1.3E-01	1.6E-01	1.1E+02	8.8E-01	1.1E+01	3.5E+00	1.1E+02	1.6E+01	1.1E+01	3.0E+02	7.7E+01	3.9E+02	1.7E+02	1198
Anode I	PA5	3.2E-03	1.1E-01	5.3E-02	7.0E-02	4.6E+01	3.7E+00	4.6E+01	7.3E+00	4.4E+01	6.5E+00	4.5E+01	1.2E+02	3.2E+01	1.6E+02	7.3E+01	591
Anode I	PA7	2.2E-03	7.3E-02	3.6E-02	4.6E-02	3.1E+01	2.5E-01	3.1E+00	9.9E-01	3.0E+01	4.4E+00	3.0E+00	8.3E+01	2.2E+01	1.1E+02	4.9E+01	337
Anode I	PA10	5.3E-04	1.8E-02	8.8E-03	1.1E-02	7.6E+00	6.0E-02	7.6E-01	2.4E-01	7.3E+00	1.1E+00	7.4E-01	2.0E+01	5.3E+00	2.7E+01	1.2E+01	82
Anode I	PA11	1.9E-04	6.5E-03	3.2E-03	4.1E-03	2.8E+00	2.2E-02	2.8E-01	8.8E-02	2.7E+00	3.9E-01	2.7E-01	7.4E+00	1.9E+00	9.8E+00	4.4E+00	30
Anode I	PA12 [1)]	1.5E-05	5.0E-04	2.5E-04	3.3E-04	2.2E-01	1.7E-02	2.2E-01	3.4E-02	2.1E-01	3.1E-02	2.1E-01	5.8E-01	1.5E-01	7.6E-01	3.4E-01	3
Anode I	PA13	1.3E-03	4.4E-02	2.2E-02	2.8E-02	1.9E+01	1.5E+00	1.9E+01	3.0E+00	1.8E+01	2.6E+00	1.8E+01	5.0E+01	1.3E+01	6.6E+01	2.9E+01	240
Anode I	PA14	1.2E-02	3.9E-01	1.9E-01	2.5E-01	1.7E+02	1.3E+01	1.7E+02	2.7E+01	1.6E+02	2.4E+01	1.6E+02	4.5E+02	1.2E+02	5.9E+02	2.6E+02	2155
Anode I	PA15	3.9E-06	1.3E-04	6.4E-05	8.2E-05	5.6E-02	4.4E-04	5.6E-03	1.8E-03	5.3E-02	7.8E-03	5.4E-03	1.5E-01	3.8E-02	2.0E-01	8.7E-02	0.6
Anode I	A1 [1)]	1.5E-02	5.0E-01	2.5E-01	3.2E-01	2.2E+02	1.7E+01	2.1E+02	3.4E+01	2.1E+02	3.0E+01	2.1E+02	5.7E+02	1.5E+02	7.6E+02	3.4E+02	2745

[1)] emission to fresh water

B.10.2.1.4 Regional background in fresh and marine surface water (including sediment)

Not for all 16 EPA PAHs EU fresh water monitoring data are available. The available data result in risk ratios > 1 for fresh water and fresh water sediment. (see Table B.10.12).

With respect to the marine environment OSPAR data gives information on 4 PAHs. Based on these monitoring data the risk quotients for water well below 1. However, the concentrations for benzo(b+k)fluoranthene and fluoranthene, result in risk ratios > 1 for marine sediment organisms (see Table B.10.13).

Table B.10.12. Ratio between the COMMPS monitoring data and PNEC for surface water and sediment organisms.

Compound	Surface water		Sediment	
	Median	90-percentile	Median	90-percentile
Naphthalene	0.03	0.84	0.05	0.67
Acenaphthene	0.00	0.11	0.03	0.42
Acenaphthylene			0.34	3.33
Fluorene			0.09	0.29
Anthracene	0.04	0.83	1.24	3.35
Phenanthrene			0.12	0.68
Fluoranthene	1.60	8.23	6.0	26.7
Pyrene			0.20	1.13
Benz(a)anthracene	1.76	6.93	5.0	22.7
Chrysene			1.8	14.7
Benzo(a)pyrene	0.32	1.24	1.7	5.3
Benzo(b)fluoranthene	0.52	2.85	2.5	10.0
Benzo(k)fluoranthene	0.26	1.46	1.4	4.8
Benzo(ghi)perylene	0.98	5.73	3.5	11.9
Indeno(123-cd)pyrene	12.41	34.67	4.9	20.6
Total	17.9	62.9	28.9	126.6

Table B.10.13. Ratio between the OSPAR monitoring data and PNEC for marine water and sediment organisms.

PAH	northern North Sea/ Skagerrak		southern North Sea		Arctic Ocean/ Iceland Sea	
	water	sediment	water	sediment	water	sediment
Benzo(a)pyrene	0.00	0.05-0.61	0.00	0.00-0.28	0.00	0.01-0.03
Fluoranthene	0.03	0.10-1.7	0.03	0.00-1.0	0.01	0.02-0.08
Benzo(b+k)fluoranthene	0.01	3.2-31	0.01	0.08-10.1	0.00	0.52-2.1
Pyrene	0.00	0.01- 0.09	0.00	0.00-0.06	0.00	0.00

B.10.2.2 Sewage treatment plant

There are insufficient data available to obtain $PNEC_{microorganism}$ values for the individual PAHs in a sewage treatment plant. However, based on the assumption that the PNECs have to be in the µg/L range or higher, it is not expected that the calculated concentrations for the CTPHT production sites (see Table B.9.66) will pose a risk for micro-organisms in a sewage treatment plant (**conclusion ii**).

The down stream users of CTPHT do not emit waste water to a sewage treatment plant.

Conclusion (i) applies to industry using CTPHT for the production of binder for coal briquetting, clay pigeons and heavy duty corrosion protection (see Section B.2.2.3). Industry is requested to provide information on the release of PAHs from production and use of these types of use.

B.10.2.3 Terrestrial compartment

The risk characterisation is based on the PECs listed in Table B.9.48 to Table B.9.52 and the PNECs listed in Table B.7.5. In accordance to the EU TGD (EC. 2003b), for all PAHs with a log K_{OW} > 5 an additional factor of 10 is applied to the $PNEC_{soil}$ in case no experimental data are available and by extension the equilibrium partitioning approach is used. The risk assessment is based on the local concentration for terrestrial compartment without taking the regional background concentration into account.

B.10.2.3.1 Production

In Table B.10.14 the risk characterisation for agricultural soil is presented for the production.

Table B.10.14. C_{local}/PNEC for agricultural soil for the different CTPHT production sites.

Substance/Site	1	3	4	5	6	7	8	9
Naphthalene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Acenaphthene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Acenaphthylene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Fluorene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Anthracene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Phenanthrene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Fluoranthene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Pyrene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Benz(a)anthracene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chrysene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Benzo(a)pyrene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Benzo(b)fluoranthene	0.3	0.2	0.1	0.0	0.1	0.1	0.2	0.2
Benzo(k)fluoranthene	0.1	0.1	0.0	0.1	0.1	0.0	0.1	0.0
Benzo(ghi)perylene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Dibenzo(a,h)anthracene	0.4	0.3	0.1	0.2	0.2	0.1	0.3	0.3
Indeno(123-cd)pyrene	0.1	0.1	0.0	0.1	0.0	0.0	0.1	0.1
Sum PAH	0.95	0.69	0.2	0.5	0.5	0.2	0.7	0.5

B.10.2.3.2 Industrial use/processing

For the industrial use the risk characterisation is listed in Table B.10.15 and Table B.10.16.

Table B.10.15. C_{local}/PNEC for agricultural soil and grassland for the ferro-alloy and graphite industry.

	agricultural soil	
Substance/Scenario	Ferro-Alloy	Graphite [1)]
Naphthalene	0.0	0.0
Acenaphthene	0.0	0.0
Acenaphthylene	0.0	0.0
Fluorene	0.0	0.0
Anthracene	0.0	0.0
Phenanthrene	0.0	0.0

	agricultural soil	
Substance/Scenario	**Ferro-Alloy**	**Graphite** [1)]
Fluoranthene	0.0	0.0
Pyrene	0.0	0.0
Benz(a)anthracene	0.1	0.1
Chrysene	0.2	0.4
Benzo(a)pyrene	0.2	0.0
Benzo(b)fluoranthene	0.7	0.5
Benzo(k)fluoranthene	-	-
Benzo(ghi)perylene	0.3	0.2
Dibenzo(a,h)anthracene	0.5	0.3
Indeno(123-cd)pyrene	0.5	0.3
Sum of PAH	2.6	1.7

See for a description of the other scenarios Table B.7.5. [1)] Production of graphite electrodes (including paste preparation using a wet process for cooling).

B.10.2.3.3 Conclusion

Conclusions to the risk assessment for the production stage[9]:

Conclusion (ii) applies to all CTPHT production sites.

Conclusions to the risk assessment for industrial use/processing:

Conclusion (i) (**on hold)** applies to the ferro-alloy industry, graphite industry, anode production industry (including prebake primary aluminium industry with on-site anode production plant) and primary aluminium industry using Søderberg technology others than mentioned above as the sum of C_{local}/PNEC is higher than 1 mainly based on PNECs which were determined with equilibrium partitioning or where additional toxicity data could refine the PNEC. Further testing is needed to elucidate the chronic toxicity for soil organisms of benz(a)anthracene[10] chrysene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenzo(a,h)-anthracene and indeno(123-cd)pyrene, benzo(ghi)perylene.

For site PA2, S3 and S4 measured benzo(a)pyrene concentrations in air were more than a factor of 10 lower than the predicted concentrations. As the contamination of soil at these sites is determined by atmospheric deposition, this would mean that the PAH concentrations in soil will deviate in the same extent, provided that the measurements are reliable and representative. The PEC/PNEC ratio for both sites is < 10. Consequently, the risk for soil organisms might also be low.

Conclusion (i) also applies to industry using CTPHT for the production of binder for coal briquetting, clay pigeons and heavy duty corrosion protection (see Section B.2.2.3). Industry is requested to provide information on the release of PAHs from production and use of these types of use.

[9] **Conclusion (i)**: There is a need for further information and/or testing. **Conclusion (ii)**: There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already. **Conclusion (iii)**: There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

[10] The PNEC for benz(a)anthracene is based on an high extrapolation factor to normalise it from 90 to 2% organic carbon content. This might be overconservative.

Table B.10.16. C_{local}/PNEC agricultural soil at primary aluminium production and anode baking sites.

Use cat.	Site	Naphthalene	Acenaphthene	Acenaphthylene	Fluorene	Anthracene	Phenanthrene	Fluoranthene	Pyrene	Benz(a)anthracene	Chrysene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(k)fluoranthene	Benzo(ghi)perylene	Dibenzo(a,h)anthracene	Indeno(123-cd)pyrene	Total C_{local}/PNEC
VSS II	S1	6.7E-04	3.6E-03	1.1E-03	1.3E-03	3.2E-03	2.0E-03	3.8E-03	3.3E-03	4.3E-02	1.4E-01	1.4E-01	5.2E-01		2.7E-01	3.7E-01	3.8E-01	1.9
VSS II	S3	1.7E-03	9.3E-03	2.9E-03	3.4E-03	8.2E-03	5.2E-03	9.8E-03	8.4E-03	1.1E-01	3.7E-01	3.7E-01	1.3E+00		7.0E-01	9.6E-01	9.9E-01	4.9
VSS II	S4	1.8E-03	9.8E-03	3.1E-03	3.6E-03	8.8E-03	5.5E-03	1.0E-02	8.9E-03	1.2E-01	3.9E-01	3.9E-01	1.4E+00		7.5E-01	1.0E+00	1.1E+00	5.2
VSS I	S5	1.9E-03	1.0E-02	3.2E-03	3.7E-03	6.4E-03	2.4E-03	4.1E-03	5.3E-03	1.1E-01	3.8E-01	4.1E-01	2.0E+00		9.3E-01	5.4E-01	1.1E+00	5.5
VSS I	S6	1.8E-03	1.0E-02	3.1E-03	3.5E-03	6.1E-03	2.3E-03	4.0E-03	5.1E-03	1.1E-01	3.7E-01	4.0E-01	1.9E+00		9.0E-01	5.2E-01	1.1E+00	5.3
Anode I	PA1	7.0E-04	3.4E-03	2.1E-05	7.9E-04	5.5E-03	2.3E-03	4.0E-03	3.8E-03	7.8E-02	2.6E-01	1.1E-01	9.3E-01		3.2E-01	1.1E+00	4.5E-01	3.2
Anode I	PA2	3.0E-04	1.4E-03	8.9E-06	3.4E-04	2.3E-03	9.7E-04	1.7E-03	1.6E-03	3.3E-02	1.1E-01	4.5E-02	3.9E-01		1.4E-01	4.6E-01	1.9E-01	1.4
Anode I	PA3	2.7E-07	1.3E-06	8.0E-09	3.1E-07	2.1E-06	8.8E-07	1.5E-06	1.5E-06	3.0E-05	1.0E-04	4.1E-05	3.6E-04		1.2E-04	4.2E-04	1.7E-04	0.001
Anode I	PA4	3.1E-05	1.5E-04	9.2E-07	3.5E-05	2.4E-04	1.0E-04	1.8E-04	1.7E-04	3.4E-03	1.2E-02	4.7E-03	4.1E-02		1.4E-02	4.8E-02	2.0E-02	0.14
Anode I	PA5	1.9E-04	9.2E-04	5.6E-06	2.1E-04	1.5E-03	6.1E-04	1.1E-03	1.0E-03	2.1E-02	7.1E-02	2.9E-02	2.5E-01		8.7E-02	2.9E-01	1.2E-01	0.9
Anode I	PA6	6.7E-04	3.3E-03	2.0E-05	7.6E-04	5.3E-03	2.2E-03	3.8E-03	3.6E-03	7.4E-02	2.5E-01	1.0E-01	8.9E-01		3.1E-01	1.0E+00	4.3E-01	3.1
Anode I	PA6	1.8E-03	8.8E-03	5.4E-05	2.1E-03	1.4E-02	5.9E-03	1.0E-02	9.8E-03	2.0E-01	6.8E-01	2.8E-01	2.4E+00		8.4E-01	2.8E+00	1.2E+00	8.4
Anode I	PA7	2.8E-05	1.3E-04	8.3E-07	3.1E-05	2.2E-04	9.0E-05	1.6E-04	1.5E-04	3.1E-03	1.0E-02	4.2E-03	3.7E-02		1.3E-02	4.3E-02	1.8E-02	0.1
Anode I	PA8	6.7E-06	3.3E-05	2.0E-07	7.6E-06	5.3E-05	2.2E-05	3.8E-05	3.6E-05	7.5E-04	2.5E-03	1.0E-03	8.9E-03		3.1E-03	1.0E-02	4.3E-03	0.031
Anode I	PA9	1.6E-02	7.7E-02	4.7E-04	1.8E-02	1.2E-01	5.1E-02	9.0E-02	8.5E-02	1.7E+00	5.9E+00	2.4E+00	2.1E+01		7.3E+00	2.4E+01	1.0E+01	73
Anode I	PA10	1.7E-04	8.5E-04	5.2E-06	2.0E-04	1.4E-03	5.7E-04	1.0E-03	9.5E-04	1.9E-02	6.6E-02	2.7E-02	2.3E-01		8.1E-02	2.7E-01	1.1E-01	0.8
Anode I	PA11	6.7E-04	3.3E-03	2.0E-05	7.6E-04	5.3E-03	2.2E-03	3.8E-03	3.6E-03	7.4E-02	2.5E-01	1.0E-01	8.9E-01		3.1E-01	1.0E+00	4.3E-01	3.1
Anode I	PA12	1.9E-05	9.2E-05	5.6E-07	2.1E-05	1.5E-04	6.1E-05	1.1E-04	1.0E-04	2.1E-03	7.1E-03	2.9E-03	2.5E-02		8.7E-03	2.9E-02	1.2E-02	0.09
Anode I	PA13	2.4E-03	1.2E-02	7.2E-05	2.7E-03	1.9E-02	7.9E-03	1.4E-02	1.3E-02	2.7E-01	9.1E-01	3.7E-01	3.2E+00		1.1E+00	3.7E+00	1.6E+00	11
Anode I	PA14	1.8E-03	8.8E-03	5.4E-05	2.1E-03	1.4E-02	5.9E-03	1.0E-02	9.8E-03	2.0E-01	6.8E-01	2.8E-01	2.4E+00		8.4E-01	2.8E+00	1.2E+00	8.4
Anode I	PA15	8.1E-07	3.9E-06	2.4E-08	9.2E-07	6.4E-06	2.6E-06	4.6E-06	4.4E-06	9.0E-05	3.0E-04	1.2E-04	1.1E-03		3.7E-04	1.2E-03	5.2E-04	0.004
Anode I	A1	9.9E-03	4.8E-02	3.0E-04	1.1E-02	7.8E-02	3.2E-02	5.6E-02	5.3E-02	1.1E+00	3.7E+00	1.5E+00	1.3E+01		4.5E+00	1.5E+01	6.3E+00	45.8

Conclusion (ii) applies to the primary aluminium plant PA3, PA4, PA5 PA7, PA8, PA10, PA12 and PA15. Conclusion (ii) also applies to all primary aluminium plants using prebaked anodes without an anode plant on site as the emission of PAHs is negligible.

B.10.2.3.4 Regional background in soil

Based on the mean values for arable land, grassland, forest and urban soil there is a potential risk for soil organism (see Table B.10.17).

Table B.10.17. Ratio between the background concentrations in different soils and the PNEC for soil organisms.

Compound	Arable land	grassland	Forest soil	Urban soil
Naphthalene	0.01	0.00	0.03	0.04
Acenaphthylene	0.06	0.01	0.01	0.06
Acenaphthene	0.25	0.58	0.05	1.50
Fluorene	0.00	0.00	0.01	0.02
Phenanthrene	0.01	0.01	0.03	0.11
Anthracene	0.02	0.01	0.07	0.45
Fluoranthene	0.04	0.03	0.08	0.54
Pyrene	0.04	0.03	0.07	0.59
Benz(a)anthracene	0.49	0.33	0.54	5.53
Chrysene	0.40	0.38	2.13	5.05
Benzo(b)fluoranthene	0.12	0.12	0.56	1.63
Benzo(k)fluoranthene	0.04	0.07	0.69	0.87
Benzo(a)pyrene	0.34	0.36	0.74	6.60
Indeno(123-cd)pyrene	0.12	0.11	0.63	2.98
Dibenzo(a,h)anthracene	0.20	0.19	0.28	1.02
Benzo(ghi)perylene	0.11	0.16	0.36	2.18
total	1.89	2.05	4.37	24.61

Background concentrations used were taken from Wilcke (2000).

B.10.2.4 Atmosphere

Due to the lack of data, no PNEC has been established for the atmospheric compartment. In the risk assessment for man indirectly exposed to the environment the exposure to air concentrations in the vicinity of the different plants is considered. It is to be expected that any precautions necessary to limit that risk will also be protective for wild life.

B.10.2.5 Secondary poisoning

In the absence of sufficient toxicity data, no $PNEC_{oral}$ for any of the PAHs can be derived. The risk assessment is also hampered by the lack of sufficient information on the bioaccumulation potential in fish. Therefore, a realistic quantitative risk assessment for secondary poisoning for the PAHs can not be made.

CTPHT has been identified as PBT and vPvB, as several PAHs, like benzo(a)pyrene, are identified as PBT and/or vPvB substances. Therefore, it is also not considered necessary to perform a full risk assessment for secondary poisoning, as companies already have to take the most effective measures to minimise the emission of PAHs to the environment with automatically will reduce the risk for secondary poisoning.

To illustrate the potential risk the following preliminary assessment for benzo(a)pyrene is made:

All BCF values for fish were not considered reliable, although a value of 600 could be used as an upper limit. For mussels reliable BCF values are available which are on average around 100,000. Based on this value as a worst case estimate for mussel-eating birds and mammals, a concentration in the water phase of > 14 ng/L will lead to concentrations in mussels that exceeds the preliminary $PNEC_{oral}$ of 1.4 mg/kg food (see Section B.7.5), which is the case for some of the uses of CTPHT.

It should be noted that the PNEC for aquatic compartment is 22 ng benzo(a)pyrene/L, indicating that it might also be protective for secondary poisoning.

B.10.2.6 Areas of uncertainty in the environmental risk assessment

B.10.2.6.1 Adsorption and bioavailability

Uncertainties exist towards the sorption and bioavailability of PAHs. As highlighted in Section B.4.2.1, PAHs can be sorbed to amorphous organic matter (traditionally referred to as organic carbon), to black carbon and other carbonaceous geosorbents, which have differential adsorption properties. Consequently, the K_{OC} value can show a high degree of variation. Hence, the fate and behaviour of PAHs will depend on how PAHs are emitted (gas or particle bound), the characteristics of particles to which the PAHs are bound and the characteristics of the soil or sediment. In addition, sorption of PAHs will also depend on the concentration. The results of the research on the particle affinity of PAHs associated with coal tar pitch (Ruus *et al.*, 2007) suggests that the K_{OC} values in sediment in the vicinity of aluminium smelters are higher than those used in the present risk assessment. However, no clear relationship could be found between the characteristics of the sediment and K_{OC} values measured and no difference with clean sediment was demonstrated, which hamper the implementation of these results in a generic approach.

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.

It should be noted that in the present risk assessment, the impact of a change in K_{OC} values will be limited as for most high molecular PAHs both the PEC and the PNEC are derived by using equilibrium partitioning. Consequently, by taking a different K_{OC} value both values will change in the same extent and in concomitant the PEC:PNEC ratio will remain the same. It should also be noted that most of the high molecular PAHs are emitted particle-bound and as such contaminate sediment via direct deposition without dissolving first and partitioning to sediment, successively.

Therefore, for a refinement of the risk assessment monitoring data for all relevant sites are needed together with information on the composition of the organic material present. In addition, it is also crucial to obtain toxicity data for sediment and soil dwelling organism for the high molecular PAHs preferable in relation to the binding to various organic carbon material present.

B.10.2.6.2 Ageing

The bioavailability may also depend on the age of the particles. 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 effects were found at an organic carbon content of standard soil (2%) and the fact that this phenomenon is not sufficiently quantified, aging is as yet not considered in the risk assessment.

B.10.2.6.3 Information on the release of the individual PAHs

Another factor of uncertainty is the emission estimated for the individual PAHs. In most cases the emissions are reported as benzo(a)pyrene only or total PAHs and not specified for the individual PAHs. As been described in Section B.9.5.2.3 for each process one general emission profile is used

to estimate the emission of the single PAHs. Consequently, the actual emission of the PAHs could deviate.

B.10.2.6.4 Prediction air concentration

The measured data shows that the modelled air concentration can be considered as a conservative prediction. A more accurate measure of air concentration is difficult to make with the generic tools available and can only be obtained by local measurements taking into account the site-specific conditions.

B.11 Summary on hazard and risk

B.11.1 Hazard assessment

B.11.1.1 Human Health

The database on possible health hazards induced by CTPHT is rather limited, implicating that a full hazard assessment for all the required endpoints is not possible. 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 striking 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 also exposed to, 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 effect, quite possibly even to zero. Therefore, in spite of the limited available data on non-carcinogenic properties of CTPHT, it is decided that in this risk characterisation for CTPHT conclusions on risks and further testing for some endpoints will be subordinated to conclusions on risks based on carcinogenic and mutagenic properties, using the best-studied PAH benzo(a)pyrene as a guidance substance.

There were no data available on the toxicokinetics of CTPHT. Some information on the toxicokinetics of selected homocyclic polycyclic aromatic hydrocarbons was available. From these data, it was concluded that PAHs are lipophilic compounds that can be absorbed through the respiratory and gastrointestinal tract and through the skin. After absorption, PAHs are widely distributed throughout the organism to almost all organs, especially the lipid-rich ones. They can cross the placenta and reach foetal tissues. The metabolism of PAHs can take place in the liver, respiratory tract, and the skin, and appears very complex leading to a variety of metabolites from a limited number of reaction types. Only a few metabolites are toxicologically relevant. Most metabolic processes result in detoxification products that are excreted in urine and faeces. However, some pathways yield reactive compounds capable of binding to DNA and initiating tumour formation (see Section B.5.1 for further details).

Skin effects were observed in animals and humans after repeated exposure to CTP(V) or combined exposure to CTP(V) and sunlight. In view of the human data on occupation exposure to CTP (fumes, volatiles and dust, not further specified) which show eye irritation and, after repeated exposure, chemosis of the conjunctiva, ulceration and infiltration of the cornea, deep staining of the cornea, and conjunctival discolouration and irritation, classification as ‘irritant’ with ‘risk of serious damage to eyes’ (Xi, R41) is proposed. Sunlight aggravated irritating effects of CTP(V) on the eyes and skin (see Section B.5.3 for further details).

There are no indications that CTP has corrosive properties. Since CTPHT may contain up to 1.5% benzo(a)pyrene (a skin sensitizer) it is proposed to classify CTPHT as a skin sensitizer(Xi;R43) (see Section B.5.1 for further details).

Only one oral repeated dose study in pigs was available to the rapporteur, which had only limited significance. In addition, only carcinogenicity was studied in repeated dose toxicity animal studies. Therefore, no NOAEL for non-carcinogenic effects could be derived from these studies (see Section B.5.6 for further details).

The data set available on the mutagenicity/genotoxicity of CTPHT does not meet the basis requirements as specified in Annex VIIA of Directive 67/548/EEC (EC, 1967), but numerous genotoxicity studies with coal tar, coal tar waste, coal tar products, and individual PAHs demonstrated the genotoxicity of these substances (ATSDR, 2002; WHO, 1998). In addition, CTPHT may contain a variable amount of mutagenic PAHs, so classification of CTPHT as a category 2 mutagen is proposed (T; R46) (see Section B.5.7 for further details).

Based on the available experimental and epidemiological data on the carcinogenicity of CTPHT and CTPVHT and the evaluation of these data by the IARC, CTPHT and CTPVHT will be classified as a category 1 carcinogen (T; R45). Based on the genotoxic and carcinogenic properties of CTPHT, for risk characterisation a non-threshold approach will be adopted (see Section B.5.8 for further details).

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTPHT. Data was available on high-boiling coal liquid, coal tar derived products and creosote (inhalation, oral and dermal route), but although some effects on fertility were reported, offspring appeared not to be affected. However, since CTPHT may contain up to 1.5% benzo(a)pyrene, which is classified for effects on reproduction (category 2; T, R.60/61), it is proposed to classify CTPHT as toxic to reproduction(T; R60/61) (see Section B.5.9 for further details).

B.11.1.2 Environment

In the hazard assessment for the environment the ecotoxicity data has been evaluated for the 16 EPA PAHs separately and subsequently predicted no effect concentrations (PNECs) for the individual PAHs have been derived (see Table B.11.1). The data from both literature and other EU risk assessment reports are used.

PAHs can be toxic via different mode of actions, such as non-polar narcosis and phototoxicity. 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.

Although it is recognized 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, it is thought that the phototoxic effects can not be ignored in the present risk assessment. Therefore these effects are also considered in deriving the PNECs for aquatic species. It should be noted that the UV exposure levels of the selected studies did not exceed the UV levels under natural sun light conditions.

For sewage treatment plants, there were not sufficient data to derive exact $PNEC_{microorganisms}$ values for the individual PAHs. Based on the solubility data given in Section B.1.3, it can however be assumed that the PNEC values will be in the range of µg/L or higher.

For the atmosphere no toxicity data are available, so no $PNEC_{air}$ can be derived.

Based on the available information $PNEC_{oral}$ values for the individual PAHs can not be derived for secondary poisoning.

Table B.11.1. The PNEC for the various PAHs for the various environmental compartments.

