COAL TAR PITCH, HIGH TEMPERATURE

CAS No: 65996-93-2

EINECS No: 266-028-2

SUMMARY RISK ASSESSMENT REPORT

Final report, April 2008

The Netherlands

Rapporteur for the risk assessment of Coal Tar Pitch high temperature is the Ministry of Housing, Spatial Planning and the Environment (VROM) and the Ministry of Social Affairs and Employment (SZW), in consultation with the Ministry of Public Health, Welfare and Sport (VWS). Responsible for the risk evaluation and subsequently for the contents of this report is the rapporteur.

The scientific work on this report has been prepared by the Netherlands Organization for Applied Scientific Research (TNO) and the National Institute of Public Health and Environment (RIVM), by order of the rapporteur.

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PREFACE

The report provides the comprehensive risk assessment of the substance coal tar pitch, high temperature (CTP(ht)). It has been prepared by the Netherlands in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances. For detailed information on the risk assessment principles and procedures followed, the underlying data and the literature references, the reader is referred to the original risk assessment report that can be obtained from European Chemicals Bureau[1]. The present summary report should preferably not be used for citation purposes.

[1] European chemicals Bureau – Existing Chemicals - http://ecb.ei.jrc.it

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1 GENERAL SUBSTANCE INFORMATION

Identification of the substance

This RAR concerns only Pitch, coal tar, high temperature (CTP(ht)) with CAS # 65996-93-2. Coal tar pitch high temperature (CTP(ht)) 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 (1976) as 'the condensation product obtained by cooling, to approximately ambient temperature, of the gas evolved in the high temperature (greater than 700 ºC (1292 ºF)) 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 CTP(ht) is heated, Coal tar pitch volatiles (CTPV(ht)) 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.

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. 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.
Because of the complexity and variability of CTP(ht), 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 CTPV and Polycyclic Aromatic Hydrocarbons (PAHs), in the air. Since PAHs are among the major components of CTP(ht), and some individual PAHs are proven animal carcinogens, PAH levels are considered as a measure of exposure to CTP(ht). Existing exposure information suggested that the airborne concentration of BaP 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 HSE has pinpointed BaP as the most suitable marker for assessing exposure to PAHs[2] for Coal Tar Pitch Volatile (CTPV) industries. As such, in conducting this exposure assessment to CTP(ht), exposure to BaP has been adopted as the primary indicator.

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

The database on possible health hazards induced by CTP(ht) 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 CTP(ht) is produced and/or used, that indicate that carcinogenicity is a striking hazard associated with CTP(ht). This is attributed to the presence of the PAHs in CTP(ht), as indicated above. Given the uncertainties with respect to the effects of other chemical constituents of CTP(ht) (and related substances), it is not completely sure that carcinogenicity is the only relevant effect of CTP(ht). 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 database, it is decided that the focus will be on the carcinogenic and mutagenic properties, using the best-studied PAH BaP as a guidance substance.

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.
Structural formula:	not applicable
Molecular weight:	not applicable
Synonyms:	anode pitch, binder pitch, clay pidgeon binder, electrode pitch, hard pitch, impregnating pitch, pitch, soft pitch, vacuum pitch

Purity/impurities, additives

Purity:	not applicable
Impurity:	not applicable
Additives:	not applicable

Physico-chemical properties

The physico-chemical properties of high-temperature coal tar pitch are listed in Table 1.1.

[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 RAR

Table 1.1 Summary of physico-chemical properties of CTP(ht)

Property	Value	Comment / Reference
Physical state	black solid	
Melting point	65-150°C	softening range; CCSG (2006a)
Boiling point	>360°C	at 1013 hPa
Density	1.15-1.4 g/cm^3	at 20°C; ASTM D 71; CCSG (2006a)
Vapour pressure	<0.1 Pa <10 Pa	at 20°C at 200°C ; OECD 104; CCSG (2006a)
Water solubility	<1 mg/L at 20°C	No test reports were available. Data are from data sheets (ACCCI, 1992; van den Bosch, 1997) (see also HEDSET).
Partition coefficient n-octanol/water (log value)	-	not applicable
Flash point	>250°C	ISO 2719; CCSG (2006a)
Flammability	non flammable	No test reports were available. Data are from data sheets (ACCCI, 1992; van den Bosch, 1997) (see also HEDSET).
Auto ignition temperature	>450°C	at 1013 hPa; DIN 51794; CCSG (2006a)
Explosive properties	not explosive	CCSG (2006a)
Oxidizing properties	not oxidising	CCSG (2006a)

Conclusion

All relevant physico-chemical data were available. None of them are substantiated with test reports. However, the data are considered as sufficiently reliable to fulfil the Annex VIIA requirements.

Classification and labelling

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)

There are insufficient data available on the sensitising properties, mutagenicity and toxicity for reproduction of CTP(ht) itself. However, it is proposed to classify CTP(ht) as a skin sensitiser, a category 2 mutagen, and as toxic to reproduction (category 2), because CTP(ht) contains substances which are classified as such (see 1.7.2.1 of Annex VI of Directive 67/548).

