

IPCS

INTERNATIONAL PROGRAMME ON CHEMICAL SAFETY



Concise International Chemical Assessment Document

62

Coal tar creosote

IOMC

INTER-ORGANIZATION PROGRAMME FOR THE SOUND MANAGEMENT OF CHEMICALS
A cooperative agreement among UNEP, ILO, FAO, WHO, UNIDO, UNITAR and OECD



World Health Organization
Geneva, 2004

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Concise International Chemical Assessment Document 62

COAL TAR CREOSOTE

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The **International Programme on Chemical Safety (IPCS)**, established in 1980, is a joint venture of the United Nations Environment Programme (UNEP), the International Labour Organization (ILO), and the World Health Organization (WHO). The overall objectives of the IPCS are to establish the scientific basis for assessment of the risk to human health and the environment from exposure to chemicals, through international peer review processes, as a prerequisite for the promotion of chemical safety, and to provide technical assistance in strengthening national capacities for the sound management of chemicals.

The **Inter-Organization Programme for the Sound Management of Chemicals (IOMC)** was established in 1995 by UNEP, ILO, the Food and Agriculture Organization of the United Nations, WHO, the United Nations Industrial Development Organization, the United Nations Institute for Training and Research, and the Organisation for Economic Co-operation and Development (Participating Organizations), following recommendations made by the 1992 UN Conference on Environment and Development to strengthen cooperation and increase coordination in the field of chemical safety. The purpose of the IOMC is to promote coordination of the policies and activities pursued by the Participating Organizations, jointly or separately, to achieve the sound management of chemicals in relation to human health and the environment.

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FOREWORD

Concise International Chemical Assessment Documents (CICADs) are the latest in a family of publications from the International Programme on Chemical Safety (IPCS) — a cooperative programme of the World Health Organization (WHO), the International Labour Organization (ILO), and the United Nations Environment Programme (UNEP). CICADs join the Environmental Health Criteria documents (EHCs) as authoritative documents on the risk assessment of chemicals.

International Chemical Safety Cards on the relevant chemical(s) are attached at the end of the CICAD, to provide the reader with concise information on the protection of human health and on emergency action. They are produced in a separate peer-reviewed procedure at IPCS. They may be complemented by information from IPCS Poison Information Monographs (PIM), similarly produced separately from the CICAD process.

CICADs are concise documents that provide summaries of the relevant scientific information concerning the potential effects of chemicals upon human health and/or the environment. They are usually based on selected national or regional evaluation documents or on existing EHCs. Before acceptance for publication as CICADs by IPCS, these documents undergo extensive peer review by internationally selected experts to ensure their completeness, accuracy in the way in which the original data are represented, and the validity of the conclusions drawn.

The primary objective of CICADs is characterization of hazard and dose-response from exposure to a chemical. CICADs are not a summary of all available data on a particular chemical; rather, they include only that information considered critical for characterization of the risk posed by the chemical. The critical studies are, however, presented in sufficient detail to support the conclusions drawn. For additional information, the reader should consult the identified source documents upon which the CICAD has been based.

Risks to human health and the environment will vary considerably depending upon the type and extent of exposure. Responsible authorities are strongly encouraged to characterize risk on the basis of locally measured or predicted exposure scenarios. To assist the reader, examples of exposure estimation and risk characterization are provided in CICADs, whenever possible. These examples cannot be considered as representing all

possible exposure situations, but are provided as guidance only. The reader is referred to EHC 170.¹

While every effort is made to ensure that CICADs represent the current status of knowledge, new information is being developed constantly. Unless otherwise stated, CICADs are based on a search of the scientific literature to the date shown in the executive summary. In the event that a reader becomes aware of new information that would change the conclusions drawn in a CICAD, the reader is requested to contact IPCS to inform it of the new information.

Procedures

The flow chart on page 2 shows the procedures followed to produce a CICAD. These procedures are designed to take advantage of the expertise that exists around the world — expertise that is required to produce the high-quality evaluations of toxicological, exposure, and other data that are necessary for assessing risks to human health and/or the environment. The IPCS Risk Assessment Steering Group advises the Coordinator, IPCS, on the selection of chemicals for an IPCS risk assessment based on the following criteria:

- there is the probability of exposure; and/or
- there is significant toxicity/ecotoxicity.