Compound	fresh water (µg/l)	marine water (µg/l)	fresh water sediment (mg/kg_{dwt})	marine sediment (mg/kg_{dwt})	soil (mg/kg_{dwt})
Naphthalene	2	2	2.9	0.29	1.0
Anthracene	0.1	0.1	0.14	0.014	0.13
Phenanthrene	1.3	1.3	5	5	1.8
Fluoranthene	0.01	0.01	0.96	0.96	1.5
Pyrene	0.023	0.023	2.8	1.4	1.0
9H-Fluorene	2.5	0.25	2.56	0.26	1.0
Acenaphthylene	1.3	0.13	0.34	0.03	0.29
Acenaphthene	3.8	0.38	1.6	0.16	0.038
Chrysene	0.07	0.007	2.79	0.28	0.55
Benzo(a)anthracene	0.012	0.0012	0.60	0.06	0.079
Benzo(b)fluoranthene	0.017 a)	0.0017 a)	1.38 a)	0.14 a)	0.28
Benzo(ghi)perylene	0.0082	0.00082	0.84	0.084	0.17
Benzo(k)fluoranthene	0.017	0.0017	1.38	0.14	0.27
Benzo(a)pyrene	0.022	0.022	1.83	1.83	0.053
Dibenzo(a,h)anthracene	0.0014	0.00014	0.27	0.027	0.054
Indeno(123*cd*)pyrene	0.0027	0.00027	0.63	0.063	0.13

a) For benzo(b)fluoranthene the PNEC is the same as for benzo(k)fluoranthene after read-across with this compound.

B.11.2 Risk assessment

B.11.2.1 Human Health

B.11.2.1.1 Workers

There is a need for information and/or testing on skin irritation, systemic toxicity after repeated exposure, and effects on reproduction, although a proper risk reduction strategy might already be sufficient to minimize these risks.

For workers there is a need for specific measures to limit the risks , because of concerns for sensitisation as a consequence of dermal exposure arising from all occupational scenarios, and concerns for mutagenicity and carcinogenicity as a consequence of inhalation and dermal exposure arising from all occupational scenarios. To limit these risks it is proposed to establish at community level occupational exposure limit values for CTPHT according to Directive 98/24/EEC (EC, 1998) or Directive 2004/37/EC (EC, 2004b) as appropriate.

B.11.2.1.2 Consumers

For consumers there is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied, because exposure of consumers is assumed not to exist.

B.11.2.1.3 Humans exposed via the environment

For humans exposed via the environment there is a need for specific measures to limit the risks, because concerns for mutagenicity and carcinogenicity as a consequence of inhalation and oral exposure are arising from all scenarios for humans exposed via the environment.

B.11.2.1.4 Human health risks based on physico-chemical properties

Based on physico-chemical properties of CTPHT there is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied. This conclusion

is reached because: the risk assessment shows that risks are not expected. Risk reduction measures already being applied are considered sufficient.

B.11.2.2 Environment

B.11.2.2.1 Aquatic system

There is a need for further information and/or testing, because there is a need for information on the release of the 16 EPA PAHs to the aquatic ecosystem from the use of CTPHT in the production and use of binder for coal briquetting, clay pigeons and heavy duty corrosion protection.

There are also concerns for the aquatic ecosystem as a consequence of exposure arising from production (Site 4) and industrial use like primary aluminium plants, ferro-alloy industry and graphite industry, which ask for specific measures to limit the risks.

B.11.2.2.2 Micro-organisms in the sewage treatment plant

For sewage treatment plants there is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied, because the risk reduction measures already being applied are considered sufficient.

B.11.2.2.3 Terrestrial ecosystem

As for the aquatic system, there is a need for further information on the release of the 16 EPA PAHs to the terrestrial compartment from the use of CTPHT in the production and use of binder for coal briquetting, clay pigeons and heavy duty corrosion protection.

B.11.2.2.4 Atmosphere

For the atmosphere there is not sufficient data available for a risk assessment.

B.11.2.2.5 Secondary poisoning

There is insufficient data available for a realistic quantitative risk assessment for secondary poisoning. However, CTPHT has been identified as a PBT-substance, which implies that companies already have to take the most effective measures to minimise the emission of PAHs to the environment with automatically will reduce the risk for secondary poisoning.

B.11.2.2.6 Summary

Table B.11.2 summarizes the risk characterisation of CTPHT for the environment.

Table B.11.2. Summary of the risk characterisation of CTPHT for the environment.

	Water [a)]	Sedi-ment [a)]	Sewage treat-ment plant [a)]	Terrestrial [a)]	Atmos-phere [c)]	Secondary poisoning [c)]	Exposure scenario
Production of CTPHT	iii	iii	ii	ii	x	x	site specific
Primary aluminium production	iii	iii	ii	ii	x	x	site specific
Graphite electrode production	ii [d)]	ii [d)]	ii	ii	x	x	generic
Ferro-alloy industry applying electric arc furnaces with Søderberg electrodes	iii	iii	ii	ii	x	x	generic
Coal briquetting	i	i	ii	i	x	x	
Clay pigeons	i	i	ii	i	x	x	
Heavy duty corrosion protection	i	i	ii	i	x	x	
Regional background [d)]	iii	iii		ii	x	x	

[a)] i = there is a need for information on the release of the 16 EPA PAHs to the different environmental compartments ii = there is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already, iii = there is a need for limiting the risks, risk reduction measures which are already applied shall be taken into account; [b)] No Predicted No Effect Concentration (PNEC) available, so no risk characterisation possible (indicated by x); [c)] No emissions to water; [d)] Significantly higher PEC than regional levels for some of the sites, but there are large differences in emissions and related risks.

C. A FIRST SCREENING OF ALTERNATIVES

There is no full overview in the risk assessment reports of alternative substances (The Netherlands, 2008ab). Considering that this is a transitional dossier and no restrictions are proposed, no additional research on alternatives has been performed.

In this section only those alternatives are discussed that are related to applications at risk (see Section B.10). For anode production for metal industry possible alternative substances and techniques are identified and discussed in relation to their availability, their risks for human health and the environment, and their technical and economical feasibility. Alternatives for other applications are briefly discussed in Annex J.7.

C.1 Anode production for metal industry

There are several techniques available for the production of anodes. The technique which is used partly determines the emissions of PAH. At some plants very low PAH-levels are found. Sometimes an explanation for the differences can be found (use of prebaked anode instead of Søderberg anodes; no emission to the water compartment), but at this stage there is no detailed understanding of the techniques and operational conditions leading to low PAH emissions. The information on available techniques in the IPPC BAT reference document (EC, 2001a) will be revised.

Since more than ten years a new technology has been developed at bench scale based on inert anodes to replace CTPHT-bound carbonated anodes, but this technology is still immature and costly. Therefore, it can be expected that CTPHT will be used for more than decades in the primary aluminium smelters. Yet, since over 70% of the total sales of CTPHT is used for anodes this application is the major contributor to the risks of CTPHT, which implies that limiting the risks in this application by using inert anodes will also strongly limit the overall risks of CTPHT.

C.2 Production of ferro-alloys

Also several techniques can be used for the production of ferro-alloys (carbo-thermic and metallo-thermic reduction) and there are three types of furnaces used for the production (electric arc furnace, electric resistance furnace, blast furnace). In the risk assessment reports (The Netherlands, 2008ab) no information is given whether the techniques and types have the same functionality, use the same amount of CTPHT and have different PAH emission levels. For the electrode production and the use in the ferro-alloy industry it is therefore not clear if alternatives are available.

D. JUSTIFICATION FOR ACTION ON A COMMUNITY-WIDE BASIS

Risk assessment of PAHs involves many (diffuse) sources, which are unlikely to be adequately controlled by the individual Member States and thus appears to require Community-wide action. However, the production and use of CTPHT only adds a small contribution to the emission of PAHs in comparison to the other sources (see *e.g.* Table B.8.1). In addition, the emission contribution of CTPHT is mainly on a local scale (water, air), which suggests that risks of CTPHT can be adequately controlled by implementation of IPPC (EC, 1996) and national legislation of the Member States. Yet, risks are being identified, which suggests that IPPC and national legislation of the Member States may not be sufficient (although adaptation of the BAT reference document appears to be sufficient to further minimize these risks). In addition, it should be noted that for the primary anode production PAH concentrations are calculated that are significantly higher than the regional background suggesting that these risks cannot be adequately controlled by the individual Member States and thus ask for Community-wide action.

In addition, for workers it is proposed to establish at community level occupational exposure limit values for CTPHT according to Directive 98/24/EEC (EC, 1998) or Directive 2004/37/EC (EC, 2004b) as appropriate (see Section H.1 for the full risk reduction strategy).

In general, it appears that implementation of IPPC and national legislation of the Member States appears to be sufficient to adequately control the risks of CTPHT, suggesting that there is no clear basis for Community-wide action for CTPHT risks other than adjustment of the BAT reference document for the IPPC legislation and the establishment of occupational exposure limit values at community level. It should be emphasized, however, that this conclusion only holds for risks associated with CTPHT. For a proper risks assessment of PAHs in general, Community-wide action appears to be inevitable, because of the many (diffuse) sources of PAHs.

E. COMPARISON OF DIFFERENT COMMUNITY-WIDE MEASURES

E.1 Identification and description of risk management options

E.1.1 Risk to be addressed-the baseline

For workers there are health concerns for CTPHT arising from all occupational scenarios, resulting in sensitisation as a consequence of dermal exposure, and mutagenicity and carcinogenicity as a consequence of inhalation and dermal exposure (see Section B.11.2.1.1). To limit these risks it has already been proposed in the risk reduction strategy for workers (The Netherlands, 2008c; see Section H.1) to establish at community level occupational exposure limit values for CTPHT according to Directive 98/24/EEC (EC, 1998) or Directive 2004/37/EC (EC, 2004b) as appropriate.

Environmental risks for CTPHT are only identified for the production of CTPHT and the main applications of CTPHT in the primary aluminium production and the production of electrodes and anodes (see Section B.10). It should be noted, however, that for the atmosphere and for secondary poisoning a lack of data hampered a proper risk assessment. As indicated in Section D, the identified risks should be adequately controlled by IPPC legislation (EC, 2008). The relevant BAT reference document for the scenarios at risk (EC, 2001a) will be revised within the next years.

In addition, registration under the REACH regulation (EC, 2006b) will provide better information about the emissions related to production and use of CTPHT, which will be beneficial for fine-tuning the risk characterisation. In this report it is assumed that for the baseline situation the current production and use of CTPHT and the emissions related to the production and is unaffected by REACH or by other developments.

E.1.2 Other Community-wide risk management options than restriction

Apart from implementation of the IPPC legislation (EC, 2008) and registration under the REACH regulation (EC, 2006b), other risk management options that can be considered on a Community-wide basis are authorisation under the REACH regulation, voluntary action, and adaptation of the IPPC legislation. The applicability of these options is summarized in Table E.1.1 for each of the applications at risk. Authorisation is only applicable to the use of substances and thus not applicable to the production of CTPHT. In the following sections each of the applicable risk management options is further discussed.

Table E.1.1. Applicability of other Community-wide risk management options than restriction.

	Authorisation	Voluntary action	Adaptation of IPPC legislation
Production of CTPHT	-	+	+
Primary aluminium production	+	+	+
Graphite electrode production	+	+	+
Ferro-alloy industry applying arc furnaces with Søderberg electrodes	+	+	+

E.1.2.1 Authorisation under REACH

Based on the hazard assessment of CTPHT (PBT, vPvB and CMR) it is possible to initiate the authorisation process under REACH. In this section it is examined whether the authorisation instrument is effective for the reduction of PAH-emissions related to CTPHT or to replace CTPHT by suitable alternatives.

According to article 59 of the REACH Regulation, a manufacturer, importer or downstream user shall not place a substance included in Annex XIV on the market for use or use it himself, unless authorisation for that use has been granted.

In this report three possible scenarios for authorisation are considered:

1) Authorisation is granted for all applications at risk, because alternatives are not available (see Section C). Risk control is based on presently available techniques (Option A1).
2) Authorisation is granted for all applications at risk, because alternatives are not available (see Section C). Risk control is enhanced by improved techniques (Option A2)..
3) There will be no exemptions for authorisation, which will result in the ban of the use of CTPHT, because no authorisation will be given for any of the uses of CTPHT (Option A3).

It is important to note that production of CTPHT (for which a risk is identified) is not covered by the authorisation instrument (see the first paragraph of Section E.1.2).

E.1.2.2 Voluntary action

Voluntary action by industry to reduce PAH emissions would be a possibility for risk reduction. To ensure that voluntary action of industry is really a risk reduction measure, it is needed that the relevant industries commit themselves to a written agreement and that this agreement is publicly available. Several companies claim to replace Søderberg by prebaked technologies in the coming years. There is a possibility that industry will voluntary improve the (prebaked) techniques, but there are no indications that this improvement will take place in the coming years. To some extent such a voluntary action could be triggered by the registration under the REACH regulation, in order to improve the level of control of the risks.

E.1.2.3 IPPC directive

Risks related to industrial sites fall within the framework of the IPPC legislation, which makes this legislation a realistic option for controlling the risks of CTPHT. It should be noted, however, that this legislation is already in place and thus implemented in the baseline (see Section E.1.1).

In spite of this, at present the relevant BAT reference document (EC, 2001a) appears to be insufficient in controlling the risks as indicated by the fact that risks are identified. Yet, within the next years this BAT reference document will be revised. Since there are production sites with very low emissions, it appears to be technically feasible to achieve a higher risk reduction at the production sites which currently have a higher emission profile. A strengthened IPPC BAT reference document is considered as possible risk management option. Information from the risk assessment report may be used for this revision for which suggestions are given in Section H.2.

E.1.3 Options for restrictions

In primary aluminium production two different types of anodes are applied, Søderberg anodes and prebaked anodes. In general, risks for Søderberg application are higher than for prebaked anodes. Therefore, a ban on Søderberg anodes could be a restriction option (Option R1). Restriction of the use of Søderberg anodes will likely result in the replacement of these anodes by prebaked anodes, which show lower risks. Another restriction option is a ban on all CTPHT electrodes and anodes (Option R2). Since the main applications of CTPHT in the primary aluminium production and the production of electrodes and anodes are very similar, it will be in theory feasible to ban all production of electrodes and anodes, rather than limiting the ban to a specific type of electrode or anode. This scenario will result in a total ban of both CTPHT production and CTPHT application in primary aluminium production and the production of electrodes and anodes, because if the use of CTPHT is banned in primary aluminium production and the production of electrodes and anodes, it is hard to find arguments to still allow the production of CTPHT (where risks are also identified). This would of course also imply a ban on other applications, although in these applications no risk is identified and therefore no further risk management was deemed necessary for these applications (see Annex J.7 for arguments).

E.2 Comparison of instruments: restriction vs. other Community-wide risk management options

It should be noted that this is a transitional dossier and that the comparison made in this section is based on information from the risk assessment reports (The Netherlands, 2008ab) only.

E.2.1.1 Risk reduction capacity

The original idea was that a risk reduction strategy for CTPHT could contribute to the development of a risk reduction strategy for PAH emissions. During the study it turned out that none of the proposed risk reduction measures has a high reduction capacity for the total PAH emissions, because CTPHT is only a minor source of PAHs. In the following paragraphs the risk reduction capacity analysis is therefore solely focussing on CTPHT.

It is obvious that listing CTPHT on Annex XIV, without giving any authorisation to industry (Option A3), or restriction of all CTPHT production and use (Option R2), will reduce the risks of CTPHT significantly. However, since there are no alternative techniques available, the risk reduction capacity of authorisation is low if a number of exemptions have to be granted. If authorisation is granted for all applications (Option A1) in effect nothing will change. If authorisation is only granted when risks are controlled by using improved techniques (Option A2), a reduction of risks could be expected. The absence of alternatives also hampers the reduction capacity of voluntary action, because it is expected that the (high) costs will discourage companies to voluntarily reduce their emissions.

IPPC legislation potentially has a high risk reduction capacity for the production of CTPHT. Since there are production sites with very low emissions, it appears to be technically feasible to achieve a higher risk reduction at the production sites which currently have a higher emission profile. Risks are likely to be sufficiently controlled by IPPC if the emission limits are strengthened in a revision of the BAT reference document.

The ban of Søderberg anodes (Option R1) will result in a reduction of the risks of CTPHT, albeit the effect will be minimal, because the use of these Søderberg anodes is limited (see Table B.2.3).

E.2.1.2 Proportionality

Because CTPHT is only a minor source of PAHs, neither of the proposed risk reduction measures is proportional to the total PAH emissions.

All authorisation and restriction risk management options require much more effort from authorities and industry compared to the voluntary action and strengthened IPPC legislation. Focussing on CTPHT, proportionality of authorisation remains low. The most probable outcome is that authorisation will be granted for all uses at risk, which raises questions whether time and money should be spend on a risk reduction measure for which the risk reduction capacity is low. Even when risks are better controlled than at present by using improved techniques, it appears to be more feasible to control this process by legal requirements already in place. If authorisation results in a ban on the use of CTPHT, it can easily be argued that this measure is not proportionate in terms of cost-effectiveness.

Since no alternatives are available it is likely that proportionality of voluntary action will be moderate in terms of cost-effectiveness. It can be expected that the use of Søderberg anodes will be phased out, but since this use is at present limited to only about 1% of the total primary aluminium production (see Table B.2.3), this will only result in a minor reduction of the risks of CTPHT.

Compared to voluntary action, it is expected that risks are further reduced because stengthened IPPC will require techniques to the level of the best operating production sites. However, the extra costs for production sites with a high emission profile will also be higher compared to voluntary

action and could hinder or even obstruct the implementability. This leads to the overall proportionality of strengthened IPPC legislation to be moderate.

Restriction of the use of Søderberg anodes will have only very limited proportionality, since this use is at present limited to only about 1% of the total primary aluminium production (see Table B.2.3) and is expected to be phased out in a few years. For a total ban on production and use of CTPHT, it can easily be argued that this measure is not proportionate in terms of cost-effectiveness.

E.2.2 Practicality: implementability, enforceability, manageability

As argued in Section E.2.1.1, if authorisation is granted and risks are controlled by using presently available techniques, in effect nothing will change, which makes this option easily implementable and enforceable. If risks are to be controlled by new (improved) methods, implementability and enforceability are likely to be more difficult, since these techniques are not readily available. Not authorising the use of CTPHT is hardly implementable due to a lack of necessary alternatives and economical feasibility, but enforceability should not be a problem.

By nature, voluntary action could easily be implemented, but is not enforceable by authorities.

For proper risk control, IPPC legislation should be adapted, which will include development of improved techniques. This limits to some extend the implementability and enforceability of this option.

A restriction on the use of Søderberg anodes is easily implementable and enforceable, because only a few sites are involved and phasing out appears to be in action already. Restriction (*i.e.* a total ban) of all production and use of CTPHT, however will be hard if not impossible to implement, due to a lack of alternatives. Enforceability in the sense that the authorities check if the relevant actors are in compliance with a proposed restriction appears to be feasible. On the other hand, the enforcement of restrictions that are hardly or even not implementable could be very difficult for the authorities.

All management options discussed appear to be manageable and are therefor not distinguishing between these options.

E.2.3 Monitorability

Monitorability of effectiveness of all proposed risk management measures is high. (Reduction of) PAH emissions can be monitored against a relatively low cost. In addition, authorisation and IPPC legislation may be good instruments to get information on the production and use of CTPHT, because such information should be provided by the industry to get permission for this production and use.

E.2.4 Overall assessment against the three criteria

The overall assessment against effectiveness, practicality and monitorability is summarized in Table E.2.1.

Authorisation will not be effective, if the applications which show a risk will not change. An improvement of the risk control is not likely to be proportional and could complicate implementability and enforceability. Not authorizing the use of CTPHT, which results in a total ban on the use of CTPHT, is not proportional to the identified risk and very hard to implement. Furthermore, when a total ban on the use of CTPHT is considered, it is easy to argue to extend such a ban to the production of CTPHT. This latter ban on production and use of CTPHT, however, shows similar proportionality and implementability problems.

A ban on the use of Søderberg anodes can easily be implemented, but its proportionality is very limited, especially considering the relatively large effort in time and money needed for such a relatively small reduction in risk.

Table E.2.1. Comparison of restriction vs. other Community-wide risk management options for CTPHT.

	Annex XIV - SVHC[a)]			Voluntary action	Strengthened IPPC	Annex XVII - Restriction[b)]	
	A1	A2	A3			R1	R2
Effectiveness							
Risk reduction capacity	0	+	+	0	+	0	+
Proportionality	low	low	low	moderate	moderate	low	low
Practicality							
Implementability	0	moderate	difficult	easy	moderate	easy	difficult
Enforceability	0	moderate	difficult	difficult	moderate	easy	difficult
Manageability	0	0	0	0	0	0	0
Monitorability	0	0	0	0	0	0	0

0: The situation will not change compared to baseline (current practice); +: The situation will improve compared to baseline. a) A1: Authorisation is granted for all applications at risk, risk control is based on presently available techniques; A2: Authorisation is granted for all applications at risk, risk control is based on improved techniques; A3: Authorisation is not granted. b) R1: Restriction of the use of Søderberg anodes; R2: Restriction of the use of all CTPHT anodes.

The risk reduction capacity of voluntary action is small and the enforceability is difficult. Strengthening the IPPC legislation has the advantage that it can be enforced and has the potention to reduce the estimated risks.

An adaptation of the IPPC legislation to further reduce PAH emissions from CTPHT production and use, therefore, appears to be most suitable to control the risks of CTPHT.

E.3 Comparison of restrictions options

Since no restriction is proposed, this paragraph is not applicable.

E.4 Main assumptions used and decisions made during analysis

Not applicable.

E.5 The proposed restriction(s) and summary of the justifications

No restrictions are proposed, because the identified risks of CTPHT are all related to industrial point sources. Emission control via IPPC or national legislation (permitting) is the first instrument to consider for such point sources. Further measures, beyond permitting via IPPC and national legislation, cannot be justified (see Section E.2).

To enhance the effectiveness of this legislation, it is suggested that a revision of the BAT reference document should incorporate a more detailed description to prevent risks that are associated with the identified emissions to the aquatic and terrestrial environment, next to the presently included emissions to the atmosphere (see also Section H.2).

F. SOCIO-ECONOMIC ASSESSMENT OF PROPOSED RESTRICTION(S)

No restrictions are proposed.

G. STAKEHOLDER CONSULTATION

The following stakeholders have been informed on this transitional report:

- European Aluminium Association
- European Carbon and Graphite Association
- Cindu Chemicals BV
- Rütger-Chemicals

H. OTHER INFORMATION

H.1 Risk reduction strategy for workers

November 2007 (endorsed at the 15th RRSM in April 2008).

H.1.1 Background

Coal tar pitch high temperature (CTPHT) is the solid fraction produced during the distillation of coal tars. Coal tars are condensation products obtained during the production of coke an/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. High-temperature coal tars (CAS # 65996-89-6) is defined in EC 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. A black viscous liquid denser than water. Composed primarily of a complex mixture of condensed ring aromatic hydrocarbons. May contain minor amounts of phenolic compounds and aromatic nitrogen bases'. The distillation of high-temperature coal tars results in tar oils (including naphthalene oil, creosote oil, anthracene oil, and creosote) and a solid fraction (coal tar pitch high temperature). When CTPHT is heated, Coal tar pitch volatiles (CTPVHT) are released. 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 it's products are heated. 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.

Classification

Current classification (According to Annex I)

Classification :Carc. Cat. 2

Symbol :T

R-phrases :45

S-phrases :53-45

Notes :H (pitch)

Proposed classification

Decisions by the Technical Committee on Classification and Labelling (TC-C&L) in October 2006 for physical and human health endpoints.

Classification :Mut. Cat 2; Carc. Cat. 1; Repro. Cat. 2

Symbol :T; Xi

R-phrases :41, 43, 45, 46, 60-61

S-phrases :53 - 45

Notes :H (pitch)

Occupational limit values

Occupational limit values for CTPV.

Country/ organization	8-hr time weighted average (mg/m³)	Remarks	References
USA			
ACGIH	0.2	As benzene soluble aerosol	ACGIH (1999)
OSHA	0.2	As benzene soluble fraction	ACGIH (2002)
NIOSH	0.1	As cyclohexane-soluble fraction	ACGIH (1999)
		Classified as A1 carcinogen, *i.e.* confirmed human carcinogen: the compound is carcinogenic to humans based on the weight of evidence from epidemiological studies.	ACGIH (2002)
The Netherlands	-	Under consideration[11]	Health Council of The Netherlands (2006)
UK	-	Listed among compounds which are currently on the work programme.	HSE (1999)
Germany	-	Listed as pyrolysis products from organic materials. They are classified among compounds which are capable of inducing malignant tumours as shown by experience with humans.	DFG (2001)
		The TRK (Technical Guide Concentration) for benzo(a)pyrene was 0.005 mg/m³ for production, loading and unloading of pencil pitch, and in the area near the ovens in coking plants and 0.002 mg/m³ for all other workplaces in Germany.	
Sweden	not listed	The occupational exposure limit for benzo(a)pyrene is 0.002 mg/m³.	NBOSH (2002)
Denmark	0.2	As benzene-soluble fraction	Arbejdstilsynet (2005)
		Coal- and oil-derived substances, as well as coal tar products, including coal tar pitch distillates with boiling point > 200 °C are listed among substances which are considered to be carcinogenic.	
Norway	-	Reference is made to polycyclic aromatic hydrocarbons for which an exposure limit of 0.04 mg/m³ has been established.	Direktoratet for arbeidstilsynet (1996)
European Union	not listed		Hunter *et al.* (1997)

The U.S.A. uses, depending on the organization, an occupational exposure limit between 0.1 and 0.2 mg/m^3. Except for Denmark no occupational exposure limit is set for CTPHT. An occupational exposure limit is deducted from PAHs, benzo(a)pyrene or other coal/oil-derived substances. The occupational exposure limit set by Denmark is within the range of that of the U.S.A. with 0.2 mg/m^3.

H.1.2 The risk assessment results

Production and use

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 1127,000 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.

[11] The Health Council advises a value of 5.6 ng/m^3 (risk level of 10^{-6}) for benzo(a)pyrene, with an upper limit of 560 ng/m^3 (risk level 10^{-4}).

Coal tar pitch is mainly used as a binding agent in the production of carbon electrodes, anodes and Søderberg 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.

Use pattern for coal tar pitch. 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.

The exposure assessment will be focussed on the emission of PAHs on a local scale for production of coal tar pitch and the main applications, primarily because lower emissions for the other sources are expected[12]. Moreover, the amounts of coal tar pitch used for roofing and road paving decrease as it is replaced by petroleum pitch on account of the lower PAH content (worker hygiene). Some manufacturers claim to produce "environmentally" friendly clay pigeons by applying petroleum pitch in order to meet the EEC environmental protection directives, or apply no binder at all. The emission of PAHs at coke ovens are not considered because coal tar is produced at this process. Coal tar is used as a feedstock for the production of coal tar pitch and therefore the coke ovens are not part of the life cycle of coal tar pitch which actually starts the production stage of coal tar pitch.

With respect to the main applications of coal tar pitch, the following point sources will be considered:

- Anode production
- Aluminium production applying prebakes (with and without) anode baking.
- Aluminium production using Søderberg technology
- Graphite electrode production
- Production of steel, silicon, etc., applying electric arc furnaces with Søderberg electrodes.

The future consumption of pitches depends not only on human health risks and environmental hazards but also on economics due to progress of science and technology.

Since more than ten years a new technology has been developed at bench scale based on inert anodes to replace CTPHT-bound carbonated anodes but this technology is still immature and costly. Therefore, it can be expected that CTPHT will be used for more than decades in the primary aluminium smelters.