2 GENERAL INFORMATION ON EXPOSURE

3 ENVIRONMENT

4 HUMAN HEALTH

4.1 EXPOSURE

4.1.1 Occupational exposure

CTP(ht) (Coal Tar Pitch high temperature) is a complex hydrocarbon produced during distillation of coal tars. To assess exposure to CTP(ht), benzo(a)pyrene (BaP) is used as the primary marker. The estimated proportion of BaP in CTP(ht) is 1%. Occupational exposure assessment has been conducted for the production of CTP(ht) in a coal tar distillation facility (most prevalent source of exposure), use as a binder and impregnation of electrodes, use as a binder in the asphalt industry and in refractories. Additionally, exposure assessment has been conducted, if possible, for the following exposure scenarios which represent a small part of the overall use of CTP(ht): use as a binder for active carbon, heavy duty corrosion protection, coal briquetting and clay pigeons.

Operators, cleaners, drivers and quality control annalists may be exposed to CTP(ht) in coal tar distillation plants during all activities of production. Based on measured inhalation data "handling of solid pitch" and "tar processing and handling of liquid pitch" are identified as two high exposure sub-scenarios for both inhalation and dermal exposure assessment. Dermal exposure during "tar processing and handling of liquid pitch" is estimated to be negligible due to the high temperature of the liquid pitch.

CTP(ht) is used as a binding agent for electrodes in the aluminium industry. In many different tasks such as stud-pulling, rack-raising, mounting of flaints and adding of anode paste, the exposure can be considerable. However, exposure concentrations are dependent on the technology used and the age of the plant. Therefore, four sub-scenarios have been identified: Søderberg potrooms (not modernised and modernised), anode bake plants and paste plants, for which inhalation and dermal exposure estimates have been assessed. If only (hot) liquid pitch is used dermal exposure is estimated to be negligible.

There is potential for inhalation and dermal exposure to CTP(ht) particulates and vapour at electrode paste plants where CTP(ht) is used as a binding agent during impregnation of electrodes with liquid pitch. Inhalation exposures are estimated for the higher exposed group: workers in mixing/grinding, baking and impregnation and maintenance and repair staff. Daily dermal exposure due to handling of hot CTP(ht) is estimated to be negligible. Workers in other areas will have lower exposures.

Workers in the road construction industry carry out road paving and recycling/resurfacing activities. These individuals can be exposed, via the skin and via inhalation, to CTPV (Coal tar pitch volatiles), when CTP(ht) is used as a binder. It should be noted that the estimate for inhalation exposure is based on one source only and the data to estimate current exposure is outdated. The extraction of data from a graph includes some degree of uncertainty.

During the laying down of a new roof or the repair of a roof with patches hot CTP(ht) is used. Volatile matter emanates from the heated asphalt resulting in possible inhalation and dermal exposure to CTP(ht) or CTPV. Removal of an old roof using hand tools may also result in inhalation and dermal exposure.

Refractories are materials that 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. Inhalation exposure and dermal exposure to CTP(ht) in refractories may occur during production and use. There is one source of inhalation exposure data for the use of CTP(ht) as a binding agent for refractories. Due to the high temperatures in the use of refractories, dermal exposure is expected not to occur repeatedly in this part of the scenario.

No measured information is available on the use of CTP(ht) as a binding agent for active carbon. At present, there is insufficient information with regard to process details and proportion of CTP(ht) used in the binder to allow for the derivation of exposure estimates using EASE modelling.

Hot-applied coal tar enamel coatings are used in heavy-duty corrosion protection. Coal tar enamels are formulated from refined CTP(ht). Inhalation exposure to CTPV(ht) is expected with the coating operator, paper latcher, breakout man, holiday patcher, end finisher and the kettle tender. Dermal exposure is expected to be low due to the high operating temperature of the process. Where the coal tar enamel may have spilled dermal exposure is expected to be incidental. Based on measured data coating operators and other workers are defined as two distinctive exposure groups for which exposure values are assessed.

During coal briquetting fine coal is compressed to form a ‘patent fuel’ or briquette where CTP(ht) is used as a binder. During the whole process there is potential for inhalation and dermal exposure. Based on measured data exposure values are assessed for two groups of workers namely cleaners and other workers.

In an artificial shooting target factory, targets (clay pigeons) are made of chalk (70-75%) and a basic binder (23-30%) like CTP(ht). There is potential for dermal and inhalation exposure during packing where packers may handle nude targets or painted targets without gloves, maintenance of presses and conveyor belts and during tasks performed by the foreman. Indications show that this scenario is a minor market.

The estimated exposure levels for CTP(ht) are presented in **Table 4.1**.