Thus, it is typical of a priority chemical that

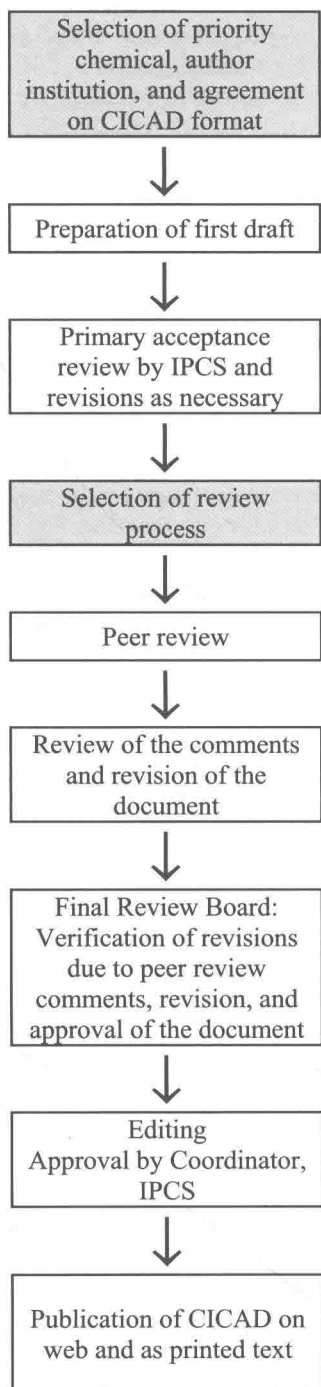
- it is of transboundary concern;
- it is of concern to a range of countries (developed, developing, and those with economies in transition) for possible risk management;
- there is significant international trade;
- it has high production volume;
- it has dispersive use.

The Steering Group will also advise IPCS on the appropriate form of the document (i.e., a standard CICAD or a *de novo* CICAD) and which institution bears the responsibility of the document production, as well as on the type and extent of the international peer review.

The first draft is usually based on an existing national, regional, or international review. When no appropriate source document is available, a CICAD may be produced *de novo*. Authors of the first draft are usually, but not necessarily, from the institution that developed the original review. A standard outline has been developed to encourage consistency in form. The

¹ International Programme on Chemical Safety (1994) *Assessing human health risks of chemicals: derivation of guidance values for health-based exposure limits*. Geneva, World Health Organization (Environmental Health Criteria 170) (also available at <http://www.who.int/pcs/>).

CICAD PREPARATION FLOW CHART



Advice from Risk Assessment Steering Group

Criteria of priority:

- there is the probability of exposure; and/or
- there is significant toxicity/ecotoxicity.

Thus, it is typical of a priority chemical that

- it is of transboundary concern;
- it is of concern to a range of countries (developed, developing, and those with economies in transition) for possible risk management;
- there is significant international trade;
- the production volume is high;
- the use is dispersive.

Special emphasis is placed on avoiding duplication of effort by WHO and other international organizations.

A usual prerequisite of the production of a CICAD is the availability of a recent high-quality national/regional risk assessment document = source document. The source document and the CICAD may be produced in parallel. If the source document does not contain an environmental section, this may be produced *de novo*, provided it is not controversial. If no source document is available, IPCS may produce a *de novo* risk assessment document if the cost is justified.

Depending on the complexity and extent of controversy of the issues involved, the steering group may advise on different levels of peer review:

- standard IPCS Contact Points
- above + specialized experts
- above + consultative group

first draft undergoes primary review by IPCS to ensure that it meets the specified criteria for CICADs.

The second stage involves international peer review by scientists known for their particular expertise and by scientists selected from an international roster compiled by IPCS through recommendations from IPCS national Contact Points and from IPCS Participating Institutions. Adequate time is allowed for the selected experts to undertake a thorough review. Authors are required to take reviewers' comments into account and revise their draft, if necessary. The resulting second draft is submitted to a Final Review Board together with the reviewers' comments. At any stage in the international review process, a consultative group may be necessary to address specific areas of the science. When a CICAD is prepared *de novo*, a consultative group is normally convened.

The CICAD Final Review Board has several important functions:

- to ensure that each CICAD has been subjected to an appropriate and thorough peer review;
- to verify that the peer reviewers' comments have been addressed appropriately;
- to provide guidance to those responsible for the preparation of CICADs on how to resolve any remaining issues if, in the opinion of the Board, the author has not adequately addressed all comments of the reviewers; and
- to approve CICADs as international assessments.

Board members serve in their personal capacity, not as representatives of any organization, government, or industry. They are selected because of their expertise in human and environmental toxicology or because of their experience in the regulation of chemicals. Boards are chosen according to the range of expertise required for a meeting and the need for balanced geographic representation.