[12] At present information on the emission of PAHs from the use of coal tar pitch as binder for refractories is very limited. However, it can not be excluded that the PAH emission from the use of this application can be significant (comparable to the main applications, depending on the abatement techniques used).

For refractories, the pitch industry now proposes pitches with a higher softening point resulting in a benzo(a)pyrene content of 300 ppm compared to currents levels in pitches ranging up to 20,000 ppm.

Most of the European countries have banned CTPHT in the road construction by law or agreement between trade unions and road building companies. In fact only very particular applications such as kerosene proof coatings for parking lots, airfields and taxi ways still use pitch as an emulsion. This market is decreasing and represents only 200 tonnes of pitch per year.

Pitch bound active carbons are more and more produced outside the EU and are anyway processed in closed vessels where the pitch is pyrolyzed to pure carbon with controlled emissions.

Roofing and corrosion protection with CTPHT-based products are declining dramatically and a phasing out of these artefacts is predicted in the next few years.

The use of pitch bound coal briquettes is forbidden in some countries (Germany and Scandinavia). This market is also linked to dedicated and captive users in mining countries (France and Belgium) where retired miners have rights on solid fuels provided by the former state owned companies. Capacities of 2,000 ktonnes/year of briquettes existing in the early 1980's in Europe are now decreased to 150 ktonnes/year, also using more environmental friendly binders like starch and molasses. Also here a full phasing out of the use of CTPHT can be expected in the next few years.

Clay pigeons manufacturers, claiming environmental protection, displaced carbopitch by petrochemical binders for more than 80% of their production and the former clay pigeons being exported outside the European Union.

In summary, the pitch market, decreasing in Europe for economical reasons, will remain only for electrodes, anodes and graphite artefacts.

With respect to the main applications of CTPHT, the following scenario's are considered to be relevant for occupational exposure assessment.

<u>Production and Use Scenarios</u>

Occupational Scenario 1: Production of CTPHT in coal tar distillation plants

Occupational Scenario 2: Use as a binding agent for electrodes

Sub-scenarios: (i) Use as a binding agent in electrodes in the aluminium industry

(ii) Use as a binding agent in graphite electrode production and impregnation of electrodes

Occupational Scenario 3: Use as a binding agent in the asphalt industry

Sub-scenarios: (i) Use as a binding agent in road construction

(ii) Use as a binding agent in roofing and waterproofing

Occupational Scenario 4: Use as a binding agent for refractories

Occupational Scenario 5: Use as a binding agent for active carbon

Occupational Scenario 6: Use in heavy duty corrosion protection

Occupational Scenario 7: Use as a binding agent in coal briquetting

Occupational Scenario 8: Use as a binding agent for clay pigeons

The database on possible health hazards induced by CTPHT is rather limited, implicating that a full risk assessment for all the required endpoints is not possible. 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 striking 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 also exposed to, 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 effect, quite possibly even to zero. Therefore, in spite of the limited available data on non-carcinogenic properties of CTPHT, it is decided in the risk assessment report for CTPHT that further testing for some endpoints will be subordinated to conclusions on risks based on carcinogenic and mutagenic properties, using the best-studied PAH benzo(a)pyrene as a guidance substance for exposure to CTPHT in various worker exposure scenarios, in line with the available meta-analyses on lung and bladder cancer where benzo(a)pyrene was also used as exposure indicator. In addition, as these analyses has collected, analysed and summarised all available data on CTPHT exposures in a scientifically sound manner, it is considered to yield the best estimates.

Summary of the conclusions

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

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

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

Conclusion (i) applies to skin irritation, systemic toxicity after repeated exposure, and effects on reproduction. The conclusion can be put 'on hold' and the necessity for further testing be revisited after a risk reduction strategy.

Conclusion (ii) applies to acute toxicity, skin sensitisation, eye irritation, and corrosivity.

Conclusion (iii) applies to mutagenicity, and carcinogenicity, effects that cannot be excluded for exposure (inhalation and dermal) arising from production and use as an intermediate.

It is noted that the excess lifetime risk under occupational conditions of exposure points to very low acceptable exposure levels with regard to carcinogenic effects, which implies a considerable reduction of the current limit values. It is expected that compliance to these low exposure levels will prevent effects other than carcinogenic effects to occur.

H.1.3 Current risk reduction measures

3.1 In all working processes an adequate level of containment is necessary, based on the workplace specific risk assessment evaluation according to Directive 89/391/EEC, 98/24/EEC and, especially, 2004/37/EC. The information in the risk assessment report should be an important input for the workplace specific risk assessment and evaluation.

3.2 The Safety Data Sheet according to Directive 91/155/EEC should contain all relevant information from the risk assessment report.

3.3 Relevant Occupational Exposure Limits has been set in the past on the present product or can be deduced from limit values on PAHs, benzo(a)pyrene or like products. However, some of these values are repealed in the mean time. At present there is no EU standard that is applicable to the present product.

H.1.4 Possible further risk reduction measures

H.1.4.1 Classification

The risk assessment report concludes to the necessity of a new, more severe classification. This proposal is subject to a separate EU decision scheme and cannot be evaluated here further.

However, this proposal to establish a new, more severe EU harmonised classification is sustained. As a result, more restrictions in use and a heavier containment regime might be necessary as a consequence of the new classification.

Conclusion: a new, more severe EU harmonised classification is sustained, but cannot be evaluated here.

H.1.4.2 Use restrictions

The risk assessment report has indicated a possible wide spread and open use of the present substance. A use restriction for a substance with the hazard characterisation of the present product is then to be considered in addition to the results from a new, more severe classification (see paragraph H.1.4.1).

As a result of recent developments however, the use is or will soon be restricted to electrode production and use. This use is in well defined industrial locations.

Furthermore, the composition of the present product is being very much ameliorated with regard to carcinogenic compounds (substitution principle in the context of Directive 2004/37/EC).

The possibility of containment is high in the industrial premises concerned (no open use).

In conclusion, there is not enough reason for EU harmonised use restrictions, especially when the recommendations under paragraphs H.1.4.1 and H.1.4.3 are followed.

Conclusion: there is not enough reason for additional EU harmonised use restrictions.

H.1.4.3 Occupational Exposure Limit

A European OEL is lacking. The establishment of a European OEL should be considered for substances with a use on many locations and in most EU countries, and with considerable risks. These requisites seem to be fulfilled.

The present substance is used in the metal producing industry. Furthermore, there are special production locations for the electrodes used in the metal producing industry. In most EU countries there are metal producing industries.

The present legal interventions within the EU differ considerably. It is desirable that more EU harmonisation is reached, especially on substances as the present one with considerable risks and use in most EU countries, under sometimes very different regimes.

Benzo(a)pyrene is used in the risk assessment report as the risk assessment marker chemical, and is probably a very good marker chemical to set the EU OEL.

Conclusion: there is a need for establishing a European OEL, probably on the marker chemical benzo(a)pyrene.

H.1.5 Assessment of further possible further risk reduction measures

H.1.5.1 EU OEL

Effectiveness

A European OEL for the present substance or the marker chemical benzo(a)pyrene will be an effective means to prevent health effects from worker exposure. Directives 98/24/EEC and 2004/37/EC prescribe that every Member State set a national OEL taking into account the EU OEL. Furthermore, the workplace specific risk assessment evaluation according to Directive 89/391/EEC, 98/24/EEC and, especially, 2004/37/EC by the employer must take into consideration (*inter alia*) this national OEL. Thereafter, the employer must take risk reduction measures to the effect that the health risk as identified in the workplace specific risk assessment will be eliminated or minimised.

Therefore, the EU OEL will have a direct impact on the workplace specific risk assessment and, moreover, on the resulting workplace specific risk reduction measures that eliminate or minimise possible health effects of worker exposure to the present substance.

Practicability

It is practical to eliminate or minimise possible health effects of worker exposure to the present substance by setting a EU OEL, probably for the marker chemical benzo(a)pyrene. In every Member State there will be, as a result of the EU OEL, a clear standard for accepted worker exposure to the present substance. This clear standard will be used by every employer to design his workplace, and by the enforcement authorities to assess the effectiveness of the workplace specific risk reduction measures. It is practical to have only one clear standard both for all workplaces and enforcement actions.

Economical impact

The employer has several options for workplace design based on the OEL. These options comprise, after first consideration of replacement: organisation of workplace and the system of work, protection equipment, number of workers exposed and duration and height of exposure. The employer may choose the appropriate measures with report to his specific conditions and possibilities. Therefore, the economic impact of a EU OEL will be acceptable as the resulting risk reduction measures can be tailored to each enterprise specific situation.

Monitorability

It is technical possible to monitor an OEL for benzo(a)pyrene. Monitoring is enforceable by the Member State authorities (normally the Labour Inspectorates).

H.1.6 Draft conclusions and recommendations

The legislation for workers' protection currently into force at Community level is generally considered to give an adequate framework to limit the risks of coal tar pitch, high temperature to the extent needed and shall apply. There is no need for additional risk reduction measures in the context of the Existing Substances Regulation. However, based on the results of the risk assessment report, it is recommended within the framework of the legislation on worker protection

- to establish at Community level an occupational exposure limit, probably for the marker chemical benzo(a)pyrene.

H.2 Suggestions for revision of the IPPC-directive

In the present document (EC, 2001a), limit values focus on emissions to the atmosphere. In a revised document a more detailed description should be incorporated to prevent risks that are associated with the identified emissions to the aquatic and terrestrial environment as well (see Section B.8.2).

Another point of concern that should be addressed, is that especially for the larger PAHs (> 4 rings) in many cases C_{local}/PNEC ratios are >1. To limit the associated risks it is proposed to set emission limits in such a way that not only C_{local}/PNEC ratios should be lower than 1, but also that the sum of these ratios for the 16 EPA-PAHs is lower than 1.

A final point of concern is that limit values in the present document (EC, 2001a) are set per production unit, while there are no restrictions to the production as such. This may lead to a situation where (very) high local PAH concentrations are identified, while emissions per production unit are well within the limits. It is therefore proposed to include emission limits per time unit (day, month, year) next to the emission limits per production unit that are already included.

I. REFERENCES

Accardi-Dey A, Gschwend PM (2003) Reinterpreting literature sorption data considering both absorption into organic carbon and adsorption onto black carbon. *Environ Sci Technol* 37: 99-106.

ACCCI (1992) Generic material safety data sheet for coal-tar pitch. Draft.: American Coke and Coal Chemicals Institute (ACCCI).

ACGIH (1999) TLVs and BEIs. Threshold Limit Values for chemical Substances and Physical Agents & Biological Exposure Indices. Cincinnati, OH, USA: American Conference of Governmental Industrial Hygienists (ACGIH).

ACGIH (2002) TLVs and BEIs. Threshold Limit Values for chemical Substances and Physical Agents & Biological Exposure Indices. Cincinnati, OH, USA: American Conference of Governmental Industrial Hygienists (ACGIH).

ACGIH (2003) TLVs and BEIs. Threshold Limit Values for chemical Substances and Physical Agents & Biological Exposure Indices. Cincinnati, OH, USA: American Conference of Governmental Industrial Hygienists (ACGIH).

Achazi RK, Chroszcz G, Duker C, Henneken M, Rothe B , Schaub K, Steudel I (1995) The effect of fluoranthene (Fla), benzo(a)pyrene (BaP) and cadmium (Cd) upon survival rate and life cycle parameter of two terrestrial annelids in laboratory test systems. *Newslett Enchytraeidae* 4: 7-14.

Albers PH, Loughlin TR (2003) Effects of PAHs on marine birds, mammals and reptiles. In: PAHs: An Ecotoxicological Perspective. Douben PET (Ed), John Wiley & Sons, West Sussex, England: 243-261.

Alcoa (2002) Aluminium Plant in Reydarfjordur, Fjardabyggd. Comparison of environmental impact of the proposed 322 tpy Alcoa-Reydaral aluminum plant and the two phase 420,000 tpy Reydaral aluminum plant.

Alexander M (1995) How toxic are toxic chemicals in soil? *Environ Sci Technol* 29: 2713-2717.

Alexandrie AK, Warholm M, Carstensen U, Axmon A, Hagmar L, Levin JO, Östman C, Rannug A (2000) CYP1A1 and GSTM1 polymorphisms affect urinary 1-hydroxypyrene levels after PAH exposure. *Carcinogenesis* 21: 669-676.

Allred PM, Giesy JP (1985) Solar radiation-induced toxicity of anthracene to *Daphnia pulex*. *Environ Toxicol Chem* 4: 219-226.

Angerer J, Mannschreck C, Gundel J (1997) Occupational exposure to polycyclic aromatic hydrocarbons in a graphite-electrode producing plant: biological monitoring of 1-hydroxypyrene and monohydroxylated metabolites of phenanthrene. *Int Arch Occup Environ Health* 69: 323-331.

Ankley GT, Erickson RJ, Sheedy BR, Kosian PA, Mattson VR, Cox JS (1997) Evaluation of models for predicting the phototoxic potency of polycyclic aromatic hydrocarbons. *Aquat Toxicol*, 37, 37-50.

Ankley GT, Burkhard LP, Cook PM, Diamond SA, Erickson RJ, Mount DR (2003) Assessing risks from photoactivated toxicity of PAHs to aquatic organisms. In: PAHs: An Ecotoxicological Perspective. Douben PET (Ed), John Wiley & Sons, West Sussex, England: 275-296.

Arbejdstilsynet (2005) Limit values for substances and materials. Copenhagen, Denmark: Arbejdstilsynet. http://www.at.dk/graphics/at/engelsk-pdf/at-vejledninger/gvlisteuk.pdf.

Arey J, Atkinson R (2003) Photochemical reactions of PAHs in the Atmosphere. In: Douben (ed.) PAHs: An Ecotoxicological Perspective., Wiley, 47-64.

Arey J, Zielinska B, Atkinson R, Winer AM (1987) Polycyclic aromatic hydrocarbon and nitroarene concentrations in ambient air during a winter-time high NOX episode in the Los Angeles basin. *Atmosph Environ* **21**: 1437-1444.

Armstrong B, Tremblay C, Baris D, Thériault G (1994) Lung cancer mortality and polynuclear aromatic hydrocarbons: a case- cohort study of aluminium production workers in Arvida, Quebec, Canada. *Am J Epidemiol* 139: 250-262.

Armstrong B, Hutchinson E, Fletcher T (2003) *Cancer risk following exposure to polycyclic aromatic hydrocarbons (PAHs): a meta-analysis.* Rep No 068. Sudbury, UK Health and Safety Executive. Available at http://www.hse.gov.uk (accessed 2005).

Armstrong B, Hutchinson E, Unwin J, Fletcher T (2004) Lung cancer risk after exposure to polycyclic aromatic hydrocarbons: A review and meta-analysis. *Environ Health Perspect* 112: 970-978.

Armstrong BG, Tremblay CG, Cyr D, Thériault GP (1986) Estimating the relationship between exposure to tar volatiles and the incidence of bladder cancer in aluminium smelter workers. *Scand J Work Environ Health* 12: 486-493.

Arnould JP, Pfohl-Leszkowicz A, Bach V, Libert JP, Belegaud J (1999) Biological monitoring exposure of workers from plant producing carbon electrodes: quantification of benzo(a)pyrene DNA-adducts in leukocytes, by a ^{32}P-postlabelling method and an immunoassay. *Hum Exp Toxicol* 18: 314-321.

ASTM (2004) *Standard Test Method for Relative Density of Solid Pitch and Asphalt* (Displacement Method), ASTM D71-94, American Society for Testing and Materials (ASTM).

Atienzar FA, Conradi M, Evenden AJ, Jha AN, Depledge MH (1999) Qualitative assessment of genotoxicity using random amplified polymorphic DNA: Comparison of genomic template stability with key fitness parameters in *Daphnia magna* exposed to benzo(a)pyrene. *Environ Toxicol Chem* 18: 2275-2282.

Atkinson R (1994) Gas-phase tropospheric chemistry of organic compounds. *J. Physic. Chem. Ref. Data* 2: 1-216.

Atkinson R, Arey J (1994) Atmospheric chemistry of gas-phase polycyclic aromatic hydrocarbons: formation of atmospheric mutagens. *Env Health Perspect*. 102: 117-126.

ATSDR (1995) *Toxicological Profile for Polycyclic Aromatic Hydrocarbons.* US Department of Health & Human Services, Agency for Toxic Substances and Disease Registry. August 1995: 1-487.

ATSDR (2002) *Toxicological Profile for Wood Creosote, Coal Tar Creosote, Coal Tar, Coal Tar Pitch, and Coal Tar Pitch Volatiles.* US Department of Health & Human Services, Agency for Toxic Substances and Disease Registry. September 2002: 1-354.

Axelman J, Naes K, Näf C, Broman D (1999) Accumulation of polycyclic aromatic hydrocarbons in semipermeable membrane devices and caged mussels (*Mytilus edulis* L.) in relation to water column phase distribution. *Environ Toxicol Chem* 18: 2454-2461.

Bannerjee DK, Fedorak PM, Hashimoto A, Masliyah JH, Pickard MA, Gray MR (1995) Monitoring the biological treatment of anthracene-contaminated soil in a rotating-drum bioreactor. *Appl Microbiol Biotechnol* 43: 521-528.

Barrows ME, Petrocelli SR, Macek KJ, Carroll JJ (1980) Bioconcentration and elimination of selected water pollutants by bluegill sunfish *Lepomis macrochirus.* In: *Dynamic, exposure, hazard assessment of toxic chemicals.* Michigan, Ann Arbor Science Publishers, 379-392.

Bastian MV, Toetz DW (1982) Effect of eight polynuclear hydrocarbons on growth of *Anabaena flos-aquae*. *Bull Environ Contam Toxicol* 29: 531-538.

Baudgrasset F, Baudgrasset S, Safferman SI (1993) Evaluation of the bioremediation of a contaminated soil with phytotoxicity tests. *Chemosphere* 26: 1365-1374.

Baumann PC, Harshbarger JC (1995) Decline in liver neoplasm in wild brown bullhead catfish after cooking plant closes and environmental PAHs plummet. *Environ Health Perspect* 103: 168-170.

Baussant T, Sanni S, Skadsheim A, Jonsson G, Børseth JF, Gaudebert B (2001) Bioaccumulation of polycyclic aromatic compounds: 2. modeling bioaccumulation in marine organisms chronically exposed to dispersed oil. *Environ Toxicol Chem* 20: 1185-1195.

Becker G, Colmsjo A, Ostman C (1999) Determination of thiaarenes and polycyclic aromatic hydrocarbons in workplace air of an aluminium reduction plant. *Environ Sci Technol* 33: 1321-1327.

Behymer TD, Hites RA (1988) Photolysis of polycyclic aromatic hydrocarbons adsorbed on fly ash. Environ. Sc. Tech., 22, 1311-1319.

Belfroid A, Sikkenk M, Seinen W, Van Gestel K, Hermens J (1994) The toxicokinetic behavior of chlorobenzenes in earthworm (*Eisenia andrei*) experiments in soil. *Environ Toxicol Chem* 13: 93-99.

Belfroid AC, Sijm DTHM, Van Gestel CAM (1996) Bioavailability and toxicokinetics of hydrophobic aromatic compounds in benthic and terrestrial invertebrates. *Environ Rev* 4: 276-299.

Bellona (2004) Mangansmelteverke i Norge. http://bellona.no/no/harde_miljoefakta/industri_og_oljevirksomhet/1282.html, at 14 June 2004.

Bentsen-Farmen RK, Botnen IV, Notø, Jacob J, Øvrebø S (1999) Detection of polycyclic aromatic hydrocarbon metabolites by high- pressure liquid chromatography after purification on immunoaffinity columns in urine from occupationally exposed workers. *Int Arch Occup Environ Health* 72: 161-168.

Berdowski JJM, Baas J, Bloos JPJ, Visschedijk AJH, Zandveld PYJ (1997) *The European Emission Inventory of Heavy Metals and Persistent Organic Pollutants for 1990*, Forschungsbericht 104 02 672/03. The Netherlands Organisation for Applied Scientific Research (TNO), TNO-MEP, Apeldoorn, the Netherlands.

Berglind L (1982) Determination of polycyclic aromatic hydrocarbons in industrial discharges and other aqueous effluents. Oslo, Central Institute for Industrial Research, 21 pp. (Nordic PAH Project, Report No. 16). Reference taken from IARC (1984).

Bidleman, TF (1988) Atmospheric processes. *Environ Sci Technol* 22: 361-367.

Bintein S, Devillers J, Karcher W (1993) Nonlinear dependence of fish bioconcentration on n-octanol/water partition coefficient. *SAR QSAR Environ Res* 1: 29-39.

Bisson M, Dujardin R, Flammarion P, Garric J, Babut M, Lamy M-H, Porcher J-M, Thybaud É, Vindimian É (2000) *Complément au SEQ-Eau: méthode de détermination des seuils de qualité pour les substances génotoxiques.* Verneuil-en-Halatte, France: Institut National de l'Environnement Industriel et des Risques (INERIS), Agence de l'eau Rhin-Meuse.

Bjørseth A, Bjørseth O, Fjeldstad PE (1981) Polycyclic aromatic hydrocarbons in the work atmosphere, Determination of area-specific concentrations and job-specific exposure in a vertical pin Søderberg aluminum plant. *Scand J Work Environ Health* 7: 223-232.

Black JA, Birge WJ, Westerman AG, Francis PC (1983) Comparative aquatic toxicology of aromatic hydrocarbons. *Fundam Appl Toxicol* 3: 353-358.

Bleeker EAJ, Wiegman S, Droge STJ, Kraak MHS, Van Gestel CAM (2003) *Towards an Improvement of the Risk Assessment of Polycyclic (Hetero)Aromatic Hydrocarbons.* Amsterdam, The Netherlands: Aquatic Ecology and Ecotoxicology, Faculty of Science, University of Amsterdam and Animal Ecology, Institute of Ecological Science, Faculty of Earth and Life Sciences, Vrije Universiteit Amsterdam. Report 2003-01 (UvA)/Report 2003-04 (VU).

Boese BL, Lamberson JO, Swartz RC, Ozretich RJ (1997) Photoinduced toxicity of fluoranthene to seven marine benthic crustaceans. *Arch Environ Contam Toxicol* 32: 389-393.

Boese BL, Lamberson JO, Swartz RC, Ozretich R, Cole F (1998) Photoinduced toxicity of PAHs and alkylated PAHs to a marine infaunal amphipod (*Rhepoxynius abronius*). *Arch Environ Contam Toxicol* 34: 235-240.

Boese BL, Ozretich RJ, Lamberson JO, Swartz RC, Cole FA, Pelletier J, Jones J (1999) Toxicity and phototoxicity of mixtures of highly lipophilic PAH compounds in marine sediment: Can the ΣPAH model be extrapolated? *Arch Environ Contam Toxicol* 36: 270-280.

Boffetta P, Jourenkova N, Gustavsson P (1997) Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. *Cancer Causes Control* 8: 444-472.

Boffetta P, Burstyn I**,** Partanen T **,** Kromhout H**,** Svane O**,** Langård S**,** Järvholm B**,** Frentzel-Beyme R**,** Kauppinen T**,** Stücker I**,** Shaham J**,** Heederik D**,** Ahrens W**,** Bergdahl IA**,** Cenée S**,** Ferro G**,** Heikkilä P**,** Hooiveld M**,** Johansen C**,** Randem BG**,** Schill W (2003) Cancer mortality among European asphalt workers: An international epidemiological study. II. Exposure to bitumen fume and other agents. *Am J Ind Med* **43:** 28-39.

Boogaard PJ, Van Sittert NJ (1994) Exposure to polycyclic aromatic hydrocarbons in petrochemical industries by measurement of urinary 1-hydroxypyrene. *Occup Environ Med* 51: 250-258.

Borovsky D, Linley JR, Kagan J (1987) Polycyclic aromatic compounds as phototoxic mosquito larvicides. *J Am Mosq Control Assoc* 3: 246-250.

Bossert ID, Bartha R (1986) Structure biodegradability relationships of polycyclic aromatic hydrocarbons in soil. *Bull Environ Contam Toxicol* 37: 490-495.

Bowmer CT (1994) *Paris Commission sediment reworker ring-test, 1993.* Technical report. TNO-report IMW-R 93/317, The Netherlands Organisation for Applied Scientific Research (TNO), Environmental and Energy Research, Institute of Environmental Sciences, Delft, the Netherlands.

Bowmer CT, Roza P, Henzen L, Degeling C (1993) *The development of chronic toxicological tests for PAH contaminated soils using the earthworm* Eisenia fetida *and the springtail* Folsomia candida. TNO-report IMW-R 92/387, The Netherlands Organisation for Applied Scientific Research (TNO), Environmental and Energy Research, Institute of Environmental Sciences, Delft, the Netherlands.

Broman D, Näf C, Lundbergh I, Zebuhr Y (1990) An *in situ* study on the distribution, biotransformation and flux of polycyclic aromatic hydrocarbons (PAHs) in an aquatic food-chain (Seston, *Mytilus edulis* L, *Somateria mollissima L*) from the Baltic-an ecotoxicological perspective. *Environ Toxicol Chem* 9: 429-442.

Brubaker WW, Hites RA (1998) OH reaction kinetics of polycyclic aromatic hydrocarbons and polychlorinated dibenzo-*p*-dioxins and dibenzofurans. *J Phys Chem* 102: 915-921.

Bruner KA, Fisher SW, Landrum PF (1994) The role of the Zebra Mussel, *Dreissena polymorpha*, in contaminant cycling: I. The effect of body size and lipid content on the bioconcentration of PCBs and PAHs. *J Great Lakes Res* 10: 725-734.

Bucheli TD, Gustafson O (2000) Quantification of the soot-water distribution coefficient of PAHs provides mechanistic basis for enhanced sorption observations. *Environ Sci Technol* 34: 5144-5151.

Buchet JP, Ferreira M, Burrion JB (1995) Tumor markers in serum, polyamines and modified nucleosides in urine, and cytogenetic aberrations in lymphocytes of workers exposed to polycyclic aromatic hydrocarbons. *Am J Indust Med* 27: 523-543.

Buchet JP, Gennart JP, Mercado-Calderon F, Delavignette JP, Cupers L, Lauwerys R (1992) Evaluation of exposure to polycyclic aromatic hydrocarbons in a coke production and a graphite electrode manufacturing plant: assessment of urinary excretion of 1-hydroxypyrene as a biological indicator of exposure. *Br J Ind Med* 49: 761-768.

Buijsman E (1999) *Assessment of air quality for Polycyclic Aromatic Hydrocarbons in the Netherlands.* RIVM Report 729999001.

Burgess RM, Ryba SA, Perron MM, Tien R, Thibodeau LM, Cantwell MG (2004) Sorption of 2,4'-Dichlorobiphenyl and fluoranthene to a marine sediment amended with different types of black carbon. *Environ Toxicol Chem* 23: 2534-2544.

Burgos WD, Berry DF, Bhandari A, Novak JT (1999) Impact of soil-chemical interactions on the bioavailability of naphthalene and 1-naphthol. *Water Res* 33: 3789-3795.

Burstyn I, Kromhout H (2000a) Are the members of a paving crew uniformly exposed to bitumen fume, organic vapor, and benzo(a)pyrene? *Risk Anal* 20: 653-663.

Burstyn I, Kromhout H (2000b) *Exposure assessment for a multicentric cohort study of cancer risk among European asphalt workers.* Utrecht University.

Burstyn I, Kromhout H, Boffetta P (2000) Literature review of levels and determinants of exposure to potential carcinogens and other agents in the road construction industry. *AIHAJ* 61: 715-726.

Cairns MA, Nebeker AV (1982) Toxicity of acenaphthene and isophorone to early life stages of fathead minnows. *Arch Environ Contam Toxicol* 11: 703-707.