Table 4.1 Summary of occupational exposure to CTP(ht); exposure expresses using BaP as primary indicator

Scenario/sub scenario	Estimated inhalation exposure level (µg/m)						Skin exposure (mg/day)	
	Full-shift (8 hour time weighted average)				Short term		Full-Shift	
	Typical	Method	RWC	Method	RWC	Method	RWC	Method
1. Production of Coal Tar Pitch (ht) 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								
a. Søderberg potrooms (not modernised)	1	measured	8	measured	17	measured	0.5	modelled
b. Søderberg potrooms (modernised)	0.20	measured	0.35	measured	0.75	measured	0.5	modelled
c. Anode bake plants	0.15	measured	0.40	measured	1.40	measured	0.5	modelled
d. 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 judg.	100	modelled
4. Use – Binder for refractories								
a. Production of refractories	0.17	measured	3.5	measured	7	expert judg.	na	-
b. Use of refractories	0.63	measured	23	measured	64	expert judg.	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 Production	670	measured	1760	measured	2200	measured	10	modelled
b. Cleaning	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	measured

Notes to summary table:
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.
RWC – reasonable worst case.
expert judg. – expert judgement

4.1.2 Consumer exposure

Consumer use was not identified by industry, not in literature nor on the Internet. Therefore the exposure to consumers to CTP(ht) can be considered negligible.

4.1.3 Man exposed indirectly via the environment

Like in the environmental risk assessment, 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. 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.

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 Kow, 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 (B(a)P) 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 B(a)P can be considered one of the most toxic PAHs. For this reason the risk assessment will be focussed on the exposure to B(a)P. In case a risk is identified already for this one PAH, the other 15 PAHs will not be considered further.

The estimated concentrations of B(a)P in air and food and the resulting estimated human daily intake are given in **Table 4.2** and **Table 4.3**, respectively.

Table 4.2 Estimated concentrations of Benzo(a)pyrene in air and food for humans

source	Air (ng/m^3)	Root crops (µg/kg)	Leaf crops (µg/kg)	Meat (µg/kg)	Milk (µg/kg)	Drinking water (ng/L)
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	92	9.7	220	510	160	1.1
S4	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

Table 4.3 Estimated human daily intake[1] of Benzo(a)pyrene via environmental routes in ng/kg bw/d

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	26	53	3800	2200	1300	0.30	7400
S4	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	589
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

Regional exposure via the environment

Since many unintentional sources contribute to the total emission of PAHs into the environment, which by extension are not related to production and use of CTP(ht), the risk characterisation will only be focussed on the PAHs emitted by producers and downstream users of CTP(ht) 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 and fresh water environment. No formal conclusions will be derived for the regional background.

4.2 EFFECTS ASSESSEMENT

The database on possible health hazards induced by CTP(ht) 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 CTP(ht) is produced and/or used, that indicate that carcinogenicity is a striking hazard associated with CTP(ht). This is attributed to the presence of the PAHs in CTP(ht). Given the uncertainties with respect to the effects of other chemical constituents of CTP(ht) and related substances also exposed to, it is not completely sure that carcinogenicity is the only relevant effect of CTP(ht). 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 CTP(ht), it is decided that in this risk characterisation for CTP(ht) 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 BaP as a guidance substance.

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 CTP(ht). 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 CTP(ht) 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 CTP(ht) default values of (in this case) 100% may be used for absorption of critical components via inhalation and oral exposure. It is emphasised 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-Directive 2001/59/EC) 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 conclude whether the observed dermal effects are caused by irritation or/and sensitisation (photosensitisation or sensitisation after repeated exposure), therefore classification of CTP(ht) 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 CTP(ht) required as specified in Annex VIIA of Directive 67/548/EEC were available. Taking the available human and animal data into account, there are no indications that CTP has corrosive properties. According to section 1.7.2.1 of Annex VI of Directive 67/548, complex substances containing more than 1% of a skin sensitising substance need to be classified as a skin sensitiser. Since CTP(ht) may contain up to 1.5% BaP (a skin sensitiser) it is proposed to classify CTP(ht) as a skin sensitiser(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 CTP(ht) 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 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 CTP(ht).

The data set available on the mutagenicity/genotoxicity of CTP(ht) does not meet the basis requirements as specified in Annex VIIA of Directive 67/548/EEC. From mutagenicity testing in *Salmonella 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 psoriasis patients (to coal-tar applications), and coke oven, and carbon plant workers.
There were no data on *in vivo* genotoxicity testing of CTP(ht) 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 section 1.7.2.1 of Annex VI of Directive 67/548, complex substances containing more than 0.1% of a category 1 or 2 mutagen need to be classified as a category 1 or 2 mutagen. CTP(ht) may contain a variable amount of mutagenic PAHs. The mutagenic effect of these individual PAHs may be considered at least additive. Since CTP(ht) may at least contain up to 1.5% BaP (a category 2 mutagen), the amount of category 2 mutagens in CTP(ht) is estimated to be more than 0.1% in nearly if not all circumstances.
Based on the amount of category 2 mutagens in CTP(ht) and the available genotoxicity data on CTP(ht), CTPV(ht), coal tar, coal tar waste, coal tar products, and individual PAHs, classification of CTP(ht) as a category 2 mutagen is proposed (T; R46).

