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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR PHENANTHRENE

Prepared for

OFFICE OF SOLID WASTE AND
EMERGENCY RESPONSE

Prepared by

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

EXECUTIVE SUMMARY

Phenanthrene is a colorless solid at ambient temperatures. It is soluble in a number of organic solvents including ethanol, benzene, toluene, carbon disulfide and ethyl ether (Verschueren, 1983; Windholz, 1983), but practically insoluble in water (Pearlman et al., 1984). The aqueous solubility of phenanthrene decreases slightly with the increase of ionic strength and decreases greatly with the lowering of water temperature (Whitehouse, 1984). It is susceptible to oxidation by ozone, perioxides and other oxidizing agents (NAS, 1972). Although this compound is not currently produced or imported into the United States (IARC, 1983; USITC, 1984; SRI, 1986), between 1.1 and 11.0 million pounds of it was produced by two U.S. companies in 1977 (U.S. EPA, 1977). Phenanthrene is produced by fractional distillation of high-boiling coal-tar oil and the subsequent purification of the crystalline solid (Hawley, 1981). This compound is used for the production of dyestuffs, explosives and drugs. It can also be used for the synthesis of phenanthrenequinone (Hawley, 1981).

The fate and transport of phenanthrene in surface waters depends on the nature of the water. The three processes that are likely to be important for the loss of phenanthrene from water are photolysis, biodegradation and volatilization. In very shallow, fast flowing and clear water, both photolysis and volatilization may be important processes. The half-life of phenanthrene in such waterbodies may be <1 day (Zepp and Schlotzhauer, 1979; Lyman et al., 1982). On the other hand, in deep eutrophic ponds, biodegradation may be the most important process for aquatic phenanthrene. Based on its biodegradation half-life in estuarine water (Lee and Ryan, 1983), the

half-life of the compound in deep eutrophic ponds may be >36 days. Phenanthrene will moderately bioconcentrate in aquatic organisms. A steady-state bioconcentration factor of 374 has been estimated for phenanthrene in Daphnia pulex (Southworth et al., 1978).

In air, phenanthrene is expected to be present both in the vapor and the particle-sorbed phase, although the vapor phase is likely to predominate (Thrane and Mikalsen, 1981). The photochemical reaction of particle-sorbed or gas-phase state phenanthrene in the atmosphere will not be important compared with its other chemical reactions (Behymer and Hites, 1985; Korfmacher et al., 1980). The half-lives for the vapor phase chemical reactions of phenanthrene with O_3 and HO radical are estimated to be ~6 hours each (Atkinson, 1985; Butkovic et al., 1982); however, these chemical reactions will be slower for particle-sorbed phenanthrene in the atmosphere (Santodonato et al., 1981). The long-range transport of phenanthrene observed by Lunde and Bjoerseth (1977) indicates that particle-sorbed phenanthrene may have a half-life of the order of days.

The fate and transport of phenanthrene in soils is not well documented. Both biodegradation and unknown chemical reactions will decrease phenanthrene in soils (Bossert et al., 1984). In sandy loam soil, the half-life of phenanthrene could be as high as 35 days (Bossert et al., 1984). Phenanthrene may not leach from most soils because of its high soil sorption coefficient (Gile et al., 1982). Leaching of phenanthrene may occur in sandy soils that have low sorptive capacities and in soils from waste disposal sites that have been depleted of phenanthrene-utilizing and cometabolizing microorganisms by high concentrations of toxic chemicals.

Phenanthrene is widely distributed in the aquatic environment and has been detected in industrial effluents, runoff waters, surface water and sediments, groundwater and drinking water. Phenanthrene concentrations of ~70 µg/ℓ were detected in the wastewater from an unspecified tire manufacturing plant (Jungclaus et al., 1976). Cole et al. (1984) reported phenanthrene in urban runoffs from five U.S. cities at a concentration range of 0.3-10.0 µg/ℓ. The frequency of detection of phenanthrene in runoff water from 15 U.S. cities was 12%. Phenanthrene was detected at trace levels in water from the Delaware River north of Philadelphia (Hites, 1979). Unseparated anthracene/phenanthrene derived from various sources and at concentrations ≤6.46.4 mg/kg was detected in a sediment sample from an estuary between England and Wales (John et al., 1979). Phenanthrene at a concentration ≤0.78 mg/ℓ was reported in groundwater in the vicinity of a wood treatment plant in Pensacola, FL (Goerlitz et al., 1985). This compound has been detected in drinking water in the United States and elsewhere in the world. The median concentration of phenanthrene in finished water from 11 U.S. water supplies was 5 ng/ℓ. Assuming this value as the average concentration of phenanthrene in U.S. drinking water, and a daily human consumption of 2 ℓ of drinking water, the average daily intake of phenanthrene for an adult in the United States is estimated as 10 ng.

Some of the known sources of phenanthrene in the atmosphere are vehicular emissions, coal and oil burning, wood combustion, coke plants, aluminum plants, iron and steel works, foundries, ferroalloy plants, municipal incinerators, synfuel plants and oil shale plants (Santodonato et al., 1981; Daisey et al., 1986; Gammage, 1983). The atmospheric concentration of phenanthrene in a Soderberg aluminum reduction plant in Norway was reported to be ≤454 µg/m³ (Bjoerseth et al., 1978). Phenanthrene exists in the

ambient air in cities around the world at various concentrations (Ligocki et al., 1985; Keller and Bidleman, 1984; Karickhoff et al., 1979; Yamasaki et al., 1982). Although data for phenanthrene was limited, Grosjean (1983) estimated that the levels of other PAHs in Los Angeles air did not significantly change during the last decade. The median concentration of atmospheric phenanthrene is estimated to be 14 ng/m^3 from the available atmospheric levels of five U.S. locations. Assuming this value as the average phenanthrene concentration in U.S. air, and that an adult inhales 20 m^3 air/day, the average daily inhalation intake of phenanthrene for a U.S. individual is estimated to be 280 ng.

Phenanthrene has been reported to be present in oysters and fishes collected from contaminated waters and in liquid smoke, smoked foods and charcoal-broiled steaks (Fazio and Howard, 1983). Marcus and Stokes (1985) reported the concentration of phenanthrene in oysters collected from contaminated waters in South Carolina ranged from not detected to 76.5 µg/ℓ. Fishes collected from contaminated U.S. waters were reported to contain <20-100 µg/kg of combined phenanthrene/anthracene (DeVault, 1985). Until data on the levels of this compound in total diet composites used by an average individual in the United States are available, it is not possible to estimate the human dietary intake of phenanthrene.

The data base for the aquatic toxicity of phenanthrene is limited. The most sensitive of four fish species tested was the rainbow trout, which experienced a 10% mortality of eggs and larvae at 1-4 µg/ℓ (Black et al., 1983). Among the nine invertebrate species tested, the lowest reported lethal concentration was 100 µg/ℓ, the 96-hour LC_{50} for D. pulex (Trucco et al., 1983). This result conflicts with the only chronic toxicity study available (Geiger and Buikema, 1982), in which no toxic effects or

reproductive success or survival of D. pulex occurred at 110 µg/ℓ. Aquatic plants appeared to be less sensitive to phenanthrene than fish and invertebrates, with EC_{50} values for inhibition of photo- synthesis ranging from 870 µg/ℓ in N. paleo (Millemann et al., 1984) to 100% saturation in S. capricornutum (Giddings, 1979). Bioconcentration and residue monitoring data indicated wide variability in potential for phenanthrene accumulation in various species (Tables 6-2 and 6-3). Bony fishes (teleosts) tended to metabolize and eliminate phenanthrene more rapidly than other aquatic organisms (Solbakken and Palmork, 1981).

Pertinent data regarding the absorption, distribution and excretion of phenanthrene could not be located in the available literature as cited in the Appendix. PAHs are, in general, highly lipid-soluble, however, and are absorbed readily from the gastrointestinal tract and lungs. Metabolites of phenanthrene identified in in vivo and in vitro studies indicate that metabolism proceeds by epoxidation at the 1-2, 3-4 and 9-10 carbons (Boyland and Wolf, 1950; Boyland and Sims, 1962; Sims, 1970; Chaturapit and Holder, 1978; Nordqvist et al., 1981). trans-Dihydrodihydroxyphenanthrenes (dihydrodiols) are the primary products, with the 9,10-dihydrodiol being the major metabolite.

Phenanthrene did not induce mammary tumors in rats when administered in single 200 mg oral treatments (Huggins and Yang, 1962) and was not tumorigenic to mice when administered in single subcutaneous injections (Steiner, 1955; Grant and Roe, 1963) or three intraperitoneal injections to neonates (Buening et al., 1979). The results of these studies were negative, but should be regarded as inconclusive because the studies are inadequate for evaluation of carcinogenicity because of limited treatment schedules.

Phenanthrene did not produce skin tumors in mice in an inadequately reported skin painting study (dose and application schedule not reported) (Kennaway, 1924). Several mouse skin initiation-promotion assays using phenanthrene have been conducted. Phenanthrene was active as a tumor initiator in one study in which TPA was used as the promoter (Scribner, 1973), but was inactive in the other studies in which TPA was used as the promoter (Wood et al., 1979; LaVoie et al., 1981), croton oil was used as the promoter (Roe, 1962), benzo[a]pyrene and croton oil were used as promoters (Roe and Grant, 1964) and benzo[a]pyrene was used as the initiator (Roe and Grant, 1964). Phenanthrene also was not active when used as an initiator by subcutaneous injection with croton oil promotion by skin application (Roe, 1962).

Phenanthrene has been tested in numerous mutagenicity and other short-term assays with generally negative responses. Phenanthrene was reported not to be mutagenic in the His^+ reversion assay using Salmonella typhimurium tester strains TA100, TA98, TA1535, TA1537 and TA1538 when assayed with or without liver metabolic activation (McCann et al., 1975; Wood et al., 1979; Buecker et al., 1979; LaVoie et al., 1981; Florin et al., 1980; Dunkel et al., 1984). One study reported phenanthrene to be mutagenic in Salmonella tester stain TA100 when assayed in the presence of a high concentration of liver S9 (Oesch et al., 1981) and another study found phenanthrene to be positive in the new frameshift sensitive tester strain Salmonella typhimurium TA97 (Sakai et al., 1985). Negative results were reported in the forward mutation assay using Salmonella typhimurium TM677 (Kaden et al., 1979; Seixas et al., 1982).

Phenanthrene was reported to induce gene mutations in human lymphoblastoid TK6 cells in vitro in the presence of a metabolic activation system (Barfknecht et al., 1981), but was reported to be negative for gene mutations at two different loci in Chinese hamster V79 cells in vitro

(Huberman an Sachs, 1979). Intraperitoneal injection of phenanthrene into Chinese hamsters produced sister chromatid exchanges, but no chromosome aberrations or micronuclei in the bone marrow cells (Bayer, 1978; Roszinsky-Kocher et al., 1979). Sister chromatid exchanges and chromosome aberrations were not produced in Chinese hamster V79-4 cells treated in vitro with phenanthrene in the presence of exogenous metabolic activation (Popescu et al., 1977).