Caldwell RS, Caldarone EM, Mallon MH (1977) Effects of a seawater-soluble fraction of Cook Inlet crude oil and its major aromatic components on larval stages of the Dungeness crab, *Cancer magister* Dana. In: *Fate and effects of petroleum hydrocarbons in marine ecosystems and organisms* (Wolfe DA, ed), Pergamon Press, New York: 210-220.

Calvert JG, Atkinson R, Becker KH, Kamens RM, Seinfeld JH, Wallington TJ, Yarwood G (2002) *The Mechanisms of Atmospheric Oxidation of Aromatic Hydrocarbons*, Oxford University Press, Oxford: 566.

Carmichael, LM, Pfaender FK (1997) Polynuclear aromatic hydrocarbon metabolism in soils-relationship to soil characteristics and preexposure. *Environ Toxicol Chem* 16: 666-675.

Carslaw N, Carpenter LJ, Plane JMC, Allan BJ, Burgess RA, Clemitshaw KC, Coe H, Penkett SA (1997) Simultaneous observations of nitrate and peroxy radicals in the marine boundary layer. *J Geoph Res* 102: 18917-18933.

Carstensen U, Hou SM, Alexandrie AK, Högstedt B, Tagesson C, Warholm M, Rannug A, Lambert B, Axmon A, Hagmar L (1999a) Influence of genetic polymorphisms of biotransformation enzymes on gene mutations, strand breaks of deoxyribonucleic acid, and micronuclei in mononuclear blood cells and urinary 8-hydroxydeoxyguanosine in potroom workers exposed to polyaromatic hydrocarbons. *Scand J Work Environ Health* 25: 351-360.

Carstensen U, Yang K, Levin JO, Ostman C, Nilsson T, Hemminki K, Hagmar L (1999b) Genotoxic exposures of potroom workers. *Scand J Work Environ Health* 25: 24-32.

CCDM (2000) Emission and waste in The Netherlands, Annual report 1998 and estimates for 1999. Report series Coordination Commission Target group monitoring, Nr. 6, November 2000.

CCSG (2006a) *Internal communication*, Coal Chemicals Sector Group/CEFIC.

CCSG (2006b) Coal Chemicals Sector Group. Several data sets and information in (e-mail) communications.

CCSG (2007) IND comments on Pitch RAR Draft of May 2007, Human Health Part (COM323_hh_IND5.doc).

Cerniglia CE (1992) Biodegradation of polycyclic aromatic hydrocarbons. *Biodegradation* 3: 351-368.

Cerniglia CE, Sutherland JB, Crow, SA (1992) Fungal metabolism of aromatic hydrocarbons. In: *Microbial Degradation of Natural Products* (Winkel G, ed), VCH Weinheim: 193-217.

Chang F, Wang L, Zhao Q, Zhu Q, Wu Y, Chen C, Syrjänen S, Syrjänen K (1992) Investigation on the carcinogenic effects of coal tar pitch in rat respiratory tract by intratracheal instillations. *Carcinogenesis* 13: 233-239.

Chepiga TA, Morton MJ, Murphy PA, Avalos JT, Bombick BR, Doolittle DJ, Borgerding MF, Swauger JE (2000) A comparison of the mainstream smoke chemistry and mutagenicity of a representative sample of the US cigarette market with two Kentucky reference cigarettes (K1R4F and K1R5F). *Food Chem Toxicol* 38: 949-962.

Cho E-A, Bailer AE, Oris JT (2003) Effect of methyl *tert*-butyl ether on the bioconcentration and photoinduced toxicity of fluoranthene in Fathead Minnow larvae (*Pimephales promelas*). *Environ Sci Technol* 37: 1306-1310.

Chung N, Alexander M (1998) Differences in sequestration and bioavailability of organic compounds in dissimilar soils. *Environ Sci Technol* 32: 855-860.

Cincinelli A, Stortinin AM, Perugini M, Checchini L, Lepri, L (2001) Organic pollutants in sea-surface microlayer and aerosol in the coastal environment of Leghorn (Tyrrhenian Sea). *Mar Chem* 76: 77-98.

Claypigeon Company Ltd. (2008) Quality. http://www.claypigeoncompany.co.uk/quality.html.

Clements WH, Oris JT, Wissing TE (1994) Accumulation and food-chain transfer of fluoranthene and benzo(a)pyrene in *Chironomus riparius* and *Lepomis macrochirus*. *Arch Environ Contam Toxicol* 26: 261-266.

Coates JD, Woodward J, Allen J, Philp P, Lovley DR (1997) Anaerobic degradation of polycyclic aromatic hydrocarbons and alkanes in petroleum-contaminated marine harbor sediments. *Appl Environ Micr* 63: 3589-3593.

Cody TE, Radike MJ, Warshawsky D (1984) The phototoxicity of benzo(a)pyrene in the green alga *Selenastrum capricornutum*. *Environ Res* 35: 122-132.

Cole FA, Boese BL, Swartz RC, Lamberson JO, DeWitt TH (2000) Effects of storage on the toxicity of sediments spiked with fluoranthene to the amphipod, *Rhepoxynius abronius*. *Environ Toxicol Chem* 19: 744-748.

Collin G, Höke H (2002) TAR AND PITCH-Processing of Coke-Oven Coal Tar. In: *Ullmann's Encyclopedia of Industrial Chemistry*, Wiley-VCH Verlag GmbH, Weinheim, Germany.

Collins WJ, Stevenson DS, Johnson CE, Derwent RG (1995) Tropospheric ozone modelled with a global scale Lagrangian model of diurnal atmospheric chemistry. Met O (APR) Turbulence and Diffusion Note 224. Meteorological Office, Bracknell.

Contox (1991a) Estudio de toxicidad aguda por via dermica. Substancia de ensayo: Brea de hulla. (suministrada por Industrias Quimicas de Nalon). Madrid, Spain: Laboratorio Contox.

Contox (1991b) Estudio de toxicidad aguda por via oral. Substancia de ensayo: Brea de hulla. (suministrada por Industrias Quimicas de Nalon). Madrid, Spain: Laboratorio Contox.

Cornelissen G, Gustafsson O, Bucheli TD, Jonker MTO, Koelmans AA, Van Noort PCM (2005) Extensive Sorption of Organic Compounds to Black Carbon, Coal, and Kerogen in Sediments and Soils: Mechanisms and Consequences for Distribution, Bioaccumulation, and Biodegradation. *Environ Sci Technol* 39: 6881 -6895.

Correa M, Coler R (1983) Enhanced oxygen uptake rates in dragonfly nymphs (*Somatochlora cingulata*) as an indication of stress from naphthalene. *Bull Environ Contam Toxicol* 30: 269-276.

Coutant RW, Brown L, Chuang JC, Riggin RM, Lewis RG (1988) Phase distribution and artifact formation in ambient air sampling for polynuclear aromatic hydrocarbons. *Atmosph Environ* 22: 403-409.

Creosote Council Europe (2004) *Report Summaries from Creosote Dossier of 28 March 2004 (Biocide Directive).*

Cullen MR, Checkoway H, Alexander BH (1996) Investigation of a cluster of pituitary adenomas in workers in the aluminium industry. *Occup Environ Med* 53: 782-786.

Culp SJ, Beland FA (1994) Comparison of DNA adduct formation in mice fed coal tar or benzo(a)pyrene. *Carcinogenesis* 15: 247-252.

Culp SJ, Gaylor DW, Sheldon WG, Goldstein LS, Beland FA (1996a) DNA adduct measurements in relation to small intestine and forestomach tumor incidence during the chronic feeding of coal tar or benzo(a)pyrene to mice. *Polycycl Aromat Comp* 11: 161-168.

Culp SJ, Gaylor DW, Sheldon WG, Goldstein LS, Beland FA (1996b) Relationship between DNA adduct levels and tumor incidence in mice fed coal tar or benzo(a)pyrene for 2 years. *Proc Am Assoc Cancer Res* 37: 101.

Culp SJ, Gaylor DW, Sheldon WG, Goldstein, LS, Beland FA (1998) A comparison of the tumors induced by coal tar and benzo(*a*)pyrene in a 2-year bioassay. *Carcinogenesis* 19: 117-124.

De Maagd PGJ (1996) *Polycyclic aromatic hydrocarbons: fate and effects in the aquatic environment.* Academic Thesis, Rijksuniversiteit Utrecht, The Netherlands.

De Maagd PGJ, Sinnige TL, Schrap SM, Opperhuizen A, Sijm DTHM (1998) Sorption coefficient of polycyclic aromatic hydrocarbons for two lake sediments: influence of the bactericide sodium azide. *Environ Toxicol Chem* 17: 1899-1907.

De Voogt P, Van Hattum B, Leonards P, Klamer JC, Govers, H (1991) Bioconcentration of polycyclic heteroaromatic hydrocarbons in the guppy (*Poecilia reticulata*). *Aquat Toxicol* 20: 169-194.

Den Besten PJ, Schmidt CA, Ohm M, Ruys MM, Van Berhem JW, Van der Guchte, C (1995) Sediment quality assessment in the deltas of the river Rhine and Meuse based on filed observations, bioassays and food chain implications. *J Aquat Ecosyst Health* 4: 257-270.

Denzer S, Herrchen M, Lepper P, Müller M, Sehrt R, Storm A, Volmer J (1999) Revised proposal for a list of priority substances in the context of the water framework directive (COMMPS procedure). Ref 98/788/3040/DEB/E1.

DeWitt TH, Hickey CW, Morrisey DJ, Nipper MG, Roper DS, Williamson RB, Van Dam L, Williams EK (1999) Do amphipods have the same concentration-response to contaminated sediment *in situ* as *in vitro*? *Environ Toxicol Chem* 18: 1026-1037.

DFG (2001) MAK- und BAT-Werte-Liste 2001. Maximale Arbeitsplatzkonzentrationen und Biologische Arbeitsstoftoleranzwerte. Senatskommission zur Prüfung gesundheitsschadlichter Arbeitsstoffe. Mitteilung 37. Weinheim, Federal Republic of Germany: Wiley-VCH Verlag GmbH.

DFG (2006) MAK- und BAT-Werte-Liste 2006. Maximale Arbeitsplatzkonzentrationen und Biologische Arbeitsstoftoleranzwerte. Senatskommission zur Prüfung gesundheitsschadlichter Arbeitsstoffe. Mitteilung 42. Weinheim, Federal Republic of Germany: Wiley-VCH Verlag GmbH.

DIN (1984) DIN 51920, Ausgabe 1984-04, Prüfung von Kohlenstoffmaterialien; Bestimmung des Erweichungspunktes nach Mettler; Binde- und Imprägniermittel. Deutsches Institut für Normung e.V. (DIN).

DIN (1991) DIN 38412, Deutsche Einheitsverfahren zur Wasser-, Abwasser- und Schlammuntersuchung. Testverfahren mit Wasserorganismen (Gruppe L) Bestimmung der Hemmwirkung von Wasserinhaltsstoffen auf Bakterien (Pseudomonas-Zellvermehrungs-Hemmtest). Deutsches Institut für Normung e.V. (DIN).

DIN (2003) DIN 51794, Ausgabe 2003-05, Prüfung von Mineralölkohlenwasserstoffen-Bestimmung der Zündtemperatur. Deutsches Institut für Normung e.V. (DIN).

Ding YS, Yan XJ, Jain RB, Lopp E, Tavakoli A, Polzin GM, Stanfill SB, Ashley DL, Watson CH (2006) Determination of 14 polycyclic aromatic hydrocarbons in mainstream smoke from U.S. brand and non-U.S. brand cigarettes. *Environ Sci Technol* 40:1133-1138.

Ding YS, Ashley DL, Watson CH (2007) Determination of 10 carcinogenic polycyclic aromatic hydrocarbons in mainstream cigarette smoke. *J Agric Food Chem* 55:5966-5973.

Direktoratet for arbeidstilsynet (1996) *Ardministrative normer for forurensning i arbeidsatmosfaere 1996.* Direktoratet for arbeidstilsynet, Oslo, Norway.

Diskowski H (2002) PHOSPHORUS-Production of White Phosphorus. In: *Ullmann's Encyclopedia of Industrial Chemistry*, Wiley-VCH Verlag GmbH, Weinheim, Germany.

DiToro DM, McGrath JA (2000) Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. *Environ Toxicol Chem* 19: 1971-1982.

DiToro DM, McGrath JA, Hansen DJ (2000) Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. I. Water and tissue. *Environ Toxicol Chem* 19: 1951-1970.

Djomo JE, Garriques P, Narbonnes JF (1996) Uptake and depuration of polycyclic aromatic hydrocarbons from sediment by zebrafish (*Brachydanio rerio*). *Environ Toxicol Chem* 15: 1177-1181.

Donato F, Monarca F, Marchinonna G, Rossi A, Cicioni C, Chiesa R, Colin D, Boffetta P (2000) Mortality from cancer and chronic respiratory diseases among workers who manufacture carbon electrodes. *Occup Environ Med* 57; 484-487.

Dor F, Dab W, Empereur-Bissonnet P, Zmirou D (1999) Validity of biomarkers in environmental health studies: the case of PAHs and benzene. *Crit Rev Toxicol* 29(2): 129-168.

Douben PET (2003) *PAHs: An Ecotoxicological perspective.* Wiley.

Droge STJ, Léon-Paumen M, Bleeker EAJ, Kraak MHS, Van Gestel CAM (2006) Chronic toxicity of polycyclic aromatic compounds to the springtail *Folsomia candida* and the enchytraeid *Enchytraeus crypticus. Environ Toxicol Chem* 25: 2423-2431.

Durant ND, Wilson LP, Bouwer EJ (1995) Microcosm studies of sunsurface PAH-degrading bacteria from a former manufactured gas plant. *J Contam Hydrol* 17: 213-223.

Dutch Expert Committee on Occupational Standards, a committee of the Health Council of the Netherlands (2006) *BaP and PAH from coal-derived sources. Health-based calculated occupational cancer risk values of benzo(a)pyrene and unsubstituted non-heterocyclic polycyclic aromatic hydrocarbons from coal-derived sources.* Report No. 2006/01OSH, The Hague, The Netherlands.

Dutton CB, Pigeon MJ, Renzi PM, Feustel PJ, Dutton RE, Renzi GD (1993) Lung function in workers refining phosphorus rock to obtain elementary phosphorus. *J Occup Med* 35(10): 1028-1033.

Duyzer JH, Plant RAJ, Bleeker A (2002) *Determination of emissions to water by atmospheric deposition.* TNO-MEP report R 2002/268, The Netherlands Organisation for Applied Scientific Research (TNO), Environment, Energy & Process Innovation, The Netherlands Organisation for Applied Scientific Research (TNO), Environment, Energy & Process Innovation, Den Helder, The Netherlands.

Duyzer JH, Vonk AW (2003) *Atmospheric deposition of pesticides, PAHs and PCBs in the Netherlands.* TNO-MEP report R 2003/255, The Netherlands Organisation for Applied Scientific Research (TNO), Environment, Energy & Process Innovation, Den Helder, The Netherlands.

EAA (2004) European Aluminium Association. Information provided by the industry. Fax/Letter.

EAA (2005) European Aluminium Association. Updated exposure data for Søderberg plants, anode plants and paste plants (several files).

EAA (2006) European Aluminium Association. Several files with updated presentations of data sets and communication in response to questions.

EAA (2007) European Aluminium Association. Information provide by the industry. Fax/Letter.

Eastmond DA, Booth GM, Lee ML (1984) Toxicity, accumulation, and elimination of polycyclic aromatic sulfur heterocycles in *Daphnia magna*. *Arch Environ Contam Toxicol* 13: 105-111.

EC (1967) Council Directive 67/548/EEC of 27 June 1967 on the approximation of laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances. *Official Journal* 196, 16/08/1967: 1-98.

EC (1976a) Council Directive 76/768/EEC of 27 July 1976 on the approximation of the laws of the Member States relating to cosmetic products. *Official Journal* L 53, 25/02/1977: 30.

EC (1976b) Council Directive 76/769/EEC of 27 July 1976 on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. *Official Journal* L 262, 27/09/1976: 201-203.

EC (1984) Commission Directive 84/449/EEC of 25 April 1984 adapting to technical progress for the sixth time Council Directive 67/548/EEC on the approximation of laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances. *Official Journal* 251, 19/09/1984: 1-223.

EC (1989) Council Directive 89/391/EEC of 12 June 1989 on the introduction of measures to encourage improvements in the safety and health of workers at work. *Official Journal* L 183, 29/06/1989: 1-8.

EC (1991) Commission Directive 91/155/EEC of 5 March 1991 defining and laying down the detailed arrangements for the system of specific information relating to dangerous preparations in implementation of Article 10 of Directive 88/379/EEC. *Official Journal* L 76, 22/03/1991: 35-41.

EC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. *Official Journal* L 84, 05/04/1993: 1-75.

EC (1996) Council Directive 96/61/EC of 24 September 1996 concerning integrated pollution prevention and control. *Official Journal L 257, 10/10/1996: 26-40.*

EC (1997) 21st Commission Directive 97/45/EC of 14 July 1997 adapting to technical progress Annexes II, III, VI and VII to Council Directive 76/768/EEC on the approximation of the laws of the Member States relating to cosmetic products (Text with EEA relevance). *Official Journal* L 196, 24/07/1997: 77-78.

EC (1998) Council Directive 98/24/EEC of 7 April 1998 on the protection of the health and safety of workers from the risks related to chemical agents at work (Fourteenth individual directive within the meaning of article 16(1) of Directive 89/391/EEC) *Official Journal* L 131, 05/05/1998: 11-23.

EC (1999a) Council directive 1999/31/EC of 26 April 1999 on the landfill of waste. *Official Journal* L 182, 16/07/1999: 1-19.

EC (1999b) Directive 1999/45/EC of the European Parliament and of the Council of 31 May 1999 concerning the approximation of the laws, regulations and administrative provisions of the Member States relating to the classification, packaging and labelling of dangerous preparations. *Official Journal* L 200, 30/07/1999: 1-68.

EC (2000a) Directive 2000/60/EC of the European Parliament and of the Council of 23 October 2000 establishing a framework for Community action in the field of water policy. *Official Journal* L 327, 22/12/2000: 1-73.

EC (2000b) Directive 2000/76/EC of the European Parliament and of the Council of 4 December 2000 on the incineration of waste. *Official Journal* L 332, 28/12/2000: 91-111.

EC (2001a) Integrated Pollution Prevention and Control (IPPC). Reference Document on Best Available Techniques in the Non Ferrous Metals Industries. European Commission, Brussels, Belgium.

EC (2001b) Ambient Air Pollution by Polycyclic Aromatic Hydrocarbons (PAH). Position Paper. Prepared by the Working Group on Polycyclic Aromatic Hydrocarbons. July, 2001.

EC (2001c) Commission Directive 2001/59/EC of 6 August 2001 adapting to technical progress for the 28th time Council Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances (Text with EEA relevance.) *Official Journal* L 225, 21/8/2001: 1-333.

EC (2001d) Commission Directive 2001/90/EC of 26 October 2001 adapting to technical progress for the seventh time Annex I to Council Directive 76/769/EEC on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations (creosote) (Text with EEA relevance) *Official Journal* L 283, 27/10/2001: 41-43.

EC (2003a) Council Decision of 19 December 2002 establishing criteria and procedures for the acceptance of waste at landfills pursuant to Article 16 of and Annex II to Directive 1999/31/EC (2003/33/EC). *Official Journal* L 11, 16/01/2003: 27-49.

EC (2003b) Technical Guidance Document on Risk Assessment in support of Commission Directive 93/67/EEC on Risk Assessment for new notified substances, Commission Regulation (EC) No. 1488/94 on Risk Assessment for existing substances and Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market. European Commission, Joint Research Centre, European Chemicals Bureau.

EC (2004a) Directive 2004/107/EC of the European Parliament and of the Council of 15 December 2004 relating to arsenic, cadmium, mercury, nickel and polycyclic aromatic hydrocarbons in ambient air. *Official Journal* L 23, 26/01/2005: 3-16.

EC (2004b) Directive 2004/37/EC of the European Parliament and of the Council of 29 April 2004 on the protection of workers from the risks related to exposure to carcinogens or mutagens at work (Sixth individual Directive within the meaning of Article 16(1) of Council Directive 89/391/EEC) (codified version) (Text with EEA relevance) *Official Journal* L 158, 30/4/2004: 50-76.

EC (2005) Final draft Technical Guidance Document (TGD) Human Health Risk Characterisation (2005). European Commission, European Communities, Italy.

EC (2006a) Commission Directive 2006/8/EC, amending, for the purposes of their adaptation to technical progress, Annexes II, III and V to Directive 1999/45/EC of the European Parliament and of the Council concerning the approximation of the laws, regulations and administrative provisions of the Member States relating to the classification, packaging and labelling of dangerous preparations. *Official Journal* L 19, 24/01/2006: 12-19.

EC (2006b) Directive 2006/121/EC of the European Parliament and of the Council of 18 December 2006 amending Council Directive 67/548/EEC on the approximation of laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances in order to adapt it to Regulation (EC) No 1907/2006 concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) and establishing a European Chemicals Agency *Official Journal* L 396, 30/12/2006: 1-856.

EC (2008) Directive 2008/1/EC of the European Parliament and of the Council of 15 January 2008 concerning integrated pollution prevention and control (Codified version). *Official Journal* L 24, 29/01/2008: 8-29.

ECGA (2004) European Carbon and Graphite Association. Information provide by the industry on the emission to air and water of B(a)P in the year 2002.

ECGA (2005) European Carbon and Graphite Association. Aggregated questionnaire occupational exposure data (aggregated questionnaire Q CTP_2.doc).

ECGA (2006a) European Carbon and Graphite Association. Information provide by the industry on the emission to air and water of B(a)P in the year 2002 and 2005.

ECGA (2006b) European Carbon and Graphite Association. Report of 2006 measurements of BaP values and communication in response to questions.

ECGA (2007) European Carbon and Graphite Association. Information provide by the industry on the emission of cooling water.

Edlund S (2001) PAH as a POP. Possibilities, implications and appropriateness of regulating global emissions of Polycyclic Aromatic Hydrocarbons through the Stockholm Convention on Persistent Organic Pollutants. IIIEE Report 2001:9. The International Institute for Industrial Environmental Economics (IIIEE), Lund University, Lund, Sweden.

Edwards NT (1983) Polycyclic aromatic hydrocarbons (PAHs) in the terrestrial environment-a review. *J Environ Qual* 12: 427-441.

EEA (2001) *EMEP / CORINAIR Emission Inventory Guidebook-3rd edition.* European Environment Agency. Copenhagen, Denmark.

EEIG (2003) Smoking and the environment: Actions and attitudes. European Opinion Research Group, Directorate General Health and Consumer Protection, European Commission, Special Eurobarometer 183/ Wave 58.2.

El-Alawi YS, Dixon DG, Greenberg BM (2001) Effects of a pre-incubation period on the photoinduced toxicity of polycyclic aromatic hydrocarbons to the luminescent bacterium *Vibrio fischeri*. *Environ Toxicol* 16: 277-286.

El-Alawi YS, Huang X-D, Dixon DG, Greenberg BM (2002) Quantative structure-activity relationship for the photoinduced toxicity of polycyclic aromatic hydrocarbons to the luminescent bacteria *Vibrio fischeri*. *Environ Toxicol Chem*, 21 (10), 2225-2232.

Emery Jr. VL, Dillon TM (1996) Chronic toxicity of phenanthrene to the marine polychaete worm, *Nereis* (*Neanthes*) *arenaceodentata*. *Bull Environ Contam Toxicol* 56: 265-270.

Emmett EA (1986) Cutaneous and ocular hazards of roofers. *Occup Med* 1; (2): 307-322.

Emmett EA, Bingham EM, Barkley W (1981) A carcinogenic bioassay of certain roofing materials. *Am J Ind Med* 2: 59-64.

EPA (1998a) *Emission Inventory of Section 112(c)(6) Pollutants.* Final Report, April 1998.

EPA (1998b) *Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter. Radiant Corporation (1995).* Report 298-130-43.

EPER (2004) The European Pollutant Emission Register (EPER). http://eper.eea.europa.eu/eper/.

Eramet (2003) *Environmental report 2002.* Eramet Comilog, Eramet Norway AS.

Eramet (2004) Information taken from the URL: http://www.eramet.fr, at 2 July 2004.

Evans MS, Landrum PF (1989) Toxicokinetics of DDE, benzo*(a)*pyrene, and 2,4,5,2',4',5'-hexachlorobiphenyl in *Pontoporeia hoyi* and *Mysis relicta*. *J Great Lakes Res* 15: 589-600.

Falk-Petersen I-B, Sæthre LJ, Lönnig S (1982) Toxic effects of naphthalene and methylnaphthalenes on marine plankton organisms. *Sarsia* 67: 171-178.

Fernandez M, L'Haridon J (1992) Influence of lighting conditions on toxicity and genotoxicity of various PAH in the newt *in vivo*. *Mutat Res* 298: 31-41.

Fielden MR, Wu Z, Sinal CJ, Jury HH, Bend JR, Hammond GL, Zacharewski TR (2000) Estrogen receptor and aryl hydrocarbon receptor-mediated activities of a coal-tar creosote. *Environ Toxicol Chem* 19: 1262-1271.

Finger SE, Little EF, Henry MG, Fairchild JF, Boyle TP (1985) Comparison of laboratory and field assessment of fluorene-Part I: Effects of fluorene on the survival, growth, reproduction, and behavior of aquatic organisms in laboratory tests. In: *Validation and predictability of laboratory methods for assessing the fate and effects of contaminants in aquatic ecosystems* (Boyle TP, ed), American Society for Testing and Materials, ASTM STP 865, Philadelphia, PA: 120-133.

Fisk AT, Norstrom RJ, Cymbalisty CD, Muir DCG (1998) Dietary accumulation and depuration of hydrophobic organ chlorines: bioaccumulation parameters and their relationship with the octanol/water partition coefficient. *Environ Toxicol Chem* 17: 951-961.

Foran JA, Holst LL, Giesy JP (1991) Effects of photo enhanced toxicity of anthracene on ecological and genetic fitness of *Daphnia magna*: A reappraisal. *Environ Toxicol Chem* 10: 425-427.

Forget-Leray J, Landriau I, Minier C, Leboulenger F (2005) Impact of endocrine toxicants on survival, development, and reproduction of the estuarine copepod *Eurytemora affinis* (Poppe). *Ecotoxicol Environ Saf* 60: 288-294.

Frank AP, Landrum PF, Eadie BJ (1986) Polycyclic aromatic hydrocarbon: Rates of uptake, depuration, and biotransformation by Lake Michigan *(Stylodrilus heringianus). Chemosphere* 15: 317-330.

Franssen MEJ, Van der Wilt GJ, De Jong PCMP, Bos RP, Arnold WP (1999) A retrospective study of the teratogenicity of dermatological coal tar products [3]. *Acta Dermato-Venereologica* 79: 390-391.

Freitag D, Geyer H, Kraus A, Viswanathan R, Kotzias D, Attar A, Klein W, Korte F (1982) Ecotoxicological profile analysis VII. Screening chemicals for their environmental behavior by comparative evaluation. *Ecotoxicol Environ Saf* 6: 60-81.

Freitag DL, Ballhorn L, Geyer H, Korte F (1985) Environmental hazard profile of organic chemicals. An experimental method for the assessment of the behaviour of organic chemicals in the ecosphere by means of simple laboratory tests with ^{14}C labelled chemicals. *Chemosphere* 14: 1589-1616.

Gerlach SA (1981) *Marine pollution. Diagnosis and Therapy.* Springer Verlag.

Gibbs GW (1985) Mortality of aluminum reduction plant workers, 1950 through 1977. *J Occup Med* 27: 761-770.

Goldbohm RA, Tielemans ELJP, Heederik D, Rubingh CM, Dekkers S, Willems MI, Kroese ED (2006) Risk estimation for carcinogens based on epidemiological data: a structured approach, illustrated by an example on Chromium. *Regul Toxicol Pharmacol* 44: 294-310.