There were no data available on the potential carcinogenicity of CTP(ht) 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 BaP resulted in increased tumour incidences in amongst others the liver, forestomach, and auditory canal in rats and forestomach and upper GI tract in mice.
Inhalation of CTP(ht) caused lung tumours in rats and mice, while dermal exposure to CTP(ht) 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 CTP(ht) 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 and by the UK Health and Safety Executive (HSE). The IARC concluded that there is sufficient evidence that coal-tar pitches are carcinogenic in humans already in 1985. 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)[4] 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. These meta-analyses estimates are considered the best estimates of the risk on lung and bladder cancer risk due to exposure of CTP(ht). 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 (CI): 1.11-1.29) per *unit* of 100 μg/m^3.year cumulative BaP exposure[5]. Furthermore, for aluminium smelters, the only industry exposed to CTPV(ht) 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 μg/m^3.year cumulative BaP exposure is forwarded to the risk characterisation. Furthermore, for aluminium smelters, the only industry exposed to CTPV(ht) 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 μg/m^3.year cumulative BaP exposure for bladder cancer. This value will be taken forward to the risk characterisation for aluminium smelters.
Based on the available experimental and epidemiological data on the carcinogenicity of CTP(ht) and CTPV(ht) and the evaluation of these data by the IARC, CTP(ht) and CTPV(ht) will be classified as a category 1 carcinogen (T; R45).
Based on the genotoxic and carcinogenic properties of CTP(ht), for risk characterisation a non-threshold approach will be adopted.

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTP(ht). 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).

[4] Armstrong B, Hutchinson,E Fletcher T. (2003) Cancer risk following exposure to polycyclic aromatic hydrocarbons (PAHs): a meta-analysis. Rep No 068. Sudbury, UKL Health and safely Executive.
Armstrong B, Hutchchinson E, Unwin J, Fletcher T. (2004) Lung Cancer Risk after Exposure to Polycyclic Aromatic Hydrocarbons: A Review and Meta-Analysis. *Envinron Helath Perspect* 112 (9): 970-978.

[5] The indicator function of BaP is rather scenario-specific: i.e. the amount of total PAHs may correlate well with the airborne concentration of BaP (in μg/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 CTP(ht) and CTPV(ht), 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, industry-specific data were only available for the aluminium smelter industry.

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 one study, 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 section 1.7.2.1 of Annex VI of Directive 67/548, complex substances 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 CTP(ht) may contain up to 1.5% BaP, which is classified for effects on reproduction (category 2; T, R.60/61), it is proposed to classify CTP(ht) as toxic to reproduction(T; R60/61).

4.3 RISK CHARACTERISATION

4.3.1 Workplace

An overview of the occupational exposure to CTP(ht) is given in **Table 4.1**.

Assuming that oral exposure is prevented by personal hygienic measures, the risk characterisation for workers is limited to the dermal and inhalation routes of exposure.

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 CTP(ht) is of no concern for workers with regard to acute systemic effects (**conclusion ii**).

Irritation and corrosivity
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 CTP(ht).
However, since it is concluded that the carcinogenic activity of CTP(ht) is the critical effect, the need for more information on local skin effects of CTP(ht) 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 CTP(ht) as irritant with risk of serious damage to eyes (Xi, R41). Although the data are insufficient for quantitative risk characterisation, it is concluded that CTP(ht) is of concern for workers. However, if the required protection is strictly adhered to, exposure will occur only incidentally, so **conclusion ii** is justifiable.

Corrosivity

No experimental data on the potential corrosivity of CTP(ht) are available, however taking the available human and animal data into account, there are no indications that CTP(ht) has corrosive properties, so **conclusion ii** is justifiable.

Sensitisation

No experimental data on the sensitisation potential of CTP(ht) are available. However, since CTP(ht) may contain up to 1.5% BaP, which is classified for skin sensitisation, it is proposed to classify CTP(ht) as a skin sensitiser (R43). The available data are insufficient for a quantitative risk characterisation. However, as sensitisation is considered a non-threshold effect, it is concluded that CTP(ht) is of concern for workers (**conclusion iii**).

Repeated dose toxicity

No valid experimental animal studies addressing the potential non-carcinogenic effects of CTP(ht) 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)). However, exposure was as well to 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 CTP(ht) is the critical effect, the need for more information on non-carcinogenic effects of CTP(ht) after repeated exposure will be revised in the light of the risk reduction strategy due to its carcinogenic properties (**conclusion i on hold**).

Mutagenicity

Based on the proposal to classify CTP(ht) as a category 2 mutagen, it is concluded that exposure to CTP(ht) is associated with a mutagenic risk: **conclusion iii**.