Board members, authors, reviewers, consultants, and advisers who participate in the preparation of a CICAD are required to declare any real or potential conflict of interest in relation to the subjects under discussion at any stage of the process. Representatives of nongovernmental organizations may be invited to observe the proceedings of the Final Review Board. Observers may participate in Board discussions only at the invitation of the Chairperson, and they may not participate in the final decision-making process.

1. EXECUTIVE SUMMARY

The first draft of this CICAD was prepared by the Fraunhofer Institute of Toxicology and Experimental Medicine, Hanover.¹ A comprehensive literature search of relevant databases was performed in June 2002. The first draft of this document was circulated for a limited peer review, and a Consultative Group was convened to finalize the document and verify that the peer review comments had been adequately dealt with. The members of the Consultative Group, who were participants in this peer review, are provided in Appendix 2. The final draft was then sent for peer review to IPCS Contact Points and Participating Institutions, as well as to further experts identified in collaboration with the IPCS Risk Assessment Steering Group. Information on the peer review of this CICAD is presented in Appendix 3. This CICAD was approved as an international assessment at a meeting of the Final Review Board, held in Varna, Bulgaria, on 8–11 September 2003. The members of the Final Review Board are listed in Appendix 4. The International Chemical Safety Card for creosote (ICSC 0572), produced by the International Programme on Chemical Safety (IPCS, 2002), has also been reproduced in this document.

1.1 Identity, physical/chemical properties, and analytical methods

This CICAD is on coal tar creosote. Wood creosote is a different product that is used mainly in pharmaceutical preparations and is not covered in this document.

Coal tar creosote is a brownish-black/yellowish-dark green oily liquid with a characteristic odour, obtained by the fractional distillation of crude coal tars. The approximate distillation range is 200–400 °C. The chemical composition of creosote is influenced by the origin of the coal and also by the nature of the distilling process; as a result, the creosote components are rarely consistent in their type and concentration.

Creosote is a mixture of several hundred, probably a thousand, chemicals, but only a limited number of them are present in amounts greater than 1%. There are six major classes of compounds in creosote: aromatic hydrocarbons, including polycyclic aromatic hydrocarbons (PAHs) and alkylated PAHs (which can constitute up to 90% of creosote); tar acids / phenolics; tar bases / nitrogen-containing heterocycles; aromatic amines; sulfur-containing heterocycles; and oxygen-containing heterocycles, including dibenzofurans. Creosote may be sold as diluted preparations, which may contain carrier oil or solvents. The composition and use of creosote are

regulated in some countries; the regulations usually focus on the content of benzo[*a*]pyrene (BaP) and phenolics.

Creosote is only slightly soluble in water and soluble in a variety of organic solvents. However, the physical and chemical properties of the individual components of creosote vary widely; some, for example, are highly soluble in water.

The analysis of creosote is complex. Different profiles of creosote chemicals are found in the different matrices: the most volatile are found in air, the most soluble in water, and those with greater sorptive capacity in sediment/soil. Depending upon the matrix (e.g., air, water, soil/sediment, biological materials) from which the sample is taken, suitable cleanup and extraction are necessary. High-resolution gas chromatography (HRGC) with a flame ionization detector (FID) or mass spectrometric (MS) detection or reversed-phase high-performance liquid chromatography (HPLC) with a fluorescence detector (FL) have been the separation and determination methods most commonly used.

Occupational exposure to airborne creosote particles has been previously monitored as coal tar pitch volatiles (CTPV). However, the CTPV method is not sensitive enough to measure low concentrations of creosote fumes. Important components such as airborne PAHs can be sampled on a polytetrafluoroethylene (PTFE) filter connected to a sorbent tube and analysed after extraction by HRGC or HPLC. Other volatile compounds from creosote can be sampled on sorbent tubes.

The urinary PAH metabolites 1-pyrenol (1-hydroxypyrene) and 1-naphthol (1-hydroxynaphthalene) have been used in the assessment of creosote exposure.

1.2 Sources of human and environmental exposure

Coal tar creosote is a wood preservative and waterproofing agent for structures on land and in marine and fresh waters and for railway crossing timbers and sleepers (railroad ties), bridge and pier decking, poles, log homes, fencing, and equipment for children's playgrounds.

The majority of creosote used in the European Union (EU) is for the pressure impregnation of wood. In the USA and many other countries, the use of coal tar creosote is limited to certified applicators.

Non-wood uses include anti-fouling applications on concrete marine pilings. Creosote can be a component of roofing pitch, fuel oil, and lamp black and a lubricant for die moulds. Other uses reported include animal and bird repellent, insecticide, animal dip, and fungicide.