Goldstein LS, Weyand EH, Safe S, Steinberg M, Culp SJ, Gaylor DW, Beland FA, Rodriguez LV (1998) Tumours and DNA adducts in mice exposed to benzo(a]pyrene and coal tars: implications for risk assessment. *Environ Health Perspect* 106: 1325-1330.

Golomb D, Ryan D, Underhill J, Wade T, Zemba S (1997) Atmospheric deposition of toxics onto Massachusetts Bay-II. Polycyclic aromatic hydrocarbons. *Atmos Environ* 31: 1361-1368.

Gorski T (1959) Experimental investigations on the carcinogenic properties of some pitches and tars produced from Silesian pit coal. *Med Pract* 5: 309-317.

Ghosh U, Zimmerman JR, Luthy RG (2003) PCB and PAH speciation among particle types in contaminated harbor sediments and effects on PAH bioavailability. *Environ Sci Technol* **37**: 2209-2217.

Gossiaux DC, Landrum PF, Fisher SW (1996) Effect of temperature on the accumulation kinetics of PAHs and PCBs in the Zebra Mussel, *Dreissena polymorpha. J Great Lakes Res* 22: 379-388.

GrafTech (2004) *GrafTech Updates Earnings Estimates, Tax Planning Activities and Strategic Initiatives*, October 2004. Retrieved from the Internet at 6/6/2005: http://www.graftech.com.

Graham R, Hester HR, Barkey W (1940) Coal-tar-pitch poisoning in pigs. *J Am Vet Med Assoc* 96: 135-149.

Grandrass J, Salomons W (eds) (2001) *Appendix to Project Report Dredged Material in the Port of Rotterdam, Interface between Rhine Catchment Area and North Sea.* GKSS Research Centre, Institute for Coastal Research.

Grant WM (1986) *Toxicology of the eye. 3rd Ed.* Charles C Thomas Publisher, Springfield IL, USA.

Groot MSM, Bronswijk JJB, Willems WJ, De Haan T, Del Castilho P (1997) *Landelijk Meetnet Bodemkwaliteit; Resultaten 1994.* RIVM report 714801017, National Institute for Public Health and the Environment (RIVM), Bilthoven, The Netherlands.

Groot MSM, Bronswijk JJB, Van Leeuwen TC (2003) *National Soil Monitoring Network; Results 1997.* RIVM report 714801029, National Institute for Public Health and the Environment (RIVM), Bilthoven, The Netherlands.

Grosjean D, Fung K, Harrison J (1983) Interactions of polycyclic aromatic hydrocarbons with atmospheric pollutants. *Environ Sci Technol* 17: 673-679.

Gundel J, Schaller KH, Angerer J (2000) Occupational exposure to polycyclic aromatic hydrocarbons in a fireproof stone producing plant: biological monitoring of 1- hydroxypyrene, 1-, 2-, 3- and 4-hydroxyphenanthrene, 3-hydroxybenz(a)anthracene and 3-hydroxybenzo(a)pyrene. *Int Arch Occup Environ Health* 73: 270-274.

Hackett PL, Rommereim DN, Sikov MR (1984) Developmental toxicity following oral administration of a high-boiling coal liquid to pregnant rats. *J Appl Toxicol* 4: 57-62.

Hagen LO (2002) *Kontrollmålinger av PAH i luft ved aluminiumverk vinteren 2001/2002.* NILU report OR 37/2002, Norwegian Institute for Air Research (NILU), Kjeller, Norway.

Halling-Sørensen B, Nyholm N, Baun A (1996) Algal toxicity test with volatile and hazardous compounds in air-tight test flasks with CO_2 enriched headspace. *Chemosphere* 32: 1513-1526.

Hannah JB, Hose JE, Landolt ML, Miller BS, Felton SP, Iwaoka WT (1982) Benzo(a)pyrene-induced morphologic and developmental abnormalities in rainbow trout. *Arch Environ Contam Toxicol* 11: 727-734.

Hansen ES (1989) Cancer mortality in the asphalt industry: a ten year follow up of an occupational cohort. *British J Indus Med* 46: 582-585.

Harkey G, Van Hoof P, Landrum P (1995) Bioavailability of polycyclic aromatic hydrocarbons from a historically contaminated sediment core. *Environ Toxicol Chem* 14: 1551-1560.

Harkey GA, Lydy MJ, Kukkonen J, Landrum PF (1994) Feeding selectivity and assimilation of PAH and PCB in *Diporeia* spp. *Environ Toxicol Chem*, 13, 1445-1455.

Hatch AC, Burton Jr. GA (1998) Effects of photoinduced toxicity of fluoranthene on amphibian embryos and larvae. *Environ Toxicol Chem* 17: 1777-1785.

Hatch AC, Burton Jr. GA (1999) Photo-induced toxicity of PAHs to *Hyalella azteca* and *Chironomus tentans*: Effects of mixtures and behavior. *Environ Pollut* 106: 157-167.

Hawthorne SB, Grabanski, CB, Miller, DJ (2006) Measured partitioning coefficients for parent and alkyl polycyclic aromatic hydrocarbons in 114 historically contaminated sediments: Part I. K_{OC} values. *Environ Toxicol Chem* 25: 2901-2911.

Health Council of the Netherlands (2006) BaP and PAH from coal-derived sources Health-based calculated occupational cancer risk values of benzo[a]pyrene and unsubstituted non-heterocyclic polycyclic aromatic hydrocarbons from coal-derived sources. Publication no. 2006/01OSH, Health Council of the Netherlands, The Hague, The Netherlands.

Healy J, Bradley SD, Northage C, Scobbie E (2001) Inhalation exposure in secondary aluminium smelting. *Ann Occup Hyg* 45: 217-225.

Heinrich U, Pott F, Mohr U, Fuhst R, König J (1986) Lung tumours in rats and mice after inhalation of PAH-rich emissions. *Exp Pathol* 29: 29-34.

Heinrich U, Peters L, Creutzenberg O, Dasenbrock C, Hoymann H.-G (1994a) Inhalation exposure of rats to tar/pitch condensation aerosol or carbon black alone or in combination with irritant gases. In: *Toxic and carcinogenic effects of solid particles in the respiratory tract (ILSI Monographs)* (Mohr U, Dungworth DL, Mauderly JL, Oberdörster G, eds). ILSI Press, Washington DC, USA: 433-441.

Heinrich U, Roller M, Pott F (1994b) Estimation of a lifetime unit lung cancer risk for benzo(a)pyrene based on tumour rates in rats exposed to coal tar/pitch condensation aerosol. *Toxicol Lett* 72: 155-161.

Hellou J, Upshall C, Ni IH, Payne JF, Huang YS (1991) Polycyclic aromatic hydrocarbons in harp seals (*Phoca groenlandica*) from the north-west Atlantic. *Arch Environ Contam Toxicol* 21: 135-140.

HERAG (2006) Project factsheet: Indirect exposure via the environment-Consumer exposure.

Herbes SE, Schwall LR (1978) Microbial transformation of polycyclic aromatic hydrocarbons in pristine and petroleum contaminated sediments. *Appl Environ Microbiol* 35: 306-316.

Heussner JC, Ward Jr. JB, Legator MS (1985) Genetic monitoring of aluminium workers exposed to coal tar pitch volatiles. *Mutat Res* 155: 143-155.

Hickey CW, Roper DS, Holland PT, Trower TM (1995) Accumulation of organic contaminants in two sediment dwelling shellfish with contrasting feeding modes: deposit- (*Macomona liliana*) and filter-feeding (*Austrovenus stutchburyi*) *Arch Environ Contam Toxicol* 29: 221-231.

Holcombe GW, Phipps GL, Fiandt JT (1983) Toxicity of selected priority pollutants to various aquatic organisms. *Ecotoxicol Environ Saf* 7: 400-409.

Holland MR, Jones HH, Berdowski J, Bleeker A, Visschedijk AJH (2001) *Economic Evaluation of Air Quality Targets for PAHs*. AEA Technology Environment, report number AEA/ENV/R0593.

Holst LL, Giesy JP (1989) Chronic effects of the photoenhanced toxicity of anthracene on *Daphnia magna* reproduction. *Environ Toxicol Chem* 8: 933-942.

Hooftman RN, Evers-de Ruiter A (1992) *Early life stage tests with* Brachydanio rerio *and several polycyclic aromatic hydrocarbons using an intermittent flow-through system (draft OECD guideline).* TNO-report IMW-R 92/210, The Netherlands Organisation for Applied Scientific Research (TNO), Environmental and Energy Research, Institute of Environmental Sciences, Delft, The Netherlands.

Horstmann M, McLachlan MS (1998) Atmospheric deposition of semivolatile organic compounds to two forest canopies. *Atmos Environ* 32: 1799-1809.

Hose JE (1985) Potential uses of sea urchin embryos for identifying toxic chemicals: Description of a bioassay incorporating cytologic, cytogenetic and embryologic endpoints. *J Appl Toxicol* 5: 245-254.

Hose JE, Hannah JB, DiJulio D, Landolt ML, Miller BS, Iwaoka WT, Felton SP (1982) Effects of benzo(a)pyrene on early development of flatfish. *Arch Environ Contam Toxicol* 11: 167-171.

Hose JE, Puffer HW, Oshida PS, Bay SM (1983) Developmental and cytogenic abnormalities induced in the purple sea urchin by environmental levels of benzo(a)pyrene. *Arch Environ Contam Toxicol* 12: 319-325.

Howard PH, Boethling RS, Jarvis WF, Meylan WM, Michelenko EM (1991) *Handbook of Environmental Degradation Rates*, Lewis Publishers, Chelsea, Michigan.

HSDB (2004) *Hazardous Substances Data Bank (HSDB), coal tar*, http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB, assessed in 2004.

HSE (1993) *Occupational Exposure Limits: 1993.* EH40/93, HSE (Health and Safety Executive) Books, Sudbury (Suffolk), UK.

HSE (1999) *Occupational Exposure Limits: 1999.* EH40/99, HSE (Health and Safety Executive) Books, Sudbury (Suffolk), UK.

HSE (2002) *Occupational Exposure Limits 2002.* EH40/2002, HSE (Health and Safety Executive) Books, Sudbury (Suffolk), UK.

Huang X-D, Dixon DG, Greenberg BM (1995) Increased polycyclic aromatic hydrocarbon toxicity following their photomodification in natural sunlight: Impacts on the duckweed *Lemna gibba* L. G-3. *Ecotoxicol Environ Saf* 32: 194-200.

Huang X-D, Krylov SN, Ren L, McConkey BJ, Dixon DG, Greenberg BM (1997a) Mechanistic quantitative structure-activity relationship model for the photoinduced toxicity of polycyclic aromatic hydrocarbons: II. An empirical model for the toxicity of 16 polycyclic aromatic hydrocarbons to the duckweed *Lemna gibba* L. G-3. *Environ Toxicol Chem* 16: 2296-2303.

Huang X-D, McConkey BJ, Babu TS, Greenberg BM (1997b) Mechanisms of photoinduced toxicity of photomodified anthracene to plants: Inhibition of photosynthesis in the aquatic higher plant *Lemna gibba* (duckweed). *Environ Toxicol Chem* 16: 1707-1715.

Hubble DH, Russel RO, Vernon HL, Marr RJ (1998) Chapter 4, Steelmaking Refractories. In: *The Making, Shaping and Treating of Steel, 11th Edition-Steelmaking and Refining Volume* (Fruehan RJ, ed), Sample pages: http://www.aise.org.

Hulzebos EM, Adema DMM, Dirven-Van Breemen EM, Henzen L, Van Dis WA, Herbold HA, Hoekstra JA, Baerselman R, Van Gestel CAM (1993) Phytotoxicity studies with *Lactuca sativa* in soil and nutrient solution. *Environ Toxicol Chem* 12: 1079-1094.

Hunter WJ, Aresini G, Haigh R, Papadopoulos P (1997) Occupational exposure limits for chemicals in the European Union. *Occup Environ Med* 54: 217-222.

IARC (1983) *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data.* IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 32. International Agency for Research on Cancer (IARC), Lyon, France.

IARC (1984) *Polynuclear aromatic Compounds, Part 3, Industrial Exposures in aluminium Production, Coal Gasification, Coke Production, and Iron and Steel Founding* IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 34. International Agency for Research on Cancer (IARC), Lyon, France.

IARC (1985) *Polynuclear aromatic Compounds, Part 4, Bitumens, Coal-tars and Derived Products, Shale Oils and Soots.* IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 35. International Agency for Research on Cancer (IARC), Lyon, France.

IARC (1987) *Silica and Some Silicates.* IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 42. International Agency for Research on Cancer (IARC), Lyon, France.

IARC (2002) *Cancer Incidence in Five Continents, Vol. VIII* (Parkin DM, Whelan SL, Ferlay J, Teppo T, Thomas DB, eds), IARC Scientific Publications No. 155, International Agency for Research on Cancer (IARC), Lyon, France.

Industry, 2006. Pitch RAR Human Part (Draft of Sep. 2005) [R323_0510_hh(10Nov05]-IND comments-08. Jan. 2006.

Institut National de Recherche et de Sécurité (2006) *Valeurs limites d'exposition professionnelle aux agents chimiques en France, Aide-mémoire technique.*

International Tar Association (2002) *Use of coal tar pitch in the EU.* Letter dd. 20/12/02. No. 0426.

ISO (1997) *Carbonaceous materials for the production of aluminium -- Pitch for electrodes -- Determination of coking value.* ISO 6998. International Organisation for Standardisation (ISO).

ISO (2002) *Determination of flash point-Pensky-Martens closed cup method.* ISO 2719. International Organisation for Standardisation (ISO).

Jager DT (1998) Mechanistic approach for estimating bioconcentration of organic chemicals in earthworms (Oligochaeta) *Environ Toxicol Chem* 17: 2080-2090.

Jager T, Baerselman R, Dijkman E, De Groot AC, Hogendoorn EA, De Jong A, Kruitbosch JAW, Peijnenburg WJGM (2003) Availability of polycyclic aromatic hydrocarbons to earthworms (*Eisenia andrei*, Oligochaeta) in field-polluted soils and soil-sediment mixtures. *Environ Toxicol Chem* 22: 767-775.

Jager T, Sanchez FAA, Buijs B, Van der Velde EG, Posthuma L (2000) Toxicokinetics of polycyclic aromatic hydrocarbons in *Eisenia andrei* (Oligochaeta) using spiked soil. *Environ Toxicol Chem* 19: 953-961.

Jimenez BD, Cirmo CP, McCarthy JF (1987) Effects of feeding and temperature on uptake, elimination and metabolism of benzo(a)pyrene in the bluegill sunfish *(Lepomis macrochirus). Aquat Toxicol* 10: 41-57.

Johnsen S, Kukkonen J, Grande M (1989) Influence of natural aquatic humic substances on the bioavailability of benzo(a)pyrene to Atlantic salmon. *Sci Total Environ* 81/82: 691-702.

Johnson BT, Long ER (1998) Rapid toxicity assessment of sediment from estuarine ecosystems: A new tandem *in vitro* testing approach. *Environ Toxicol Chem* 17: 1099-1106.

Jongeneelen FJ, Scheepers PT, Groenendijk A, Van Aerts LA, Anzion RB, Bos RP, Veenstra SJ (1988) Airborne concentrations, skin contamination, and urinary metabolite excretion of polycyclic aromatic hydrocarbons among paving workers exposed to coal tar derived road tars. *Am Ind Hyg Assoc J* 49: 600-607.

Jonker MTO, Koelmans AA (2001) Polyoxymethylene solid phase extraction as a partitioning method for hydrophobic organic chemicals in sediment and soot. *Environ Sci Technol* 35: 3742-3748.

Jonsson G, Bechmann RK, Bamber SD, Baussant T (2004) Bioconcentration, biotransformation, and elimination of polycyclic aromatic hydrocarbons in sheepshead minnows (*Cyprinodon variegatus*) exposed to contaminated seawater. *Environ Toxicol Chem* 23: 1538-1548.

Juhasz AL, Naidu R (2000) Bioremediation of high molecular weight polycyclic aromatic hydrocarbons: a review of the microbial degradation of benzo(a)pyrene. *Intern Biodet Biodeg* 45: 57-88.

Kaag NHBM, Foekema EM, Scholten MCT, Van Straalen NM (1997) Comparison of contaminant accumulation in three species of marine invertebrates with different feeding habits. *Environ Toxicol Chem* 16: 837-842.

Kadar R, Nagy K, Fremstad D (1980) Determination of polycyclic aromatic hydrocarbons in industrial waste water at the ng/ml. level. *Talanta* 27: 227-303.

Kagan J, Kagan ED, Kagan IA, Kagan PA, Quigley S (1985) The phototoxicity of non-carcinogenic polycyclic aromatic hydrocarbons in aquatic organisms. *Chemosphere* 14: 1829-1834.

Kagan J, Kagan ED, Kagan IA, Kagan PA (1987) Do polycyclic aromatic hydrocarbons, acting as photosensitizers, participate in the toxic effects of acid rain? In: *Photochemistry of environmental aquatic systems* (Zika RG, Cooper WJ, eds). ACS Symposium Series 327. Washington, DC: American Chemical Society: 191-204.

Kalaitzoglou M, Samara C (2006) Gas/particle partitioning and yield levels of polycyclic aromatic hydrocarbons and *n*-alkanes in the mainstream cigarette smoke of commercial cigarette brands. *Food Chemical Toxicol* 44: 1432-1442.

Kamens RM, Guo Z, Fulcher JN, Bell DA (1988) Influence of humidity, sunlight, and temperature on the daytime decay of polyaromatic hydrocarbons on atmospheric soot particles. *Environ Sci Technol* 22: 103-108.

Kamens RM, Zhishi G, Fulcher JN, Bell DA (1991) The influence of humidity on the daytime decay of PAH on atmospheric soot particles. In: *Polynuclear aromatic hydrocarbons: Measurement, means and metabolism* (Cook M, Loening K, Merritt J, eds), Battelle Press, Columbus, Ohio: 435-451.

Kan CA, Traag WA, Hoogenboom LAP (2003) *Voorkomen van PAK's in voer, omgeving van dieren, melk- en zuivelproducten alsmede een oriënterende studie in melkvee.* Report 03/0027745, Animal Sciences Group, Wageningen. In Dutch.

Kanaly RA, Harayama S (2000) Biodegradation of high-molecular-weight polycyclic aromatic hydrocarbons by bacteria. *J Bacteriol* 182: 2059-2067.

Kane Driscoll S, Landrum PF (1997) A comparison of equilibrium partitioning and critical body residue approaches for predicting toxicity of sediment-associated fluoranthene to freshwater amphipods. *Environ Toxicol Chem* 16: 2179-2186.

Kane Driscoll S, Harkey GA, Landrum PF (1997a) Accumulation and toxicokinetics of fluoranthene in sediment bioassays with freshwater amphipods. *Environ Toxicol Chem* 16: 742-753.

Kane Driscoll S, Landrum PF, Tigue E (1997b) Accumulation and toxicokinetics of fluoranthene in water-only exposures with freshwater amphipods. *Environ Toxicol Chem* 16: 754-761.

Kane Driscoll S, Linda B, Schaffner C, Dickhut RM (1998) Toxicokinetics of fluoranthene to the amphipod, *Leptocherius plumulosus* in water-only and sediment exposures. *Mar Environ Res* 45: 269-284.

Karickhoff, SW (1981) Semi-empirical estimation of sorption of hydrophobic pollutants on natural sediments and soil. *Chemosphere* 10: 883-846.

Karickhoff SW, Brown DS, Scott TA (1979) Sorption of hydrophobic pollutants on natural sediments. *Water Res* 13: 241-248.

Kästner M, Breuer-Jammali M, Mahro B (1994) Enumeration and characterization of the soil microflora from hydrocarbon-contaminated soil sites able to mineralize polycyclic aromatic hydrocarbons (PAH). *Appl Microbiol Biotechnol* 41: 267-273.

Kästner M, Streibich S, Beyrer M, Richnow HH, Fritsche W (1999) Formation of bound residues during microbial degradation of [^{14}C]anthracene in soil. *Appl Environ Microbiol* 65: 1834-1842.

Kauss PB, Hutchinson TC (1975) The effects of water-soluble petroleum components on the growth of *Chlorella vulgaris* Beijerinck. *Environ Pollut* 9: 157-174.

Kirchmann H, Åström H, Jönsäll G (1991) Organic pollutants in sewage sludge 1. Effect of toluene, naphthalene, 2-methylnaphthalene, 4-*n*-nonylphenol and di-2-ethylhexyl phthalate on soil biological processes and their decomposition in soil. *Swed J Agric Res* 21: 107-113.

Kireeva IS (1968) On the carcinogenic properties of coal pitch and petroleum bitumen used as bonding agents in coal briquettes. *Gig Sanit* 33: 35-41.

Koelmans AA, Jonker MTO, Cornelissen G, Bucheli TD, Van Noort PCM, Gustafsson O (2006) Black carbon: The reverse of its dark side. *Chemosphere* 63: 365-377.

Korte F, Freitag D, Geyer H (1978) Ecotoxicologic profile analysis. A concept for establishing ecotoxicologic priority lists for chemicals. *Chemosphere* 7: 79-102.

Kroese ED, Muller JJA, Mohn GR, Dortant PM, Wester PW (2001) *Tumorigenic effects in Wistar rats orally administered benzo(a)pyrene for two years (gavage studies). Implication for human cancer risks associated with oral exposure to polycyclic aromatic hydrocarbons.* RIVM Report No. 658603010. National Institute for Public Health and the Environment (RIVM), Bilthoven, The Netherlands.

Kukkonen J, Landrum PF (1994) Toxicokinetics and toxicity of sediment-associated pyrene to *Lumbriculus variegatus* (Oligochaeta). *Environ Toxicol Chem* 13: 1457-1468.

Kukkonen J, Landrum PF (1995) Measuring assimilation efficiencies for sediment-bound PAH and PCB congeners by benthic organisms. *Aquat Toxicol* 32: 75-92.

Lafontaine M, Limasset JC, Truy S (1987) Exposure to polycyclic aromatic hydrocarbons in the coal briquetting industry. *Cahiers de notes documentaires Securite et Hygiene du travail* 126: 57-62.

Lafontaine M, Payan JP, Delsaut P, Morele Y (2000) Polycyclic aromatic hydrocarbon exposure in an artificial shooting target factory: assessment of 1-hydroxypyrene urinary excretion as a biological indicator of exposure. *Ann Occup Hyg* 44: 89-100.

Lamoureux EM, Brownawell B (2004) Influence of soot on hydrophobic organic contaminant desorption and assimilation efficiency. *Environ Toxicol Chem* 23: 2571-2577.

Lampi MA, Gurska J, McDonald KIC, Xie F, Huang X-D, Dixon DG, Greenberg BM (2006) Photoinduced toxicity of polycyclic aromatic hydrocarbons to *Daphnia magna*: ultraviolet-mediated effects and the toxicity of polycyclic aromatic hydrocarbon photoproducts. *Environ Toxicol Chem* 25: 1079-1087.

Landrum PF (1982) Uptake, depuration and biotransformation of anthracene by the scud *Pontoporeia hoyi. Chemosphere* 11: 1049-1057.

Landrum PF, Scavia D (1983) Influence of sediment on anthracene uptake, depuration, and biotransformation by the amphipod *Hyalella azteca. Can J Fish Aquat Sci* 40: 298-305.

Landrum PF (1988) Toxicokinetics of organic xenobiotics in the amphipod *Pontoporeia hoyi*: Role of physiological and environmental variables. *Aquat Toxicol* 12: 245-271.

Landrum PF, Poore R (1988) Toxicokinetics of selected xenobiotics in *Heagenia limbata. J Great Lakes Res* 14: 427-437.

Landrum P, Eadie B, Faust W (1992) Variation in the bioavailability of polycyclic aromatic hydrocarbons to the amphipods *Diporeia* (spp.) with sediment aging. *Environ Toxicol Chem* 11: 1197-1208.

Landrum PF, Lotufo GR, Gossiaux DC, Gedeon ML, Lee J-H (2003) Bioaccumulation and critical body residue of PAHs in the amphipod, *Diporeia* spp.: Additional evidence to support toxicity additivity for PAH mixtures. *Chemosphere* 51: 481-489.

Larson BA (1978) Occupational exposure to coal tar pitch volatiles at pipeline protective coating operations. *Am Ind Hyg Assoc J* 39: 250-255.

Laurén DJ, Rice S (1985) Significance of active and passive depuration in the clearance of naphthalene from the tissues of *Hemigrapsus nudus* (Crustacea: Decapoda). *Mar Biol* 88: 135-142.

Lech JJ, Vodicnik MJ (1985) Biotransformation. In: *Fundamentals of Aquatic Toxicology; Methods and Applications.* (Rand GM, Petrocelli SR, eds) Hemisphere Publishing Corporation, New York, USA: 526-557.

Lemaire P, Den Besten PJ, O'Hara SCM, Livingstone DR (1993) Comparative metabolism of benzo(a)pyrene by microsomes of hepatopancreas of the shore crab *Carcinus maenas* L., and digestive gland of the common mussel *Mytilus edulis* L. *Polycycl Aromat Comp* 3(suppl): 1133-1140.

Leppänen M (1995) The role of feeding behaviour in bioaccumulation of organic chemicals in benthic organisms. *Ann Zool Fennici* 32: 247-255.

Leslie TJ, Dickson KL, Jordan JA, Hopkins DW (1987) Effects of suspended solids on the water column biotransformation of anthracene. *Arch Environ Contam Toxicol* 16: 637-642.

Leversee GJ, Landrum PJ, Bartell S, Gerould S, Bruno M, Spacie A, Bowling J, Haddock J, Fannin T (1981) Disposition of benzo*(a)*pyrene in aquatic system components: periphyton, chironomids, daphnia, fish. In: *Polynuclear aromatic hydrocarbons: Chemical analysis and biological fate.* (Cooke M, Dennis AJ, eds) , Battelle Press, Columbus, Ohio: 357-367.

Levin JO, Rhen M, Sikstrom E (1995) Occupational PAH exposure: urinary 1-hydroxypyrene levels of coke oven workers, aluminium smelter pot-room workers, road pavers, and occupationally non-exposed persons in Sweden. *Sci Total Environ* 163: 169-177.

Leyval C, Binet P (1998) Effect of polyaromatic hydrocarbons in soil on arbuscular mycorrhizal plants. *J Environ Qual* 27: 402-407.

Linder G, Bergman H, Meyer J (1985) Anthracene bioconcentration in rainbow-trout during single-compound and complex-mixture exposures. *Environ Toxicol Chem* 4: 549-558.

Lireko (2002) A clay pigeon, really made of clay! http://www.lireko.nl/lirekoenglish.html.

Liu N, Wang Z, Dong D, Chen K, Qin L (1997) Cancer mortality among carbon workers in China: Retrospective cohort study. *J Occup Health* 39: 325-330.

Lohmann R, Burgess RM, Cantwell MG, Ryba SA, MacFarlane JK, Gschwend PM (2004) Dependency of polychlorinated biphenyl and polycyclic aromatic hydrocarbons bioaccumulation in *Mya arenaria* on both water column and sediment bed chemical activities. *Environ Toxicol Chem* 23: 2551-2562.

Loibner AP, Szolar OHJ, Braun R, Hirmann D (2004) Toxicity testing of 16 priority polycyclic aromatic hydrocarbons using lumistox®. *Environ Toxicol Chem* 23: 557-564.

Lotufo GR (1997) Toxicity of sediment-associated PAHs to an estuarine copepod: Effects on survival, feeding, reproduction and behavior. *Mar Environ Res* 44: 149-166.

Lotufo GR (1998) Lethal and sublethal toxicity of sediment-associated fluoranthene to benthic copepods: application of the critical-body-residue approach. *Aquat Toxicol* 44: 17-30.