Carcinogenicity

Based on the available experimental and epidemiological data and the evaluation of these data by the IARC, it is concluded that CTP(ht) and CTPV(ht) should be classified as category 1 carcinogens. Human data are mainly available on lung and bladder cancer risk in occupationally CTPV(ht)-exposed cohorts. Although a considerable number of epidemiological studies on CTPV(ht) 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. Recently, a meta-analysis on lung and bladder cancer risk after exposure to PAHs has been published by Armstrong et al. (2003; 2004). As exposure to BaP has been adopted as the primary indicator of exposure to CTPV(ht) at the workplace 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 CTPV(ht).

In this meta-analysis, unit relative risks (URRs) for lung and bladder cancer were estimated by fitting a log-linear model to the data. An overall URR per unit of 100 μg/m^3.year cumulative BaP 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 URR estimates in aluminium smelters, the only industry exposed to CTPV(ht) 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 BaP 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. A linear relative risk model ($RR = 1 + bx$) 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. In the meta-analysis also a linear model was fitted, resulting in an overall URR 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 URRs derived from the two models revealed that studies in industries with relatively low exposure, for example tar distillation, had very high URRs in the log-linear model, but lower URRs in the linear model. The explanation is that the benchmark exposure of 100 μg/m^3.year cumulative BaP 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 URRs are overestimated using the results from the log-linear model due to extrapolation. See figure 1 for an illustration with a hypothetical example. Therefore industry-specific URRs estimated with the log-linear model should not be used for industries for which the benchmark exposure (100 μg/m^3.year cumulative BaP) is far higher than the observed exposure range.

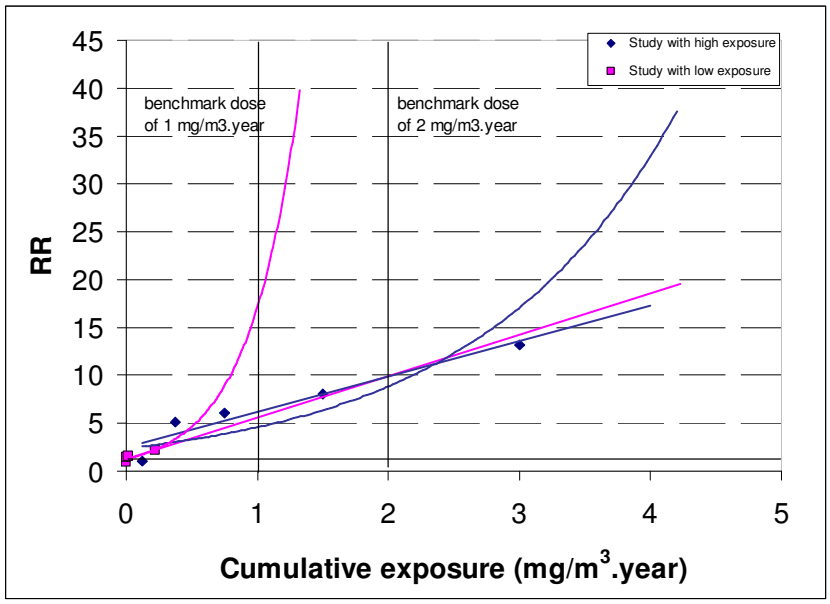

Figure 1. Influence of the choice of a benchmark dose on predicted relative risk: a hypothetical example of fitting log-linear and linear slopes to exposure-risk data points from studies with relatively low exposure (below 1 mg/m^3.yr, red) to studies with relatively high exposure 0-3 mg/m^3.yr, blue). 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.

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 URRs 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 URRs 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 BaP exposure) should not be compared with the URR from the log-linear model, but with that from the linear model instead. This URR was 1.19 for lung cancer (no confidence interval and no URR for bladder cancer were presented). At exposures within the range of the data from which the URRs were estimated, the log-linear and linear models will give similar estimates.

Excess lifetime risk (ELR) was calculated from the RR at the reasonable worst case (RWC) exposure estimated for the specified exposure scenarios (see **Table 4.1**) with the formula: ELR = RR*P –P, in which P denotes the background risk in the exposed target population (i.e., the population figuring in the exposure scenario) (see **Table 4.4** and **Table 4.5**).
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 (TGD Human Health Risk Characterisation, 2005)): for this reason typical exposure values are included as well.
The RR at the exposure level specified in the exposure scenario, was calculated from the URR at 100 µg/m^3.year cumulative BaP derived from the log-linear model as follows: $RR_x = URR^{x/100}$ and from the linear model as follows: RRx = 1+ (URR – 1) x/100. 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.
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). 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.
Because only data on airborne concentrations are available from the epidemiological studies, the (8-hour Time Weighted Average of the) airborne concentration of BaP (in $\mu g/m^3$) is used for risk assessment. It is assumed that in the epidemiological studies, the effects of combined exposure (inhalation and dermal) were studied. Assuming a constant (linear) relation 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.