Phenanthrene did not produce positive responses in other assays indicative of DNA damage using bacteria mammalian cells in vitro, and yeast (i.e., differential growth inhibition, DNA repair and mitotic recombination tests) (McCarrol et al., 1981; Rosenkranz and Poirier, 1979; Lake et al., 1978; Probst et al., 1981; Simmon, 1979).

Neoplastic transformation was not induced in mouse prostate C3HG23 cells, C3H/10T1/2 clone 8 mouse embryo fibroblasts, Syrian hamster embryo cells, mouse BALB/3T3 cells or guinea pig fetal cells by in vitro treatment with phenanthrene or in hamster embryo cells following intraperitoneal injection of phenanthrene in pregnant females (Quarles et al., 1979; Marquardt et al., 1972; Pienta et al., 1977; Kakunaga, 1973; Evans and DiPaolo, 1975; Peterson et al., 1981).

Data regarding teratogenicity or other reproductive effects, or the chronic or subchronic toxicity of phenanthrene, could not be located in the available literature. Single intraperitoneal injections of 150 mg/kg produced evidence of slight hepatotoxicity in rats (Yoshikawa et al., 1985); these included gross congestion and distinct lobulation and small increases in the activities of SGOT and serum GGTP.

Data were insufficient to derive an RfD, RQ, q_1^* or F factor for phenanthrene. This chemical was placed in EPA Group D, that is, not classified, and no direct ranking under CERCLA is possible.

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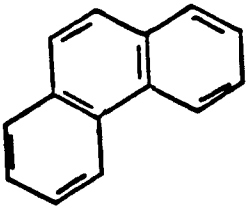
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
BUN	Blood urea nitrogen
CAS	Chemical Abstract Service
DMSO	Dimethyl sulfoxide
DNA	Deoxyribonucleic acid
EC_{50}	Concentration effective to 50% of recipients
GGTP	Gamma glutamyl transpeptidase
K_{oc}	Soil sorption coefficient
K_{ow}	Log octanol/water partition coefficient
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
LDH	Lactate dehydrogenase
MED	Minimum effective dose
PAH	Polycyclic aromatic hydrocarbons
ppm	Parts per million
RfD	Reference dose
RQ	Reportable quantity
RV_d	Dose-rating value
RV_e	Effect-rating value
SGOT	Serum glutamic oxaloacetic transaminase
SGPT	Serum glutamic pyruvic transaminase
TLV	Threshold-limit value
TPA	12-O-Tetradecanoylphorbol-13-acetate
TWA	Time-weighted average

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Phenanthrene is a member of a class of chemicals called polycyclic aromatic hydrocarbons (PAH). The structure, empirical formula, molecular weight and CAS Registry number for this chemical are as follows:



Empirical formula: $C_{14}H_{10}$

Molecular weight: 178.22

CAS Registry number: 85-01-8

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Phenanthrene is a colorless crystalline solid at ambient temperatures. It is practically insoluble in water but is soluble in a number of organic solvents including ethanol, benzene, toluene, carbon disulfide and ethyl ether (Verschueren, 1983; Windholz, 1983). The relevant physical properties of phenanthrene are listed below:

Melting point:	101°C	Santodonato et al., 1981
Boiling point:	340°C	Santodonato et al., 1981
Density at 25°C:	1.179 g/cm³	Windholz, 1983
Water solubility:		
distilled water at 25°C	1.28 mg/ℓ	Pearlman et al., 1984
distilled water at 25°C	1.10 mg/ℓ	Whitehouse, 1984
distilled water with 36.5‰ salinity at 25.3°C	1.00 mg/ℓ	Whitehouse, 1984
distilled water at 4.6°C	0.36 mg/ℓ	Whitehouse, 1984

Log K_{ow}:	4.45-4.57	Mackay et al., 1985; Miller et al., 1985
Vapor pressure:	6.8×10^{-4} mm Hg	Santodonato et al., 1981
	5.2×10^{-4} to 8.3×10^{-4} mm Hg at 25°C	Bidleman, 1984
Henry's Law constant:	9.5×10^{-5} atm•m³/mol^{-1} (estimated based on solubility of 1.28 mg/ℓ and vapor pressure of 5.2×10^{-4} mm Hg)	

It can be concluded from the above tabulated data that the solubility of phenanthrene in water decreases slightly with the increase in salt content. The solubility, however, is greatly dependent upon the water temperature. PAHs are reactive chemically and can undergo substitution and addition reactions. In addition, these compounds are susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972).

1.3. PRODUCTION DATA

According to the TSCA production file (U.S. EPA, 1977), two U.S. companies produced between 1.1 and 11.0 million pounds of phenanthrene in 1977. Currently, it is neither commercially produced nor imported into the United States (IARC, 1983; USITC, 1984; SRI, 1986). Phenanthrene is produced by fractional distillation of high-boiling coal-tar oil. The distillate is crystallized and the phenanthrene is purified by recrystallization from alcohol (Hawley, 1981).

1.4. USE DATA

Phenanthrene can be used in the production of dyestuffs, explosives and drugs. It can also be used for the synthesis of phenanthrenequinone (Hawley, 1981).

1.5. SUMMARY

Phenanthrene is a colorless solid at ambient temperatures. It is soluble in a number of organic solvents including ethanol, benzene, toluene, carbon disulfide and ethyl ether (Verschueren, 1983; Windholz, 1983), but practically insoluble in water (Pearlman et al., 1984). The aqueous solubility of phenanthrene decreases slightly with the increase of ionic strength and decreases greatly with the lowering of water temperature (Whitehouse, 1984). Phenanthrene is susceptible to oxidation by ozone, perioxides and other oxidizing agents (NAS, 1972). Although this compound is not currently produced or imported into the United States (IARC, 1983; USITC, 1984; SRI, 1986), between 1.1 and 11.0 million pounds of it was produced by two U.S. companies in 1977 (U.S. EPA, 1977). Phenanthrene is produced by fractional distillation of high-boiling coal-tar oil and the subsequent purification of the crystalline solid (Hawley, 1981). This compound can be used for the production of dyestuffs, explosives and drugs. It can also be used for the synthesis of phenanthrenequinone (Hawley, 1981).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Photodegradation. The photodegradation of phenanthrene in water by natural sunlight was studied by Zepp and Schlotzhauer (1979). The near-surface half-life for direct photochemical transformation of phenanthrene at 40° N latitude by midday, midsummer sun was estimated to be 8.4 hours. Because of light attenuation and sediment-water partitioning, the photolysis rate decreases with the increase of water depth and suspended sediment concentration. The photolysis half-life of phenanthrene in river water 5 m deep with a suspended sediment concentration of 20 mg/ℓ during a summer day at 40° N latitude was estimated to be 69 days (Zepp and Schlotzhauer, 1979). Zepp and Schlotzhauer (1983) showed that certain green and blue-green algae found in many natural waters, accelerate the phototransformation of several compounds, probably through sensitized photoreaction. In the case of phenanthrene, only one of six species of algae slightly accelerated the sunlight-induced photoreaction, but all the other five species slightly lowered the phototransformation rate. Therefore, the presence of algae may not significantly affect the phototransformation of phenanthrene in most natural waters.

2.1.2. Chemical Reactions. The rate of oxidation of phenanthrene with singlet oxygen ($^1O_2$) was reported by Zepp and Schlotzhauer (1979). Assuming the near surface steady-state concentration of singlet oxygen in natural waters in summer to be 6×10^{-12} M, these authors estimated the half-life for this reaction to be 10^9 hours. (The source of the rate constant value used in determination of the half-life is not clear.) Therefore, this reaction was concluded not to be a significant fate-determining process for phenanthrene in water. The reaction of

phenanthrene with ozone was reported by Kuo and Barnes (1985) and Butkovic et al. (1983). The rate of this reaction was reported to be higher in neutral solution than in strongly acidic solution, and the rate was enhanced at higher temperatures. The rate constants for this reaction at 25°C and a pH of 1 and 7 were reported to be (1.33-1.94)x10^4 and (1.57-4.75)x10^4 ℓ/mol-sec, respectively. At an ozone concentration of 10^{-4} M, these correspond to half-lives of <1 second. Therefore, the ozone reaction may be important for complete oxidation of phenanthrene if ozonation is used as a method of disinfection of drinking water.

2.1.3. Biodegradation. The biodegradability of phenanthrene has been studied with pure cultures of microorganisms, mixed microorganisms and in natural water and sediments. Several pure cultures of microorganisms including Flavobacterium sp., Pseudomonas aeruginosa, Pseudomonas putida, Beijerinckia sp., Pseudomonas sp., Alcaligenes faecalis, Achromobacter sp., Aeromonas sp. and Nocardia sp. (Kobayashi and Rittman, 1982; Shiaris and Cooney, 1983; Fuhs, 1961; Kiyohara et al., 1982; McKenna, 1977; Gibson, 1977; Cerniglia, 1981; Ribbons and Eaton, 1982) degraded phenanthrene. Although these pure culture studies do not simulate environmental conditions for biodegradation, they are useful in establishing biodegradation pathways of chemicals. The proposed pathway for microbial catabolism of phenanthrene is shown in Figure 2-1.