Lotufo GR, Fleeger JW (1996) Toxicity of sediment-associated pyrene and phenanthrene to *Limnodrilus hoffmeisteri* (Oligochaeta: Tubificidae). *Environ Toxicol Chem* 15: 1508-1516.

Lutz S, Feidt C, Monteau F, Rychen G, Le Bizec B, Jurjanz S (2006) Effect of exposure to soil-bound polycyclic aromatic hydrocarbons on milk contaminations of parent compounds and their monohydroxylated metabolites. *J Agric Food Chem* 54: 263-268.

Lyons BP, Pascoe CK, McFadzen IRB (2002) Phototoxicity of pyrene and benzo(a)pyrene to embryo-larval stages of the pacific oyster *Crassostrea gigas*. *Mar Environ Res* 54: 627-631.

Ma W, Van Kleunen A, Immerzeel J, De Maagd PG (1998) Bioaccumulation of polycyclic aromatic hydrocarbons by earthworms: assessment of the equilibrium partitioning theory in *in situ* studies and water experiments. *Environ Toxicol Chem* 17: 1730-1737.

Machado ML, Beatty PW, Fetzer JC, Glickman, AH, McGinnis EL (1993) Evaluation of the relationship between PAH content and mutagenic activity of fumes from roofing and paving asphalts and coal tar pitch. *Fundam Appl Toxicol* 21: 492-499.

Mackay D, Shiu WY, Ma KC (1992) *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate of Organic Chemicals.* Lewis Publishers, Boca Raton, FL.

Mackay D, Shiu WY, Ma KC (2000) *Physical-Chemical Properties and Environmental Fate Handbook*, Chapman & Hall.

MacKenzie KM, Angevine DM (1981) Infertility in mice exposed *in utero* to benzo(a)pyrene. *Biol Reprod* 24: 183-191.

Main HH, Friedlander SK (1990) Dry deposition of atmospheric aerosols by dual tracer method, part I. point source. *Atmos Environ* 24A: 103-108.

Malkin R, Kiefer M, Tolos W (1996) 1-Hydroxypyrene levels in coal-handling workers at a coke oven. *J Occup Environ Med* 38: 1141-1144.

McCarthy JF, Jimenez BD (1985) Reduction in bioavailability to Bluegills of polycyclic aromatic hydrocarbons bound to dissolved humic material. *Environ Toxicol Chem* 4: 511-521.

McCarthy JF, Jimenez BD, Barbee T (1985) Effect of dissolved humic material on accumulation of polycyclic aromatic hydrocarbons: Structure-activity relationships. *Aquat Toxicol* 7: 15-24.

McElroy AE, Sisson JD (1989) Trophic transfer of benzo(a)pyrene metabolites between benthic marine organisms. *Mar Environ Res* 28: 265-270.

McGillivray AR, Shiaris MP (1994) Microbial ecology of polycyclic aromatic hydrocarbon (PAH) degradation in coastal sediments. In: *Biological Degradation and Bioremediation of Toxic Chemical* (Chaudry GR, ed), Chapman & Hall, Portland, Oregon: 125–147.

McLeese DW, Burridge LE (1987) Comparative accumulation of PAHs in four marine invertebrates. In: *Oceanic processes in marine pollution* (Capuzzo IM, Kester DR, eds) , Kirger RE, Malabar, Florida: 109-118.

McLeod PM, Van den Heuvel-Greve MJ, Allen-King RM, Luoma SN, Luthy RG (2004) Effects of particulate carbonaceous matter on the bioavailability of benzo(a)pyrene and 2,2',5,5'-tetrachlorobiphenyl to the clam, *Macoma balthica. Environ Sci Technol* 38: 4549-4556.

McVetty B, Hites R (1988) Atmospheric deposition of polycyclic aromatic hydrocarbons to water surfaces: A mass balance approach. *Atmos Environ* 22: 511-536.

Meador JP (2003) Bioaccumulation of PAHs in marine invertebrates. In: *PAHs: An Ecotoxicological Perspective* (Douben PET, ed), Wiley: 147-172.

Meador JP, Stein JE, Reichert WL, Varanasi U (1995) A review of bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. *Rev Environ Contam Toxicol* 143: 79-165.

Meharg AA, Wright J, Dyke H, Osborn D (1998) Polycyclic aromatic hydrocarbon (PAH) dispersion and deposition to vegetation and soil following a large scale chemical fire. *Environ Pollut* 99: 29-36.

Melancon MJ, Lech JJ (1978) Distribution and elimination of naphthalene and 2-methylnaphthalene in rainbow trout during short- and long-term exposures. *Arch Environ Contam Toxicol* 7: 207-220.

Melikian AA, Djordjevic MV, Chen S, Richie Jr J, Stellman SD (2007) Effect of delivered dosage of cigarette smoke toxins on the levels of urinary biomarkers of exposure. *Cancer Epidemiol Biomarkers Prev* 16:1408-1415.

Milleman RE, Birge WJ, Black JA, Cushman RM, Daniels KL, Franco PJ, Giddings JM, McCarthy JF, Stewart AJ (1984) Comparative acute toxicity to aquatic organisms of components of coal-derived synthetic fuels. *Trans Am Fisher Soc* 113: 74-85.

Millward RN, Bridges TS, Ghosh U, Zimmerman JR, Luthy RG (2005) Addition of activated carbon sediments to reduce PCB bioaccumulation by polycheate (*Neanthes arenaceodentata*) and an amphipod (*Leptocheirus plumulosus*). *Environ Sci Technol* 39: 2880-2887.

Mitchell RL, Burchett MD, Pulkownik A, McCluskey L (1988) Effects of environmentally hazardous chemicals on the emergence and early growth of selected Australian plants. *Plant Soil* 112: 195-199.

Moermond CTA, Zwolsman JJG, Koelmans AA (2005) Black carbon and ecological factors affect *in situ* biota to sediment accumulation factors for hydrophobic organic compounds in flood plain lakes. *Environ Sci Technol* 39: 3101-3109.

Moles A, Bates S, Rice SD, Korn S (1981) Reduced growth in coho salmon fry exposed to two petroleum components, toluene and naphthalene, in fresh water. *Trans Am Fisher Soc* 110: 430-436.

Moles A, Rice SD (1983) Effects of crude oil and naphthalene on growth, caloric content, and fat content of pink salmon juveniles in seawater. *Trans Am Fisher Soc* 112: 205-211.

Montizaan GK, Kramers PGN, Janus JA, Posthumus R (1989) Integrated criteria document polynuclear aromatic hydrocarbons (PAH): effects of 10 selected compounds. Appendix to RIVM Report no. 75847007. National Institute of Public Health and Environment (RIVM), Bilthoven, the Netherlands.

Moore JW, Ramamoorthy S (1984) *Organic chemicals in natural waters.* Springer Verlag New York Inc, New York.

Moy FE, Walday M (1996) Accumulation and depuration of organic micro-pollutants in marine hard bottom organisms. *Mar Pollut Bull* 33: 56-63.

Munn SJ, Allanou R, Berthault F, De Bruijn J, Musset C, O'Connor S, Pakalin S, Pellegrini G, Scheer S, Vegro S (eds) (2003) European Union Risk Assessment Report Naphthalene, Volume 33. Report nr: EUR 20763 EN, Office for Official Publications of the European Communities, Luxembourg, Luxembourg.

NAEI (2001) *UK Emissions of Air Pollutants 1970 to 1999.* National Atmospheric Emission Inventory (NAEI).

Naes K, Knutzen J, Berglind L (1995) Occurence of PAH in marine organisms and sediments form smelter discharge in Norway. *Sci Total Environ* 163: 93-106.

Naes K, Oug E (1998) The distribution and environmental relationships of polycyclic aromatic hydrocarbons (PAHs) in sediments from Norwegian smelter-affected fjords. *Chemosphere* 36: 561-576.

Naes K, Oug E, Knutzen J (1998) Source and species-dependent accumulation of polycyclic aromatic hydrocarbons (PAHs) in littoral indicator organisms from Norwegian smelter-affected marine waters. *Mar Environ Res* 45: 193-207.

Nam K, Chung N, Alexander M (1998) Relationship between organic matter content of soil and the sequestration of phenanthrene. *Environ Sci Technol* 32: 3785-3788.

NBOSH (2002) Occupational exposure limit values and measures against air contaminants. Ordinance AFS 2000:3. National Board of Occupational Safety and Health (NBOSH), Solna, Sweden.

Neff JM (1979) *Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fate and biological effects.* Applied Science Publishers, London.

Negri E, La Vecchia C (2001) Epidemiology and prevention of bladder cancer. *Eur J Canc Prev* 10: 7-14.

Neiderfellner J, Lenoir D, Matuschek G, Rehfeldt F, Utschick H, Bruggeman R (1997) Description of vapour pressures of polycyclic aromatic hydrocarbons by graph theoretical indices. *Quant Struct-Act Relat* 16: 38-48.

The Netherlands (2008a) Coal Tar Pitch, High Temperature, Risk Assessment Report Human Health (RAR323_0804_HH), National Institute for Public Health and the Environment (RIVM), Bilthoven, the Netherlands.

The Netherlands (2008b) Coal Tar Pitch, High Temperature, Risk Assessment Report Environment (RAR323_2805_ENV), National Institute for Public Health and the Environment (RIVM), Bilthoven, the Netherlands.

The Netherlands (2008c) Coal Tar Pitch, High Temperature, Risk Reduction Strategy Workers (ES/31b/2008), National Institute for Public Health and the Environment (RIVM), Bilthoven, the Netherlands.

Newsted JL, Giesy Jr. JP (1987) Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to *Daphnia magna,* Strauss (Cladocera, Crustacea). *Environ Toxicol Chem* 6: 445-461.

Nieman JKC, Sims RC, Sims JL, Sorensen DL, McLean JE, Rice JA (1999) [^{14}C]pyrene-bound residue evaluation using MIBK fractionation method for creosote-contaminated soil. *Environ Sci Technol* 33: 776-781.

Niimi AJ, Dookran GP (1989) *PAHs and related compounds.* The Handbook of Environmental Chemistry, vol 3, Part J, Springer Verlag, Berlin.

Nikkilä A, Penttinen S, Kukkonen JVK (1999) UV-B induced acute toxicity of pyrene to the water flea *Daphnia magna* in natural freshwaters. *Ecotoxicol Environ Saf* 44: 271-279.

NIOSH (1987) *NIOSH Respirator Decision Logic, DHHS.* National Institute for Occupational Safety and Health (NIOSH) Publication no. 87-108.

NIOSH (2007) *Pocket Guide to Chemical Hazards.* National Institute for Occupational Safety and Health (NIOSH) Publication No. 2005-149.

OECD (1984) *Activated Sludge, Respiration Inhibition Test.* OECD Guideline for the Testing of Chemicals No. 209, Organisation for Economic Co-operation and Development (OECD).

OECD (1995) *Vapour Pressure.* OECD Guideline for the Testing of Chemicals No. 104, Organisation for Economic Co-operation and Development (OECD).

OECD (1996) *Bioconcentration: Flow-through Fish Test.* OECD Guideline for the Testing of Chemicals No. 305, Organisation for Economic Co-operation and Development (OECD).

OECD (2002) *Guidance Document on Aquatic toxicity Testing of Difficult Substances and Mixtures.* OECD Series on Testing and Assessment No. 23, Organisation for Economic Co-operation and Development (OECD).

Oja V, Suuberg EM (1998) Vapor pressures and enthalpies of sublimation of polycyclic aromatic hydrocarbons and their derivatives. *J Chem Eng Data* 43: 486-492.

Olufsen SB (1980) Polynuclear aromatic hydrocarbons in Norwegian drinking water resources. In: *Polynuclear aromatic hydrocarbons: Chemistry and biological effects* (Bjorseth A, Dennis AJ, eds), Battelle Press, Columbus, Ohio: 333-343.

Oris JT, Giesy Jr. JP (1987) The photo-induced toxicity of polycyclic aromatic hydrocarbons to larvae of the fathead minnow (*Pimephales promelas*). *Chemosphere* 16: 1395-1404.

Oris JT, Winner RW, Moore MV (1991) A four-day survival and reproduction test for *Ceriodaphnia dubia*. *Environ Toxicol Chem* 10: 217-224.

OSHA (2007) *Limits for air contaminants-1910.1000 Table Z-1.* Occupational Safety and Health Administration (OSGA) http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=9992.

OSPAR (2000) *Quality Status Report 2000 for the North-East Atlantic.* OSPAR Commission.

OSPAR (2001) *Polycyclic Aromatic Hydrocarbons (PAHs)* Priority Substances Series. OSPAR Commission.

OSPAR (2002) *Emissions and Discharges of Polycyclic Aromatic Hydrocarbons (PAHs) from Primary Aluminium Electrolysis Soederberg Technology.* Hazardous Substances Series. OSPAR Commission.

Ott FS, Harris RP, O'Hara SCM (1978) Acute and sublethal toxicity of naphthalene and three methylated derivatives to the estuarine copepod, *Eurytemora affinis*. *Mar Environ Res* 1: 49-58.

Øvrebø S, Haugen A, Fjeldstad, PE, Hemminki K, Szyfter K (1994) Biological monitoring of exposure to polycyclic aromatic hydrocarbons in an electrode paste plant. *J Occup Med* 36: 303-310.

Palmork KH, Solbakken JE (1981) Distribution and elimination of [9-^{14}C]phenanthrene in the horse mussel (*Modiola modiolus*). *Bull Environ Contam Toxicol* 26: 196-201.

Pankow JF (1987) Review and comparative analysis of the theories on portioning between the gas and aerosol particulate phases in the atmosphere. *Atmos Environ* 21: 2275-2283.

Park KS, Sims RC, Dupont RR, Doucette WJ, Matthews JE (1990) Fate of PAH compounds in two soil types: Influence of volatilization, abiotic loss and biological activity. *Environ Toxicol Chem* 9: 187-195.

Parkerton TF (1993) Estimating toxicokinetic parameters for modeling the bioaccumulation of non-ionic organic chemicals, PhD Thesis, Rutgers University, New Brunswick, New Jersey.

Partanen T, Boffetta P (1994) Cancer risk in asphalt workers and roofers: review and meta-analysis of epidemiologic studies. *Am J Ind Med* 26: 721-740.

Patton JF, Dieter MP (1980) Effect of petroleum hydrocarbons on hepatic function in the duck. *Comp Physiol Biochem* 65: 33-36.

Peachey RL, Crosby DG (1996) Phototoxicity in tropical reef animals. *Mar Environ Res* 42: 359-362.

Pechiney (2002) *Milieujaarverslag 2001.* (Environmental report 2001). In Dutch.

Pelletier MC, Burgess RM, Ho KT, Kuhn A, McKinney RA, Ryba SA (1997) Phototoxicity of individual polycyclic aromatic hydrocarbons and petroleum to marine invertebrate larvae and juveniles. *Environ Toxicol Chem* 16: 2190-2199.

Peters J, Seifert B (1980) Losses of benzo(a)pyrene under conditions of high volume sampling. *Atmos Environ* 14: 117-119.

Petersen GI, Kristensen P (1998) Bioaccumulation of lipophilic substances in fish early life stages. *Environ Toxicol Chem* 17: 1385-1395.

Petry T, Schmid P, Schlatter C (1996a) Airborne exposure to polycyclic aromatic hydrocarbons (PAHs) and urinary excretion of 1-hydroxypyrene of carbon anode plant workers. *Ann Occup Hyg* 40: 345-357.

Petry T, Schmid P, Schlatter C (1996b) The use of toxic equivalency factors in assessing occupational and environmental health risk associated with exposure to airborne mixtures of polycyclic aromatic hydrocarbons (PAHs). *Chemosphere* 32: 639-648.

Pitts JN, Paur H-R, Zielinska B, Arey J, Winer AM, Ramdahl T, Mejia V (1986) Factors influencing the reactivity of polycyclic hydrocarbons adsorbed on filters and ambient POM with ozone. *Chemosphere* 15: 675-685.

Poerschmann J, Kopinke F (2001) Sorption of very hydrophobic organic compounds (VHOCs) on dissolved humic organic matter (DOM). 2. Measurement of sorption and application of a Flory-Huggins concept to interpret the data. *Environ Sci Technol* 35: 1142-1148.

PORG (1997) *Ozone in the United Kingdom.* Fourth report of the Photochemical Oxidants Review Group (PORG). Department of the Environment, Transport and the Regions, Air and Environmental Quality Division, London, UK.

Preuss B, Roßbach B, Müller J, Angerer J (2003) Eine deutschlandweite Studie zur inneren und äußeren Exposition gegenüber polycyclischen aromatischen Kohlenwasserstoffen (PAK) am Arbeitsplatz. [A Germany-wide study of internal and external exposure to polycyclic aromatic hydrocarbons (PAH) at the workplace]. *Gefahrstoffe-Reinhaltung der Luft* 63: 7-13.

Randem BG, Burstyn I, Langård S, Svane O, Järvholm B, Kauppinen T, Bergdahl IA, Johansen C, Hansen J, Partanen T, Kromhout H, Ferro G, Boffetta P (2004) Cancer incidence of Nordic asphalt workers. *Scand J Work Environ Health* **30**: 350-355.

Ren L, Zeiler LF, Dixon DG, Greenberg BM (1996) Photoinduced effects of polycyclic aromatic hydrocarbons on *Brassica napus* (Canola) during germination and early seedling development. *Ecotoxicol Environ Saf* 33: 73-80.

Richards DJ, Shieh WK (1986) Biological fate of organic priority pollutants in the aquatic environment. *Water Res* 20: 1077-1190.

Richardson BJ, Tse ES-C, De Luca-Abbott SB, Martin M, Lam PKS (2005) Uptake and depuration of PAHs and chlorinated pesticides by semi-permeable membrane devices (SPMDs) and green-lipped mussels (*Perna viridis*). *Mar Pollut Bull* 51: 975-993.

Riley RT, Shirazi MA, Swartz RC (1981) Transport of naphthalene in the oyster *Ostrea edulis. Mar Biol* 63: 325-330.

Rogerson A, Shiu WY, Huang GL, Mackay D, Berger J (1983) Determination and interpretation of hydrocarbon toxicity to ciliate protozoa. *Aquat Toxicol* 3: 215-228.

Romundstad P, Andersen A, Haldorsen T (2000a) Cancer incidence among workers in six Norwegian aluminium plants. *Scand J Work Environ Health* 26: 461-469.

Romundstad P, Haldorsen T, Andersen A (2000b) Cancer incidence and cause specific mortality among workers in Two Norwegian aluminium reduction plants. *Am J Indus Med* 37: 175-183.

Ronneberg A, Langmark F (1992) Epidemiologic evidence of cancer in aluminium reduction plant workers. *Am J Ind Med* 22: 573-590.

Ronneberg A, Andersen A (1995) Mortality and cancer morbidity in workers from an aluminium smelter with prebaked carbon anodes--Part II: Cancer morbidity. *Occup Environ Med* 52: 250-254.

Ronneberg A, Haldorsen T, Romundstad PR, Andersen A (1999) Occupational exposure and cancer incidence among workers from an aluminium smelter in western Norway. *Scand J Work Environ Health* 25: 207-214.

Rossi SS, Neff JM (1978) Toxicity of polynuclear aromatic hydrocarbons to the polychaete *Neanthes arenaceodentata*. *Mar Pollut Bull* 9: 220-223.

Routschka G, Granitzki KE (2002) REFRACTORY CERAMICS-Types of refractory ceramics. In: *Ullmann's Encyclopedia of Industrial Chemistry*. Wiley-VCH Verlag GmbH, Weinheim, Germany.

Rust AJ, Burgess RM, McElroy AE, Cantwell MG, Brownawell BJ (2004a) Influence of soot carbon on the bioaccumulation of sediment-bound polycyclic aromatic hydrocarbons by marine benthic invertebrates: An interspecies comparison. *Environ Toxicol Chem* 23: 2594-2603.

Rust AJ, Burgess RM, McElroy AE, Cantwell MG, Brownawell BJ (2004b) Role of source in the bioavailability of polycyclic aromatic hydrocarbons to deposit-feeding benthic invertebrates. *Environ Toxicol Chem* 23: 2604-2610.

Rütgers VFT (1997) *Wasserverunreinigung durch Flüssigpechkontamination.*

Rütgers VFT (1999a) *Untersuchung zur Extrahierbarkeit von EPA-PAK's aus Pechstaub.*

Rütgers VFT (1999b) *Untersuchung zur Extrahierbarkeit von EPA-PAK's aus Pechstaub. Fortsetzung der Versuche des Zwischenberichts vom 23.2.99.*

Rütgers VFT (2000) *Extrahierbarkeit von EPA-PAK's aus granuliertem Steinkohlteerpech (Bx95, CAS-Nr. 65996-93-2).*

Ruus A, Bøyum O, Grung M, Naes K (2007) *Particle affinity and bio-availability of PAHs associated with coal tar pitch.* NIVA report O-26066. Norwegian Institute for Water Research (NIVA), Oslo, Norway.

Sabljic A, Gusten H, Verhaar H, Hermens J (1995) QSAR modelling of soil sorption. Improvements and systematics of log K_{OC} vs. log K_{OW} correlations. *Chemosphere* 31: 4489-4514

Sæthre LJ, Falk-Petersen I-B, Sydnes LK, Lřnnig S, Naley AM (1984) Toxicity and chemical reactivity of naphthalene and methylnaphthalenes. *Aquat Toxicol* 5: 291-306.

Sanborn HR, Malins DC (1977) Toxicity and metabolism of naphthalene: A study with marine larval invertebrates. *Proc Soc Exp Biol Med* 154: 151-155.

Sauter DH, Reimert R (2002) COAL-Agglomeration. In: *Ullmann's Encyclopedia of Industrial Chemistry*, Wiley-VCH Verlag GmbH, Weinheim, Germany.

Schimberg RW, Pfaffli P, Tossavainen A (1980) Polycyclic aromatic hydrocarbons in foundries. *J Toxicol Environ Health* 6: 1187-1194.

Schipper IA (1961) Toxicity of wood preservatives for swine. *Am J Vet Res* 22: 401-405.

Schnell JV, Gruger EH, Malins DC (1980) Monooxygenase activities of coho salmon (*Oncorhynchus mykiss*) liver microsomes using three polycyclic aromatic hydrocarbons substrates. *Xenobiotica* 10: 229-234.

Schulte A, Ernst H, Peters L, Heinrich U (1994) Induction of squamous cell carcinomas in the mouse lung after long-term inhalation of polycyclic aromatic hydrocarbon-rich exhausts. *Exp Toxicol Pathol* 45: 415-421.

SCOEL (2007) Scientific Committee on Occupational Exposure Limits. http://ec.europa.eu/employment_social/health_safety/occupational_en.htm.

Sheu H-L, Lee W-J, Su C-C, Choa H-R, Fan Y-C (1996) Dry deposition of polycyclic aromatic hydrocarbons in ambient air. *J Environ Eng* 122: 1101-1109.

Shootingworld (2002) *Target Production Factory.* http:www.shootingworld.com.

Sijm DTHM, Opperhuizen A (1989) Biotransformation of organic chemicals by fish: enzyme activities and reactions. In: *Handbook of Environmental Chemistry Reactions and Processes*, Volume 2E (Hutzinger, O, ed.), Springer, Berlin, Germany: 163-235.

Sim M, Benke G (2003) World at work: Hazards and controls in aluminium potrooms. *Occup Environ Med* 60: 989-992.

Sims RM, Overcash MR (1983) Fate of polynuclear aromatic compounds (PNAs) in soil-plant systems. *Res Rev* 88: 1-68.

Sjardin M (2003) *CO_2 Emission Factors for Non-Energy Use in the Non-Ferrous Metal, Ferroalloys and Inorganics Industry.* Copernicus Institute, Department of Science, Technology and Society, University of Utrecht, Utrecht, The Netherlands.

Skarphéðinsdóttir H, Ericson G, Dalla Zuanna L, Gilek M (2003) Tissue differences, dose-response relationship and persistence of DNA adducts in blue mussels (*Mytilus edulis* L.) exposed to benzo(a)pyrene. *Aquat Toxicol* 62: 165-177.

Slooff W, Janus JA, Matthijsen AJCM, Montizaan GK, Ros JPM (1989) *Integrated Criteria Document PAHs.* RIVM report nr. 758474011. National Institute for Public Health and the Environment (RIVM), Bilthoven, the Netherlands.

Smith SB, Savino JF, Blouin MA (1988) Acute toxicity to *Daphnia pulex* of six classes of chemical compounds potentially hazardous to Great Lakes aquatic biota. *J Great Lakes Res* 14: 394-404.

Social Economic Council (2007) http://www.ser.nl.

Solorzano MF, Barcon OD, Lopez ME (1993) *Informe sobre caracterizacion de residuos (residuo: brea).* (1/13/0578 no 2). Anencia del Medo Ambiente.

Southworth GR (1979) The role of volatilization in removing aromatic hydrocarbons from aquatic environments. *Bull Environ Contam Toxicol* 21: 507-514.

Southworth GR, Beauchamp JJ, Schmieder PK (1978) Bioaccumulation potential of polycyclic aromatic hydrocarbons in *Daphnia pulex. Water Res* 12: 973-977.

Spacie A, Landrum PF, Leversee GJ (1983) Uptake, depuration, and biotransformation of anthracene and benzo(a)pyrene in bluegill sunfish. *Ecotoxicol Environ Saf* 7: 330-341.

Spehar RL, Poucher S, Brooke LT, Hansen DJ, Champlin D, Cox DA (1999) Comparative toxicity of fluoranthene to freshwater and saltwater species under fluorescent light. *Arch Environ Contam Toxicol* 37: 496-502.

Spinelli JJ, Band PR, Svirchev LM, Gallagher RP (1991) Mortality and cancer incidence in aluminium reduction plant workers. *J Occup Med* 33: 1150-1155.

Springer DL, Poston KA, Mahlum DD, Sikov MR (1982) Teratogenicity following inhalation exposure of rats to a high-boiling coal liquid. *J Appl Toxicol* 2: 260-264.

Springer DL, Hackett PL, Miller RA, Buschbom RL (1986a) Lung development and postnatal survival for rats exposed *in utero* to a high-boiling coal liquid. *J Appl Toxicol* 6: 129-133.

Springer DL, Miller RA, Weimer WC, Ragan HA, Buschbom RL, Mahlum DD (1986b) Effects of inhalation exposure to a high-boiling (288 to 454 °C) coal liquid. *Toxicol Appl Pharmacol* 82: 112-131.

Springer DL, Miller RA, Wright CW, Ragan HA, Buschbom RL, Mahlum DD (1987) Effects of subchronic inhalation exposure of mice to a high-boiling coal liquid. *Fund Appl Toxicol* 9: 227-286.

SRC (2004). Interactive PhysProp Database at URL: Demo http://www.syrres.com/esc/physdemo.htm. Syracuse Research Corporation (SRC).

Steinhauser KG (1997) *Bewertung wassergefahrender Stoffe. Stoffname: Seinkohlenteerpech mit Erweichungspunkt > 80 °C, geschmolzen oder stuckig (Korngröße <1cm).* Datenblatt-Nr: 1446.

Stellman JM (ed) (1998a) *Guide to Chemicals: Properties of the Polyaromatic Hydrocarbons. Encyclopaedia of Occupational Health and Safety. Vol. III.* Chp 84. 4th ed. Geneva, International Labour Office.

Stellman JM (ed) (1998b) *Guide to Chemicals: Properties of the Polyaromatic Hydrocarbons. Encyclopaedia of Occupational Health and Safety. Vol. III.* Chp 74. 4th ed. Geneva, International Labour Office.

Stern FB, Ruder AM, Chen G (2000) Proportionate mortality among unionized roofers and waterproofers. *Am J Ind Med* 37: 478-492.