Table 4.4 Occupational lung and bladder cancer risk characterisation workers using RWC exposure values

Exposure scenario	Cancer type	Estimated RWC exposure (TWA[1] of airborne concentration) (µg/m³ BaP)	Estimated RWC cumulative exposure [2] (µg/m³ BaP.year)	Estimated unit relative risk (URR) (per 100 µg/m³ BaP.year)	Model	Calculated relative risk at the estimated cumulative exposure level	Order of magnitude of estimated excess lifetime risk (ELR)	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 electode 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	Linear estimate n.a.[3]			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

Exposure scenario	Cancer type	Estimated RWC exposure (TWA[1] of airborne concentration) (µg/m³ BaP)	Estimated RWC cumulative exposure [2] (µg/m³ BaP.year)	Estimated unit relative risk (URR) (per 100 µg/m³ BaP.year)	Model	Calculated relative risk at the estimated cumulative exposure level	Order of magnitude of estimated excess lifetime risk (ELR)	Conclusion
	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	Linear estimate n.a. [3]			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	Linear estimate n.a. [3]			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	Linear estimate n.a. [3]			10^{-2}	iii
7 a. Use – Binder in coal briquetting - Production	lung	1760	70400	1.19	linear	135	$>10^{-1}$	iii
	bladder	1760	70400	Linear estimate n.a. [3]			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	Linear estimate n.a. [3]			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] TWA: Time Weighted Average over 8 hours;
[2] TWA x 40 year;
[3] Linear URR estimates were not available for bladder cancer (indicated by n.a). In these cases, the ELR for bladder cancer was assumed to be approximately one third of that for lung cancer as the ELR values for bladder cancer are about one third of the ELR value for lung cancer for each scenario for which the log-linear method was used.

Table 4.5 Occupational lung and bladder cancer risk characterisation workers using typical exposure values

Exposure scenario	Cancer type	Estimated RWC exposure (TWA[1] of airborne concentration) (µg/m³ BaP)	Estimated RWC cumulative exposure [2] (µg/m³ BaP.year)	Estimated unit relative risk (URR) (per 100 µg/m³ BaP.year)	Model	Calculated relative risk at the estimated cumulative exposure level	Order of magnitude of estimated excess lifetime risk (ELR)	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	lung	1	40	1.16 (CI): 1.05-1.28)	log-linear	1.06 (CI: 1.02-1.10)	10^{-2}	iii
Not modernised	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	lung	0.20	8	1.16 (CI): 1.05-1.28)	log-linear	1.01 (CI: 1.0-1.02)	10^{-3}	iii
Modernised	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 electode 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
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	Linear estimate n.a.[3]			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

Exposure scenario	Cancer type	Estimated RWC exposure (TWA[1] of airborne concentration) (µg/m³ BaP)	Estimated RWC cumulative exposure [2] (µg/m³ BaP.year)	Estimated unit relative risk (URR) (per 100 µg/m³ BaP.year)	Model	Calculated relative risk at the estimated cumulative exposure level	Order of magnitude of estimated excess lifetime risk (ELR)	Conclusion
	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	Linear estimate n.a. [3]			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	Linear estimate n.a. [3]			10^{-2}	iii
7 b. Use - Binder in coal briquetting - Cleaning	lung	14	560	1.19	linear	2.06	$>10^{-1}$	iii
	bladder	14	560	Linear estimate n.a. [3]			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] TWA: Time Weighted Average over 8 hours;
[2] TWA x 40 year;
[3] Linear URR estimates were not available for bladder cancer (indicated by n.a). In these cases, the ELR for bladder cancer was assumed to be approximately one third of that for lung cancer as the ELR values for bladder cancer are about one third of the ELR value for lung cancer for each scenario for which the log-linear method was used.

Table 4.6 Comparison of occupational lung and bladder cancer risk characterisation for workers using RWC and typical exposure values

Exposure scenario	Cancer type	Estimated **RWC** cumulative exposure (µg/m³ BaP.year)	Order of magnitude of estimated excess lifetime risk (ELR)	Conclusion		Estimated **typical** cumulative exposure (µg/m³ BaP.year)	Order of magnitude of estimated excess lifetime risk (ELR)	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	lung	320	10^{-2}	iii		40	10^{-2}	iii
Not modernised	bladder	320	10^{-2}	iii		40	10^{-3}	iii
2 i ab. Søderberg potroom	lung	14	10^{-3}	iii		8	10^{-3}	iii
Modernised	bladder	14	10^{-2}	iii		8	10^{-3}	iii
2 i bc. Anode bake and past 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 electode 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

Exposure scenario	Cancer type	Estimated **RWC** cumulative exposure (µg/m³ BaP.year)	Order of magnitude of estimated excess lifetime risk (ELR)	Conclusion		Estimated **typical** cumulative exposure (µg/m³ BaP.year)	Order of magnitude of estimated excess lifetime risk (ELR)	Conclusion
	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
7 a. Use – Binder in coal briquetting - Production	lung	70400	$>10^{-1}$	iii		26800	$>10^{-1}$	iii
	bladder	70400	10^{-2}	iii		26800	10^{-2}	iii
7 b. Use - Binder in coal briquetting - Cleaning	lung	1600	10^{-1}	iii		560	$>10^{-1}$	iii
	bladder	1600	10^{-2}	iii		560	10^{-2}	iii
8. Binder for clay pigeons	lung	120	10^{-2}	iii		40	10^{-2}	iii
	bladder	120	10^{-2}	iii		40	10^{-3}	iii

All ELR values listed in **Table 4.4** and **Table 4.5** are equal or higher than an additional risk level of 1 x 10^{-4} (see also **Table 4.6**). 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 CTP(ht) and CTPV(ht) 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.