The biodegradability of phenanthrene with mixed microorganisms was studied by several investigators. Thom and Agg (1975) reported that phenanthrene is biodegradable by biological sewage treatment, provided that suitable acclimatization can be achieved. With settled domestic wastewater as microbial inoculum and a static-culture flask-screening procedure, 100% of the phenanthrene was found to be biodegradable in 7 days at an initial

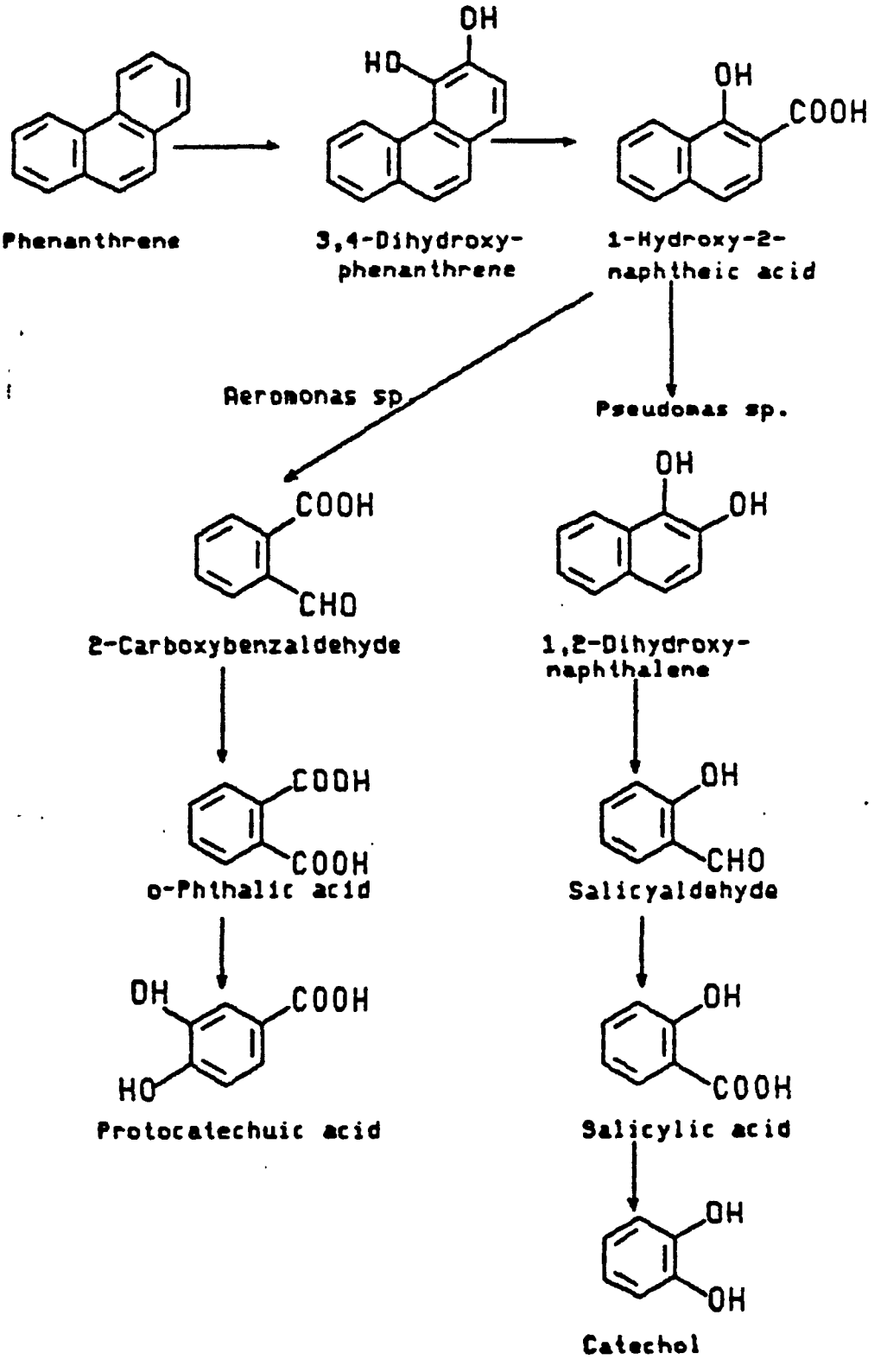


FIGURE 2-1

Proposed Pathway for Microbial Degradation of Phenanthrene

Sources: Cerniglia, 1981; Van der Linden and Thijsse, 1965; McKenna and Kallio, 1965

concentration of 5 ppm (Tabak et al., 1981). Lutin et al. (1965) used activated sludge from three municipal treatment plants as microbial inoculum and the Warburg method for the estimation of the rate of biooxidation. Phenanthrene was reported to be biodegradable with all three activated sludges. The removal of phenanthrene in a municipal facility using activated sludge was reported to be 91% at an influent concentration of 3.2 µg/ℓ, but the removal was 0% at an influent concentration of 3 µg/ℓ in an industrial facility using aerated lagoon treatment (Patterson and Kodukala, 1981).

The biodegradability of phenanthrene with natural waters (Lee and Ryan, 1983; Sherrill and Sayler, 1980) has also been reported. The biodegradation of phenanthrene in water is controlled by the temperature, state of acclimatization of the microorganisms and its concentration. The biodegradation rates were linearly higher as the temperature was raised from 15-37°C. Phenanthrene biodegradation was virtually not detected at the extreme temperatures of 5 and 45°C (Sherrill and Sayler, 1980). Similarly, higher biodegradation rates were observed with microorganisms acclimatized with PAH, possibly phenanthrene (Sherrill and Sayler, 1980; Lee and Ryan, 1983). The acclimatization time for phenanthrene-degrading microorganisms was probably <3 days (Sherrill and Sayler, 1980). Higher concentrations of phenanthrene were found to increase the biodegradation rates. Increasing the phenanthrene concentration from 100-1000 µg/ℓ increased the relative biodegradation rate 3-fold (Sherrill and Sayler, 1980). The optimum concentration at which phenanthrene may be toxic to the microorganisms was not reported. The half-life for phenanthrene biodegradation in water was reported to be 12 days in fresh water at 25°C (Sherrill and Sayler, 1980) and 19-36 days in estuarine water at 27-28°C (Lee and Ryan, 1983).

2.1.4. Volatilization. Using the liquid and gas-phase exchange coefficients for computing the overall liquid-phase mass transfer coefficient, Lyman et al. (1982) estimated the half-life of evaporation for phenanthrene from water 1 m deep to be 31 hours at a wind speed of 3 m/sec and a water current of 1 m/sec. The volatility of phenanthrene from a laboratory-scale waste stabilization pond was reported by Davis et al. (1983). Although the experimental volatilization half-life was 300 hours, the predicted half-life from the same pond by the Liss and Slater model was 2.1 hours. The authors concluded that the sorption of phenanthrene onto biota and silt was responsible for the difference between the experimental and predicted half-life values (the predictive models do not consider the effect of sorption on volatilization). From their waste stabilization pond study, Davis et al. (1983) estimated that only 0.2% of the applied phenanthrene dose was lost by volatilization; losses of 93.5 and 3% were due to degradation and sedimentation, respectively. The remainder was lost in the effluent or remained in the water column as residual.

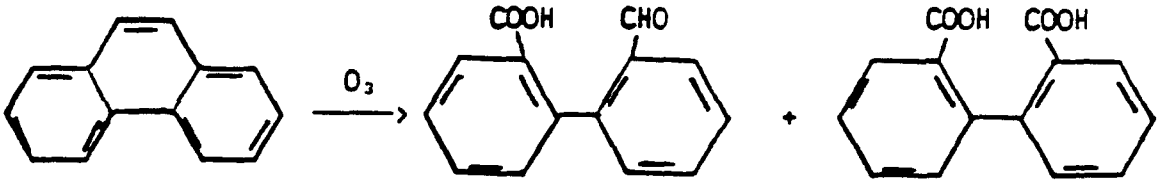
2.1.5. Adsorption. The adsorption of phenanthrene to suspended particulate matter and sediment can be predicted from its K_{oc}. The K_{oc} value for phenanthrene is estimated to be 23,000 (Karickhoff et al., 1979). This is indicative of the possibility of strong sorption of phenanthrene onto suspended particles and sediments in water. As in the case of anthracene (estimated K_{oc} of 26,000) where the removal through adsorption constitutes only negligible to 18% (Southworth, 1979) of the overall removal processes, the contribution of sorption in water is expected to be low in the case of phenanthrene as well. In their model waste stabilization pond study, Davis et al. (1983) estimated that only 3% of total phenanthrene removal was attributable to sedimentation.

2.1.6. **Bioconcentration.** The BCF for phenanthrene in algae (species unspecified) was reported to be 4552 (Davis et al., 1983). Southworth et al. (1978) examined the bioaccumulation potential of several PAH including phenanthrene in Daphnia pulex, a representative component of the aquatic food web. A 24-hour BCF of 325 was reported for phenanthrene in filtered spring water. Using uptake and elimination rates, these authors estimated a steady-state BCF of 374 for phenanthrene. The bioconcentration of phenanthrene in aquatic organisms may be species-dependent. Species that contain microsomal oxidase/mixed function oxidase activity that allows metabolism of the parent compounds will tend to lower the BCF (Santodonato et al., 1981). It was also reported by McCarthy (1983) that the BCF for hydrophobic organic pollutants are considerably less in natural water than measured values in laboratories using particle-free water because of nonavailability of sorbed-state compounds for uptake by organisms.

2.2. AIR

The fate and transport of phenanthrene in the atmosphere is less documented than its water fate. The reactivity of atmospheric phenanthrene will depend on the state in which it exists in the atmosphere. The reactivity of vapor phase phenanthrene is expected to be faster than in the adsorbed state (Santodonato et al., 1981). Thrane and Mikalsen (1981) suggest that phenanthrene will be present predominantly in the vapor phase in the atmosphere. The heterogenous reaction of gas-phase O_3 and NO_2 with phenanthrene coated on sodium chloride was reported by Niessner et al. (1985).

The heterogenous reaction of phenanthrene was negligible with NO_2, but reaction with O_3 was significant. The reaction with O_3 produced the following products:



The atmospheric half-life of phenanthrene resulting from reaction with O_3 was reported by Butkovic et al. (1983). Assuming the rate constant for this reaction as 1.5×10^4 ℓ/mol-sec (the same as in water) and the tropospheric O_3 concentration as 2×10^{-9} M in clear air, these authors estimated a half-life of 6 hours. The rate constant for the gas phase reaction of phenanthrene with HO radical at 25°C was reported to be 34×10^{-12} cm^3/molecule-sec (Atkinson, 1985). If the concentration of HO radical in the atmosphere is assumed to be 10^6 radicals/cm^3, the half-life of this reaction is ~6 hours.

Korfmacher et al. (1980) reported that phenanthrene was resistant to photodecomposition in cyclohexanone solution. When phenanthrene vapors adsorbed on fly ash were irradiated with a xenon lamp for 3.3 hours, no significant decomposition was observed (Korfmacher et al., 1980). The photodegradation of particle-bound phenanthrene was found to be highly dependent on the substrate to which it was adsorbed (Behymer and Hites, 1985). For example, the half-lives of phenanthrene irradiated with medium pressure mercury arc lamps in a rotary photoreactor were 150, 40, 49 and $\geq$1000 hours when the adsorption media were silica gel, alumina, fly ash and carbon black, respectively. Therefore, it can be concluded that photodegradation of phenanthrene in the atmosphere will be less significant than its reactions with O_3 and HO radical.

The removal of atmospheric phenanthrene through wet and dry deposition can also occur. Eisenreich et al. (1981) reported that in the Great Lakes ecosystem both dry deposition of the vapor and particle-bound phenanthrene and wet deposition through rain and snow occurred; dry deposition, however, was found to be more important. Ligocki et al. (1985) concluded from their experimental observation that particle scavenging was less important than gas scavenging of atmospheric phenanthrene. The half-lives for these physical removal mechanisms were not provided in either study. The removal of atmospheric phenanthrene through these physical processes appear to be less significant than its removal through chemical processes. Finally, Lunde and Bjorseth (1977) reported that the concentration of phenanthrene in air trajectories that originated from Western Europe (polluted air) contained >8 times more phenanthrene than air samples with trajectories from northern Norway or stationary air from southern Norway (less polluted air). This result suggests that phenanthrene is capable of undergoing long-distance transport in the atmosphere.