¹ This CICAD has been prepared *de novo*, and there is no actual source document.

Creosote production in the USA falls into two categories: distillate (100%) creosote and creosote in coal tar solution. Distillate production in 1992 was 240 000 tonnes; production of creosote in coal tar solution was 110 000 tonnes. The production of creosote in the EU has been estimated to be approximately 60 000–100 000 tonnes per year.

During pressure impregnation of wood products, excess creosote may be released from the treated materials. Leaching of spilled wastes from these application sites has been common. Creosote is also released to the environment from facilities through air emissions.

1.3 Environmental transport, distribution, and transformation

The environmental transport and distribution of creosote are complex processes, depending on the physicochemical properties of creosote constituents and their interaction with matrix properties, as well as environmental conditions. Generally, creosote is distributed within all environmental compartments (air, water, sediment, soil, biota). However, the major environmental sinks of creosote components are sediment, soil, and groundwater.

Generally, phenolic compounds, low-molecular-weight PAHs, and some heterocycles tend to be predominantly in the gaseous phase. Creosote constituents may also occur in the atmosphere as particulate matter.

Volatilization of creosote from water surfaces is not considered to be a significant process.

The movement of creosote within aquatic systems is dependent upon the aqueous solubility, affinity to organic phases, and sorptive capacity of the components. Generally, the highly soluble fraction includes phenolic and heterocyclic compounds and low-molecular-weight PAHs. The high-molecular-weight aromatic compounds, with relatively low solubilities and high adsorptive capacities, dominate the associated sediments. However, movement of high-molecular-weight compounds may occur by co-transport of colloid-sorbed contaminants.

Field observations and laboratory leaching experiments have shown losses of creosote components from wooden creosoted constructions during water immersion. Leachability of creosote components was higher in fresh water than in seawater. The rate of migration increased with increasing temperature and decreased with the age of the pilings. Nitrogen-containing heterocycles leached faster than PAHs and dibenzofuran.

The rate of vertical or horizontal transport of creosote components in soil is dependent upon their

physicochemical properties as well as the soil properties and environmental conditions. Laboratory model and field experiments (simulating creosote spills) showed a high retardation of transport of high-molecular-weight compounds coupled with a fast downward migration of low-molecular-weight compounds. Some of the creosote compounds released from treated wood products into surrounding soil may persist for decades.

Creosote PAHs are taken up to a small degree by terrestrial plants and animals. No quantitative data on uptake of creosote compounds are available for farm animals. A number of aquatic invertebrates and fish monitored in field and laboratory studies showed significant uptake of creosote-derived PAHs. Transfer to the human food supply is possible via contaminated seafood.

The biodegradability of creosote constituents is variable. Generally, the efficacy of aerobic degradation is greater than that of anaerobic degradation. Phenolic compounds are relatively easily degraded. Within PAHs, degradability appears to be inversely related to the number of aromatic rings. Some heteroaromatic compounds are quickly removed, whereas others are recalcitrant. Biotransformation of creosote components appears to dominate over mineralization. In some cases, the intermediates formed can be more persistent, mobile, or toxic than their parent compounds.

Besides structural features of the chemicals, a number of other factors, such as bioavailability, microbial adaptation, oxygen supply, and nutrient availability, influence their degradation or transformation *in situ*.

Although little examined, fish appear to metabolize creosote PAHs more rapidly than aquatic invertebrates.

Photochemical transformation seems to be the most important abiotic mechanism by which creosote constituents, such as PAHs and heterocyclic and phenolic compounds, are transformed in the atmosphere and, to a lesser extent, in water and soil. Photo-oxidation prevails over direct photolysis. A study performing irradiation of selected PAHs separately or of the same PAHs present in a creosote mixture showed that there was a trend of decreased photoreactivity in the mixture compared with the individual tests.

Aquatic invertebrates and fish bioaccumulate creosote components, as has been demonstrated mainly for PAHs by field monitoring studies at creosote-contaminated sites, relocation experiments, and laboratory or microcosm studies. Generally, PAH profiles in insects and crayfish were close to that found in sediments, whereas fish had greatly altered ratios for low/high-molecular-weight PAHs. Bioconcentration factors (BCFs) in connection with creosote exposure

have rarely been reported. However, BCFs for PAH components from creosote-contaminated sediments have been estimated to range from 0.3 to 73 000.

A number of remediation strategies have been developed, mainly for creosote-contaminated ground-water and soils. Most of the treatments achieved significant reductions for certain substances, but were not or only partially successful in reducing the toxic potential of the treated matrices.