Toxicity for reproduction
No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTP(ht). 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 CTP(ht) may contain up to 1.5% BaP (classified as toxic for effects on reproduction (category 2)) it is proposed to classify CTP(ht) as toxic to reproduction (category 2). Although the data are insufficient for quantitative risk characterisation, it is concluded that CTP(ht) is of concern for workers. However, since it is concluded that the carcinogenic activity of CTP(ht) is the critical effect, the need for more information on the reproductive toxicity of CTP(ht) will be revised in the light of the risk reducing strategy due to its carcinogenic properties (**conclusion i on hold**).

4.3.2 Consumers

Since there is no consumer exposure, no risk characterisation is performed.

4.3.3 Man indirectly 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 CTP(ht), attributable to the presence of PAHs in CTP(ht), and that B(a)P is the best-studied PAH and one of the most toxic ones. Therefore, as a practical solution B(a)P is chosen as the 'leading' PAH on which the risk characterisation will focuss. Although carcinogenicity may not be the only relevant effect of CTP(ht), 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.

Repeated dose toxicity
No valid experimental animal studies or human data addressing the potential non-carcinogenic effects of CTP(ht) were available to the rapporteur. However, since it is concluded that the carcinogenic activity of CTP(ht) is the critical effect, the need for more information on non-carcinogenic effects of CTP(ht) after repeated exposure will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion **i on hold**).

Mutagenicity
Based on the classification of CTP(ht) as a category 2 mutagen, it is concluded that exposure to CTP(ht) is associated with a mutagenic risk: conclusion **iii**.

Carcinogenicity
CTP(ht) and CTPV(ht) are classified as category 1 carcinogens. For quantitative risk assessment, valid human data (mainly in occupationally CTPV(ht)-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 CTP(ht) can be determined using B(a)P 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 characterisation for workers, starting points for the risk characterisation for humans exposed inhalatory via the environment are the airborne concentrations of B(a)P from table 4.2 and the unit relative risks (URRs) 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 B(a)P as indicator of exposure. For lung cancer, the overall URR per unit of 100 µg/m^3.year cumulative B(a)P exposure was 1.20 (95% confidence interval: 1.11-1.29), 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 RRs at the (cumulative) exposure level were calculated from the URRs at 100 µg/m^3.year cumulative B(a)P as follows: $RR_x = URR^{x/100}$. Subsequently, excess lifetime risks (ELR) were calculated from the RRs with the formula: ELR = RR*P –P, in which 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.

With a few exceptions (sites PA3 and PA15), all ELR values were equal to or higher than an additional risk level of 1 x 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 CTP(ht) 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 B(a)P 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 characterisation for humans exposed orally via the environment are the intake estimates for B(a)P from table 4.3 and the overall dose descriptor T25 derived for B(a)P from the oral carcinogenicity studies in mice and rats.

The lowest, overall T25 of 1 mg/kg bw/d is used for the risk characterisation. From this T25 a human T25 (HT25) of 0.14 mg/kg bw/d is calculated by applying an overall assessment factor of 7 to the T25. 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 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 HT25 using the formula: eLR = exposure/(HT25/0.25). The calculated eLRs (point estimates) were rounded to the nearest order of magnitude.

All eLR values were equal to or higher than an additional risk level of 1 x 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 B(a)P, 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 B(a)P alone.

Exposure via air and food and water - Regional

As indicated in section 4.1.3, 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 B(a)P concentrations found in air (0.02 and 39 ng/m^3, respectively) and for the resulting lowest and highest total daily B(a)P intake (1.6 and 3100 ng/kg bw/d, respectively), in the same way as described above for the local exposures. The results are presented in **Table 4.7**.

Table 4.7 Cancer risk characterisation for humans exposed via the environment – regional

Regional	ELR	eLR	Conclusion
Air concentration of B(a)P			
0.02 ng/m^3	10^{-7} (lung)		iii[a]
	10^{-7} (bladder)		iii[a]
39 ng/m^3	10^{-4} (lung)		iii
	10^{-4} (bladder)		iii
Total daily intake of B(a)P			
1.6 ng/kg bw/d		10^{-6}	iii
3100 ng/kg bw/d		10^{-2}	iii

[a] Low concern

Toxicity for reproduction

No valid experimental animal studies were available which addressed the potential reproduction toxicity of CTP(ht). 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 CTP(ht) may contain up to 1.5% BaP (classified as toxic for effects on reproduction (category 2)), CTP(ht) is classified as toxic to reproduction (category 2). Although the data are insufficient for quantitative risk characterisation, it is concluded that CTP(ht) is of concern for humans exposed indirectly via the environment. However, since it is concluded that the carcinogenic activity of CTP(ht) is the critical effect, the need for more information on the

reproductive toxicity of CTP(ht) will be revised in the light of the risk reduction strategy due to its carcinogenic properties (conclusion **i on hold)**.