2.3. SOIL

The fate of phenanthrene in soils is even less documented than its fate in the atmosphere. Predictions, however, can be made from the knowledge of its fate in water. The three processes that are important in the loss of phenanthrene from water are photolysis, biodegradation and volatilization. Because of light attenuation and scattering, photolysis cannot be an important process for the loss of phenanthrene beyond the surface layer of soils. Bossert et al. (1984) incorporated oily sludge containing phenanthrene in a sandy loam soil and observed the loss of phenanthrene in sterile and non-sterile soils. Because multiple applications of sludge to soil were made at various intervals with intervening nonapplication periods, it is difficult to estimate from the data the degradation half-life of phenanthrene in soil.

On the basis of the loss of the chemical after its first application in non-sterile soil, the overall degradation half-life is estimated to be ~35 days. The authors concluded from sterilized and nonsterilized soil studies that both biodegradation and undefined chemical processes accounted for the observed loss in phenanthrene concentrations. The chemical processes were responsible for <50% of the loss. The loss of phenanthrene from volatilization was speculated to be insignificant.

The leaching of phenanthrene from soil to groundwater depends on the soil characteristics. The K_{oc} for phenanthrene was estimated to be 23,000 (Karickhoff et al., 1979), indicating that phenanthrene will be strongly adsorbed to most soils and degrade before it reaches groundwater. The terrestrial microcosm experiment performed by Gile et al. (1982) predicted that leaching of phenanthrene from soil to groundwater will not normally occur. Leaching of phenanthrene may occur in sandy soil that has a low sorptive capacity and in soils from waste disposal sites that have been depleted of phenanthrene-utilizing and cometabolizing microorganisms.

2.4. SUMMARY

The fate and transport of phenanthrene in surface waters depends on the nature of the water. The three processes that are likely to be important for the loss of phenanthrene from water are photolysis, biodegradation and volatilization. In very shallow, fast-flowing and clear water, both photolysis and volatilization may be important processes. The half-life of phenanthrene in such waterbodies may be <1 day (Zepp and Schlotzhauer, 1979; Lyman et al., 1982). On the other hand, in deep eutrophic ponds, biodegradation may be the most important process for aquatic phenanthrene. Based on its biodegradation half-life in estuarine water (Lee and Ryan, 1983), the

half-life of the compound in deep eutrophic ponds may be >36 days. Phenanthrene will moderately bioconcentrate in aquatic organisms. A steady-state bioconcentration factor of 374 has been estimated for phenanthrene in Daphnia pulex (Southworth et al., 1978).

In air, phenanthrene is expected to be present both in the vapor and the particle-sorbed, although the vapor phase is likely to predominate (Thrane and Mikalsen, 1981). The photochemical reaction of particle-sorbed or gas-phase state phenanthrene in the atmosphere will not be important compared with its other chemical reactions (Behymer and Hites, 1985; Korfmacher et al., 1980). The half-lives for the vapor phase chemical reactions of phenanthrene with O_3 and HO radical are estimated to be ~6 hours each (Atkinson, 1985; Butkovic et al., 1982); however, these chemical reactions will be slower for particle-sorbed phenanthrene in the atmosphere (Santodonato et al., 1981). The long-range transport of phenanthrene observed by Lunde and Bjorseth (1977) indicates that particle-sorbed phenanthrene may have a half-life of the order of days.

The fate and transport of phenanthrene in soils is not well documented. Both biodegradation and unknown chemical reactions will degrade phenanthrene in soils (Bossert et al., 1984). In sandy loam soil, the half-life of phenanthrene could be as high as 35 days (Bossert et al., 1984). Phenanthrene may not leach from most soils because of its high soil sorption coefficient (Gile et al., 1982). Leaching of phenanthrene may occur in sandy soils that have low sorptive capacities and in soils from waste disposal sites that have been depleted of phenanthrene-utilizing and cometabolizing microorganisms by high concentrations of toxic chemicals.

3. EXPOSURE

3.1. WATER

Phenanthrene is widely distributed in the aquatic environment. It has been detected in industrial effluents, in run-off water, in surface water and sediments, in groundwater and in drinking water. Phenanthrene at a concentration of ~70 µg/ℓ was detected in the wastewater from an unspecified tire manufacturing plant (Jungclaus et al., 1976). Concentrations of phenanthrene in the secondary effluent from a Scandanavian sewage treatment plant were reported to range from 72-117 ng/ℓ (Kveseth et al., 1982). The aqueous effluents from unspecified coke plants reportedly contained <30-1300 ng/ℓ of phenanthrene (Griest, 1980; Walters and Luthy, 1984). The U.S. EPA has detected phenanthrene at a frequency of 5% in ~1288 effluents collected since 1980 from different sources, with a median concentration of <10 µg/ℓ (Staples et al., 1985). Phenanthrene was also detected in urban runoff waters. The annual inputs of phenanthrene by urban runoff to the upper Narragansett Bay, RI, watershed were estimated to be 1.7, 2.1, 32.4 and 32.2 kg/year from residential, commercial, industrial and highway runoffs, respectively (Hoffman et al., 1984). Cole et al. (1984) detected phenanthrene in urban runoffs from five U.S. cities at a concentration range of 0.3-10.0 µg/ℓ and at a frequency of 12%. Phenanthrene was detected at trace levels in water from a small segment of the Delaware River north of Philadelphia (Hites, 1979). The U.S. EPA has collected 865 ambient water samples since 1980 and has detected phenanthrene in 5% of these samples, with a median concentration of <10 µg/ℓ (Staples et al., 1985). Phenanthrene was also reported in surface water in England (Fielding et al., 1981). Several investigators reported the detection of phenanthrene in surface water sediments and attempted to establish the sources and modes of

transportation of this compound in water (Jungclaus et al., 1978; Boehm and Farrington, 1984; Sportstol et al., 1983; Windsor and Hites, 1979; Eadie et al., 1982; Tan and Heit, 1981). Unseparated anthracene/phenanthrene derived from various sources and at concentrations as high as 6.4 mg/kg were reported in sediment samples from an estuary between England and Wales (John et al., 1979).

Rostad et al. (1985) qualitatively detected phenanthrene in groundwater from a coal tar waste aquifer in St. Louis Park, MN. Goerlitz et al. (1985) monitored groundwater from several sites in the vicinity of a wood treatment plant at Pensacola, FL, and reported phenanthrene concentrations as high as 0.78 mg/ℓ; however, phenanthrene was not detected in groundwater beyond a depth of 18 m. Phenanthrene has been detected in drinking water in the United States and elsewhere in the world. Kveseth et al. (1982) detected phenanthrene in tap water from Scandinavia in the concentration range of 0.2-64 ng/ℓ. In Tsukuba, Japan, the concentration of phenanthrene in tap water was reported to be 0.34-1.41 ng/ℓ (Shiraishi et al., 1985). Tap water from Kitakyushu, Japan, contained concentrations of combined phenanthrene/anthracene at 1.7 µg/ℓ (Shinohara et al., 1981). The concentration of phenanthrene in drinking water from Ottawa was reported to be >0.5-1.1 ng/ℓ (Benoit et al., 1979). The combined concentrations of anthracene/phenanthrene (unseparable) in Canadian drinking water derived from the Great Lakes reportedly ranged between 0.6 and 1269 ng/ℓ (Williams et al., 1982). The highest concentration was obtained in the water from Sault Ste Marie collected during the summer. Fielding et al. (1981) monitored 14 treated water samples in England and qualitatively detected phenanthrene in 7 of these samples. The concentrations of phenanthrene detected in several U.S. finished and distributed waters (passed through transmission /distribution pipes) are given in Table 3-1.

TABLE 3-1

Concentrations of Phenanthrene in U.S. Finished and Distributed Waters (ng/ℓ)*

City	Finished	Distributed
Cape Girardeau, MO	5	NR
Cincinnati, OH	10	NR
Colorado Springs, CO	3	29
Columbus, OH	3	17
Jefferson Parish, LA	14	NR
Ludlow, MA	2	3
Miami, FL	14	NR
New Orleans, LA	NR	14
Portland, OR	8	3300
Seattle, WA	2-10	32
Standish, ME	5	57
Wheeling, WV	4	NR

*Source: Sorrell et al., 1980

NR = Not reported

The distributed water from some cities (e.g., Portland, OR) shows much higher concentrations of phenanthrene than the treated water because phenanthrene is leached from the coating materials of the distribution pipes. Assuming the average concentration of phenanthrene in U.S. drinking water to be the same as the median value of the concentration of the finished water given in Table 3-1 (5 ng/ℓ), and that human consumption of drinking water is 2 ℓ/day, the average daily intake of phenanthrene for an adult in the United States is 10 ng.

3.2. AIR

The sources of PAH including phenanthrene in the atmosphere are vehicular emissions, coal and oil burning, wood combustion, coke plants, aluminum plants, iron and steel works, foundries, ferroalloy plants and municipal incinerators (Santodonato et al., 1981; Daisey et al., 1986; Gammage, 1983). More recent sources of phenanthrene may be synfuel and oil shale plants. The atmospheric concentration of phenanthrene in an aluminum reduction plant (Soderberg) in Norway was reported to be as high as 454 μg/m³ (Bjoerseth et al., 1978). Personnel sampling of the Soderberg plant showed particulate phenanthracene concentration of none detected for tappers to 60.4 μg/m³ for pin pullers (Bjoerseth et al., 1978). The concentrations of atmospheric combined anthracene/phenanthrene inside a Solvent Refined Coal Pilot plant facility at Fort Lewis, WA, was reported to vary between 1.8 and 43.2 μg/m³ (Gammage, 1983). Personal air samples taken in the coal preparation area of the plant showed combined anthracene/phenanthrene concentrations of none detected to 15.7 μg/m³ (Gammage, 1983). The simulated incineration of polyvinylchloride at temperatures between 800°C and 950°C was qualitatively shown to produce phenanthrene (Hawley-Fedder et al., 1984). The concentrations of phenanthrene in the atmosphere of woodheated saunas varied from 2.3-122 μg/m³ (Hasanen et al., 1984).

The ambient atmospheric levels of phenanthrene in various locations around the world are shown in Table 3-2. Although suitable data were not available for phenanthrene, Grosjean (1983) estimated that the levels of other PAHs in the air in Los Angeles did not significantly change during the last decade. Assuming that the mean levels of phenanthrene concentration in urban U.S. air is similar to the median value of all the U.S. ambient atmospheric levels (14 ng/m^3) given in Table 3-2, and that an adult inhales 20 m^3/day, the average daily inhalation intake of phenanthrene for a U.S. individual would be 280 ng.