Creosote-treated wood does not decay in the environment, and therefore its disposal is problematic. Creosote-treated wood should not be incinerated under uncontrolled conditions, as toxicants such as PAHs and halogenated dioxins and furans may be produced.

1.4 Environmental levels and human exposure

The very few data available for ambient air concentrations refer to concentrations of selected PAHs in the vicinity of creosote facilities. A maximum concentration of 90 ng/m³ has been reported for naphthalene at a distance of 2000 m. Concentrations decreased with increasing distances from creosoting plants: for example, from 64 ng/m³ at 500 m to 1.6 ng/m³ at 5000 m for fluoranthene or from 5 ng/m³ at 100 m to 0.6 ng/m³ at 2000 m for BaP.

Groundwater samples near creosote waste sites in several countries have been found to contain creosote-related PAHs and phenolic, heterocyclic, and BTEX (benzene, toluene, ethylbenzene, and xylene) compounds. Monitoring data from 44 Danish creosote sites showed concentrations (90th percentiles) of 30 µg/litre for BaP and 50 µg/litre for chrysene. Highest concentrations of several individual heterocyclic, phenolic, or BTEX compounds detected in the vicinity of several creosote waste sites were in the range of 10–80 mg/litre.

Concentrations in the mg/litre range have been found for some individual PAHs in river water affected by a creosote spill 10 years earlier. Twelve individual PAHs were monitored in water samples of a drainage stream near a creosote works. Maximum concentrations ranged from 0.02 µg/litre (benzo[*b*]- and benzo[*k*]fluoranthene) to 153 µg/litre (naphthalene), with BaP concentrations of up to 0.05 µg/litre.

Elevated PAH concentrations have also been observed in small waterways, where banks were protected with creosoted wood constructions, or in railway ditches, where creosote-treated power or telecommunication line poles were erected. The maximum BaP concentration measured was 2.5 µg/litre. The mean total PAH concentration in the ditches was about 600 µg/litre.

In the vicinity of wood-preserving facilities, maxima for total PAHs in sediments amounted to about 20 000–30 000 mg/kg dry weight; maxima for total nitrogen heterocycles were in the order of 1000 mg/kg dry weight. BaP concentrations as high as several hundred mg/kg dry weight have been measured. The most abundant heterocycle was carbazole (18 mg/kg dry weight). Sediments near creosoted wooden constructions (pilings, bank protection, poles/sleepers) showed total PAH concentrations of up to 1200 mg/kg dry weight, with mean BaP concentrations of about 2 mg/kg dry weight.

Elevated concentrations of creosote-derived compounds have been documented in soils near abandoned creosote-producing/using facilities in several countries, with maximum concentrations of several thousand mg/kg dry weight for total PAHs and of nearly 100 mg/kg for total phenols. Concentrations of “creosote oil contents” up to 90 000 mg/kg dry weight have been reported around creosote-treated poles. Soil from a storage area for impregnated railway ties and playground sand from sandboxes made of old impregnated railway ties contained total PAHs at concentrations up to 20 mg/kg and up to about 2 mg/kg dry weight, respectively. BaP concentrations found in soils near wood treatment/storage sites reached a maximum of 390 mg/kg dry weight, those near creosoted posts, 6 mg/kg, and those from playground sand, 0.2 mg/kg.

Creosoted wood products can contain high concentrations of PAHs, even after several decades of use; phenolic and heterocyclic compounds may also be present. For example, mean concentrations (mg/kg wood) ranging from 1510 (quinoline) to 11 990 (phenanthrene) have been found to occur in creosoted wood. Wooden sleepers installed in playgrounds showed BaP concentrations of up to 1570 mg/kg shavings.

Edible fish and seafood captured from creosote-contaminated areas or held in creosoted cages have been found to contain increased concentrations of PAHs and PAH metabolites. The mean concentration of BaP in tail meat of commercial market lobsters increased from 0.6 to 79 µg/kg wet weight after about 3 months of impoundment.

Creosote-derived PAHs have been detected at concentrations significantly over background levels in several classes of aquatic fauna, including insects, molluscs, crustaceans, and fish collected at various creosote-contaminated sites of freshwater or estuarine/marine environments. In general, concentrations were highest in invertebrates (up to several hundred mg/kg dry weight). Concentrations of total PAHs in liver of fish living on creosote-contaminated sediment and in their invertebrate food organisms were as high as 1 and 84 mg/kg dry weight, respectively (compared with

0.1 and 0.5 mg/kg dry weight in controls). Heterocyclic compounds in snails (*Thais haemastoma*) from a bay near a wood-preserving facility were found to be present at concentrations up to about 10 µg/kg wet weight, and PAHs were present at concentrations up to about 200 µg/kg wet weight.