Stewart KM, Thompson RS (1995) Fluoranthene as a model toxicant in sediment studies with *Chironomus riparius*. *J Aquat Ecosyst Health* 4: 231-238.

Stoye D, Funke D, Hoppe L, Hasselkus J, Curtis LG, Hoehne K, Zech H-J, Heiling P (2002) PAINTS AND COATINGS-Types of Paints and Coatings (Binders). In: *Ullmann's Encyclopedia of Industrial Chemistry*, Wiley-VCH Verlag GmbH, Weinheim, Germany.

Suedel BC, Rodgers Jr. JH, Clifford PA (1993) Bioavailability of fluoranthene in freshwater sediment toxicity tests. *Environ Toxicol Chem* 12: 155-165.

Suedel BC, Boraczek JA, Peddicord RK, Cliffort PA, Dillon TM (1994) Trophic transfer and biomagnification potential of contaminants in aquatic ecosystems. *Rev Environ Contam Toxicol* 136: 21-89.

Suedel BC, Rodgers Jr. JH (1996) Toxicity of fluoranthene to *Daphnia magna*, *Hyalella azteca*, *Chironomus tentans*, and *Stylaria lacustris* in water-only and whole sediment exposures. *Bull Environ Contam Toxicol* 57: 132-138.

Sundelin B, Wiklund A-KE, Lithner G, Gustafsson Ö (2004) Evaluation of the role of black carbon in attenuating bioaccumulation of polycyclic aromatic hydrocarbons from field-contaminated sediments. *Environ Toxicol Chem* 23: 2611-2617.

Suzuki J, Hagino T, Suzuki S (1987) Formation of 1-nitropyrene by photolysis of pyrene in water containing nitrite ion. *Chemosphere* 16: 859-867.

Sverdrup LE, Kelley AE, Krogh PH, Nielsen T, Jensen J, Scott-Fordsmand JJ, Stenersen J (2001) Effects of eight polycyclic aromatic compounds on the survival and reproduction of the springtail *Folsomia fimetaria* L. (Collembola, Isotomidae). *Environ Toxicol Chem* 20: 1332-1338.

Sverdrup LE, Ekelund F, Krogh PH, Nielsen T, Johnson K (2002a) Soil microbial toxicity of eight polycyclic aromatic compounds: Effects on nitrification, the genetic diversity of bacteria, and the total number of protozoans. *Environ Toxicol Chem* 21: 1644-1650.

Sverdrup LE, Jensen J, Kelley AE, Krogh PH, Stenersen J (2002b). Effects of eight polycyclic aromatic compounds on the survival and reproduction of *Enchytraeus crypticus* (Oligochaeta, Clitellata). *Environ Toxicol Chem* 21: 109-114.

Sverdrup LE, Jensen J, Krogh PH, Stenersen J (2002c) Studies on the effect of soil aging on the toxicity of pyrene and phenanthrene to a soil-dwelling springtail. *Environ Toxicol Chem* 21: 489-492.

Sverdrup LE, Krogh PH, Nielsen T, Stenersen J (2002d) Relative sensitivity of three terrestrial invertebrate tests to polycyclic aromatic compounds. *Environ Toxicol Chem* 21: 1927-1933.

Sverdrup LE, Nielsen T, Krogh PH (2002e) Soil ecotoxicity of polycyclic aromatic hydrocarbons in relation to soil sorption, lipophilicity, and water solubility. *Environ Sci Technol* 36: 2429-2435.

Sverdrup LE, Krogh PH, Nielsen T, Kjær C, Stenersen J (2003) Toxicity of eight polycyclic aromatic compounds to red clover (*Trifolium pratense*), ryegrass (*Lolium perenne*), and mustard (*Sinapsis alba*). *Chemosphere* 53: 993-1003.

Swartz RC, Kemp PF, Schults DW, Lamberson JO (1988) Effects of mixtures of sediment contaminants on the marine infaunal amphipod, *Rhepoxynius abronius*. *Environ Toxicol Chem* 7: 1013-1020.

Swartz RC, Ferraro SP, Lamberson JO, Cole FA, Ozretich RJ, Boese BL, Schults DW, Behrenfeld M, Ankley GT (1997) Photoactivation and toxicity of mixtures of polycyclic aromatic hydrocarbon compounds in marine sediment. *Environ Toxicol Chem* 16: 2151-2157.

Swartz RC, Schults DW, DeWitt TH, Ditsworth GR, Lamberson JO (1990) Toxicity of fluoranthene in sediment to marine amphipods: A test of the equilibrium partitioning approach to sediment quality criteria. *Environ Toxicol Chem* 9: 1071-1080.

Swedish Work Environment Authority (2007) Occupational exposure limit values and measures against air contaminants. AFS 2005:17. http://www.av.se/dokument/inenglish/legislations/eng0517.pdf.

Tadokoro H, Maeda M, Kawashima Y, Kitano M, Hwang D-F, Yoshida T (1991) Aquatic toxicity testing for multicomponent compounds with special reference to preparation of test solution. *Ecotoxicol Environ Saf* 21: 57-67.

Tang JX, Alexander M (1999) Mild extractability and bioavailability of polycyclic aromatic hydrocarbons in soil. *Environ Toxicol Chem* 18: 2711-2714.

Thierrin J, Davis GB, Barber C, Patterson BM, Pribac F, Power TR, Lambert M (1993) Natural degradation rates of BTEX compounds and naphthalene in a sulphate reducing groundwater environment. *Hydrol Sci J* 38: 309-322.

Thomann RV, Connolly JP, Parkerton TF (1992) An equilibrium model of organic chemical accumulation in aquatic food webs with sediment interaction. *Environ Toxicol Chem* 11: 615-629.

Thomann RV, Komlos J (1999) Model of biota-sediment accumulation factor for polycyclic aromatic hydrocarbons. *Environ Toxicol Chem* 18: 1060-1068.

Thrane KE, Mikalsen A (1981) High-volume sampling of airborne polycyclic aromatic hydrocarbons using glass fibre filters and polyurethane foam. *Atmos Environ* 15: 909-918.

Tinfos (2004) Company Information from: http://www.tinfos.no, assessed at 11/06/2004.

Toraason M, Hayden C, Marlow D, Rinehart R, Mathias P, Werren D, Olsen LD, Neumeister CE, Mathews ES, Cheever KL, Marlow KL, DeBord DG, Reid TM (2001) DNA strand breaks, oxidative damage, and 1-OH pyrene in roofers with coal-tar pitch dust and/or asphalt fume exposure. *Int Arch Occup Environ Health* 74: 396-404.

Tremblay C, Armstrong B, Thériault G, Brodeur J (1995) Estimation of risk of developing bladder cancer among workers exposed to coal tar pitch volatiles in the primary aluminium industry. *Am J Ind Med* 27: 335-348.

Trucco RG, Engelhardt FR, Stacey B (1983) Toxicity accumulation and clearance of aromatic hydrocarbons in *Daphnia pulex*. *Environ Pollut Ser A* 31: 191-202.

Trust KA, Fairbrother A, Hooper MJ (1994) Effects of 7,12-dimethylbenz(a]anthracene on immune function and mixed-function oxygenase activity in the European starling. *Environ Toxicol Chem* 13: 821-830.

Tsibulsky V (2001) *Polycyclic Aromatic Hydrocarbons Emission Inventories and Emission Expert Estimates.* MSC-E Contribution to the HM and POP Emission Inventories. Technical Note 7/2001, June 2001. Meteorological Synthesizing Centre-East, Moscow.

UBA (1997) *Untersuchungen zum Elutionsverhalten von Steinkohlenteerpech.* Umweltbundesamt (UBA), Germany.

Ullmann (2000) *Ullmann's Encyclopedia of Industrial Chemistry*, Wiley-VCH Verlag GmbH, Weinheim, Germany.

US-DOE (1997) *Energy and Environmental Profile of the U.S. Aluminium Industry.* U.S. Department of Energy, Office of Industrial Technologies.

US-DOE (2003) *Supporting Industries, Energy and Environmental Profile*. U.S. Department of Energy (DOE), October 2003.

US-EPA (1995) *Compilation of Air Pollutant Emission Factors, AP-42, Fifth Edition, Volume I: Stationary Point and Area Sources*, Chapter 11 Mineral Products Industry, Section 11.5 Refractory Manufacturing. Available at http://www.epa.gov/ttn/chief/ap42.

US-EPA (2001) *Emergency Planning and Community Right-to-know Act-Section 313: Guidance for Reporting Toxic Chemicals: Polycyclic Aromatic Compounds Category.* United States Environmental Protection Agency, Office of Environmental Information, Washington DC, EPA 260-B-01-03.

Valerio F, Antolini E, Lazzarotto A (1987) A model to evaluate half-lives of PAHs naturally occurring on airborne particulate. *Int J Environ Anal Chem* 28: 185-196.

Van Brummelen TC, Stuijfzand SC (1993) Effects of benzo(a)pyrene on survival, growth and energy reserves in the terrestrial isopods *Oniscus asellus* and *Porcellio scaber*. *Sci Total Environ* Suppl 1993: 921-930.

Van Brummelen TC, Van Gestel CAM, Verweij RA (1996a) Long-term toxicity of five polycyclic aromatic hydrocarbons for the terrestrial isopods *Oniscus asellus* and *Porcellio scaber*. *Environ Toxicol Chem* 15: 1199-1210.

Van Brummelen TC, Verweij RA, Wedzinga SA, Van Gestel CAM (1996b) Polycyclic aromatic hydrocarbons in earthworm and isopods from contaminated forest soils. *Chemosphere* 32: 315-341.

Van Delft JHM, Steenwinkel M-JST, Van Asten JG, Van Es J, Kraak A, Baan RA (1998) Monitoring of occupational exposure to polycyclic aromatic hydrocarbons in a carbon-electrode manufacturing plant. *Ann Occup Hyg* 42: 105-114.

Van der Meer JR, De Vos WM, Harayama S, Zehnder AJB (1992) Molecular mechanisms of genetic adaptation to xenobiotic compounds. *Microbiol Rev* 56: 677-694.

Van der Oost R, Heida H, Opperhuizen A, Vermeulen NPE (1991) Bioaccumulation of organic micropollutants in different aquatic organisms: sublethal toxic effects on fish, In: *Aquatic Toxicology and Risk Assessment* (Mayes MA, Baron MG, eds) ASTM STP 1124, vol 14, American Society for Testing and Materials, Philadelphia: 166-180.

Van der Oost R, Beyer J, Vermeulen NPE (2003) Fish bioaccumulation and biomarkers in environmental risk assessment: a review. *Environ Toxicol Pharmacol* 13: 57-149.

Van Harmelen AK, Coenen PWHG, Draaijers GPJ, Leneman H, Nagelhout D, Rood GA, De Vries DJ, Zonneveld EA (1999) *Emissies en afval in Nederland. Jaarrapport 1997 en ramingen 1998.* In Dutch.

Van Hattum B, Leonards P, Burgers I, Van der Horst A (1993) *Biomonitoring of Micropollutants in Foodchain of the Nieuwe Merwede and the Dortsche Biesbosch.* Report E-92/19. Institute for Environmental Studies, Vrije Universiteit, Amsterdam, The Netherlands.

Van Hattum B, Burgers I, Swart K, Van der Horst A, Wegener JW, Leonards P, Rijkeboer M, Den Besten PJ (1996) *Biomonitoring of Micropollutants in Foodchain of Hollandsch Diep, Dortsche Biesbosch and Brabantsche Biesbosch.* Report E-96/12. Institute for Environmental Studies, Vrije Universiteit, Amsterdam, The Netherlands.

Van Hattum B, Burgers I, Swart K, Van der Horst A, Wegener JWM, Den Besten PJ (1998) *Biomonitoring of Micropollutants in Foodchain in the Amer and the Haringvliet.* Report E-98/08. Institute for Environmental Studies, Vrije Universiteit, Amsterdam, The Netherlands.

Van Rooij JG, Bodelier-Bade MM, De Looff AJ, Dijkmans AP, Jongeneelen FJ (1992) Dermal exposure to polycyclic aromatic hydrocarbons among primary aluminium workers. *Med Lav* 83: 519-529.

Van Rooij JG, Bodelier-Bade MM, Jongeneelen FJ (1993) Estimation of individual dermal and respiratory uptake of polycyclic aromatic hydrocarbons in 12 coke oven workers. *Br J Ind Med* 50: 623-632.

Van Rooij JG, Vinke E, De Lange J, Bruijnzeel PL, Bodelier-Bade MM, Noordhoek J, Jongeneelen FJ (1995) Dermal absorption of polycyclic aromatic hydrocarbons in the blood-perfused pig ear. *J Appl Toxicol* 15:193-200.

Van Schooten FJ, Jongeneelen FJ, Hillebrand MJX, Van Leeuwen FE (1995) Polycyclic aromatic hydrocarbon-DNA adducts in white blood cell DNA and 1-hydroxypyrene in the urine from aluminium workers: relation with job category and synergistic effect of smoking. *Cancer Epidemiol Biomarkers Prev* 4: 69-77.

Van Straalen NM, Verweij RA (1991) Effects of benzo(a)pyrene on food assimilation and growth efficiency in *Porcellio scaber* (Isopoda). *Bull Environ Contam Toxicol* 46: 134-140.

Vermeulen NPE, Donné-Op den Kelder G, Commandeur JNM (1992) Formation and protection against toxic reactive intermediates. In: *Perspectives in Medicinal Chemistry* (Testa B, Kyburz E, Fuhrer W, Giger R, eds), Verlag Helvetica Chimica Acta, Basel, Switzerland: 573-593.

Verrhiest G, Clément B, Blake G (2001) Single and combined effects of sediment-associated PAHs on three species of freshwater macroinvertebrates. *Ecotoxicology* 10: 363-372.

Vinturella AE, Burgess RM, Coull BA, Thompson KM, Shine JP (2004) Importance of black carbon in distribution and bioaccumulation models of polycyclic aromatic hydrocarbons in contaminated marine sediments. *Environ Toxicol Chem* 23: 2578-2586.

Vohler O, Nutsch G, Collin G, Von Sturm F, Wege E, Frohs W, Henning K-D, Von Kienle H, Voll M (2002) CARBON-Carbon and Graphite Materials. In: *Ullmann's Encyclopedia of Industrial Chemistry*, Wiley-VCH Verlag GmbH, Weinheim, Germany.

Volkering F, Breure AM (2003) Biodegradation and general aspects of bioavailability. In: *PAHs: An Ecotoxicological Perspective* (Douben PET, ed), Wiley: 81- 98.

Vu Duc T, Huynh CK (1991) Photodecomposition rates of adsorbed PAH mutagenic activity of irradiated synthetic mixtures. In: *Polynuclear aromatic hydrocarbons: Measurement, means and metabolism* (Cook M, Loening K, Merritt J, eds) , Battelle Press, Columbus, Ohio: 979-994.

Wallcave L, Garcia H, Feldman R, Lijinsky W, Shubik P (1971) Skin tumorigenesis in mice by petroleum asphalts and coal-tar pitches of known polynuclear aromatic hydrocarbon content. *Toxicol Appl Pharmacol* 18: 41-52.

Wang, X, Yu X, Bartha R (1990) Effect of bioremediation of polycyclic aromatic hydrocarbon residues in soil. *Environ Sci Technol* 24: 1086-108.

Wang X, Wang WX (2006) Bioaccumulation and transfer of benzo(a)pyrene in a simplified marine foodchain. *Mar Ecol Prog Ser* 312: 101-111.

Wania F, Mackay D (1996) Tracking the distribution of persistent organic pollutants. *Environ Sci Technol* 30: 390-396.

Ward GS, Parrish PR, Rigby RA (1981) Early life stage toxicity test with a saltwater fish: Effects of eight chemicals on survival, growth, and development of sheepshead minnows (*Cyprinodon variegatus*). *J Toxicol Environ Health* 8: 225-240.

Wassenberg DM, Swails EE, Di Guilio RT (2002) Effects of single and combined exposures to benzo(a)pyrene and 3,3'4,4'5-pentachlorobiphenyl on EROD activity and development in *Fundulus heteroclitus*. *Mar Environ Res* 54: 279-283.

WATCH (2000) *Extract from the Minutes of the 17 May meeting of the Working Group on the Assessment of Toxic Chemicals* (WATCH/21/2000).

Watts R, Wallingford K, Williams R, House D, Lewtas J (1998) Airborne exposures to PAH and PM2.5 particles for road paving workers applying conventional asphalt and crumb rubber modified asphalt. *J Expo Anal Environ Epidemiol* 8: 213-229.

Webster L, Fryer RJ, Megginson C, Dalgarno EJ, McIntosh AD, Moffat, CF (2004) The polycyclic aromatic hydrocarbon and geochemical biomarker composition of sediments from sea lochs on the west coast of Scotland. *J Environ Monit* 6: 219-228.

Weinstein JE, Oris JT (1999) Humic acids reduce the bioaccumulation and photoinduced toxicity of fluoranthene to fish. *Environ Toxicol Chem* 18: 2087-2094.

Weinstein JE, Polk KD (2001) Phototoxicity of anthracene and pyrene to glochidia of the freshwater mussel *Utterbackia imbecilis*. *Environ Toxicol Chem* 20: 2021-2028.

Wernersson A-S (2003) Predicting petroleum phototoxicity. *Ecotoxicol Environ Saf* 54: 355-365.

Wernersson A-S, Dave G (1997) Phototoxicity identification by solid phase extraction and photoinduced toxicity to *Daphnia magna*. *Arch Environ Contam Toxicol* 32: 268-273.

Weyand EH, Wu Y, Patel S, Goldstein L (1994) Biochemical effects of manufactured gas plant residue following ingestion by B6C3F1 mice. *J Toxicol Environ Health* 42: 89-107.

White JC, Kelsey JW, Hatzinger PB, Alexander M (1997) Factors affecting sequestration and bioavailability of phenanthrene in soils. *Environ Toxicol Chem* 16: 2040-2045.

WHO (1998) *Selected non-heterocyclic polycyclic aromatic hydrocarbons.* Environmental Health Criteria, 202, International Programme on Chemical Safety (IPCS), World Health Organization, Geneva, Switzerland.

WHO (2000) Polycyclic aromatic hydrocarbons. In: *Air Quality Guidelines for Europe,* 2nd Edition (Theakston F, ed), WHO Regional Office for Europe, Copenhagen, Denmark: 92-96.

Widdows J, Morre SL, Clarke KR, Donkin P (1983) Uptake, tissue distribution and elimination of [1-^{14}C] naphthalene in the mussel *Mytilus edulis*. *Mar Biol* 76: 109-114.

Wilcke W (2000) Polycyclic aromatic hydrocarbons (PAHs) in soil - a review. *J Plant Nutr Soil Sci* 163: 229-248.

Wilcoxen SE, Meier PG, Landrum PF (2003) The toxicity of fluoranthene to *Hyalella azteca* in sediment and water-only exposures under varying light spectra. *Ecotoxicol Environ Saf* 54: 105-117.

Wild SR, Berrow ML, Jones KC (1991) The persistence of polynuclear aromatic hydrocarbons (PAHs) in sewage sludge amended agricultural soils. *Environ Pollut* 72: 141-157.

Wild, SR, Jones KC (1993) Biological and abiotic losses of polynuclear aromatic hydrocarbons (PAHs) from soils freshly amended with sewage sludge. *Environ Toxicol Chem* 12: 5-12.

Wild SR, Jones KC (1995) Polynuclear aromatic hydrocarbons in the United Kingdom environment: a preliminary source inventory and budget. *Environ Pollut* 88: 91-108.

WMO (1999) WMO/EMEP/UNEP Workshop on Modelling of Atmospheric Transport and Deposition of Persistent Organic Pollutants and Heavy Metals. World Meteorological Organisation Global Atmosphere Watch, Report No. 136 (Volume 1) (WMO/TD No. 1008), World Meteorological Organisation, Geneva, Switzerland.

Yoshioka Y, Ose Y (1993) A quantitative structure-activity relationship study and ecotoxicological risk quotient for the protection from chemical pollution. *Environ Toxicol Water Qual* 8: 87-101.

Zangar RC, Springer DL, Buschbom RL, Mahlum DD (1989) Comparison of fetotoxic effects of a dermally applied complex organic mixture in rats and mice. *Fundam Appl Toxicol* 13: 662-669.

Zepp RG, Schlotzhauer PF (1979) Photoreactivity of selected aromatic hydrocarbons in water. In: *Polynuclear aromatic hydrocarbons. Carcinogenesis and mutagenesis* (Jones PW, Leber P, eds) , Ann Arbor Science Publishers, Ann Arbor, Michigan: 141-158.

Zimmerman JR, Ghosh U, Millward RN, Bridges TS, Luthy RG (2004) Addition of carbon sorbents to reduce PCB and PAH bioavailability in marine sediments: Physicochemical tests. *Environ Sci Technol* 38: 5458-5464.

Zimmerman JR, Werner D, Ghosh U, Millward RN, Bridges TS, Luthy RG (2005) Effects of dose and particle size on activated carbon treatment to sequester polychlorinated biphenyls and polycyclic aromatic hydrocarbons in marine sediments. *Environ Toxicol Chem* 24: 1594-1601.

J. ANNEXES

J.1 Abbreviations

ATP	Adaptation to Technical Progress
B	Bioaccumulation
BCF	Bioconcentration Factor
bw	body weight
CAS	Chemical Abstract Services
CMR	Carcinogenic, Mutagenic and toxic to Reproduction
dwt	dry weight
DIN	Deutsche Industrie Norm (German norm)
DNA	Deoxyribo-Nucleic Acid
DOC	Dissolved Organic Carbon
DT_{50}	Degradation half-life or period required for 50 percent dissipation / degradation
EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties (Model]
EC	European Community
EC_{10}	Effect Concentration measured as 10% effect
EC_{50}	median Effect Concentration
EEC	European Economic Community
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
EU	European Union
EUSES	European Union System for the Evaluation of Substances (software tool in support of the Technical Guidance Document on risk assessment)
HELCOM	Helsinki Commission - Baltic Marine Environment Protection Commission
HPLC	High Pressure Liquid Chromatography
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
ISO	International Organisation for Standardisation
IUCLID	International Uniform Chemical Information Database (existing substances)
IUPAC	International Union for Pure and Applied Chemistry
K_{OC}	organic carbon normalised distribution coefficient
K_{OW}	octanol/water partition coefficient
K_p	solids-water partition coefficient
LC_{50}	median Lethal Concentration
LD_{50}	median Lethal Dose
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
NOAEL	No Observed Adverse Effect Level
NOEC	No Observed Effect Concentration
OECD	Organisation for Economic Cooperation and Development
OSPAR	Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic
P	Persistent
PAH	Polycyclic aromatic hydrocarbons
PBT	Persistent, Bioaccumulative and Toxic
PEC	Predicted Environmental Concentration
pH	logarithm (to the base 10) of the hydrogen ion concentration $[H^+]$
PNEC	Predicted No Effect Concentration
POP	Persistent Organic Pollutant
PPE	Personal Protective Equipment

QSAR	Quantitative Structure-Activity Relationship
R phrases	Risk phrases according to Annex III of Directive 67/548/EEC
R41	Risk of serious damage to eyes (Risk phrase according to Annex III of Directive 67/548/EEC)
R43	May cause sensitisation by skin contact (Risk phrase according to Annex III of Directive 67/548/EEC)
R45	May cause cancer (Risk phrase according to Annex III of Directive 67/548/EEC)
R46	May cause heritable genetic damage (Risk phrase according to Annex III of Directive 67/548/EEC)
R50	Very toxic to aquatic organisms (Risk phrase according to Annex III of Directive 67/548/EEC)
R53	May cause long-term adverse effects in the aquatic environment (Risk phrase according to Annex III of Directive 67/548/EEC)
R60	May impair fertility (Risk phrase according to Annex III of Directive 67/548/EEC)
R61	May cause harm to the unborn child (Risk phrase according to Annex III of Directive 67/548/EEC)
RAR	Risk Assessment Report
RPE	Respiratory Protective Equipment
RWC	Reasonable Worst Case
S phrases	Safety phrases according to Annex III of Directive 67/548/EEC
S45	In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible) (Safety phrase according to Annex III of Directive 67/548/EEC)
S53	Avoid exposure - obtain special instructions before use (Safety phrase according to Annex III of Directive 67/548/EEC)
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange
T	Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
TGD	Technical Guidance Document
UNEP	United Nations Environment Programme
US EPA	Environmental Protection Agency, USA
UV	Ultraviolet Region of Spectrum
vB	very Bioaccumulative
vP	very Persistent
vPvB	very Persistent and very Bioaccumulative
WHO	World Health Organization
wwt	wet weight
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)

J.2 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.

J.3 Available ecotoxicity data on PAHs

The tables with the available ecotoxicity data on PAHs, the notes to the tables and list of references are provided in separate pdf files:

- Appendix J.3.1. Table aquatic toxicity data PAHs
- Appendix J.3.2. Notes to table aquatic toxicity data PAHs
- Appendix J.3.3. Table benthic toxicity data PAHs
- Appendix J.3.4. Table terrestrial toxicity data PAHs
- Appendix J.3.5. Notes to table terrestrial and benthic toxicity data PAHs
- Appendix J.3.6. Reference list toxicity data PAHs

J.4 Summaries of studies for human health assessment.

Table J.4.1. Summary of the epidemiological studies on tar distillation.

Reference	Study population	Reference population	Exposure groups	observation period (of outcome)	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	1970-1980	non-significant increases in both lung and bladder cancer death. Standardised Mortality Ratio (SMR)men > 45 years: lung cancer: 143 (95% CI: 82-232) (16 cases) bladder cancer: 301 (95% CI: 98-703) (5 cases) SMR men > 45 years old + 5 year 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 *et al.*, 2003)	907 tar distillery Dutch workers employed at least one half-year between January 1947 and January 1980.	national population	exposed/ non-exposed	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 *et al.*, 2003)	255 British tar distillery workers employed on 1 January 1967 31 December to 1983	regional and national population	exposed/ non-exposed	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)

Table J.4.2. Summary of studies on aluminium production workers.