3.3. FOOD

Phenanthrene reportedly is present in oysters, liquid smoke, smoked foods and charcoal-broiled steaks (Fazio and Howard, 1983). The levels of phenanthrene detected in different foods are given in Table 3-3. Because data on the levels in total diet composites used by an individual in the United States are not available, it is not possible to estimate the human intake of phenanthrene through food consumption.

3.4. SUMMARY

Phenanthrene is widely distributed in the aquatic environment and has been detected in industrial effluents, runoff waters, surface water and sediments, groundwater and drinking water. Phenanthrene concentrations of ~70 µg/ℓ were detected in the wastewater from an unspecified tire manufacturing plant (Jungclaus et al., 1976). Cole et al. (1984) reported phenanthrene in urban runoffs from five U.S. cities at a concentration range of 0.3-10.0 µg/ℓ. The frequency of detection of phenanthrene in runoff water from 15 U.S. cities was 12%. Phenanthrene was detected at trace levels in water from the Delaware River north of Philadelphia (Hites, 1979).

TABLE 3-2

Ambient Atmospheric Levels of Phenanthrene in Various World Locations

Location	Years Sampled	Phenanthrene Concentration (ng/m^3)	Reference
Portland, OR	1984	27	Ligocki et al., 1985
Columbia, SC	1981-1982	14 to $\geq$140	Keller and Bidleman, 1984
Savannah River plant, SC	1981-1982	6 to $\geq$14	Keller and Bidleman, 1984
Gainesville, FL	NR[a]	10	Kerkhoff et al., 1985
Jacksonville, FL	NR[a]	20	Kerkhoff et al., 1985
Osaka, Japan	1977-1978	52.1-294.5[b]	Yamasaki et al., 1982
Osaka, Japan	1981-1982	0.79-2.64 (1.63)[c]	Matsumoto and Kashimoto, 1985
Budapest, Hungary	1971-1972	3.8-17.4 (10)[c]	Kertsz-Saringer and Morlin, 1975

[a]The year of sampling was not reported but appears to be 1982.

[b]Combined anthracene/phenanthrene values

[c]Mean concentration values

TABLE 3-3

Phenanthrene Levels in Different Foods

Food	Phenanthrene Concentration (µg/kg)	Reference
Oysters from Arkansas and Galvaston Bay	ND	Fazio and Howard, 1983
Coffee roasted dark and very dark	ND	Fazio and Howard, 1983
Roasted coffee soots	130-300	Fazio and Howard, 1983
Electric-broiled Japanese horse mackerel	1-9	Fazio and Howard, 1983
Gas-broiled Japanese horse mackerel	8-11	Fazio and Howard, 1983
Charcoal-broiled steaks	21	Fazio and Howard, 1983
Barbecued ribs	58	Fazio and Howard, 1983
Fish (U.S.)	<20 to 100*	DeVault, 1985
Mussel (Greece) (M. galloprovincialis)	9	Iosifidou et al., 1982
Fresh water fish (preserved) from Nigeria	9-189.3	Afolabi et al., 1983
Mussel composite (U.S.) (M. edulis and M. californianus)	7.9-32*	Galloway et al., 1983
Oyster (Crassostrea virginica) from South Carolina	ND-76.5	Marcus and Stokes, 1985

*Combined anthracene/phenanthrene levels

ND = Not detected

Unseparated anthracene/phenanthrene derived from various sources and at concentrations ≤6.46.4 mg/kg was detected in a sediment sample from an estuary between England and Wales (John et al., 1979). Phenanthrene at a concentration ≤0.78 mg/ℓ was reported in groundwater in the vicinity of a wood treatment plant in Pensacola, FL (Goerlitz et al., 1985). This compound has been detected in drinking water in the United States and elsewhere in the world. The median concentration of phenanthrene in finished water from 11 U.S. water supplies was 5 ng/ℓ. Assuming this value as the average concentration of phenanthrene in U.S. drinking water, and a daily human consumption of 2 ℓ of drinking water, the average daily intake of phenanthrene for an adult in the United States is estimated as 10 ng.

Some of the known sources of phenanthrene in the atmosphere are vehicular emissions, coal and oil burning, wood combustion, coke plants, aluminum plants, iron and steel works, foundries, ferroalloy plants, municipal incinerators, synfuel plants and oil shale plants (Santodonato et al., 1981; Daisey et al., 1986; Gammage, 1983). The atmospheric concentration of phenanthrene in a Soderberg aluminum reduction plant in Norway was reported to be ≤454 μg/m³ (Bjoerseth et al., 1978). Phenanthrene exists in the ambient air in cities around the world at various concentrations (Ligocki et al., 1985; Keller and Bidleman, 1984; Karickhoff et al., 1979; Yamasaki et al., 1982). Although data for phenanthrene was limited, Grosjean (1983) estimated that the levels of other PAHs in Los Angeles air did not significantly change during the last decade. The median concentration of atmospheric phenanthrene is estimated to be 14 ng/m³ from the available atmospheric levels of five U.S. locations. Assuming this value as the average phenanthrene concentration in U.S. air, and that an adult inhales 20 m³ air/day, the average daily inhalation intake of phenanthrene for a U.S. individual is estimated to be 280 ng.

Phenanthrene has been reported to be present in oysters and fishes collected from contaminated waters and in liquid smoke, smoked foods and charcoal-broiled steaks (Fazio and Howard, 1983). Marcus and Stokes (1985) reported the concentration of phenanthrene in oysters collected from contaminated waters in South Carolina ranged from not detected to 76.5 µg/ℓ. Fishes collected from contaminated U.S. waters were reported to contain <20-100 µg/kg of combined phenanthrene/anthracene (DeVault, 1985). Until data on the levels of this compound in total diet composites used by an average individual in the United States is available, it is not possible to estimate the human dietary intake of phenanthrene.

4. PHARMACOKINETICS

4.1. ABSORPTION

Pertinent data regarding the gastrointestinal or pulmonary absorption of phenanthrene could not be located in the available literature as cited in the Appendix. Data from other structurally related PAHs suggest, however, that phenanthrene is absorbed readily from the gastrointestinal tract (Rees et al., 1971) and lungs (Kotin et al., 1969; Vainio et al., 1976). In general, these compounds are highly lipid-soluble and can pass across epithelial membranes (U.S. EPA, 1980a).

4.2. DISTRIBUTION

Pertinent data regarding the distribution of phenanthrene could not be located in the available literature as cited in the Appendix.

4.3. METABOLISM

Evidence from in vivo and in vitro studies indicate that metabolism of phenanthrene occurs by epoxidation at the 1-2, 3-4 and 9-10 carbons, with trans-dihydrodihydroxyphenanthrenes (dihydrodiols) as primary metabolites and the 9,10-dihydrodiol as the major metabolite.

Phenanthrene-9,10-, -1,2- and -3,4-dihydrodiol were identified unaltered or as glucuronic acid conjugates in the urine of rats and rabbits that were given intraperitoneal injections of phenanthrene (Boyland and Wolf, 1950; Boyland and Sims, 1962). The glucuronic acid conjugates of 1-, 2-, 3- and 4-hydroxyphenanthrene, 1,2-dihydroxyphenanthrene and 3,4-dihydroxyphenanthrene were also identified in these urines (Boyland and Sims, 1962). The above metabolites as well as phenanthrene-9,10-oxide and 1,2-diol-3,4-epoxide were detected in in vitro studies with guinea pig, rat and mouse liver preparations (Sims, 1970; Chaturapit and Holder, 1978; Nordqvist et al., 1981).

4.4. EXCRETION

Metabolites of phenanthrene have been detected in the urine of intraperitoneally treated rodents as indicated in Section 4.3. Additional information regarding the elimination of phenanthrene could not be located in the available literature as cited in the Appendix.

4.5. SUMMARY

Pertinent data regarding the absorption, distribution and excretion of phenanthrene could not be located in the available literature as cited in the Appendix. PAHs are, in general, highly lipid-soluble, however, and are absorbed readily from the gastrointestinal tract and lungs. Metabolites of phenanthrene identified in in vivo and in vitro studies indicate that metabolism proceeds by epoxidation at the 1-2, 3-4 and 9-10 carbons (Boyland and Wolf, 1950; Boyland and Sims, 1962; Sims, 1970; Chaturapit and Holder, 1978; Nordqvist et al., 1981). trans-Dihydrodihydroxyphenanthrenes (dihydrodiols) are the primary products, with the 9,10-dihydrodiol as the major metabolite.

5. EFFECTS

5.1. CARCINOGENICITY

Single oral doses of 200 mg phenanthrene (purity unspecified) in sesame oil vehicle were administered by gavage to ten 50-day-old female Sprague-Dawley rats (Huggins and Yang, 1962). The rats were examined for development of mammary tumors by palpation for 60 days following treatment. No mammary tumors were observed. Tissues other than the mammary gland were not examined as this was a comparative study of mammary tumor induction. Mammary tumors occurred in 100% of 700 rats that were administered 20 mg 7,12-dimethylbenz[a]anthracene under the same conditions.

Phenanthrene has been tested for carcinogenicity in an inadequately reported skin application study with mice (dose and schedule not specified) (Kennaway, 1924), in several mouse skin initiation-promotion assays, in single subcutaneous injection studies with adult (Steiner, 1955) or newborn (Grant and Roe, 1963) mice and in a three-injection intraperitoneal study with newborn mice (Buening et al., 1979) (Table 5-1). The results of the skin application and injection studies were negative, but interpretation is complicated by the inadequate reporting and single- or three-injection protocols. Phenanthrene was active as a tumor initiator in one study in which TPA was used as the promoter (Scribner, 1973), but it was inactive in other studies with TPA as a promoter (Wood et al., 1979; LaVoie et al., 1981), with croton oil as a promoter (Salaman and Roe, 1956; Roe, 1962), with benzo[a]pyrene and croton oil as promoters (Roe and Grant, 1964) and inactive as a promoter with benzo[a]pyrene used as an initiator (Roe and Grant, 1964). Phenanthrene was also not active when used as an initiator by subcutaneous injection with croton oil promotion by skin application (Roe, 1962).