The general population can be exposed to creosote or creosote components by handling creosote or products containing creosote and by contact with creosote-contaminated air, water, soil, or food. Routes of exposure include inhalation, drinking/ingestion, and skin contact.

Due to the complexity of creosote and the many different exposure situations, exposure may vary both qualitatively and quantitatively. Nevertheless, some estimations using BaP as a marker substance and based on several assumptions have been performed for two important exposure scenarios. As a result, a daily exposure through skin contact of about 2 ng BaP/kg body weight has been assessed for children playing on creosoted playground equipment. The daily intake of BaP from consumption of vegetables and fruits from gardens in the vicinity of creosoting plants has been estimated to range from 1.4 to 71.4 µg/kg body weight.

There is one study providing internal monitoring data for people living in the vicinity of a creosote impregnation plant. Excretion values of 1- and 2-naphthol were significantly higher in the exposed residents than in controls. For example, the mean concentrations of 1-naphthol in morning urine samples were 2.5 µmol/mol creatinine for the exposed and 1.2 µmol/mol creatinine for the non-exposed group. The 1-pyrenol excretion did not differ significantly.

Occupational exposure to creosote may occur during manufacture, use, transport, or disposal of creosote or creosoted wood products. Most data are available for wood-preserving workers.

Creosote aerosol concentrations monitored as the CTPV by similar methods in wood impregnation plants reached maxima of up to 9700 µg/m³. Total time-weighted average (TWA) concentrations of creosote vapours ranged from 0.5 to 9.1 mg/m³, with peaks up to 71 mg/m³, at wood impregnation plants and from 0.1 to 11 mg/m³ at workplaces where creosoted wood was handled. The mean concentrations of particulate PAHs ranged from 0.2 to 106 µg/m³ in the impregnation plants and from 0.8 to 46 µg/m³ in the handling of impregnated wood. The proportion of particulate-bound PAHs relative to total PAHs appeared to be less than 4%.

Prevailing compounds of the vapour phase of wood impregnation plants were naphthalene, methylnaphthalenes, indene, acenaphthene, and fluorene; the main

PAHs of the particulate phase included fluorene, phenanthrene, anthracene, and pyrene. Maximum concentrations of the marker substances naphthalene and BaP (the latter mainly particle-bound) were as high as 41 mg/m³ and 1 µg/m³, respectively. An abundant heterocyclic PAH was benzothiophene, showing concentrations of up to 2800 µg/m³. Concentrations of phenol, biphenyl, and methyl styrenes did not exceed 2000, 1000, and 3000 µg/m³, respectively. Air monitoring during cleanup operations of highly creosote-contaminated soil revealed exposure concentrations of up to 0.9 mg/m³ for volatile PAHs, 0.2 mg/m³ for particulate PAHs, and <0.002 mg/m³ for BaP.

An important route of occupational exposure to creosote is via skin. It has been estimated that over 90% of pyrene and 50–70% of naphthalene enters via the skin. A mean total pyrene contamination on the skin of creosote impregnation workers was approximately 1 mg/day in workers without protective clothing. Protective clothing reduced the pyrene contamination on the workers' skin by about 35%, on average.

Concentrations of two PAH metabolites, 1-naphthol and 1-pyrenol, have been monitored as internal markers of creosote exposure. For example, the mean urinary concentrations of 1-naphthol in Finnish wood impregnation plant workers and in assemblers handling treated wood were 1350 and 1370 µmol/mol creatinine, respectively. The mean urinary concentration of 1-pyrenol was about 10 times higher in these wood impregnators (64 µmol/mol creatinine) than in the assemblers. An increase in urinary 1-pyrenol values during the workshift has also been observed in workers involved in the production of creosote or the cleanup of creosote-contaminated soil. The 1-pyrenol concentrations correlated well with differences in pyrene skin contamination, but poorly with differences in pyrene breathing-zone air concentrations.

Exposure calculations on the basis of excreted metabolites (plus air and/or skin monitoring data) suggested a total daily uptake of 15 or 16 mg/worker (assembler or impregnator) for naphthalene. Estimations for pyrene did not exceed 5 mg/worker per day.