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Canadian studies						
Theriault *et al.*, 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 aluminium plants in Quebec (using both Søderberg and prebake pots)	225 controls, currently or previously employed for at least one year in one of the five aluminium 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: Søderberg 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)
Armstrong *et al.*, 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

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Gibbs, 1985	5406 male workers employed in 1950 at 2 Søderberg smelter plants in Quebec	population of the province	four cumulative exposure categories (relative exposure intensity of the job multiplied by number of years exposed)	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) cancer of stomach and oesophagus: 153 (50) (p<0.05) Hodgkin's disease: 179 (5) (p>0.05) leukemias: 112 (9/)(p>0.05) cancer of the pancreas: 105 (14) (p>0.05) cancer of the larynx: 131 (7) (p>0.05) cancer of the brain: 109 (8/)(p>0.05)
Armstrong *et al.*, 1994	338 lung cancer cases, employed for at least one year between 1950 and 1970 in a manual job in an aluminium plant in Quebec (using both Søderberg 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 aluminium plant in Quebec.	cumulative exposure (estimated PAH level based on industrial hygiene data x job years)	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 $\geq$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, $\geq$300 BaP µg/m^3-years: 1.00, 1.48 (1.09-2.00), 2.23 (1.46-3.39), 2.10 (1.40-3.15) and 1.87 (1.05-3.33) The data were compatible with a linear relation with cumulative exposure (RR=1 + 0.031 mg/m^3-years BSM, while a curved relation (RR=1 + 0.098 mg/m^3-years BSM$^{0.7}$) fitted somewhat better

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Tremblay *et al.*, 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	see above	see above	smoking-adjusted bladder cancer rate ratios (RR) (95% CI) <1, 1-9, 10-19, 20-29 and ≥30 BSM mg/m^3 -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). smoking-adjusted bladder cancer rate ratios (95% CI) <10, 10-99, 100-199, 200-299, ≥300 BaP µg/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) The data were best described by a linear model using BaP cumulative exposure an a lag time before diagnosis of 10 years (RR=1 + 0.0166 µg/m^3-years BaP.
Spinelli *et al.*, 1991	4213 men employed for at least 5 years between 1954 and 1985 in a Søderberg plant in British Columbia.	the population of the same province in Canada.	four cumulative exposure categories (estimated BSM level based on recent plant monitoring x job years)	1970-1985		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) ($p<0.05$) 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) 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)
Norwegian studies						

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Andersen, 1982 cited in Ronneberg & Langmark, 1992 and IARC, 1984	7410 men working for at least 18 months prior to 1970 in 4 aluminium plants in Norway (using both Søderberg and prebake pots)	national population	old plant workers/new plant workers/non-aluminium workers	1953-1970	no information on smoking	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 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 O/E leukemia mortality all workers: 17/12.6 old plant workers: 9/5.9 O/E kidney cancer mortality all workers: 18/14.8 O/E cancer of other sites all workers: 104/94.4
Ronneberg & Andersen, 1995	1137 men hired between 1922 and 1975, employed for at least 6 months at a time in a prebake aluminium smelter in Norway	national male population	semi-quantitative exposure estimates (from job exposure matrix)	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 $\geq$3 years employed cancer of all sites: 0.93 (0.77-1.11) bladder cancer: 1.58, 14 cases

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Ronneberg *et al.*, 1999	5908 workers of a Norwegian aluminium smelter (using both Søderberg 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)	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 haemotopoietic tissue.	Standardized 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 mesotheliama 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	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Romundstad *et al.*, 2000b	5627 workers employed for at least 6 months two Norwegian aluminium reduction plants	national male population	cumulative exposure (exposure intensity (2->3000 µg/m³ PAH x duration of each job held in the smelter)	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 haemotopoietic cancer.	Standardised Incidence Ratio (SIR) (95% CI) bladder cancer (30 year lag period): <50 µg/m³PAH × year: 1.29 (0.83-1.92) 50-500 µg/m³PAH × year: 1.04 (0.21-3.03) 500-2000 µg/m³PAH × year: 1.16 (0.32-2.97) >2000 µg/m³PAH × year: 4.08 (1.32-9.51) SIR (95% CI) pancreatic cancer (10 year lag): <50 µg/m³PAH × year: 0.43 (0.05-1.57) 50-500 µg/m³PAH × year: 1.14 (0.14-4.11) >500 µg/m³PAH × year: 0.81 (0.81-3.36) SIR (95% CI) lung cancer (20 year lag period): <50 µg/m³PAH × year: 0.94 (0.60-1.14) 50-500 µg/m³PAH × year: 1.72 (0.92-2.95) 500-2000 µg/m³PAH × year: 0.78 (0.34-1.54) >2000 µg/m³PAH × year: 0.28 (0.03-1.02)
Romundstad *et al.*, 2000a	11103 workers employed for more than 3 years in six Norwegian aluminium plants	national population and local county	cumulative exposure (exposure intensity (0-3400 µg/m³ PAH x duration of each job held in the smelter)	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 µg/m³PAH × year: 1 (ref) 0-499 µg/m³PAH × year: 1.0 (0.7-1.9) 500-1999 µg/m³PAH × year: 1.3 (0.8-2.0) > 2000 µg/m³PAH × year: 2.0 (1.1-2.8) Rate ratio (95% CI) for lung cancer (30 year lag time): 0 µg/m³PAH × year: 1.0 (ref) 0-499 µg/m³PAH × year: 1.0 (0.6-1.6) 500-1999 µg/m³PAH × year: 1.1 (0.7-1.7) > 2000 µg/m³PAH × year: 0.4 (0.1-1.0) Rate ratio (95% CI) for pancreatic cancer (30 year lag time): 0 µg/m³PAH × year: 1.0 (ref) 0-499 µg/m³PAH × year: 1.0 (0.4-2.7) 500-1999 µg/m³PAH × year: 1.4 (0.6-3.3) > 2000 µg/m³PAH × year: 1.5 (0.5-4.6)
US studies						

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Milham, 1979 cited in HSE, 1993 and Armstrong *et al.*, 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	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 statistically significant) pancreas cancer: 1.80 (O/E: 9/5, not statistically significant) prostate cancer: 1.62 (O/E: 8/5, not statistically 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)

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Rockette & Arena, 1983 cited in Ronneberg & Langmark, 1992 and Armstrong *et al.*, 2003	21,829 men who were employed in 14 aluminium plants for at least 5 years between 1949 and 1977 (using both Søderberg and prebake pots)	national population	length of employment and work area	1950-1977		Observed/Expected Ratios (number of deaths) mortality prebake plant workers: all causes: 0.91 (2433) ≥15 years employed: lung cancer: 1.0 (30) (not statistically significant) pancreas cancer : 2.2 (12 deaths) (p<0.05) kidney cancer: 19/13 (not statistically significant) ≥ 25 years employed in caron area: lung cancer: 3.75 (6) O/E Ratio (number of death) Søderberg 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 statistically significant) lung cancer: 0.8 (23) (not statistically significant) ≥15 years employed: bladder cancer: 2.0 (6) (not statistically significant) lung cancer: 1.0 (19) (not statistically significant)
Enterline, 1982 cited in HSE, 1993	deaths of male US aluminium 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	aluminium workers/non-aluminium workers	1946-1953		No non-accidental excess mortality was reported for the aluminium workers. Standardised Mortality Ratio (SMR) lung cancer: 0.79 compared with US males. Results were difficult to interpret because of limited data.

Reference	Study population	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Equitable environmental Health Incl., 1977 cited in HSE, 1993	23033 men working 5 years or more during the period 1946 to 1973	US population	aluminium workers/ non-aluminium workers	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	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 aluminium 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 *et al.*, 1987 cited in HSE, 1993 and Armstrong *et al.*, 2003	6455 workers employed for at least one year in the period 1950-1976 in one of the 11 aluminium plants in France (both Søderberg and prebake plants)	national population	aluminium workers/ non-aluminium workers	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	Reference population	Exposure group	observation period (of outcome)	correction for confounding and effect modification	Findings
Moulin *et al.*, 2000	2133 workers employed for at least 1 year between 1950 and 1994 in a French aluminium plant	regional population	job type and duration of employment	1968-1994	adjusted for gender, age and calendar time	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 neurodegenerative 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 neurodegenerative diseases: 2.39 (0.88-5.21)
Italian study						
Giovannazzie & D'Andrea, 1981 cited in Ronneberg & Langmark, 1992	494 men employed between 1965 and 1979 in a Søderberg reduction plant in Italy	regional population	potroom workers/ regional population	1965-1979		increased mortality in potroom workers, due to cardiovascular diseased, liver cirrhosis and lung cancer.
USSR studies						
Konstantinov & Kuz'minykh, 1971 cited in IARC, 1984	workers of two aluminium production plants in the USSR	regional population	Søderberg workers/regional population	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 *et al.*, 1974 cited in IARC, 1984	potroom workers in three aluminium plants	regional population	Søderberg workers/ regional population			Results were difficult to interpret because of the absence of information both on the study population and the reference population.

Table J.4.3 Summary of the epidemiological studies on the use of CTPHT as a binder and impregnation of electrodes.

Reference	Study population	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 aluminium plant and 6 carbon plants	11470 other steel workers employed in rough rolling mills	4 exposure categories	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	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	plant A: 1975-1985 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.

Table J.4.4. Summary of studies on asphalt workers and roofers.

Reference	Study population	Exposure groups	Observation period (for outcome)	Correction for confounding and effect modification	Findings
Cohort studies					
Hammond *et al.*, 1976 cited in Partanen & Boffetta, 1994 and Armstrong *et al.*, 2003	5339 US roofers and waterproofers	> 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 *et al.*, 1976 cited in Partanen & Boffetta, 1994	2000 US roofers	roofers vs. other occupations	1968-1973		Standardised Mortality Ratio (SMR) lung cancer: 5.0 (2.5-8.9); 11 deaths
Maizlish *et al.*, 1988 cited in Partanen & Boffetta, 1994	1570 deaths	California Highway Maintenance Workers (HMW)	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 *et al.*, 1988 cited in Partanen & 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, $p < 0.05$
Bender *et al.*, 1989 cited in Partanen & 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) leukaemia: 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

Reference	Study population	Exposure groups	Observation period (for outcome)	Correction for confounding and effect modification	Findings
Hansen, 1989; Hansen *et al.*, 1991 cited in Partanen & 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)	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 oesophagus: 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 & 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 & Boffetta, 1994	52 deaths	roofers and slaters (US)	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	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 & Boffetta, 1994	National population of Finland	Finish asphalt workers	1971-1985	adjusted for social class and age	Standardised Incidence Ratio (SIR) lung cancer: 2.7 (1.6-4.1) 20 cases
Milham *et al.*, 1993 cited in Partanen & Boffetta, 1994	7266 deaths	US Graders, pavers, operators and excavators (Washington State)	1950-1989	co-exposure to crystalline silica was suggested	Proportional Mortality Ratio (PMR) lung cancer: 1.2 (1.1-1.3); 558 deaths
Swaen & 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	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.

Reference	Study population	Exposure groups	Observation period (for outcome)	Correction for confounding and effect modification	Findings
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)
Randem *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 & 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
Vineis *et al.*, 1988 cited in Partanen & 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

Reference	Study population	Exposure groups	Observation period (for outcome)	Correction for confounding and effect modification	Findings
Zahm *et al.*, 1989 cited in Partanen & 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 & 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 & 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 & 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 & Boffetta, 1994	96 cases of renal pelvis tumours + 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 & 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 cancer 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

J.5 Measured Emission Profiles of 16 PAHs

Table J.5.1. Measured emission profile for 16 PAHs to air.

nr	16-EPA	PAH compound	particulate	gaseous	total
1	x	Phenanthrene	7.2	66.0	40.2
2	x	Anthracene	1.9	13.2	8.2
3	x	Fluoranthene	18.7	13.9	16.0
4	x	Pyrene	11.8	6.9	9.1
5		Benzo(a)fluorene	2.5	0.0	1.1
6		Benzo(b)fluorene	3.5	0.0	1.5
7	x	Benz(a)anthracene	6.5	0.0	2.9
8/9	**x**	**Chrysene/Triphenylene**	13.3	0.0	5.8
10		Benzo(bjk]fluoranthene	16.2	0.0	7.1
11		Benzo(e]pyrene	6.1	0.0	2.7
12	x	*Benzo(a)pyrene*	4.5	0.0	2.0
13	x	indeno(123-cd)pyrene	1.6	0.0	0.7
14	x	Dibenz(a,h)anthracene	0.4	0.0	0.2
15	x	benzo(ghi)perylene	2.0	0.0	0.9
16		1,2,4,5-dibenzopyrene	3.5	0.0	1.6

Values represent the average of six Norwegian primary aluminium production plants and includes the contribution to the particulate, gaseous and total emission. Data originate from OSPAR (2002).

Table J.5.2. Measured profiles for the emission of 16 PAHs to water at three Norwegian primary aluminium production plants.

nr	16-EPA	PAH compound	Site 1	Site 2	Site 3	Average
1	x	Phenanthrene	20.0	16.9	13.6	16.8
2	x	Anthracene	2.9	1.0	1.0	1.6
3	x	Fluoranthene	23.8	27.4	33.2	28.2
4	x	Pyrene	15.7	19.8	16.2	17.2
5		Benzo(a)fluorene	5.2	6.0	7.4	6.2
6		Benzo(b)fluorene	2.5	3.4	3.4	3.1
7	x	Benz(a)anthracene	4.7	4.7	5.1	4.8
8/9	**x**	**Chrysene/Triphenylene**	8.6	7.9	8.9	8.5
10		Benzo(bjk]fluoranthene	6.1	5.2	4.9	5.4
11		Benzo(e]pyrene	2.9	2.4	2.4	2.6
12	x	*Benzo(a)pyrene*	2.5	1.8	1.5	1.9
13	x	indeno(123-cd)pyrene	1.0	0.9	0.9	0.9
14	x	Dibenz(a,h)anthracene	0.5	0.3	0.3	0.3
15	x	benzo(ghi)perylene	1.4	0.9	0.5	0.9

Data originate from OSPAR (2002). Benzo(a)pyrene = 1.

J.6 Estimated Emission Profiles of the 16 EPA PAHs

Table J.6.1. Estimated (emission) profiles of the 16 EPA PAHs to air for the Vertical Stud Søderberg (VSS) primary aluminium production process.

PAH compound	PR1	PR2	PR3	PR4	PR5 [a)]	PR6 [b)]	Average [c)]	St. dev.
Naphthalene	17.75	32.21	6.43	9.58	16.5 [d)]	16.5 [d)]	16.5	9.97
Acenaphthene	4.84		1.19	1.35	2.5 [d)]	2.5 [d)]	2.5	1.68
Acenaphthylene	17.34		0.81	0.77	6.3 [d)]	6.3 [d)]	6.3	7.80
Fluorene	10.87	14.64	2.62	1.79	7.5 [d)]	7.5 [d)]	7.5	5.45
Anthracene	4.71	3.85	0.71	0.75	1.16	2.0	2.5	
Phenanthrene	52.93	51.33	66.67	25.00	7.22	19.5	49.0	
Fluoranthene	16.81	21.81	66.67	22.92	3.73	11.0	32.1	
Pyrene	10.18	13.16	33.33	3.75	2.89	5.5	15.1	
Benz(a)anthracene	2.68	1.67	2.24	1.38	0.95	1.0	2.0	
Chrysene	5.21		4.76	5.63	1.76	2.0	5.2	
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.00	1.0	1.0	
Benzo(b)fluoranthene			2.86	2.08	4.08	3.0 [e)]	2.5 [e)]	
Benzo(k)fluoranthene			1.05	1.02			1.0	0.01
Benzo(ghi)perylene	0.74	0.59	1.12	1.10	0.65	0.5	0.9	
Dibenzo(a,h)anthracene			1.07	1.04	0.11	0.2	1.1	
Indeno(123-cd)pyrene	0.72		1.05	0.98	0.51	0.5	0.9	

Shaded columns are use in the scenario calculations; PR1: Bjørseth *et al.* (1981); PR2: IARC (1984); PR3 & PR4: EPA (1998b) and PR5 & PR6: EAA (2005); [a)] this profile is applicable to facilities with wet scrubbers; [b)] this profile is applicable to facilities with dry scrubbers. [c)] The average is calculated for profiles 1-4, excluding profile number 5; [d)] calculated average value; [e)] This is for benzo(b)fluoranthene and benzo(k)fluoranthene together.

PR 1 is based on occupational exposure data. It is the average concentration measured in three potrooms in a VSS primary aluminium production plant in Norway; PR 2 is based on occupational exposure data and is referred to as coming from Bjørseth *et al.* (1978); PR 3 is based on reported emission factors from a VSS production plant with dry scrubbers; PR 4is based on emission factors from a facility with dry and wet scrubbers; PR 5 and six are based on emission measurements at VSS electrolysis plants applying wet scrubbers and dry scrubbers respectively

Table J.6.2. Estimated profiles for the emission of the 16 EPA PAHs to air for the anode baking process.

PAH compound	PR1	PR2	PR3	PR4	PR5	PR6	Average [a]
Naphthalene			2.0	4.0		29.0	11.7
Acenaphthene			3.3	4.6		3.0	3.6
Acenaphthylene			0.1	0.1		0.1 [b]	0.1
Fluorene			1.6	2.4		5.0	3.0
Anthracene		43.5	0.8	0.7		2.0	1.2
Phenanthrene		270.5	2.6	2.7		24.0	10.8
Fluoranthene	1.5	58.5	2.4	2.5	6.0	10.0	7.7
Pyrene	3.1	30.0	2.0	2.1	4.3	5.0	5.5
Benz(a)anthracene	0.8		1.0	1.1	0.8	2.0	1.5
Chrysene	0.9		1.2	1.3	2.8	5.0	2.0
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Benzo(b)fluoranthene						8.0 [c]	4.0
Benzo(k)fluoranthene			0.9	0.8			
Benzo(ghi)perylene	1.5		0.7	0.8	0.4	1.0	0.8
Dibenzo(a,h]anthracene			0.2	0.2		1.0	0.2
Indeno(1.2.3-cd)pyrene	0.7		0.7	0.7	0.8	1.0	0.7

The shaded column is used in the scenario calculations; PR1: Van Schooten *et al.* (1995); PR2: Bentsen-Farmen *et al.* (1999); PR3: Petry *et al.* (1996a); PR4: Petry *et al.* (1996b); PR5: Øvrebø *et al.* (1994) and PR6: EAA (2005). [a] The average value is calculated from profiles 1, 3, 4, and 5, excluding profile 2. [b] Calculated average value. [c] This is for benzo(b)fluoranthene and benzo(k)fluoranthene together.

PR 1 is based on occupational exposure measurements at anode baking (green paste preparation; PR 2 is based on occupational exposure measurements in an anode paste plant (green paste preparation and baking); PR 3 is based on occupational exposure data from an anode plant (including anode baking); PR 4 is based on occupational exposure data from an anode plant using petroleum pitch (including baking); PR 5 is based on exposure measurements in an electrode paste plant. PR 6 is the profile for anode baking provided by the industry.

Table J.6.3. Profiles for the emission of the 16 EPA PAHs to air for green paste production process.

PAH compound	PR1	PR2	Maximum
Naphthalene		2.00	2.0
Acenaphthene			3.6 [a)]
Acenaphthylene			0.1 [a)]
Fluorene		9.00	9.0
Anthracene	15.29	8.00	15.3
Phenanthrene	48.00	25.00	48.0
Fluoranthene	34.14	23.00	34.1
Pyrene	21.00	15.00	21.0
Benz(a)anthracene	4.29	4.00	4.3
Chrysene	4.57	4.00	4.6
Benzo(a)pyrene	1.00	1.00	1.0
Benzo(b)fluoranthene	5.00 [b)]	4.00 [b)]	5.0
Benzo(k)fluoranthene			
Benzo(ghi)perylene	0.00	0.00	0.0
Dibenzo(a,h)anthracene	0.00	0.00	0.0
Indeno(123-cd)pyrene	0.00	0.00	0.0

The shaded column is used in the scenario calculations; PR1: Elkem (1999); PR2: EAA (2005); [a)] Calculated average from anode baking; [b)] This is for benzo(b)fluoranthene and benzo(k)fluoranthene together.

PR 1 is based on occupational exposure in a silicon carbide production plant. The total PAH concentration is measured (gaseous and particulate); PR 2 is based on a profile for a class for similar processes of which silicon carbide production. The total PAH concentration is measured (gaseous and particulate).

Table J.6.4. Estimated profiles for the emission of the 16 EPA PAHs to air for the graphite electrode baking process.

PAH compound	PR1	PR2	PR3	PR4	PR 5	Maximum [a)]
Naphthalene	11.60	4.82			77.31	11.60
Acenaphthene				0.52	28.33	0.52
Acenaphthylene				0.40	16.95	0.40
Fluorene	0.81	1.07		1.18	10.88	1.18
Anthracene	0.70	1.31		0.51	9.62	1.31
Phenanthrene	6.63	3.76	13.68	9.04	131.45	13.68
Fluoranthene	3.46	1.78	4.75	2.25	90.13	4.75
Pyrene	1.79	1.44	4.05	3.93	49.83	4.05
Benz(a)anthracene	1.71	0.89	2.10	6.69	6.31	6.69
Chrysene	1.98	1.33	2.24	30.27	17.23	30.27
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.00	1.0
Benzo(b)fluoranthene			2.24		8.90	2.24
Benzo(k)fluoranthene				0.89	4.62	0.89
Benzo(ghi)perylene	0.63	0.56		1.18	0.88	1.18
Dibenzo(a,h)anthracene	0.32	0.44	0.16	0.87	0.48	0.87
Indeno(123-cd)pyrene				0.40	1.07	0.40

The shaded column is used in the scenario calculations; PR1: Buchet *et al.* (1995); PR2: Buchet *et al.* (1992); PR3: Angerer *et al.* (1997); PR4: Petry *et al.* (1996a); PR 5: ECGA (2005); [a)] Maximum value from profile 1-4.

PR 1 is based on occupational exposure in a graphite electrode manufacturing facility using coal tar pitch (vapour and particulate phase); PR 2 is based on occupational exposure measurements in a graphite electrode production plant; PR 3 is based on occupational exposure is a graphite electrode production plant (particulate and gaseous); PR 4 is base on indoor concentrations (vapour and particulate) is a graphite plant.

Table J.6.5. Profiles for the emission to water of the 16 EPA PAHs for primary aluminium production facilities using the Søderberg technology.

PAH compound	PR1	PR2	PR3	PR4	PR5	PR6	PR7	Average [a]
Naphthalene						0.3	0.3 [b]	0.25
Acenaphthene						1.3	1.3 [b]	1.25
Acenaphthylene						0.3	0.3 [b]	0.25
Fluorene						0.8	0.8 [b]	0.75
Anthracene	1.2	0.6	0.7	0.4	0.1	2.5	0.8	0.90
Phenanthrene	7.9	9.5	8.9	4.3	0.1	8.5	8.8	6.54
Fluoranthene	9.4	15.4	21.8	3.8	0.6	31.0	14.8	13.65
Pyrene	6.2	11.1	10.7	2.4	0.5	19.0	9.1	8.31
Benz(a)anthracene	1.9	2.6	3.3	0.9	1.1	2.8	2.5	2.10
Chrysene	3.4	4.4	5.8				4.5	4.55
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Benzo(b)fluoranthene					1.6	2.3	3	1.91
Benzo(k)fluoranthene								
Benzo(ghi)perylene	0.6	0.5	0.3		0.6	0.5	0.5	0.50
Dibenzo(a,h)anthracene	0.2	0.1	0.2			0.3	0.2	0.19
Indeno(123-cd)pyrene	0.4	0.5	0.6				0.5	0.49

The shaded column is used in the scenario calculations; PR1-PR3: OSPAR (2002); PR4: Kadar *et al.*(1980); PR5: Olufsen, (1980); PR6: Berglind (1982) and PR7: EAA (2005); [a] Average of profiles 1-6; [b] Based on the maximum values (value of profile 6).

Table J.6.6. Profiles for the emission to water of the 16 EPA PAHs for primary green anode production (paste preparation).

PAH compound	PR1	PR2	Average
Naphthalene			0.3 [a]
Acenaphthene			1.3 [a]
Acenaphthylene			0.3 [a]
Fluorene			0.8 [a]
Anthracene	10.0	10.6	10.3
Phenanthrene	36.0	37.9	36.9
Fluoranthene	22.0	25.6	23.8
Pyrene	16.0	17.8	16.9
Benz(a)anthracene	4.0	4.4	4.2
Chrysene	3.0	3.6	3.3
Benzo(a)pyrene	1.0	1.0	1.0
Benzo(b)fluoranthene	2.0 [b]	2.2 [b]	2.1 [b]
Benzo(k)fluoranthene			
Benzo(ghi)perylene	0.4	0.1	0.3
Dibenzo(a,h)anthracene	0.4	0.4	0.4
Indeno(123-cd)pyrene	0.4	0.3	0.4

The shaded column is used in the scenario calculations; PR1: EAA (2005); PR 2 Elkem (1999); [a] Based on the values for the Søderberg primary aluminium production profile (Table J.6.5); [b] This value is the sum of benzo(b)fluoranthene and benzo(k)fluoranthene.

J.7 Additional information on alternatives

In addition to the information on alternatives for anode production for metal industry presented in Section C, this section briefly discusses alternatives for other applications. Based on the use pattern for coal tar pitch (see Table B.2.2), the following applications are considered:

- refractories
- active carbon
- clay pigeons
- heavy duty corrosion protection
- road construction and roofing
- coal briquetting

For each of these applications possible alternative substances and techniques are identified and discussed in relation to their availability, their risks for human health and the environment, and their technical and economical feasibility.

J.7.1 Refractories

For refractories, the pitch industry now proposes pitches with a higher softening point resulting in a benzo(a)pyrene content of 300 ppm compared to currents levels in pitches ranging up to 20,000 ppm. Such pitches are already available. Yet, although benzo(a)pyrene concentrations in the alternative pitches is lower, the main constituents of these pitches still are PAHs, suggesting that many of the human health and environmental risks identified in this report will still exist, although they might be lower. In addition, as indicated in Section B.1.2, despite the high potential carcinogenic risk of PAHs other severe effects of pitch can still not be ruled out.

Furthermore, the PBT assessment for many PAHs indicates that they are either PBT or vPvB in the environment (see Section B.8). The associated environmental risks are not necessarily significantly reduced when the benzo(a)pyrene content of the pitch used is minimized. As an example, this can be visualized when binder pitch is compared to impregnation pitch (see Table B.1.2 for composition of the two pitches). The benzo(a)pyrene content in binder pitch is ca. 20% lower, but the anthracene content is almost 80% higher.

Yet, there are no risks identified associated with this application of CTPHT, which hampers justification of risk management measures other than those already in place.

J.7.2 Active carbon

Pitch bound active carbons are more and more produced outside the EU and are anyway processed in closed vessels where the pitch is pyrolyzed to pure carbon with controlled emissions. This suggests that although no alternative is available, the risks of CTPHT in this application are limited, which makes it questionable whether time and money should be spent on restriction of a problem that may solve itself in the near future[13], especially since no risks are identified for this application.

J.7.3 Clay pigeons

Clay pigeons manufacturers, claiming environmental protection, displaced carbopitch by petrochemical binders for more than 80% of their production and the former clay pigeons being exported outside the European Union. As an even better alternative, at least one manufacturer claims to apply a mixture of several clays only, without any binder at all (Lireko, 2002). From a risk reduction management point of view this latter option appears to be the recommended choice, because the potential risks of a mixture of different natural clays can be expected to be (close to)

[13] It will take about two years before a decision on restrictions is taken and about four years before the measure is implemented. For authorisation it will also take at least about two years before a decision is taken.

zero, while the risks of petrochemical binders are likely to still be significant (although lower than those of CTPHT). Yet, since in this application CTPHT is being replace by other binders, it is questionable whether time and money should be spent on restriction of a CTPHT problem that may solve itself in the near future[14], especially since no risks are identified for this application.

J.7.4 Heavy duty corrosion protection

According to industry, corrosion protection with CTPHT-based products are declining dramatically and a 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. Yet, since no risks are identified for this application there appears to be no need for risk management measures for CTPHT in this application other than those already in place.

J.7.5 Road construction and roofing

Most of the European countries have banned CTPHT in the road construction by law or agreement between trade unions and road building companies. It is replaced by petroleum pitch on account of the lower PAH content. In fact only very particular applications such as kerosene proof coatings for parking lots, airfields and taxi ways still use pitch as an emulsion. This market is decreasing and represents only 200 tonnes of pitch per year.

Also for roofing CTPHT is replaced by petroleum pitch.

In both these applications, however, it should be noted that although the replacement of CPTHT by petroleum pitch reduces the amount of PAHs involved, it does not reduce the risks to zero. Yet, further measures considering the use of CTPHT in this application are unnecessary.

J.7.6 Coal briquetting

The use of pitch bound coal briquettes is forbidden in some countries (Germany and Scandinavia). This market is also linked to dedicated and captive users in mining countries (France and Belgium) where retired miners have rights on solid fuels provided by the former state owned companies. Capacities of 2,000 ktonnes/year of briquettes existing in the early 1980s in Europe are now decreased to 150 ktonnes/year, also using more environmental friendly binders like starch and molasses. Also here a full phasing out of the use of CTPHT can be expected in the next few years, making further restrictions unnecessary.

[14] It will take about two years before a decision on restrictions is taken and about four years before the measure is implemented. For authorisation it will also take at least about two years before a decision is taken.