TABLE 5-1

Dermal and Injection Carcinogenicity Assays of Phenanthrene

Route	Species/Strain	No.*/Sex	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/NR	100/NR	NR	dose and application schedule not specified; 90% solution in benzene	9 months	No skin tumors	Kennaway, 1924
	mouse/S	20/NR	NR	0.3 mℓ of 18% solution in acetone 3 times/week for 10 applications; starting 25 days later, 18 weekly applications of croton oil in acetone (0.3 mℓ): 1 of 0.17%, 2 of 0.085% and 15 of 0.17%	25 weeks	Skin papillomas in 5/20 (12 total tumors) vs. 4/19 in controls treated only with croton oil (4 total tumors)	Salaman and Roe, 1956
	mouse/albino	10/M, 10/F	high	300 µg in acetone on days 0, 2, 6 and 8; weekly application of 0.25 mℓ of 0.1% croton oil in acetone from day 21 for 20 weeks	24 weeks	Skin papillomas in 4/19 vs. 2/20 in controls treated only with croton oil	Roe, 1962
	mouse/CD-1	30/F	TLC purified	single application of 10 µmol in benzene, followed 1 week later with applications of 5 µmol TPA, 2 times/week for 34 weeks	35 weeks	Skin papillomas in 12/30 (40%) vs. 0/30 in controls treated with 10 µmol TPA alone; 100% survival	Scribner, 1973
	mouse/Swiss Ha/ICR	20/F	>99.5%	100 µℓ of 1.0% solution in acetone 10 times on alternate days; starting 10 days later, applications of 2.5 µg TPA, 3 times/week for 20 weeks	NR	No skin tumors; high incidence of tumors induced by benzo[a]-pyrene	LaVoie et al., 1981
	mouse/Charles River CD-1	30/F	>98%	single application of 10 µmol in acetone; starting 1 week later, applications of 16 nmol TPA, 2 times/week for 35 weeks	36 weeks	Skin papillomas in 5/30 vs. 2/30 in controls treated with acetone only; incidences were 4/29 vs. 2/30 in a second identical experiment	Wood et al., 1979

TABLE 5-1 (cont.)

Route	Species/Strain	No.*/Sex	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/NR	NR/NR	NR	unspecified initiating dose of benzo[a]pyrene followed by applications of 5% phenanthrene in unspecified solvent, 3 times/week for 1 year	1 year	No skin tumors; primary report not available	Roe and Grant, 1964
	mouse/NR	NR/NR	NR	unspecified initiating dose of benzo[a]pyrene followed by 12 applications of 5% phenanthrene in unspecified solvent; application schedule not reported	NR	No skin tumors	Roe and Grant, 1964
	mouse/NR	NR/NR	NR	12 unspecified applications of 5% phenanthrene in unspecified solvent followed by a single unspecified dose of benzo[a]pyrene and weekly applications of 0.1% croton oil in acetone for an unspecified duration	NR	No skin tumors	Roe and Grant, 1964
Subcutaneous	mouse/C57Bl	40/mixed	NR	single interscapular injection of 5 mg in tricaprylin	28 months	No local tumors; 27/40 surviving at 4 months	Steiner, 1955
	mouse/stock albino	10 M, 10 F	NR	300 µg in 3% aqueous gelatin on days 0, 2, 4, 6 and 8; once weekly with 0.25 mL 0.1% croton oil in acetone from day 21 for 20 weeks	24 weeks	Skin papillomas in 3/17 treated vs. 2/20 in controls treated with acetone only	Roe, 1962

TABLE 5-1 (cont.)

Route	Species/Strain	No.*/Sex	Purity	Treatment	Duration	Effects/Comments	Reference
Subcutaneous	neonatal mouse/ stock albino	60/mixed	high	40 µg in 1% aqueous gelatin, single injection	62 weeks	Incidences of pulmonary adenomas, hepatomas and skin papillomas comparable with two solvent control groups; 10 mice/group sacrificed after 52 weeks; similar results in experiments of same design in which phenanthrene (20 or 40 µg) was mixed with benzo[a]pyrene (20 or 40 µg)	Grant and Roe, 1963
Intraperitoneal	neonatal mouse/ Blu-HA (ICR) Swiss-Webster	100/mixed	>98%	35 µg on day 1, 70 µg on day 8 and 140 µg on day 15; DMSO vehicle	36-40 weeks	Pulmonary adenomas in 6/35 (17%) vs. 9/59 (15%) in DMSO controls; incidences in survivors at 42 weeks of age; major organs examined grossly and those with suspected pathology were examined histologically	Buening et al., 1979

*Numbers in treated and control (if used) groups unless specified otherwise.

5.2. MUTAGENICITY

Phenanthrene has been tested in numerous mutagenicity and other short-term assays with generally negative responses. The discussion that follows is not a comprehensive review of all the published literature regarding the mutagenicity of phenanthrene, but a review of selected articles that provides a representative assessment of its mutagenic potential. The reader may wish to refer to other reviews on the mutagenicity of phenanthrene (IARC, 1983; Nishi, 1984; Brookes, 1977).

Phenanthrene was reported not to be mutagenic in the His^+ reversion assay using Salmonella typhimurium tester strains TA100, TA98, TA1535, TA1537 and TA1538 when assayed with or without liver metabolic activation (McCann et al., 1975; Wood et al., 1979; Buecker et al., 1979; LaVoie et al., 1981; Florin et al., 1980). In a mutagenicity test program on the interlaboratory reproducibility of chemicals tested in the standard Ames assay, phenanthrene was found to be predominantly negative in tester strains TA98, TA100, TA1535, TA1537, TA1538 and in Escherichia coli WP2 uvrA by four different laboratories when assayed with or without various rodent liver S9 mixes (Dunkel et al., 1984). However, one study reported phenanthrene to be mutagenic in Salmonella tester strain TA100 when assayed in the presence of a high concentration of liver S9 (Oesch et al., 1981). Phenanthrene also showed a positive response on Salmonella typhimurium TA97 (Sakai et al., 1985). TA97 is a new frameshift strain that is similar to and appears to be more sensitive than TA1537. Negative results were reported in the forward mutation assay using Salmonella typhimurium TM677 (Kaden et al., 1979; Seixas et al., 1982).

Phenanthrene was reported to induce mutation to trifluorothymidine resistance in human lymphoblastoid TK6 cells in vitro in the presence of a metabolic activation system (Barfknecht et al., 1981), but was reported to

be negative for the induction of 8-azoguaine and ouabain resistance in Chinese hamster V79 cells in vitro (Huberman and Sachs, 1979). Intraperitoneal injection of phenanthrene into Chinese hamsters produced sister chromatid exchanges, but no chromosome aberrations or micronuclei in the bone marrow cells (Bayer, 1978; Roszinsky-Kocher et al., 1979). Sister chromatid exchanges and chromosome aberrations were not produced in Chinese hamster V79-4 cells treated in vitro with phenanthrene in the presence of exogenous metabolic activation (Popescu et al., 1977).

Phenanthrene did not produce positive responses in other assays indicative of DNA damage with bacteria (Bacillus subtilis rec^+/rec^-, Escherichia coli $polA^+/polA^-$), mammalian cells in vitro (unscheduled DNA synthesis in human foreskin epithelial cells and primary rat hepatocytes), and yeast (mitotic recombination in Saccharomyces cerevisiae D3) (McCarrol et al., 1981; Rosenkranz and Poirier, 1979; Lake et al., 1978; Probst et al., 1981; Simmon, 1979).

5.2.1. Cell Transformation Studies. Neoplastic transformation was not induced in mouse prostate C3HG23 cells, C3H/10T1/2 clone 8 mouse embryo fibroblasts, Syrian hamster embryo cells, mouse BALB/3T3 cells or guinea pig fetal cells by in vitro treatment with phenanthrene or in hamster embryo cells following intraperitoneal injection of phenanthrene in pregnant females (Quarles et al., 1979; Marquardt et al., 1972; Pienta et al., 1977; Kakunaga, 1973; Evans and DiPaolo, 1975; Peterson et al., 1981).

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of phenanthrene could not be located in the available literature as cited in the Appendix.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding the other reproductive effects of phenanthrene could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the effects of chronic or subchronic exposure to phenanthrene could not be located in the available literature as cited in the Appendix.

5.6. OTHER RELEVANT INFORMATION

A single dose intraperitoneal LD_{50} of 700 mg/kg has been reported for mice (Simmon, 1979). A single intraperitoneal injection of 150 mg/kg phenanthrene dissolved in DMSO produced gross pathological alterations in the livers of six male Sprague-Dawley rats after 24 and 72 hours (Yoshikawa et al., 1985); these included congestion and a distinct lobular pattern. Gross effects in other unspecified tissues were not indicated. Small but significant increases in SGOT and serum GGTP levels were observed 24 but not 72 hours after treatment, and effects on SGPT and serum LDH, bilirubin, glucose, BUN and creatine were not indicated.

5.7. SUMMARY

Phenanthrene did not induce mammary tumors in rats when administered in single 200 mg oral treatments (Huggins and Yang, 1962) and was not tumorigenic to mice when administered in single subcutaneous injections (Steiner, 1955; Grant and Roe, 1963) or three intraperitoneal injections to neonates (Buening et al., 1979). The results of these studies were negative, but should be regarded as inconclusive concerning carcinogenicity because of limited treatment schedules.

Phenanthrene did not produce skin tumors in mice in an inadequately reported skin painting study (dose and application schedule not reported) (Kennaway, 1924). Several mouse skin initiation-promotion assays with phenanthrene have been conducted. Phenanthrene was active as a tumor initiator in one study in which TPA was used as the promoter (Scribner, 1973), but was inactive in the other studies in which TPA was used as the promoter (Wood et al., 1979; LaVoie et al., 1981), croton oil was used as the promoter (Roe, 1962), benzo[a]pyrene and croton oil were used as promoters (Roe and Grant, 1964) and benzo[a]pyrene was used as the initiator (Roe and Grant, 1964). Phenanthrene also was not active when used as an initiator by subcutaneous injection with croton oil promotion by skin application (Roe, 1962).

Phenanthrene has been tested in numerous mutagenicity and other short-term assays with generally negative results. Positive responses occurred in S. typhimurium TA100 in the presence of a high concentration of metabolic activation preparation (Oesch et al., 1981), but not in strains TA100, TA98, TA1535, TA1537, TA1538 or TM677 when tested with activation in other studies. Phenanthrene also induced mutation to trifluorothymidine resistance in human lymphoblastoid TK6 cells in vitro (Barfknecht et al., 1981) and sister chromatid exchanges in hamster bone marrow cells in vivo (Bayer, 1978; Roszinsky-Kocher et al., 1979). Phenanthrene did not produce chromosome aberrations or micronuclei in hamster bone marrow cells in vivo, sister chromatid exchanges or chromosome aberrations in hamster bone marrow cell in vitro, DNA damage in bacteria or mammalian cells in vitro, mitotic recombination in yeast, mutation to 8-azoguanine and ouabain resistance in hamster V79 cells in vitro or neoplastic transformation in various mouse, hamster and guinea pig systems.