1.5 Comparative kinetics and metabolism in laboratory animals and humans

There are no laboratory animal or human studies measuring the specific rate and extent of coal tar creosote absorption following oral, inhalation, or dermal exposure. However, evidence for a significant absorption of creosote components comes from detection of creosote PAH metabolites in urine of creosote-exposed workers or volunteers and from detection of PAH-DNA adducts in animal or human tissues following creosote exposure. Indirect evidence also comes from the toxic

effects elicited by creosote in laboratory animals or humans. Additionally, single-component studies show a significant absorption potential of individual PAHs, although their predictive value for the quantitative absorption kinetics after exposure to the mixture is limited.

Specific distribution studies on coal tar creosote have not been performed.

In accordance with principal PAH metabolic pathways, hydroxy metabolites of PAHs such as 1-naphthol and 1-pyrenol have been measured in urine of creosote-exposed humans.

In general, PAHs (metabolized or unmetabolized) can be excreted into bile, faeces, and urine as well as into breast milk, regardless of the route of absorption. However, specific studies on the elimination and excretion of coal tar creosote are confined to the determination of PAH metabolites in human urine. Elevated urinary levels of 1-naphthol and 1-pyrenol have been found in workers of several wood creosoting plants and in assemblers handling creosote-impregnated wood. Comparisons between the estimated daily uptake of naphthalene/pyrene by inhalation and the urinary excretion of 1-naphthol/1-pyrenol indicated a remarkable contribution of non-inhalation routes of uptake, especially for pyrene. The relevance of dermal uptake for 1-pyrenol excretion has also been demonstrated in workers using protective clothing, which resulted in a significant reduction of skin contamination and 1-pyrenol excretion. Topical treatment of volunteers with a single dose of creosote significantly enhanced the basal excretion of 1-pyrenol.

Elimination half-lives for 1-naphthol and 1-pyrenol were in the range of hours or days.

Most studies on interactions of creosote with cellular components refer to interactions of creosote PAHs with nucleic acids. PAH-DNA adducts have been detected in mice, rats, and fish after experimental or environmental exposure to creosote.

1.6 Effects on laboratory mammals and *in vitro* test systems

Based on limited studies, creosotes are of low to moderate acute toxicity in experimental animals. The lowest LD₅₀ value, 433 mg/kg body weight, was reported for mice after oral exposure. There is little reliable information on effects of creosotes after short-term exposure. Body weight losses have been observed in rats, sheep, and calves following oral creosote doses.

Some earlier limited studies with mice indicated a carcinogenic activity of creosotes after topical

application. Types of tumours included not only skin carcinomas and papillomas, but also lung carcinomas. A more recent epicutaneous mouse study performed with two different coal tar creosote preparations (CTP1: BaP content of 10 mg/kg; CTP2: BaP content of 275 mg/kg) confirmed the carcinogenic potential of creosotes with respect to induction of skin tumours. There was a linear dose-effect relationship between tumour incidence and BaP content of both creosotes. The creosotes were about 5 times more potent than expected from pure BaP treatments. Non-neoplastic effects observed in this long-term (78 weeks) study included skin ulcerations and decreases in life span.

Several creosotes have been shown to be skin irritants in animals. Data on eye irritancy are conflicting.

There are no adequate animal studies on the reproductive or developmental toxicity of creosotes. However, creosote has been shown to be able to elicit estrogen-mediated activities *in vitro*, indicating some potential for endocrine disruption. Adverse reproductive effects have also been reported in fish exposed to creosote.

A number of *in vitro* tests based upon bacterial and mammalian systems have shown creosote to be genotoxic. The pattern of genotoxicity observed was similar to that found in PAHs. Creosote was also genotoxic in an *in vivo* micronucleus test in mice.

Tests with fish cells in culture showed that the cytotoxicity of creosote is enhanced by irradiation with ultraviolet (UV) light. This is consistent with the known phototoxic potential of some PAHs.

Creosote has been shown to be a hepatic microsomal enzyme inducer in laboratory mammals.

1.7 Effects on humans

1.7.1 General population

Information on the effects of coal tar creosote in the general population is scarce.

Creosote has been involved in incidental or accidental poisoning incidents, mainly due to its use as a pesticide. Deaths occurred following ingestion of about 1–2 g (children) or about 7 g (adults). Symptoms included salivation, vomiting, respiratory difficulties, vertigo, headache, loss of pupillary reflexes, hypothermia, cyanosis, convulsion, etc., accompanied by oropharyngeal, intestinal, pericardial, liver, and kidney damage.

Increased occurrence of skin rashes in people residing in or near an abandoned wood creosoting plant in the USA has been suggested.