4.4 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

Based on the available information, CTP(ht) is not flammable, not explosive and not oxidising. Therefore, CTP(ht) is expected to be of no concern for human health regarding physico-chemical properties (**conclusion ii**).

5 OVERALL RESULTS OF THE RISK ASSESSMENT

5.1 ENVIRONMENT

5.2 HUMAN HEALTH

5.2.1 Human health (toxicity)

Workers

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, eye irritation, and corrosivity.

Conclusion (iii) applies to:
- skin sensitisation, the substance is considered a skin sensitiser and occupational dermal exposure cannot be excluded in several scenarios;
- mutagenicity and carcinogenicity, effects that cannot be excluded for exposure (inhalation and dermal) arising from production and use as an intermediate.

Consumers

Not applicable, since there is no consumer exposure.

Humans exposed via the environment

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

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 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 (iii) applies to mutagenicity and carcinogenicity, effects that cannot be excluded for exposure (inhalation and oral) via the environment.

5.2.2 Human health (risks from physico-chemical properties)

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.

GLOSSARY

Standard term Abbreviation	Explanation/Remarks and Alternative Abbreviation(s)
Ann.	Annex
AF	assessment factor
BCF	bioconcentration factor
bw	body weight / *Bw*, *b.w.*
°C	degrees Celsius (centigrade)
CAS	Chemical Abstract System
CEC	Commission of the European Communities
CEN	European Committee for Normalisation
CEPE	European Council of the Paint, Printing Ink and Artists' Colours Industry
d	day(s)
d.wt	dry weight / dw
DG	Directorate General
DT_{50}	period required for 50 percent dissipation (define method of estimation)
DT_{50lab}	period required for 50 percent dissipation under laboratory conditions (define method of estimation)
DT_{90}	period required for 90 percent dissipation (define method of estimation)
$DT_{90field}$	period required for 90 percent dissipation under field conditions (define method of estimation)
EC	European Communities
EC	European Commission
EC_{50}	median effective concentration
EEC	European Economic Community
EINECS	European Inventory of Existing Commercial Chemical Substances
EU	European Union
EUSES	European Union System for the Evaluation of Substances
f_{oc}	Fraction of organic carbon
G	gram(s)

PNEC(s)	Predicted No Effect Concentration(s)
$PNEC_{water}$	Predicted No Effect Concentration in Water
(Q)SAR	Quantitative Structure Activity Relationship
STP	Sewage Treatment Plant
TGD	Technical Guidance Document[6]
UV	Ultraviolet Region of Spectrum
UVCB	Unknown or Variable composition, Complex reaction products or Biological material
v/v	volume per volume ratio
w/w	weight per weight ratio
w	gram weight
GLP	Good Laboratory Practice
h	hour(s)
ha	Hectares / *h*
HPLC	High Pressure Liquid Chromatography
IARC	International Agency for Research on Cancer
C_{50}	median immobilisation concentration or median inhibitory concentration 1 / *explained by a footnote if necessary*
ISO	International Standards Organisation
IUPAC	International Union for Pure Applied Chemistry
kg	kilogram(s)
kPa	kilo Pascals
K_{oc}	organic carbon adsorption coefficient
K_{ow}	octanol-water partition coefficient
Kp	Solids water partition coefficient
l	litre(s)
log	logarithm to the basis 10
$L(E)C_{50}$	Lethal Concentration, Median
LEV	Local Exhaust Ventilation
m	Meter

[6] Commission of the European Communities, 1996. Technical Guidance Documents in Support of the Commission Directive 93/67/EEC on risk assessment for new substances and the Commission Regulation (EC) No 1488/94 on risk assessment for existing substances. Commission of the European Communities, Brussels, Belgium.
ISBN 92-827-801[1234]

µg	microgram(s)
mg	milligram(s)
MAC	Maximum Accessibility Concentration
MOS	Margins Of Safety
NOAEL	No Observed Adverse Effect Level
NOEC	No Observed Effect Concentration
NOEL	No Observed Effect Level
OEL	Occupational Exposure Limit
OECD	Organisation for Economic Co-operation and Development
OJ	Official Journal
pH	potential hydrogen -*logarithm* (to the base 10) of the hydrogen ion concentration $\{H^+\}$
pKa	-*logarithm* (to the base 10) of the acid dissociation constant
pKb	-*logarithm* (to the base 10) of the base dissociation constant
Pa	Pascal unit(s)
PEC	Predicted Environmental Concentration
STP	Sewage Treatment Plant
WWTP	Waste Water Treatment Plant