Data regarding teratogenicity or other reproductive effects, or the chronic or subchronic toxicity of phenanthrene, could not be located in the available literature as cited in the Appendix. Single intraperitoneal injections of 150 mg/kg produced evidence of slight hepatotoxicity in rats (Yoshikawa et al., 1985); these included gross congestion and distinct lobulation and small increases in the activities of SGOT and serum GGTP.

6. AQUATIC TOXICITY

6.1. ACUTE

The available information concerning acute toxicity of phenanthrene to aquatic organisms is presented in Table 6-1. Rainbow trout, Salmo gairdneri, were the most sensitive fish species tested, with concentrations as low as 1-4 µg/ℓ causing some mortality of eggs and larvae (Black et al., 1983). Eggs and larvae of largemouth bass, Micropterus salmoides, were also quite sensitive, experiencing 32% mortality at 68 µg/ℓ (Black et al., 1983). Among invertebrates, the lowest reported lethal concentration was 100 µg/ℓ, the 96-hour LC_{50} for Daphnia pulex (Trucco et al., 1983).

6.2. CHRONIC

The only data concerning the chronic toxicity of phenanthrene were provided by Geiger and Buikema (1982), who exposed D. pulex to phenanthrene for their lifetime (~50 days). Daphnia exposed to 360 µg/ℓ had fewer broods/animal; fewer live young/brood and delayed maturation. There were no statistically significant effects on Daphnia exposed to 110 µg/ℓ.

6.3. PLANTS

Bastian and Toetz (1982) reported that the standing crop of the blue-green alga, Anabaena flos-aquae, was reduced by phenanthrene at 96% saturation but not ≤48% saturation. Giddings (1979) found that a 100% saturated phenanthrene solution inhibited photosynthesis in the green alga, Selenastrum capricornutum. Millemann et al. (1984) reported 4-hour EC_{50} values for inhibition of photosynthesis of 940 and 870 µg/ℓ in S. capricornutum and the diatom, Nitzschia palea, respectively. Hutchinson et al. (1980) reported EC_{50} values of 945 and 1212 µg/ℓ for inhibition of photosynthesis in the green algae, Chlamydomonas angulosa and Chlorella vulgaris, respectively.

TABLE 6-1

Acute Toxicity of Phenanthrene to Aquatic Organisms

Species	Concentration (µg/ℓ)	Effect	Reference
FISH			
Sea lamprey *Petromyzon marinus*	5000	nonlethal, 24 hours	Applegate et al., 1957
Rainbow trout *Salmo gairdneri*	5000	lethal, 12 hours	Applegate et al., 1957
	40	96-hour LC_{50}, eggs and larvae	Black et al., 1983
	30	27-day LC_{50}, eggs and larvae	Millemann et al., 1984
	1-4	10% mortality, eggs and larvae	Black et al., 1983
Largemouth bass *Micropterus salmoides*	250	7-day LC_{50}, eggs and larvae	Millemann et al., 1984
	180	96-hour LC_{50}, eggs and larvae	Black et al., 1983
	68	32% mortality, eggs and larvae	Black et al., 1983
Bluegill *Lepomis macrochirus*	5000	lethal, 12 hours	Applegate et al., 1957
CRUSTACEANS			
Water flea *Daphnia magna*	700	48-hour LC_{50}	Millemann et al., 1984
	1160	48-hour LC_{50}	Bobra et al., 1983
	843	LC_{50}	Eastmond et al., 1984
Water flea	960-1280	48-hour LC_{50}	Geiger and Buikema, 1982
	100	96-hour LC_{50}	Trucco et al., 1983
Amphipod *Gammarus minus*	460	48-hour LC_{50}	Millemann et al., 1984

TABLE 6-1 (cont.)

Species	Concentration (µg/ℓ)	Effect	Reference
INSECTS			
Midge *Chironomus tentans*	490	48-hour LC_{50}	Millemann et al., 1984
MOLLUSCS			
Marine snail *Littorina littorea*	40	lysosome labilization, 3 days	Moore et al., 1985
Mussel *Mytilus edulis*	50-200	decreased lysosomal stability	Moore and Ferrar, 1985
ANNELIDS			
Polychaete *Neanthes arenaceodentata*	600	96-hour LC_{50}	Rossi and Neff, 1978
PROTOZOA			
Ciliate *Colpidium colpoda*	saturated	nontoxic, saturated solution	Rogerson et al., 1983
Ciliate *Tetrahymena elliotl*	saturated	nontoxic, saturated solution	Rogerson et al., 1983

6.4. RESIDUES

Data from bioconcentration experiments with phenanthrene are presented in Table 6-2. The highest reported BCF was 23,800 for the alga, S. capricornutum (Casserly et al., 1983). In some species, uptake from food may also be an important route of accumulation. Such species include D. pulex (Trucco et al., 1983) and the benthic amphipod, Pontoporeia hoyi (Eadie et al., 1983).

Once inside the organism, phenanthrene tends to accumulate in certain tissues. In fish, the liver seems to be the principal site of accumulation (Solbakken et al., 1979, 1982). In the horse mussel, Modiola modiolus, phenanthrene accumulated in the hepatopancreas and kidney (Palmork and Solbakken, 1981).

Bony fishes tend to metabolize and eliminate phenanthrene more quickly than other aquatic organisms (Gerhart et al., 1981; Solbakken and Palmork, 1981). The primary metabolite formed by flounder, Platichthys flosus, and rainbow trout, S. gairdneri, was 1,2-dihydro-1,2-dihydroxyphenanthrene (Solbakken and Palmork, 1981). Fathead minnows, Pimephales promelas, eliminated phenanthrene rapidly, with no detectable residues remaining after 24 hours depuration (Gerhart et al., 1981). D. magna eliminated phenanthrene somewhat more slowly, with an initial elimination half-time of 9 hours (Eastmond et al., 1984). D. pulex eliminated 80-92% of its body burden in 24 hours of depuration (Trucco et al., 1983).

Phenanthrene monitoring data are presented in Table 6-3. In some cases, aquatic organisms can accumulate body burdens of phenanthrene in the ppm range and, therefore, could be an important route of exposure if consumed by humans. Dunn and Fee (1979) pointed out that lobsters, Homarus americanus,

TABLE 6-2

Bioconcentration Data for Phenanthrene in Aquatic Organisms

Species	Concentration (µg/ℓ)	BCF	Remarks	Reference
FISH				
Fathead minnow *Pimephales promelas*	NR	3,100-5,100	28-day BCF	Carlson et al., 1979
	NR	1,000-12,000	14-day BCF	Gerhart et al., 1981
CRUSTACEANS				
Water flea *Daphnia magna*	60	600	peak BCF, 20-30 hours	Eastmond et al., 1984
Water flea *Daphnia pulex*	NR	325	24-hour BCF	Southworth et al., 1978
	NR	1,032-1,424	24-hour BCF	Trucco et al., 1983
MOLLUSCS				
Clam *Macoma inquinata*	NR	10.3	7-day BCF, water	Roesijadi et al., 1978
		0.2	7-day BCF, sediment	
Mussell *Mytilus edulis*	0.3	68	8-hour BCF	Hansen et al., 1978
	1.9	81	8-hour BCF	

TABLE 6-2 (cont.)

Species	Concentration (µg/ℓ)	BCF	Remarks	Reference
PLANTS				
Algae several species	NR	4,552	natural pond	Davis et al., 1983
Green alga *Selenastrum capricornutum*	NR	23,800	24-hour BCF	Casserly et al., 1983
Green alga *Chlorella fusca*	NR	1,760	24-hour BCF	Geyer et al., 1984

NR = Not reported; BCF = bioconcentration factor

TABLE 6-3

Monitoring Data for Phenanthrene in Aquatic Organisms

Species	Location	Tissue	Concentration (ng/g)	Reference
FISH				
White sucker *Catostomus commersoni*	eastern Lake Erie	stomach contents	23-43	Maccubbin et al., 1985
English sole *Parophrys vetulus*	Puget Sound	stomach contents	56-1400	Malins et al., 1985
CRUSTACEANS				
Brown shrimp *Penaeus aztecus*	central Gulf of Mexico	whole body	10	Nulton and Johnson, 1981
Lobster *Homarus americanus*	eastern Canada, Atlantic Ocean	whole body	32	Dunn and Fee, 1979
MOLLUSCS				
Mussell *Mytilus edulis*	Norway, polluted areas	whole body	41-792	Knutzen and Sortland, 1982
Periwinkle *Littorina littorea*	Norway, polluted areas	whole body	115-258	Knutzen and Sortland, 1982
Limpet *Patella vulgata*	Norway, polluted areas	whole body	55-2542	Knutzen and Sortland, 1982

TABLE 6-3 (cont.)

Species	Location	Tissue	Concentration (ng/g)	Reference
MISCELLANEOUS INVERTEBRATES				
Starfish *Asterias rubens*	Norway, polluted areas	whole body	32-50	Knutzen and Sortland, 1982
Sponge *Halichondria panicea*	Norway, polluted areas	whole body	71	Knutzen and Sortland, 1982
Polychaetes (unspecified)	New York bight	whole body	ND-14	Farrington et al., 1986
PLANTS				
Bladder wrack *Fucus vesiculosus*	Norway, polluted areas	whole body	31-325	Knutzen and Sortland, 1982
Toothed wrack *Fucus serratus*	Norway, polluted areas	whole body	109-146	Knutzen and Sortland, 1982
Knotted wrack *Ascophyllum nodosum*	Norway, polluted areas	whole body	45-431	Knutzen and Sortland, 1982
Laminaria saccharina	Norway, polluted areas	whole body	87	Knutzen and Sortland, 1982
Ceramium rubrum	Norway, polluted areas	whole body	34	Knutzen and Sortland, 1982

ND = Not detected

maintained in enclosures made of creosote-treated wood could significantly increase their PAH body burden. Phenanthrene levels averaged 32 ng/g in freshly caught lobsters and 100 ng/g in impounded lobsters.

6.5. SUMMARY

The data base for the aquatic toxicity of phenanthrene is limited. The most sensitive of four fish species tested was the rainbow trout, which experienced a 10% mortality of eggs and larvae at 1-4 µg/ℓ (Black et al., 1983). Among the nine invertebrate species tested, the lowest reported lethal concentration was 100 µg/ℓ, the 96-hour LC_{50} for D. pulex (Trucco et al., 1983). This result conflicts with the only chronic toxicity study available (Geiger and Buikema, 1982), in which no toxic effects or reproductive success or survival of D. pulex occurred at 110 µg/ℓ. Aquatic plants appeared to be less sensitive to phenanthrene than fish and invertebrates, with EC_{50} values for inhibition of photosynthesis ranging from 870 µg/ℓ in N. paleo (Millemann et al., 1984) to 100% saturation in S. capricornutum (Giddings, 1979). Bioconcentration and residue monitoring data indicated wide variability in potential for phenanthrene accumulation in various species (see Tables 6-2 and 6-3). Bony fishes (teleosts) tended to metabolize and eliminate phenanthrene more rapidly than other aquatic organisms (Solbakken and Palmork, 1981).