Evidence of cancer incidence following environmental exposure is limited to a report on breast and gastrointestinal cancer in females of a population exposed to a creosote-contaminated water supply in the USA. However, it could not clearly be demonstrated whether creosote or confounding risk factors were responsible.

1.7.2 Occupational exposure

Most reports on the effects of coal tar creosote on humans refer to occupational exposure, resulting mainly from dermal and/or inhalational contact with creosote or creosoted wood.

The most apparent effects included irritations or lesions of skin and eyes, including phototoxic or photo-allergic reactions, sometimes accompanied by general symptoms such as depression, weakness, headache, slight confusion, vertigo, nausea, increased salivation, or vomiting. Photosensitization (sensitization of the skin to UV light by creosote) has been observed in workers exposed to creosote.

Increased risks of developing lip and skin cancers have been observed in cohort studies of Swedish and Norwegian wood impregnators and in Finnish round timber workers. The possible interaction with sunlight exposure has not been adequately addressed. The mortality for cancer of the scrotum was elevated among brick-makers exposed to creosote.

Single epidemiological studies suggested a possible risk for bladder cancer, multiple myeloma, and lung cancer due to exposure to creosote. Two case-control studies suggested an increased risk of brain tumours and neuroblastoma among offspring of male workers with possible creosote exposure.

All of the epidemiological studies were based on qualitative estimations of exposure rather than on measurements.

1.8 Effects on other organisms in the laboratory and field

EC₅₀ values (15 min) determined using the Microtox test (inhibition of bioluminescence from *Photobacterium phosphoreum* or *Vibrio fischeri*) by different coal tar creosotes (in acetone solutions) ranged from 0.38 to 0.63 mg/litre. Significant decreases in bioluminescence compared with controls have also been found for several creosote-contaminated environmental samples, such as sediments (including their elutriates and pore waters) and groundwater. Furthermore, a strong inhibition of nitrification by creosote-contaminated leachate has been observed.

Creosote induced signs of stress and abnormal growth in experimentally exposed aquatic plants. Visual changes in *Myriophyllum spicatum* could be seen at nominal creosote concentrations as low as 1.5 mg/litre. EC₅₀ values for a decrease in node production, shoot lengths, and dry weight were calculated to be 86, 55, and 33 mg/litre, respectively. Additionally, membrane ion leakage was significantly and dose-dependently increased at creosote concentrations ranging from 0.1 to 92 mg/litre. The phototoxic potential of creosote has been demonstrated in *Lemna gibba*: EC₅₀ values (nominal) for reduction in growth rate decreased from 54 mg/litre (under laboratory visible light) to 12 mg/litre under simulated solar radiation.

Creosote EC₅₀/LC₅₀ values for aquatic invertebrates have been measured in the range of 0.02–4.3 mg/litre. Larval stages proved to be more sensitive than adult stages. Lifetime exposure of *Daphnia pulex* to water-soluble fractions of creosote resulted in decreased growth rates and reproductive impairment.

An increase in susceptibility to infections has been observed in eastern oysters (*Crassostrea virginica*) exposed to 15% and 30% dilutions of creosote-contaminated sediment. Increased mortalities have been noted in many crustacean species exposed in the laboratory to matrices environmentally contaminated by creosote. Sublethal effects, such as decreases in dry weight gain and in proportion of gravid females, have been recorded in *Mysidopsis bahia* (crustacean); the 7-day EC₅₀ for these more subtle effects was 0.015 µg total identified aromatic hydrocarbons/litre.

Acetone extracts from creosote-contaminated sediments showed an acute toxicity to *Nitocra spinipes* (crustacean) comparable to that of creosote.

Creosote is acutely toxic to fish, with the lowest LC₅₀ reported to be 0.7 mg/litre.

Creosote-contaminated groundwater, water, or sediments (including associated waters) have been shown to cause adverse reproductive and developmental effects in fish. The LC₅₀ for hatching success was calculated to be 0.05 mg creosote/litre. LC₅₀ values determined in spot (*Leiostomus xanthurus*) decreased with increasing duration of exposure to creosote-contaminated sediment during 7–28 days of exposure.

Data on the effects of creosote exposure on terrestrial organisms are limited. A root elongation test of different creosotes with *Allium cepa* resulted in 96-h EC₅₀ values (for reduction of root length) ranging from 18 to 34 mg/litre. Earthworms (*Eisenia foetida*) exposed to creosote-contaminated soil (e.g., about 1000 mg total PAHs/kg dry weight) died within a few days.