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

OSHA set an 8-hour TWA concentration limit of 0.2 mg/m³ for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo[a]-pyrene, phenanthrene, acridine, chrysene, pyrene) (OSHA, 1985). NIOSH (1977) recommended a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances of 0.1 mg/m³ of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. NIOSH (1977) concluded that these specific coal tar products, as well as coke oven emissions, are carcinogenic and can increase the risk of lung and skin cancer in workers. NIOSH (1977) also recommends a ceiling limit for exposure to asphalt fumes of 5 mg airborne particulates/m³ of air.

U.S. EPA (1980a) recommended a concentration limit of 28 ng/ℓ for the sum of all carcinogenic PAH in ambient water. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with benzo[a]pyrene, and acknowledges the conservative assumption that all carcinogenic PAH are equal in potency to benzo[a]pyrene. On the basis of the animal bioassay data, daily consumption of water containing 28 ng/ℓ of carcinogenic PAH over an entire lifetime is estimated to keep the lifetime risk of cancer development <1 chance in 100,000.

U.S. EPA (1980a) acknowledged that data suitable for quantitative risk assessment of noncarcinogenic PAH are essentially nonexistent, and an ambient water quality criterion has not been recommended.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of phenanthrene in particular could not be located in the available literature as cited in the Appendix; however, U.S. EPA (1980b) noted that acute toxicity to saltwater life occurred at concentrations as low as 300 µg/ℓ of polynuclear aromatic hydrocarbons in general and would occur at lower concentrations in species more sensitive than tested. U.S. EPA (1980b) also determined that the data base was inadequate to recommend criteria or draw conclusions about chronic or acute toxicity to freshwater biota.

8. RISK ASSESSMENT

Phenanthrene has been tested for carcinogenicity in a single-treatment (200 mg) gavage study in which rats were examined for development of mammary tumors for 60 days following treatment (Huggins and Yang, 1962). The tumorigenicity of phenanthrene was also evaluated in single-treatment (40 μg or 5 mg) subcutaneous (Steiner, 1955; Grant and Roe, 1963) and three-treatment (35, 70 and 140 μg at weekly intervals) intraperitoneal (Buening et al., 1979) studies with mice. The results of these studies were negative but should be regarded as inconclusive (see Table 5-1) because limited treatment schedules make the studies inadequate for evaluation of carcinogenicity. Phenanthrene reportedly did not produce skin tumors in mice in an inadequately reported study in which the dose and application schedule was not specified (Kennaway, 1924). Phenanthrene was active as a tumor initiator in one study in which TPA was used as the tumor promoter (Scribner, 1973), but was inactive in other mouse skin initiation-promotion studies in which TPA was used as the promoter (Wood et al., 1979; LaVoie et al., 1981), croton oil was used as the promoter (Roe, 1962), benzo[a]pyrene and croton oil were used as promoters (Roe and Grant, 1964) and benzo[a]pyrene was used as the initiator (Roe and Grant, 1964). Phenanthrene also was not active when used in mice as an initiator by subcutaneous injection with croton oil promotion by skin application (Roe, 1962).

Phenanthrene has been tested in numerous mutagenicity and other short-term assays with predominant negative responses. Point mutation tests in bacteria have generally been negative (McCann et al., 1975; Wood et al., 1979; Buecker et al., 1979; LaVoie et al., 1981; Florin et al., 1980; Dunkel et al., 1984; Kaden et al., 1979; Seixas et al., 1982) with the exception of one study showing positive results for Salmonella typhimurium TA100 when

assayed in the presence of a high concentration of liver S9 (Oesch et al., 1981) and another study reporting a positive response in the new frameshift tester strain TA97 with liver metabolic activation (Sakai et al., 1985). Phenanthrene was reported to induce gene mutations in human lymphoblastoid cells in vitro in the presence of a metabolic activation system (Barfknecht et al., 1981), but was reported to be negative for gene mutations at two different loci in Chinese hamster V79 cells in vitro (Huberman and Sachs, 1979). Intraperitoneal injection of phenanthrene into Chinese hamsters produced sister chromatid exchanges, but no chromosome aberrations or micronuclei in the bone marrow cells (Bayer, 1978; Roszinsky-Kocher et al., 1979). Sister chromatid exchanges and chromosome aberrations were not produced in Chinese hamster V79-4 cells treated in vitro with phenanthrene in the presence of exogenous metabolic activation (Popescu et al., 1977). Phenanthrene did not produce positive responses in other assays indicative of DNA damage using bacteria mammalian cells in vitro, and yeast (i.e., differential growth inhibition, DNA repair and mitotic recombination tests) (McCarrol et al., 1981; Rosenkranz and Poirier, 1979; Lake et al., 1978; Probst et al., 1981; Simmon, 1979).

The oral, subcutaneous, intraperitoneal and dermal carcinogenicity studies of phenanthrene are inadequate for evaluation of carcinogenicity because of deficiencies in treatment schedules and reporting. Phenanthrene was active as an initiator in one mouse skin study that used TPA as the promoter (Scribner, 1973), but this effect was not corroborated in other studies that used the same or different promoters or benzo[a]pyrene as the initiator. Mutagenicity and clastogenicity (sister chromatid exchange) of phenanthrene was reported in several assays, but the preponderance of data

from numerous short-term genotoxicity tests is negative. The available evidence is therefore inadequate to evaluate the carcinogenicity of phenanthrene.

Information regarding the chronic or subchronic toxicity, teratogenicity or other reproductive effects of phenanthrene could not be located in the available literature as cited in the Appendix. Calculation of an RfD (formerly ADI) is therefore precluded, as it was at the time of an earlier health effects assessment for phenanthrene (U.S. EPA, 1984).

9. REPORTABLE QUANTITY

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Information regarding the chronic or subchronic toxicity, teratogenicity or other reproductive effects of phenanthrene could not be located in the available literature as cited in the Appendix. Calculation of an RQ ranking for phenanthrene based on chronic toxicity is therefore precluded, as it was in an earlier RQ document for phenanthrene (U.S. EPA, 1983) by the lack of appropriate data.

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Phenanthrene has been tested for carcinogenicity in a single-treatment (200 mg) gavage study in which rats were examined for development of mammary tumors for 60 days following treatment (Huggins and Yang, 1962). The tumorigenicity of phenanthrene was also evaluated in single-treatment (40 µg or 5 mg) subcutaneous (Steiner, 1955; Grant and Roe, 1963) and three-treatment (35, 70 and 140 µg at weekly intervals) intraperitoneal (Buening et al., 1979) studies with mice. The results of these studies were negative but should be regarded as inconclusive because limited treatment schedules make the studies inadequate for evaluation of carcinogenicity. Phenanthrene reportedly did not produce skin tumors in mice in an inadequately reported study in which a dose and application schedule was not specified (Kennaway, 1924). As detailed in Table 5-1, phenanthrene was active as a tumor initiator in one study in which TPA was used as the tumor promoter (Scribner, 1973), but was inactive in other mouse skin initiation-promotion studies in which TPA was used as the promoter (Wood et al., 1979; LaVoie et al., 1981), croton oil was used as the promoter (Roe, 1962), benzo[a]pyrene and croton oil were used as promoters (Roe and Grant, 1964) and benzo[a]pyrene was used

as the initiator (Roe and Grant, 1964). Phenanthrene also was not active when used in mice as an initiator by subcutaneous injection with croton oil promotion by skin application (Roe, 1962).

Phenanthrene has been tested in numerous mutagenicity and other short-term assays with generally negative results. These include assays for DNA repair, mutagenesis and clastogenicity in bacterial and mammalian cells *in vitro* and *in vivo* and neoplastic transformation in mammalian cells. Positive responses occurred in *S. typhimurium* strain TA100 in the presence of a high concentration of metabolic activation preparation (Oesch et al., 1981), but not in strains TA100, TA98, TA1535, TA1537, TA1538 or TM677 with activation in other studies. Phenanthrene also induced mutation to trifluorothymidine resistance in human lymphoblastoid TK6 cells *in vitro* (Barfknecht et al., 1981) and sister chromatid exchanges in hamster bone marrow cells *in vivo* (Bayer, 1978; Rosziusky-Kocher et al., 1979).

The oral, subcutaneous, intraperitoneal and dermal carcinogenicity studies of phenanthrene are inadequate for evaluation of carcinogenicity because of the differences in treatment schedule and reporting. Phenanthrene was active as an initiator in one mouse skin study that used TPA as the promoter (Scribner, 1973), but this effect was not corroborated in other studies that used the same or different promoters or benzo[a]pyrene as the initiator. Mutagenicity and clastogenicity (sister chromatid exchange) of phenanthrene was reported in several assays, but the preponderance of data from numerous short-term genotoxicity tests is negative. The available evidence is therefore inadequate to evaluate the carcinogenicity of phenanthrene.

IARC (1983) reported that there was insufficient evidence regarding the carcinogenic risk to humans and experimental animals associated with oral or inhalation exposure to phenanthrene. Applying the EPA criteria for evaluation of the overall weight of evidence for the carcinogenic potential for humans (U.S. EPA, 1986), phenanthrene is most appropriately designated a Group D - Not Classified chemical. Direct hazard ranking of phenanthrene under CERCLA is therefore not possible.

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Zepp, R.G. and P.F. Schlotzhauer. 1979. Photoreactivity of selected aromatic hydrocarbons in water. In: Polynuclear hydrocarbons, P.W. Jones and P. Leber, Ed. Ann Arbor Science Publishers, Inc., Ann Arbor, MI. p. 141-158.

Zepp, R.G. and P.F. Schlotzhauer. 1983. Influence of algae on photolysis rates of chemicals in water. Environ. Sci. Technol. 17: 462-468.

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985-1986. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1985-1986. Cincinnati, OH. 114 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-1983. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Littleton, MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. WHO, IARC, Lyons, France.

Jaber, H.M., W.R. Mabey, S.T. Liu, T.W. Chow and H.L. Johnson. 1984. Data aquisition for environmental transport and fate screening for compounds of interest in the Office of Solid Waste. EPA 600/6-84-010. NTIS PB84-243906. SRI International, Menlo Park, CA.

NTP (National Toxicology Program). 1986. Toxicology Research and Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, N.I. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1984. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1985. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call in Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985. CSB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (U.S. International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982, USITC Publ. 1422, Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California, State Water Quality Control Board. Publ. No. 3-A.

Pimental, D. 1971. Ecological Effects of Pesticides on Non-Target Species. Prepared for the U.S. EPA, Washington, DC. PB-269605.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1: Toxicology Data. Office of Pesticide Programs, U.S. EPA, Washington, DC. EPA 540/9-79-003. NTIS PB 80-196876.